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The relationship between profiles and transitions of problem behaviour in elementary-school children and engagement in health-risk behaviours in early adolescence

by

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Abstract

Data from the National Longitudinal Survey of Children and Youth (NLSCY), a nationally representative and prospective cohort, was used to explore patterns of problem behaviour in elementary school aged children across time and their association to adolescent health-risk outcomes. Latent profile analyses identified four profiles of problem behaviour: (1) low on all problem behaviours, (2) moderate on all problem behaviours, (3) high on all problem behaviours, and (4) high on hyperactivity/inattention and internalizing. This measurement invariant trend was observed at ages 6/7, 8/9, and 10/11. Transition patterns between these profiles of problem behaviour from ages 6/7 to 10/11 were also identified (n=8,266). The association of these profiles and transition patterns with health-risk outcomes were computed using logistic regression modelling. While patterns of persisting problem behaviour were associated with suicidal thoughts, substance use, and delinquency, they did not differ from the profile at age 10/11 years, where the “Moderate all” and “High all” profiles of problem behaviour predicted the most health-risk outcomes in adolescence. The most recent assessment of problem behaviour in adolescence was as good of a predictor of adolescent health-risk outcomes relative to patterns of problem behaviour across time.

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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

Emotional and behavioural problems occur early in the life course and include feelings of negative affect, hyperactivity, and aggression.[1–3] While some of these behaviours can be considered developmentally normative, subsets of children have displayed patterns of emotional and behavioural problems that persist from childhood, through adolescence, and into adulthood.[4–7] While a wealth of research has been conducted that investigated patterns of problem behaviours in children, and how they change across time, less research has looked at how these temporal sequences of problem behaviour relate to health-risk outcomes in adolescence, such as suicidality, substance use, and delinquent behaviours. Identification of patterns of childhood problem behaviours, and how they change across time, may serve as useful predictors of health-risk outcomes and highlight potential target groups for intervention.

The adolescent period is a high-risk period in the development of many health-risk outcomes including suicidality (i.e. thoughts and behaviours), substance use, and other types of delinquency. Between the ages of 15-24, suicide is the second and third leading cause of death for individuals in Canada and the United States, respectively.[8] When looking at suicidal thoughts, it has been estimated that approximately 1 in 5 early adolescents have experienced suicidal thoughts.[9] Adolescence is also the period in which individuals first begin experimenting with alcohol, nicotine, cannabis, and sometimes, harder substances.[10–12] Approximately half of adolescents have tried alcohol, one tenth

have reported being current cigarette smokers, and a quarter of adolescent's report trying cannabis.[10–12] In relation to other types of delinquency, overall rates have been decreasing, but youth still account for the highest rates of criminality out of any age group.[13]

As these findings demonstrate, youth are a vulnerable population for suicidality, substance use, and criminal activity. It is important for researchers to investigate etiological and predictive markers in the development of these behaviours, so that they can be targeted to intervene and prevent health-risk outcomes. Modern longitudinal methodology, in tandem with large datasets of health surveys, offer the ability to explore patterns of emotional/behavioural problems in children throughout development that predict negative health-risk outcomes in adolescence. The present study will investigate patterns of problem behaviour at three timepoints in childhood. Furthermore, how these patterns of problem behaviour change across time will be explored, as well as how these temporal sequences of problem behaviour relate to negative outcomes in adolescence.

The following literature review will first outline special considerations when identifying problem behaviour in childhood and then briefly discuss the manifestations of internalizing and externalizing behaviour in childhood and how stable these problems remain throughout childhood. Lastly, the negative outcomes in adolescence associated with childhood problem behaviour will be discussed.

1.2 Problem Behaviour in School-Aged Children

In the broadest sense, childhood problem behaviour has been empirically divided into two, sometimes co-occurring, domains which are *internalizing problem behaviour* and *externalizing problem behaviour.*[14] Internalizing problem behaviours are expressed through anxious and depressive behaviours such as social withdrawal, behavioural inhibition, and low self-esteem.[14] In contrast, externalizing problem behaviours are expressed as aggression, hyperactivity, inattention, and defiance.[14] Externalizing behaviour has been studied more extensively than internalizing behaviour in children.[15] This is largely due to the fact that externalizing is easier to identify and has a more detrimental effect on classrooms and family dynamics, as a whole, compared with internalizing disorders, which have a more detrimental effect on the individual themselves.[16] These behaviours are often described separately, but are not mutually exclusive, and can occur in tandem.[17,18] This two-dimension categorization of problem behaviour is utilized in many structured assessments of childhood emotions and behaviour. The most notable example is the Child Behaviour Checklist (CBCL), one of the most widely used measures of childhood behaviour and a primary measure in the current investigation.[17]

Due to the frequency of developmental changes occurring throughout childhood, it has been argued that children's problem behaviour is developmentally normative.[19–21] It has been suggested that the development of pathological problem behaviour, especially internalizing pathology, cannot be reliably identified until pubertal changes have occurred and that, for the majority

of children, problem behaviour will decrease across development.[22,23] Indeed, some research has shown that children do frequently move in and out of pathological states of behaviour during the course of development.[21,24] There is also, however, a dearth of literature that has identified persistent pathological behaviour (both externalizing and internalizing) from early childhood, into adolescence and adulthood.[4–7,25–30]

Prior to delving into research on childhood problem behaviour, it is important to acknowledge discrepancies in how these behaviours are classified. Historically, these types of behaviours have been explored as occurring on a continuum, where the severity of the behaviours is considered, or these types of behaviour have been explored categorically, where cutoff scores are applied to a continuum and individuals are classified into dichotomous or categorical groupings. For the present investigation and in order to better capture the range of problem behaviour, continuous measures of problem behaviour were explored. This is further supported by the fact that subthreshold symptoms of diagnosed mental illness have been shown to be associated with health-risk outcomes, such as suicide and substance use, as well as subsequently meeting clinical diagnoses for mental illness.[31–33] The subsequent literature review, however, will also describe categorical classifications of problem behaviour/mental illness, as this taxonomy structure is used frequently in the field and has relevant findings to the present investigation.

1.2.1 Internalizing Problem Behaviour

When evaluating children, internalizing behaviour is often divided into depressive behaviours and anxious behaviours.[14,34] Research has shown these two emotional problems to be highly related with one another and have been studied in tandem, as well as separately. For the sake of this review, and to more specifically cover the literature in children, depression and anxiety will be described distinctly, as well as comorbidity.

1.2.1.1 Anxiety

In general, anxiety can be defined as unpleasant feelings of angst and worry, which manifest through patterns of sympathetic nervous system arousal (e.g. increased heartrate, tremors/shaking), withdrawal, and overwhelming feelings of dread.[35,36] It is important to make the distinction between fear and anxiety. While both can manifest similar behavioural and cognitive reactions, as well as lead to pathological states, the source of these reactions is different. Anxiety and fear are normal human emotions, but when they become severely impairing in relation to anticipated or hypothetical events, that are not realistic, they can be defined as problematic.[37]

Anxious behaviours have been observed across the entirety of childhood, and as early as the toddler period.[2,38–41] However, the types of problematic anxiety do seem to change as a function of age. Separation anxiety, characterized by feelings of anxiety related to being away from the home/caregiver, are most common in early- and mid-childhood, with most cases emerging before the age of 12.[2,40–42] Contrary to this, symptoms of generalized anxiety disorder,

characterized by excessive feelings of anxiety and worry about everyday events, are more common in adolescents and young adults.[43] Specific phobic disorders show less age-specificity and are observed across the entirety of childhood.[40] Prevalence rates of generalized anxiety disorder, social phobia, panic disorder, and agoraphobia seem to increase across childhood and adolescence, whereas less change is observed in specific phobias and separation anxiety.[40] Despite these age differences, it is important to note that factor analyses have been conducted and differentiation of multiple anxiety disorders such as generalized anxiety disorder, separation anxiety disorder, social anxiety disorder, obsessive-compulsive symptomatology, and phobic anxieties have all been identified in early childhood.[44,45]

The overall prevalence of any anxiety disorder in childhood has been estimated to be between 2.6% – 17.7%.[40,46] Anxiety disorders have also been observed to be highly comorbid with one another in children.[47] More specifically, generalized anxiety disorder has been found to be highly comorbid with social phobia (22-67%), separation anxiety disorder (10-51%) and specific phobias (7-33%).[47]

Although DSM criteria for anxiety disorders are largely the same for children and adults (with the exception of separation anxiety disorder), identifying the difference between developmentally normative behaviour and pathological anxiety symptoms in children can be difficult and must be considered as a potential limitation in this type of research.[15] The novelty of life and the imagination of children can result in the exacerbation of fears in things such as the

dark, characters in media, and unknown situations, but this is often normal developmental behavior.[15] It is also to be expected that children feel a moderate amount of stress when separated from their home and caregivers.[15] It is important to identify the degree of impairment, controllability, and persistence of the fears when making decisions on what is normal and what is problematic behaviour.[15,48] When considering prevalence rates of anxiety disorders/symptoms in children, these criteria must be considered as it has been shown that when the criteria of functional impairment is included in defining childhood anxiety, the prevalence of the disorder(s) goes down.[46] Despite these caveats when measuring anxious behaviours in children, empirically derived measures have been shown to accurately identify psychopathological symptoms in children.[49]

1.2.1.2 Depression

Depression is characterized by low/negative affect, as well as reduced desire to partake in activity.[37] While states of depression can occur in healthy individuals, especially relative to loss and failure, when the feelings of depression are persistent and unrealistic, across most situations, they become problematic and potentially pathological. Symptoms of clinical depression include persistent low mood, low interest in enjoyable activities, feelings of pain, as well as sleep disturbances and hyper-/hypo-phagia (i.e. over-/under-eating).

Depressive psychopathology can be observed throughout childhood, but the manifestations of it do change based on an individual's cognitive, social and physiological standing, and thus, as a function of age.[3] Major depressive disorder is the most common mood disorder observed in children.[3,50] Bipolar disorders and

depression with psychotic features are rarely observed in childhood.[50] Similar to anxiety, depressive symptoms can be observed early in the lifecourse.[46,51,52] Research on preschoolers has shown that subsets of them can display all DSM-IV symptoms of major depressive disorder and that they occur in greater frequency (with the exception of irritability) than in healthy preschoolers, as well as preschoolers diagnosed with attention deficit disorder or disruptive disorders.[34,53,54] This population has also been shown to have the same neurological and cortisol dysregulation as is seen in depressed adults, further supporting the notion that the etiology of pathologic depression, to some extent, is rooted in childhood.[55,56] The diagnosis of major depressive disorder is similar in children as it is in adults.[50,57] Symptoms of negative affect, anhedonia, irritability, as well as cognitive and vegetative symptomatology can all be observed in children.[57] It has also been reported that the duration and severity of major depression is similar in children as in adults.[57]

The epidemiology of major depression has been explored extensively from preschool to adolescence.[46,58–60] Overall, prevalence rates of major depression are lower than those of anxiety. It has been estimated that approximately 1% of preschoolers have major depression and that the rate doubles to about 2% in prepubertal children and do not differ by gender.[58–60] The prevalence of major depression seems to increase dramatically throughout childhood, evidenced by the fact that rates of major depression reach upwards of 20% in late adolescence.[46,60] This demonstrates that the stability of depression is lower across childhood, as many children without depression shift into depressive states as they age.

1.2.2 Externalizing Problem Behaviour

Externalizing problem behaviour in children is often expressed through defiance of authority, hyperactivity, and both physical and relational forms of aggression.[61] As was seen with internalizing problem behaviour, externalizing problem behaviour can also be observed early in the life course in children, as early as preschool.[5,51,52,62,63] Similar problems also exist in determining the difference between normative and pathological behaviour. For example, aggressive outbursts when not getting one's way or hyperactive behaviour are quite common in young children. There does appear to be evidence that sometimes engaging in externalizing problem behaviour is a developmental phase, rather than a stable quality of a child.[64–67] In fact, it has been shown that the highest peak of externalizing problem behaviour in children occurs at the age of two and as children become more socially and cognitively developed, these behaviours decrease.[66,67] There does, however, exist subsets of children who exhibit severe levels of aggression, hyperactivity, and defiance that seem to be less developmentally transient and more persistent, resulting in a range of negative outcomes that will be discussed later in this review.[52] Research has emerged that demonstrates that these early life problem behaviours are associated with future externalizing DSM disorders.[21,68,69]

1.2.2.1 Hyperactive/Inattentive Behaviours

Of the various types of externalizing problem behaviour, hyperactivity is the most common in children and Attention Deficit Hyperactivity Disorder (ADHD) has been studied extensively.[68,70] It has been estimated that approximately 2% of

children between the age of 3 and 5 years old meet diagnostic criteria for ADHD.[71] The prevalence of ADHD seems to increase from preschool into the elementary school-age period and the prevalence rate cited in the DSM-IV is estimated as being between 3-7%.[72] Community sample research conducted at the Centers for Disease Control has reported the prevalence of ADHD to be up to 11% in children ages 4-17, with the average age of diagnosis being 7 years.[73] Other research has found estimates as high as 1 in 5 elementary school child meeting the criteria for ADHD.[74] In fact, elementary school aged children have been estimated to have the highest rates of ADHD out of any age group.[74,75] A moderate level of agreement has been observed between parent and teacher reports of ADHD diagnoses, with parents reporting a slightly higher prevalence of ADHD.[74] In addition, issues surrounding the identification of ADHD have been discussed extensively in the literature, and it has been theorized that diagnosis of ADHD may be prone to increased rates of false positives, as well as false negatives.[70,76–81] Despite issues with identifying ADHD symptoms and diagnosing, a qualitative review by Sciutto & Eisenberg (2007) concluded that, "ìt [did] not appear that there [was] sufficient evidence to support the public perception that ADHD is systematically overdiagnosed".[81]

1.2.2.2 Conduct Problems

Childhood conduct problems are characterized by a variety of antisocial, aggressive, dishonest, delinquent, defiant and disruptive behaviours. These problems can vary in severity and induce distress in caregivers, educators, and the peers of the child presenting with the behaviour. Due to the severely negative

impact that these problem behaviours have on others, the research into them is extensive. Conduct problem behaviours can be pathologized as conduct disorder and oppositional defiant disorder.[37] A high degree in overlap between symptoms exists between conduct disorder and oppositional defiant disorder, and while both are characterized by patterns of antisocial behaviour, the former features problem behaviour symptoms of violating the rights of others through aggression.[82] The course of conduct problems has consistently documented two distinct subtypes of conduct problem behaviour: childhood-onset and adolescent onset.[83] The former of these groups is characterized by conduct problems being observed as early as preschool with stability or progression of symptoms continuing from childhood through adolescence and adulthood. A second group of conduct problem individuals, is observed to begin in adolescence, and seems to be more closely tied to rebelliousness and deviant peer affiliations.

The epidemiology of conduct problems in children has been studied extensively and relative to internalizing pathology, is easier to identify.[84] Like ADHD, the disruptive nature of conduct problems are easier to recognize relative to internalizing symptoms associated with depression and anxiety. Epidemiological studies typically estimate the prevalence of conduct disorder to be between 1-3% for elementary school aged children (5-12 years old).[82,85–88] When looking at oppositional defiant disorders in a similar age group, rates are usually higher than conduct disorder and have been estimated to be between 1.9-16%.[82,85–88] Rates of conduct disorder seem to peak in adolescence, while rates of oppositional defiant disorder peak earlier, in childhood.[82] Differences in the rates

of conduct problems are contingent on who provides the data and it has been noted that teachers and caregivers report differing rates of conduct problem, with it being suggested that caregivers underreport conduct problems.[82,86] There is also a noteworthy sex difference in children, with males usually having twice as high rates of conduct problems, as compared to females.[84] This gap does seem to decline as individuals age, however, and by the time males and females reach adolescence, the sex difference is less marked.

1.2.3 Concurrent Problem Behaviours in Children

While it is possible for problem behaviours and psychopathology to present in isolation, it has been widely recognized that often, psychopathology presents as a concurrence of multiple problem behaviours, spanning different domains. While research on patterns of comorbidity and concurrent psychopathology has been studied more extensively in adult populations, research has identified similar patterns in children and adolescents.

1.2.3.1 Anxiety and Depression

Depression and anxiety often coincide with one another and have the highest co-occurrence of all mental illness.[89] It has been estimated that anxiety disorders are present in 15 – 75% of depressed children and youth.[90–94] It is more commonly reported that children with a primary diagnosis of depression have a secondary diagnosis of anxiety (25-50%), than it is that children with primary anxiety have a secondary diagnosis of depression (10-15%).[85,95,96] Despite this, children with anxiety disorders still present with significantly higher rates of subthreshold

depression relative to those without anxiety disorders, suggesting that, at the minimum, depressive symptomatology is common in anxiety disorders.[94]

Longitudinal research has tried to identify pathways in this development, and it has been theorized that depression and anxiety may share a causal relationship with one another. One recognized pathway in the development of the comorbidity is that anxious symptomatology occurs first, eventually impacting affect and inducing depressive symptomatology.[90,97–100] The opposite, to a lesser extent, has also been observed, whereby chronic depressive symptoms, and subsequent impairment, induces the development of comorbid anxiety.[100,101] Individuals with comorbid anxiety and depression have been consistently identified as having higher symptom severity than either pathology group in isolation.[94,102]

When measuring anxiety and depression, the two pathologies have been combined into the broader construct of internalizing disorder.[17,18] This has been warranted by the observation of substantial comorbidity and other important overlaps. Oftentimes, measures of anxiety and depression have overlapping items and even when those items are removed, the measures are still found to be significantly correlated with one another.[94,103,104] Both pathologies have also been recognized as having similar etiological influences, with a key risk factor being the traits of negative emotionality and neuroticism.[105,106] The treatment of one of the two pathologies has also been shown to reduce symptoms of the other.[107,108] Interestingly, latent modelling using the CBCL items of depression and anxiety has been conducted, and while mild, moderate, and severe comorbid classes have

been identified, limited evidence for a pure depression or anxiety class have been documented.[109,110] Due to this close relationship between the two pathologies, the present study will utilize the broader measure of internalizing problem behaviour, rather than separating depression and anxiety.

1.2.3.2 ADHD and Depression

While internalizing disorders are often comorbid with each other, they have also been observed to be comorbid with hyperactive and inattentive problem behaviours, such as ADHD.[79,111–117] A meta-analysis conducted by Meinzer et al. (2014), consolidated all studies that investigated the co-occurrence of ADHD and unipolar depression in children and adolescents.[118] The overall finding was that there was a positive relationship between the two pathologies, with the caveat that a large amount of heterogeneity was observed between the studies. There was also evidence that individuals with ADHD had higher rates of subthreshold depressive symptoms.[113] The strongest evidence for a true association between the two pathologies came from cross-sectional research that operationalized ADHD based on DSM-criteria and that did not use teacher reports.[79,111–113,115] While not all longitudinal research showed a significant association, there were some that did.[114,116,117] While some studies did not observe a significant relationship between the two pathologies, the authors concluded that there was sufficient evidence to alert clinicians about a possible risk of depression in individuals with ADHD.[118]

1.2.3.3 ADHD and Anxiety

Anxiety disorders have also been observed to co-occur with ADHD in children and youth.[119–129] This comorbidity has been identified to start as early as preschool.[130] It has been estimated that 15 – 40% of individuals with either an anxiety disorder or ADHD, can be expected to have a comorbidity of both pathologies.[121,126,131,132] The symptoms of the comorbid individuals have been shown to be similar to those experienced in older children and adolescents.[133] Multiple theories have been postulated for the development of this comorbidity, such as performance shortcomings associated with ADHD leading to anxious symptoms or the etiological foundations of both types of problem behaviour being similar, supported by an overlap in symptoms of restlessness and poor focus.[124] In contrast, evidence has also shown that ADHD and anxiety can develop independently, as the presence of one of the pathologies did not seem to change the pattern of the other.[133] A review conducted in the field consolidated the research and determined that further longitudinal research is needed to better understand these pathways.[134]

1.2.3.4 ADHD and Conduct/Defiant Problem Behaviour

Hyperactive and inattentive behaviours clearly share some degree of relationship with internalizing, but research shows they have an even higher rate of concurrence with externalizing behaviours of conduct disorder and operational defiant disorder.[95,129,135,136] Community and clinical samples of children and adolescents have identified the prevalence of comorbid ADHD and other disruptive behavioural disorders to be between 30-67%.[135–138] While an

association does indeed exist with conduct disorder, individuals with ADHD are two to three times more likely to have an operational defiant disorder compared to a conduct disorder.[136,139] Similar to patterns of other comorbid problem behaviour, individuals with comorbid ADHD and other externalizing behaviours present with greater overall symptom severity relative to individuals with only one problem behaviour.[140] ADHD and conduct disorder groups have the highest symptoms of delinquency, overt aggression and ADHD symptom severity, followed by ADHD with operational defiant disorder, followed last by ADHD only groups.[140]

1.2.3.5 Other Internalizing and Externalizing Comorbidities

Other patterns of comorbid internalizing and externalizing have also been well documented and include patterns of internalizing and conduct problems, as well as overall dysregulation, or comorbidity of many problem behaviour domains.[95,141–148] Individuals who show substantial dysregulation demonstrate problems of hyperactivity, inattentiveness, conduct, and defiance. Research has shown this comorbidity pathway to begin during early childhood, and throughout childhood and adolescence, they show substantial dysregulation across all domains, and higher severities of symptoms than any other problem behaviour group. The opposite has also been observed, in that internalizing symptoms can occur before the onset of externalizing symptomatology.[142,143] This pathway seems to have a later onset and the comorbidity occurs in adolescence when depressive states become more severe and individuals lash out, externally.[142,143] Regardless of the directionality in the development of the comorbidity, negative

feedback between internalizing and externalizing problem behaviours acts to further exacerbate problem behaviour in both domains.

Research conducted using latent variable modelling has also identified a profile of dysregulation in 6 year olds, characterized by high levels of emotional reactivity, internalizing, somatic complaints, withdrawal, attention problems, and aggressive behaviour.[149] This has been further confirmed in research on school-aged children, where latent class analyses have also identified a dysregulation profile, characterized by high CBCL scores of anxiety/depression, attention problems, and aggressive behaviour.[150–152]

1.3 Stability and Transitions of Problem Behaviour across Childhood

As discussed above, multiple patterns of internalizing and externalizing problem behaviour have been observed throughout childhood, but current research is being conducted to identify the stability of these behaviours from infancy to adolescence, and beyond. Angold et al. defined two types of problem behaviour stabilities.[95] One of these types is *homotypic stability*, where the same type of problem behaviour persists over time, and the other being *heterotypic stability*, where the type of problem behaviour changes over time. One study found that infants and toddlers with problem behaviours showed recurring, homotypic problem behaviour one year later.[153] This study showed that 38%, 50%, and 75% of toddlers had stable internalizing, externalizing, and co-occurring problem behaviour, respectively, at one year follow up.[153] Other studies have also found homotypic stability in individuals from as young as 18 months old until adolescence.[149,154–159] Evidence has supported that individuals who are high on

both internalizing and externalizing behaviour show the greatest amount of homotypic stability across time and persistence of problems.[149,153,160]

Heterotypic stability has also been observed across childhood development. Children are more likely to transition from externalizing problem behaviour to internalizing problem behaviour than the reverse.[52] In fact, some research has shown that the behavioural inhibition qualities that come with being an internalizer have protective factors for children when it comes to hyperactivity, defiance, and aggression.[52] Children who exhibit externalizing behaviour at a young age are more likely to alienate their peers and without positive peer groups, they are at an increased risk of becoming isolated and lonely.[161] This sense of loneliness may exacerbate internalizing problem behaviour such as helplessness and hopelessness. These individuals may also remain highly reactive, thus showing how they can transition heterotypically from an externalizer into comorbidity for both internalizing and externalizing problem behaviour.

One final point to highlight in relation to the stability of problem behaviour is that problem behaviour can occur in early children as a result of individuals lacking social and cognitive sophistication.[22,23] As individuals develop and become aware of societal norms, there is a large subset of problem behaviour children whose behaviour declines into a normative range. Despite this, there is still an important proportion of children who experience persistent problem behaviour and are at high risk for negative outcomes throughout their life-course.

1.4 The Relationship between Childhood Problem Behaviour and Negative Outcomes in Adolescence & Early Adulthood

As was described earlier, problem behaviour in childhood can place individuals at a higher risk for future problems with their mental health, as well as other aspects of life. It is therefore important to consider the notion that children with persistent problem behaviour are at increased risk for future mental illness and may have a similar increased risk for suicide and negative health-risk outcomes seen in any individual with symptoms of mental illness. For example, a child with persistent internalizing has increased risk for meeting the criteria for major depressive disorder in adolescence.[162] Due to the fact that major depressive disorder is so closely associated with suicide, this long-term internalizer is now at greater risk for suicidality.

1.4.1 Homotypic Internalizing Behaviour

Internalizing problem behaviour in childhood has been shown to be closely related to anxiety and mood disorders in adolescence and adulthood.[6,162,163] Individuals who show persistent internalizing throughout childhood are at an increased risk of meeting diagnostic criteria for clinical diagnoses such as major depressive disorder and/or anxiety disorders in the adolescent period.[162] The risk for suicidality is higher in all individuals with major depression, relative to the general population, and it has been estimated that between 15-25% of individuals with major depressive disorder will die by suicide. Those with major depression are at 42-150% higher risk of mortality by suicide, as compared to the general population.[164,165] Notably, research has shown individuals with earlier onset of

major depressive disorder to be at increased risk for suicidality, relative to those with a later onset.[166] This, compounded with chronic depression being associated with greater risk for mortality, provides evidence that homotypic internalizing from childhood into adolescence and adulthood could place individuals at an increased risk for suicidality.[167–169]

Internalizing behaviour also seems to be closely tied to substance use in adolescence and adulthood.[97,170,171] Research has shown that persistent anxiety pathology across development predicts substance use disorders in adolescence and early adulthood.[97,171–173] Individuals with depressive disorders, as well as suicidal ideation are also at approximately 4-fold odds to be binge drinkers, 7-fold odds to be regular cannabis users, and 4-fold odds to smoke cigarettes.[174] Research points towards this relationship being causative, rather than of a shared etiology.[175]

1.4.2 Homotypic Externalizing Behaviour

Externalizing children's difficult nature often results in rejection from their peers, as well as poor academic performance early in the life-course. Over time, this results in a drift away from positive peer relations towards deviant peer relations.[52,176,177] These individuals are at increased risk for problematic behaviours including substance use & abuse, high risk sexual behaviour, violence, theft, and school dropout. These individuals are also more likely to be diagnosed with ODD and CD.[68,177,178] These problematic behaviours and pathology may have cyclical results, whereby these individuals behaviour further marginalizes them from society making behavioural change difficult.

Suicide, due to the low sense of self-worth associated with it, is more closely tied with internalizing than externalizing. There are groups of individuals who experience comorbid externalizing and internalizing, therefore it can be hard to tease apart which of the two is increasing risk for suicidality. However, forensic research has identified a specific group of externalizing adolescents and adults who die by suicide due anger and impulsivity, rather than low affect.[179,180] Due to the possible confounding nature of prison populations, Verona, Sachs-Ericsson, and Joiner conducted similar research in the community, whilst controlling for internalizing behaviour.[181] The researchers found similar results to the forensic research, showing that externalizing individual's rates of suicide were comparable to those of individuals with internalizing disorders. This research demonstrates that externalizing problem behaviour has its own unique relationship with suicidality.

1.4.3 Persistent Externalizing & Internalizing Behaviour and Heterotypic Transitions

As would be expected, individuals who have consistently high symptoms of externalizing and internalizing behaviour are at the greatest risk for negative outcomes in adolescence and throughout their lives. Individuals high on both of these problem behaviours have been shown to have higher rates of suicide, psychopathology, and criminal involvement relative to either of the problem behaviours on its own.[182–185] When it comes to heterotypic transitions, individuals seem to be at the aforementioned risks associated with being an internalizer or externalizer in adolescence/adulthood. That is to say that the predominant domain

of problem behaviour being exhibited in adolescence will have its corresponding impact on negative outcomes.[52] This means that if an individual is high on externalizing problem behaviour in early life and transitions into being high on internalizing problem behaviour, they will be at risk for the negative outcomes associated with internalizing problem behaviour.

1.5 Rationale

A wide body of research has been conducted looking at the association between symptoms of mental illness and adolescent health-risk outcomes such as suicide, substance use, and delinquency. However, much of this research has focused on psychopathology present *during* the adolescent period and its association with these behaviours. Less research has focused on how problem behaviours in childhood relate to these negative outcomes, and even fewer studies have aimed to assess the association between childhood heterotypic and homotypic trajectories of problem behaviour, leading into adolescence, and how these trajectories relate to health-risk outcomes. Due to the large detriment that suicidality, substance use, and delinquency have on the individual and society, it is warranted to conduct research that helps to identify earlier predictors of these behaviours to inform intervention and policy.

Suicide is one of the leading causes of death in Canada and accounts for approximately 4,000 deaths annually.[8] When looking at the transition period into adolescence, suicide becomes the fourth leading cause of death at age 12.[186] This highlights adolescence as a high-risk period in the development of suicidality.[187–190] Directly relevant to this is the fact that suicidal thoughts often precede and are

strongly associated with suicide attempts.[191,192] Research that followed individuals from grades seven through nine found that approximately 1 in 5 early adolescents reported suicidal thoughts.[9] Similar findings have also been replicated in other research.[193] While sex differences can be observed in all types of suicidality (i.e. thoughts, attempts, and deaths), the association between suicidal thoughts and suicide attempts is the same across sexes.[194,195] Due to the progressive nature from suicidal thoughts to suicide attempts, identifying behavioural antecedents of suicidal thoughts may highlight targets for intervention to deter the progression of suicidality, and in turn, reduce deaths by suicide. Also of importance is that a significant proportion of first suicide attempts are fatal, further highlighting the necessity of identifying risk factors of suicidal thoughts before they progress to suicide attempts.[196–198]

In 2009 the rate of suicide in Canada was 11.5 per 100,000 people[8]. That year, the direct and indirect economic burden of suicidality in Canada was estimated to be nearly 3 billion dollars.[199] Furthermore, the non-economic costs of suicide on survivors, their families, communities, and society as a whole, are vast. Survivors of suicide face social stigma from their peers and family members, and are at an elevated risk of future ideation and attempts.[200–203] Families who lose a member to suicide have also been shown to face stigma and blame within their communities.[203,204] The impact of suicide on peers and family members has also been shown to impact wellbeing and mental health.[204,205] There is also the phenomena of suicide contagion, a term coined by Higgins & Range (1996), whereby rates of suicide, especially in adolescents and young adults, increase

after exposure to the death of a peer by suicide.[206–211] These findings show the detrimental impact of adolescent suicide on survivors, families, and communities, further justifying the necessity to better understand developmental pathways that lead to these tragic outcomes.

Relative to other age groups, adolescents are at an elevated risk to die by suicide or to experience a suicide, but they are also at an elevated risk for substance use and delinquency, relative to the general population.[10–13] An annual, nationwide tobacco report in Canada estimated that approximately 10.7% of adolescents aged 15-19 years old were current smokers.[11] In relation to youth alcohol use, the age of first drink in Canada has been increasing with 50% of youth having reported trying their first alcoholic drink between the ages of 12 and 14.[12] By grades 10-12 approximately 46% of adolescents reported that they had partaken in binge drinking behaviour.[12] When it comes to other illicit substance use, youth have higher rates of use than any other age group in Canada.[10] One in four youth reported using Cannabis and between 2-5% reported using hallucinogens, ecstasy, or cocaine.[10] While partaking in adolescent substance use does not guarantee the development of substance disorders, research has shown adolescent substance use to be a risk factor for future substance abuse and addiction.[212,213] While adolescence is a risk period for the development of addiction, and associated physiological harm, it is also a risk period for engagement in other types of non-substance delinquency.[13]

In relation to non-substance delinquency, the adolescent period is a high-risk age group for involvement in these behaviours. A report released in 2016 by

Public Safety Canada reported that, overall, the frequency and severity of youth crime had been on the decline over the past decade.[13] However, youth still account for higher crime rates than any other age group. One in three youth have reported engaging in violent behaviour, as well as destruction of property. Approximately 150,000 youths were accused of committing a crime in 2010 and 42% of those accusations resulted in criminal charges or police recommendation of criminal charges. Although crime seems to occur disproportionately throughout Canada's provinces, youth account for a significant proportion of criminal activity, nationwide.

While theft and damage to property have immediate costs for taxpayers and victims, they also can result in youths being incarcerated. In 2015/16, approximately 8,500 Canadian youth between the age of 12 to 17 were being supervised in custody or a community program on any given date.[214] The cost of secure custody of youth has been estimated at $51,742 per youth and $65,526 per contact.[215] It has been estimated that the average cost per offence, for youth populations, is $34,782.[215] Overall, youth crime has been on the decline, but they still contribute a significant proportion of the costs of delinquent behaviours. The cyclical effects of involvement in the criminal justice system on subsequent arrests and imprisonment further exacerbate these costs at the individuals and societal level. As with suicide, earlier identification into the problem behaviour patterns that predict these costly outcomes may offer the opportunity to intervene and prevent them.

1.6 Objectives and Hypotheses

The present study has three primary objectives:

1. Identification of latent profiles of problem behaviour at ages 6-7, 8-9, and 10-11 years old.
    - Hypothesis: It is hypothesized that four profiles of problem behaviour will be identified and that these patterns will be present at all three timepoints:
        i. *Predominantly internalizing problem behaviour*
        ii. *Predominantly externalizing problem behaviour*
        iii. *Internalizing and externalizing problem behaviour*
        iv. *Low/no problem behaviour*
2. Identification of problem behaviour transitions from ages 6 to 11 years old
    - Hypothesis: Based on the research described in the introduction, the following transition groups are predicted to be the most predominant
        i. *Homotypic internalizing problem behaviour*
        ii. *Homotypic externalizing problem behaviour*
        iii. *Homotypic internalizing and externalizing problem behaviour*
        iv. *Heterotypic externalizing to internalizing problem behaviour*
        v. *Overall decreasing problem behaviour*
        vi. *Stable low problem behaviour*

3. Assessing the association between problem behaviour stability groups from ages 6-11 years old and suicidality, substance use, and delinquent behaviour at ages 12-15 years old

    - Hypothesis:

        i. *Individuals with stable, internalizing problem behaviour from ages 6-11 years old will be at increased odds of all negative outcomes at ages 12-15 years old, as compared to individuals with stable, non-problem behaviour and declining problem behaviour*
        ii. *Individuals with stable, externalizing problem behaviour from ages 6-11 years old will be at increased odds of all negative outcomes at ages 12-15 years old, as compared to individuals with stable, non-problem behaviour and declining problem behaviour*
        iii. *Individuals with stable, internalizing and externalizing problem behaviour from ages 6-11 years old will be at increased odds of all negative outcomes at ages 12-15 years old, as compared to individuals with stable, non-problem behaviour and declining problem behaviour, as well as individuals stable on only one domain (internalizing or externalizing)*

Chapter 2 - Methods

2.1 Study Design

The overall approach for the present study used secondary data from a large Canadian longitudinal survey, that followed participants prospectively. The study design was a longitudinal cohort study that implemented the mixture modelling technique known as latent profile analysis to explore profiles of problem behaviour in childhood. These profiles of problem behavior were then used as independent variables and their association with suicidal thoughts and other health risk outcomes in adolescence were explored using multivariate regression analyses.

2.2 The National Longitudinal Survey of Children and Youth

The present study used the National Longitudinal Survey of Children and Youth (NLSCY), which is a longitudinal survey that assessed wellbeing and development from birth to early adulthood. The NLSCY was conducted by Statistics Canada and sponsored by Human Resources and Skills Development Canada (HRSDC). Data were collected biannually across eight cycles, starting in 1994/95 (Cycle 1) and following until 2008/09 (Cycle 8). Participant ages ranged from 0-11 years old at the time of selection and the first cycle of the NLSCY consisted of 22,831 children, sampled from 13,439 households.[216]

Initial participants in Cycle 1 were selected from the 1994 Statistics Canada's Labour Force Survey with the option to participate.[216] A household roster for each household was established and consisted of details of the relationship of each household member to everyone else in the household, along

with demographics and dwelling conditions.[216] Next, a computer randomly selected a maximum of 4 children per household, along with their respective Person Most Knowledgeable about the child (PMK).[217] The PMK could be any legal guardian, but was predominantly the biological mother (91.3% of PMKs).[218] The PMK was then tasked with completing three sets of questionnaires: General Questionnaire, Parent Questionnaire, and Children's Questionnaire). The General Questionnaire surveyed information about socio-economic status, such as education, labour type, and household/individual income. The Parent Questionnaire surveyed information on the parent's general health status, along with the health status of the PMK's spouse. Information on the household environment, including details of social support and family functioning were also enquired about. Lastly, the Children's Questionnaire was completed and enquired details about the child's/children's health, behaviour, and education. An additional self-completed questionnaire was implemented when the child was 10-11 and was a questionnaire filled out by the children, themselves. The PMK first provided consent for the child to complete their own survey and then the child completed their questionnaire in private and to encourage honest responses, the PMK was not allowed to see their child's responses.[216]

A few noteworthy changes to the NLSCY survey happened across time. In Cycle 2 some participants were dropped from follow-up because of budgetary cutbacks.[217] This was achieved by randomly reducing the number of participants that could be selected per household from 4 to 2, to reduce survey demands per family. This reduced the initial sample of 22,381 to a sample of 16,903 who were

part of the longitudinal cohort. In Cycle 4, individuals who had failed to respond for 2 or more cycles were dropped.[219] Individuals who did not respond for one cycle followed by a temporarily moved status were also removed. These removal rules were applied to all remaining survey cycles (Cycle 5-8).[220–223]

2.2.1 Study Sample

The present study sample used children who were between 0-7 years old, during Cycle 1 (unweighted N=11,952).[216] Being this age at Cycle 1 meant that the child should have survey data completed at all study timepoints, unless missing due to the participants themselves. Due to the present study being primarily interested in elementary-school age children, inclusion criteria required that PMK responded to each of the following surveys when their child was: (1) 6/7 years old, (2) 8/9 years old, and (3) 10/11 years old, and (4) that the child had a self-completed survey between ages 12-15 years old. Ages 6-11 were selected to identify patterns of problem behaviour in children across the majority of elementary school.[216,217,219,224] Ages 12-15 were selected as the age of investigation for study outcomes in early adolescence.[219–223]

Justifications for the selected study age range are that (1) elementary-school children have problem behaviour patterns that are better established and potentially less developmentally transient compared to pre-school children, whose problem behaviour frequently shifts to normalcy as they move out of the pre-school period.[1,69,225–228] (2) suicidal thoughts and other health risk behaviour (e.g. substance and non-substance delinquency) increase substantially during the transition from late childhood to early adolescence[229], and (3) it is desired that the

study outcomes are not too temporally distal from the exposure, to strengthen any inference of study findings. Parent reported measures of problem behaviour were selected as opposed to teacher reported measures, to increase study power due to a much smaller subsample of the data being collected from teacher reports.

2.3 Measures

2.3.1 Primary Independent Variables: Childhood Problem Behaviour

The three types of emotional/behavioural problems that were assessed throughout childhood in the NLSCY include (1) *conduct disorder/physical aggression* (6 items), (2) *hyperactivity/inattention* (7 items), and (3) *emotional disorder/anxiety* (7 items). All three problem behaviour measures are derived from items used in the Ontario Child Health Study (OCHS).[230,231] The items from the OCHS were drawn from the Child Behavior Checklist (CBCL)[17], which is a measurement tool designed to correspond closely to Diagnostics and Statistics Manual (DSM)-III and DSM-IV criteria for mental illness.[232] The CBCL subscales have been studied extensively in terms of their psychometric properties and these measures have been used extensively in pediatric epidemiological research.[230,233–237] Reliability tests for these problem behaviour scales in 4-11 year olds have been found to have good reliability, with Cronbach's Alpha ranging from 0.76-0.78, 0.78-0.84, and 0.69-0.79, for conduct disorder/physical aggression, hyperactivity/inattention, and emotional disorder/anxiety, respectively.[238]

Responses and coding for each item are as follows: "Never or not true", "Sometimes or somewhat true", "Often or very true", "Don't know", or "Refusal". Each of the three scales is derived as the sum of these responses, where

each item is scored from zero (“Never or not true”) to two (“Often or very true”), with a maximum score of 12 for conduct disorder/physical aggression and a maximum score of 14 for hyperactivity/inattention and emotional disorder/anxiety. Items corresponding to each problem behaviour scale are listed below:

Conduct disorder/physical aggression (6 items)
Gets into many fights?
When another child accidentally hurts him/her (such as by bumping into him/her), assumes that the other child meant to do it, and then reacts with anger and fighting?
Physically attacks people?
Threatens people?
Is cruel, bullies, or is mean to others?
Kicks, bites, hits other children?
Hyperactivity/inattention (7 items)
Can’t sit still, is restless, or hyperactive?
Is distractible, has trouble sticking to any activity?
Can’t concentrate, can’t pay attention for long?
Is impulsive, acts without thinking?
Has difficulty awaiting turn in games or groups
Cannot settle to anything for more than a few moments?
Is inattentive?
Emotional disorder/anxiety (7 items)
Seems to be unhappy, sad, or depressed?
Is not as happy as other children?
Is too fearful or anxious?
Is worried?
Cries a lot?
Is nervous, highstrung or tense?

Has trouble enjoying him/herself?

2.3.2 Primary Outcome Variable: Adolescent Suicidal Thoughts

Operationalization of the suicidal thoughts was conducted using a variable which asked the participant the following, "In the past 12 months, did you seriously consider attempting suicide?" The participant can respond in the following ways: "Yes", "No" or "Refusal". This question corresponds to how other similar studies that have used the NLSCY have also operationalized suicidal thoughts.[239,240] In the present investigation this outcome was treated as dichotomous and was computed with a response of "No" (absence of suicidal thoughts) being coded as a 0 and a response of "Yes" being coded as a 1 (presence of suicidal thoughts). A "Refusal" response was coded as missing. The outcome was operationalized based on two timepoints - when the adolescent was 12/13 and when the adolescent was 14/15. If the participant responded "Yes" at either of the timepoints, they were considered as a 1 in the operationalized outcome variable. If an individual was missing data on one of the questionnaires, but had a "Yes" response on the other, they were also coded as a 1. If an individual had missing data on one of the questionnaires, and had a "No" response on the other, they were coded as missing since it was not possible to determine if they had had suicidal thoughts at either time.

2.3.3 Secondary Outcome Variables: Substance and Non-Substance Delinquency

After individuals in the NLSCY reach the age of 10/11 years, a series of questions were asked that enquire about youth health-risk behaviours. The number of

questions increased as the youth got older, to better capture health-risk behaviours that become more prevalent as youths age. The health-risk behaviours used in the present study pertain to substance use and non-substance delinquency. These behaviours were far more prevalent at ages 14/15 years, and research shows the onset of such behaviours to have a later onset in development relative to suicidal thoughts, these outcomes were drawn from only one cycle– when the adolescent was 14/15.[10,11,13,241]

Measures of non-substance delinquency were taken from the 'Risky Behaviour' questionnaire in the NLSCY.[219–223] This questionnaire was adapted from the National Longitudinal Survey of Ohio State University and the Western Australia Child Health Survey.[216,242,243] The items consisted of four possible responses that ranged from 'never' to 5 times or more' and are listed below. Due to the serious nature of the behaviours for early adolescence and to increase study power, all non-substance delinquency items were dichotomized, where a response of 'never' was categorized as a 0 and anything greater than 'never' was categorized as 1 ('yes').

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 sold any drugs?
Have you stolen something from outside of your own home?

Youth were also asked about their use of substances, and to what degree they used them. These substances included smoking cigarettes, alcohol, cannabis, hallucinogens, off-label prescription medication use, glue/inhalants, and other hard drugs (i.e. heroin, cocaine, ecstasy). Items from the Youth Smoking Survey, the WHO Survey on Health Behaviours in School Children, and the Western Australia Child Health Survey were adapted for the smoking questions.[219–223] Questions enquiring about alcohol use were adapted from the Western Australia Child Health Survey and from questions provided by Dr. Richard Tremblay from the University of Montreal.[219–223,243] Questions about all other substance use were adapted from the Northwest Territories Health Attitudes, Knowledge and Behaviours Study.[219–223] The Smoking, Drinking, and Drugs questions are provided in the table below.

Due to the use of smoking, alcohol, and cannabis occurring at a higher frequency, and based on previous research with the NLSCY, these variables were turned into three categories, rather than being dichotomized.[244] These three categories included never used, occasional use (‘I have not used in the past year’, ‘I have had a few (puffs/sips)’, ‘not anymore’, ‘a few times per year’), and regular use (≥ 1-2 times per month). Due to other substance use being rarer and to increase study power, the remaining substances of hallucinogens, glue/solvents, off-label prescription use, and hard drugs (e.g. cocaine, ecstasy, and heroin) were

each created into one variable called “other illicit substances”. A response of “never” for all of these substances was coded as a 0 and the use of any of these substances was coded as a 1.

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’
Drinking		
	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’
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 using hallucinogens in the past 12 months?	6 responses, ranging from ‘I have never done it’ to ‘10 times or more’
	Which of the following best describes your experience with using glue/solvents in the past 12 months?	6 responses, ranging from ‘I have never done it’ to ‘10 times or more’
	Which of the following best describes your experience with using prescription drugs without a prescription in the past 12 months?	6 responses, ranging from ‘I have never done it’ to ‘10 times or more’

	Which of the following best describes your experience with using other drugs (e.g., ecstasy, crack, heroin) in the past 12 months?	6 responses, ranging from 'I have never done it' to '10 times or more'

2.4 Additional Covariates

2.4.1 Gender

Gender differences have been observed in the prevalence of several problem behaviours across the course of childhood.[245–247] Differing rates and patterns of suicidality and delinquency have also been observed between males and females, across adolescence.[244,248] Furthermore, gender differences have been identified in respect of predictors and risk factors for these outcomes as well.[249–253] Due to this, gender was adjusted for in all modelling and was explored as a potential effect modifier in the primary analysis on suicidal thoughts.

2.4.2 Socioeconomic Status

Research has identified individuals from low socioeconomic status (SES) backgrounds to be at an increased risk of emotional/problem behaviour.[254–257] Low SES has also been linked with suicidality, as well as delinquency.[258] Due to the possible confounding nature of low SES on the association of interest, low SES was adjusted for in multivariate logistic regression modelling. Within the NLSCY, a variable was created by Statistics Canada using five variables[216], standardized to have a mean of 0 and a standard deviation of 1, were used to derive a SES score:

- level of education of the PMK
- level of education of the spouse/partner

- prestige of the PMK's occupation
- prestige of the spouse/partner's occupation
- household income

This variable was only measured at baseline, in cycle 1, and therefore the study sample would have ranged from ages 0-7 at this cycle. Despite this, SES has been shown to remain stable across time and therefore this item was deemed to be appropriate for inclusion.[259] Due to interest in low SES, in particular, this variable was dichotomized and those ≤10th percentile were classified as having low SES.

2.4.3 Stressful Life Events

Research has shown stress to be associated with both internalizing and externalizing problem behaviour, as well as an increase in suicidality in adolescents.[260–265] There is also evidence to suggest that high levels of stress in the adolescent period increase the risk of substance use, as well as some evidence showing a link between stress and delinquent behaviours.[266,267] Due to this, an item in the NLSCY on stressful life events, measured prior to the outcome period, at age 10/11, was adjusted for. This item was responded for by the PMK and asked them if in the past 2 years, there have been any events in their child's life that caused the child a great amount of worry or unhappiness. This variable was already dichotomized into a "Yes" or "No" format and therefore included as such in the analyses. A follow up question enquired about which category the stressor fell under: 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.[216] Due to potential variability in what individuals find stressful, and to ensure adequate cell counts, the presence of any of these events was captured as a "Yes" and adjusted for in subsequent analyses. A similar approach to operationalizing stressful life events with the NLSCY has been used in other psychiatric research.[234,268,269]

2.4.4 High Maternal Depressive Symptoms

Research has consistently shown symptoms of maternal depression to increase the risk for earlier engagement in health risk behaviours in children and adolescents.[244] Maternal depression has also been shown to be associated with mental illness in the offspring, as well as suicidality in the offspring.[244,268,270] Due to this, maternal depressive symptoms, when offspring were aged 10/11, was controlled for when measuring the association between childhood problem behaviour transitions and all outcomes of interest. The variable of maternal depressive symptoms in the NLSCY is a scale based on the following questions, which pertain to experiences over the past week:

- "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?"

Each item is responded to on a 4-point scale ranging from "rarely or none of the time" to "most or all of the time". The scale is derived from a shortened form of the widely used and validated Center for Epidemiologic Studies Depression Scale (CES-D).[271–274] A summative score, which ranged from a minimum of 0 to a maximum of 36, was calculated for each mother, at each cycle. Based on previous dichotomization of this variable in the NLSCY, conducted by Weeks et al., (2014), individuals with scores at or below the 10th percentile were deemed to have high maternal depressive symptoms in the present study.[235] While this variable is described as being maternal depressive symptoms, there were also different types of PMK's (e.g. fathers, grandparent, etc.) captured in this variable, however 91.3% of the cases were mothers.

2.4.5 High Maternal Alcohol Use

Maternal alcohol use has been shown to share a positive association with emotional/problem behaviour in their offspring.[275] Furthermore, adolescents who have mothers with problematic drinking have also been shown to be at an increased risk for suicidal thoughts and earlier onset of substance use, and subsequent substance disorders.[276–278] In the NLSCY, the mothers were asked questions relating to their alcohol use. One of the questions asked the mother about the frequency of days in the past year in which they had 5 or more drinks on

one occasion.[219–221,224] One drink was equivalent to 12 oz. of regular beer or 5 oz. of wine or 1.5 oz. of distilled spirits. This information was collected prior to the outcome period, when the child was 10/11 years of age. Overall, the frequency was low, but in order to explore the relationship between high maternal alcohol use and the outcomes of interest, the variable was dichotomized and mothers who reported having 5 or more drinks on one occasion, more than once per month, were determined to have high maternal alcohol use. The National Institute on Alcohol Abuse and Alcoholism defines binge drinking as the consumption of 4 or more drinks on one occasion for females and 5 or more drinks on one occasion for males.[279] While the question in the NLSCY seems to be more closely aligned with the guideline for males, it can still be useful for identifying potentially problematic alcohol use in females and has been used similarly in other NLSCY psychiatric research.[244] While this variable is described as being maternal alcohol use, there were also different types of PMK's (e.g. fathers, grandparent, etc.) captured in this variable, however 91.3% of the cases were mothers.

2.4.6 Parental Status of Home

Research has demonstrated that unstable home environments can contribute to mental illness and that, In particular, individuals coming from one-parent homes have been shown to be at an increased risk for problems in childhood.[280,281] Additional research has also shown that these children are at an increased risk for suicidal behaviours, substance use/abuse, and to be involved in the criminal justice system, later on in adolescence.[282–287] As such, the present study chose to include this as a covariate in subsequent analyses. This was done with an NLSCY

variable that asked the child's caregiver what the parental status of the home was. Three options were possible and included: (1) Two parent household, (2) Single parent household, and (3) other family member/no parent household. A dichotomized "Non-two parent household" was created whereby a response of a two parent household was categorized as a "0" or a "No" and the latter two options were categorized as a "1" or a "Yes".

2.5 Statistical Methodology

The primary independent variables used in this study were profiles of problem behaviour, generated using latent profile analyses, and transition groups of those problem behaviours across time (i.e. the changing status of problem behaviour through childhood). Briefly, latent profile analyses were conducted at three timepoints, when the child was 6/7, 8/9, and 10/11 and each used the three measures of problem behaviour as latent profile indicator variables (i.e. internalizing, hyperactivity/inattention, and conduct problems). Measurement invariance was then tested for using a latent transition analysis. When studying change it is important that the measure does not change so that observed differences through time can be attributed to true changes in behaviour, rather than changes in the way we are addressing something – measurement invariance means that the measure has not changed or is invariant across time. The transition groups were then further divided into subgroup transition clusters (e.g. homotypic, heterotypic) to be used as subsequent independent variables. These independent variables were then used in multivariate logistic regression modelling

to measure their association with suicidal thoughts, delinquency behaviours, and substance use behaviours.

2.5.1 Latent Profile Analyses

In order to identify unobserved patterns of problem behaviour in childhood, the finite mixture modelling technique known as latent profile analysis was selected.[288] The latent profile analysis can be used to identify different subgroups within a broader population. More specifically, it uses distributional assumptions to identify unobserved homogeneous subgroups (i.e. latent profiles) within a broader heterogeneous sample, using a set of measured continuous variables (i.e. indicator variables). Within each latent profile, the indicator variables are assumed to be independent and conditionally Gaussian. Latent profile analyses estimate the posterior probability that an observation belongs to each profile subgroup. Cases will never be perfectly grouped into latent profiles, however, when multiple fit indices are used to select the best-fitting models, this measurement error can be reduced. The key distinction between latent profile analysis and the more commonly used latent class analysis is that the former uses continuous indicators, while the latter uses categorical indicators.[288] Due to the present study being interested in problem behaviours as opposed to clinically diagnosed pathology, continuous indicators were selected to better capture the wide continuum that problem behaviour can occur on.

In the present study, latent profiles were identified at age 6/7, 8/9, and 10/11 using *Mplus* software (Appendix A).[289] The correct, or best-fitting, number of latent profiles was informed by comparison of multiple posterior fit indices.

Model fit was determined by using multiple indices that include: (1) bootstrapped likelihood ratio testing, (2) Bayesian information criterion (BIC), (3) entropy, (4) profile sample sizes, and (5) a rule that an additional profile should differ considerably in shape to other profiles. The bootstrapped likelihood ratio test compares the model fit between a model with *k* profiles (alternative model) and a model with *k-1* profiles (null model). Significance testing for the bootstrapped likelihood ratio test was conducted with an alpha of $p<0.05$. Comparison of BIC values between models was also implemented to inform model selection. BIC identifies the model that is more likely to have generate the observed data. The change in BIC values from one model to another approximates the Bayes factor. The formula for the model fit of the BIC log Bayes factor is as follows:

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

Where B_{10} is the Bayes factor and ΔBIC is the difference between the k profile model minus the k-1 profile model. The model with the smaller BIC is preferred. Mplus reports the relative entropy of a model, which is a rescaled version of traditional entropy scores. Relative entropy can range from 0 to 1 and can be interpreted as a measure of certainty in classifying your model, or, how well it is that you have good delineation of profiles.[290] A score of 0 represents poor delineation of classes and a score of 1 represents excellent delineation of classes.[290] Entropy has been suggested as a useful indice for mixture model selection in textbooks and has been used in similar research.[149,291] In order to maintain sufficient power for subsequent analyses, latent profiles had to consist of a minimum of 1% of the overall sample. Lastly, it was required that in order for

an additional profile to be added to the model, it had to differ in severity and structure, relative to the other profiles. In line with guidelines from Muthén & Muthén (2000), this was done by evaluating centroid and descriptive information to ensure substantive meaning in each profile.[292] Similar research that has investigated patterns of childhood problem behaviour, using latent profile analyses, have also applied rules about the size of each profile and a requirement that profiles differ in structure and severity.[149] A maximum likelihood estimator robust for skewness was incorporated into each model. The inclusion of this maximum likelihood estimator also allowed for semi-complete cases to be included in each latent profile analysis, with the exception that participants could not be missing on all three problem behaviour indices.

2.5.2 Measurement Invariance

In order to assess for measurement equivalence across the three timepoints that comprise the exposure groups/independent variables, measurement variant and measurement invariant models were explored. A measurement invariant model assumes that the relationship between indicator variables and the latent variable are equivalent across time, and thus comparable in a meaningful way.[293] This was done by imposing rules to the models and comparing models where the latent variables and indicators were allowed to vary across time versus where they were not allowed to vary across time.[294] In order to set up a measurement invariant model, and as instructed by Muthen & Asparouhov (2013), first a model was fit in which all classes were uncorrelated with one another.[295] This was done for each of

the three latent profile analyses in order to obtain the starting values which represent the item intercepts of the scales that the latent variable is based on.

A latent transition analysis model was then computed in which the starting values were included as parameters (measurement invariant) and compared to the model in which they were not (measurement variant). A latent transition analysis can be viewed as an extension of a latent profile analysis.[293] Where a latent profile analysis can identify latent subgroups within multiple timepoints, when using longitudinal data, a latent transition analysis can be used to identify the transition between these subgroups, across time. In relation to the present research, a latent transition analysis can be used to compare group structures across time and apply parameters in order to assess the variant or invariant structure of latent groupings. The BIC fit indices were compared between models to identify whether a measurement invariant or variant model was a better fit.

2.5.3 Problem Behaviour Transition Groups

Following the identification of a best-fitting model of invariance/variance, participant's posterior probabilities of belonging to a latent profile at each timepoint were exported from *Mplus* to *SAS.*[289,296] Participants were grouped into each of their respective latent profiles, at each timepoint, based on which profile they had the highest probability of belonging to. The best-fitting models within each timepoint, were selected to have a high entropy score (>0.8). This has been supported in the literature as an appropriate cutoff for hard-grouping latent classes and profiles.[290,297]

Transition groups were created based on profile belongingness at each of the three timepoints. Three measurement timepoints resulted in a high number of transition groups, thus potentially limiting power, so consequently a two timepoint transition group (i.e., from timepoint 1 to timepoint 3) was considered for subsequent analyses and in order to ease interpretation.

2.5.4 Multivariate Logistic Regression

In order to investigate the association between transition groups and the outcomes of interest, multivariate logistic regression modelling was conducted using the *PROC LOGISTIC* procedure in *SAS.*[296] Models were built to have the maximum sample size available for each outcome. Some of the items used as outcomes had more missing data than others, so models were built to have the highest sample size available for each outcome. Two models were computed for each of the outcomes of interest – (1) an unadjusted model, without covariates included, and (2) an adjusted model, with all covariates included. The *GLOGIT* option was used in *SAS* when outcomes had more than two categories – this function allows for the comparison of multiple categories of an outcome to a specified reference, in this case, a response of "Never".

Due to suicidal thoughts being derived from two waves of data for each individual, a sensitivity analysis was conducted on individuals who had a missing response on one cycle questionnaire and a response of "No" on the other. The initial analysis considered these individuals as missing because it cannot be assumed that if they said "No" on one questionnaire, that they would do so on the other. The sensitivity analysis, however, was conducted based on this assumption,

that if they were missing at one timepoint and responded as “No” on the other, that they would be a 0 or a “No” on the operationalized variable.

2.5.5 Missing Data

Due to the longitudinal nature of the data, missingness was an obstacle in the present study. When modelling latent profiles at each of the three timepoints, a maximum likelihood estimator was used. Rather than imputational methods, maximum likelihood estimation uses each observation in the data to compute the parameter that is most likely to have resulted in the observed data. This method requires that the observation have data for at least one of the indicator variables, therefore individuals with missing data on all three indicators, within each timepoint, were removed. The likelihood is calculated separately for individuals with complete data on all three indicators and for individuals with incomplete data (complete data on one or two indicators). In order to avoid introducing bias, individuals missing data on outcomes of interest were removed and those who were removed were compared on sociodemographics, as well as baseline problem behaviour scores.

2.5.6 Weighting

The NLSCY contains a longitudinal weight variable which was taken from cycle 1 and was included to take into account the complexity of the NLSCY and the unique Canadian sample and design effects.[216] Study survey weights were normalized by dividing the survey weight of each individual by the average weight value of the study sample. The nature of the weights needed for correct estimation of the NLSCY was not compatible with the options available in *Mplus*

(where the sum of weights cannot be less than the number of rows in the dataset). This meant it was necessary to fit the final analytical models in SAS.

Chapter 3 – Results

3.1 Latent Profile Analyses

3.1.1 Indicator Descriptive Statistics

Table 1 shows the sample sizes, mean scores and standard deviations of each problem behaviour (i.e. indicator variables), at each timepoint. The mean scores of internalizing were lowest at age 6/7, increasing slightly in both 8/9-year olds and 10/11-year olds. Overall, the mean scores of conduct problems and hyperactivity/inattention seemed to decrease slightly across time. Hyperactivity/Inattention was the most variable indicator variable at all three timepoints. At age 6/7 years of age, a total of 10,234 individuals had complete data on at least one of the three indicators of problem behaviour. By age 8/9 years, 7.2% of the original sample were missing (n=732 missing) and at age 10/11 years, 15.4% of the original sample were missing (n=1,575).

Table 1. Descriptive Statistics for Latent Profile Indicator Variables at ages 6/7, 8/9, and 10/11 years

	Age 6/7			Age 8/9			Age 10/11		
	n	Mean	SD	n	Mean	SD	n	Mean	SD
Hyperactivity/ Inattention*	10234	4.56	3.42	9502	4.28	3.42	8659	3.67	3.28
Internalizing*	10245	2.40	2.40	9503	2.59	2.49	8663	2.54	2.48
Conduct †	10234	1.43	1.87	9502	1.36	1.83	8652	1.19	1.74

n: sample size
SD: standard deviation
*Values range from 0 – 14
†Values range from 0 – 12

3.1.2 Model Fit Indices

Table 2 shows the fit indices that were used to identify the best fitting number of latent profile categories at ages 6/7, 8/9, and 10/11, respectively. At all three timepoints, a 4-profile model was selected as the best fitting model. At ages 6/7, a

5-profile model featured a category which had a prevalence of less than 1% and although a 6-profile model featured groups all above 1%, this model did not differ in entropy from the 4-profile model and also introduced a profile group that was similar to another profile, with only a slight difference in severity. At ages 8/9, each subsequently larger model above a 4-profile model had a profile which was too small and therefore the 4-profile model was selected. At ages 10/11, a 5-profile model also introduced a group that was too similar to one of the other groups, and due to the fit indices being quite similar to the 4-profile model, the 4-profile model was preferred.

Table 2. Latent Profile Analyses Model Fit Indices at ages 6/7, 8/9, and 10/11 years

Timepoint 1 – Age 6/7 (n=10,234)				
Model	BLRT	BIC	Entropy	Smallest profile
1 profile	--	143176.99	--	--
2 profile	P<0.001	136826.92	0.90	14.4%
3 profile	P<0.001	134878.59	0.89	4.9%
4 profile	P<0.001	133491.72	0.87	3.6%
5 profile	P<0.001	132589.90	0.87	<1.0%
6 profile	P<0.001	131867.10	0.87	1.6%
7 profile	P<0.001	130862.44	0.90	<1.0%
Timepoint 2 – Age 8/9 (n=9,502)				
Model	BLRT	BIC	Entropy	Smallest profile
1 profile	--	133069.78	--	--
2 profile	P<0.001	126773.96	0.92	13.9%
3 profile	P<0.001	125445.87	0.82	11.4%
4 profile	P<0.001	123801.92	0.87	3.8%
5 profile	P<0.001	122875.56	0.87	<1.0%
6 profile	P<0.001	122235.25	0.87	<1.0%
7 profile	Model failed to converge	--	--	--
Timepoint 3 – Age 10/11 (n=8,659)				
Model	BLRT	BIC	Entropy	Smallest profile
1 profile	--	119628.25	--	--
2 profile	P<0.001	113505.99	0.93	12.0%
3 profile	P<0.001	111755.74	0.84	9.4%
4 profile	P<0.001	110174.68	0.87	3.9%
5 profile	P<0.001	109528.39	0.87	2.9%
6 profile	Model failed to converge	--	--	--

BIC: Bayesian information criterion
BLRT: Bootstrapped likelihood-ratio testing

3.1.3 Selected Latent Profiles and Measurement Invariance

Table 3 and Figures 1a – 1c show the 4-profile models that were selected for each timepoint, with the respective mean scores of each indicator variable (i.e. hyperactivity/inattention, internalizing, conduct). Similar profiles of problem behaviour were identified at each of the three timepoints. The first profile, defined as the "low all" group, was the largest profile at each time point (72.1% - 73.7%) and was characterized by individuals who scored low on all three indicators of problem behaviour. The second profile, defined as the "Moderate all" group, was the second largest profile at each timepoint (11.9% - 13.8%). These individuals were characterized by moderate scores of all three indicators of problem behaviour. The third and least prevalent profile (3.6% - 3.9%) was defined as the "High all" group and was characterized by high scores of all three indicators of problem behaviour. Lastly, the final profile, and third most prevalent (8.7% - 11.6%), was defined as the "Hyperactivity/inattention and internalizing (Hyper./Inatt. & Int.)" group. These individuals showed patterns of high hyperactivity/inattentiveness, high internalizing, but lower levels of conduct problems.

Based on the identification of similar profiles across each timepoint, and to reduce the amount of transition groups in subsequent analyses, tests of measurement invariance and transition grouping were conducted for ages 6/7, 8/9 and 10/11. After comparing a latent transition model in which constraints were applied (measurement invariant model) and in which constraints were not applied (measurement variant model), the BIC indice for the measurement invariant

model across all three timepoints (BIC = 36,075.766) was favored over the measurement variant model (BIC = 44,759.697). As such, the profiles identified at each of the timepoints were deemed structurally invariant, and treated as the same constructs. With these measurement invariant constraints applied, these individuals were grouped into their most-likely profile of belongingness based on posterior probabilities.

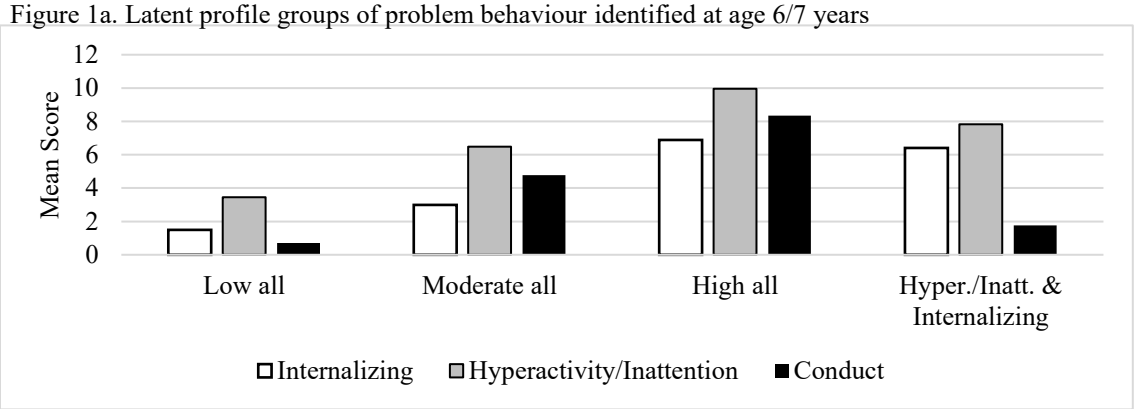
Table 3. Selected Latent Profiles at ages 6/7, 8/9, and 10/11 years

Timepoint 1 – Age 6/7 (n=10,234):		
Profile	Mean	SD
Profile 1 (Low all) [n=7375]		
Hyperactivity/Inattention	3.46	2.72
Internalizing	1.49	1.48
Conduct	0.61	0.80
Profile 2 (Moderate all) [n=1440]		
Hyperactivity/Inattention	6.48	3.10
Internalizing	3.00	1.92
Conduct	4.10	1.03
Profile 3 (High all) [n=366]		
Hyperactivity/Inattention	9.95	3.39
Internalizing	6.88	2.80
Conduct	7.15	1.75
Profile 4 (Hyper./Inatt. & Int.) [n=1053]		
Hyperactivity/Inattention	7.83	3.25
Internalizing	6.41	1.90
Conduct	1.52	1.09
Timepoint 2 – Age 8/9 (n=9,502):		
Profile	Mean	SD
Profile 1 (Low all) [n=7001]		
Hyperactivity/Inattention	3.22	2.65
Internalizing	1.70	1.60
Conduct	0.58	0.78
Profile 2 (Moderate all) [n=1309]		
Hyperactivity/Inattention	6.04	3.20
Internalizing	3.56	2.17
Conduct	4.01	0.97
Profile 3 (High all) [n=365]		
Hyperactivity/Inattention	9.89	3.47
Internalizing	7.18	3.05
Conduct	6.91	1.70
Profile 4 (Hyper./Inatt. & Int.) [n=827]		
Hyperactivity/Inattention	8.04	3.23
Internalizing	6.62	2.07
Conduct	1.24	0.92
Timepoint 3 – Age 10/11 (n=8,659):		
Profile	Mean	SD
Profile 1 (Low all) [n=6293]		

Hyperactivity/Inattention	2.41	2.15
Internalizing	1.74	1.76
Conduct	0.45	0.67
Profile 2 (Moderate all) [n=1028]		
Hyperactivity/Inattention	5.00	2.97
Internalizing	3.54	2.34
Conduct	3.79	0.84
Profile 3 (High all) [n=334]		
Hyperactivity/Inattention	8.73	3.49
Internalizing	6.77	3.21
Conduct	6.81	1.56
Profile 4 (Hyper./Inatt. & Int.) [n=1004]		
Hyperactivity/Inattention	8.55	2.60
Internalizing	5.19	2.57
Conduct	1.25	0.88

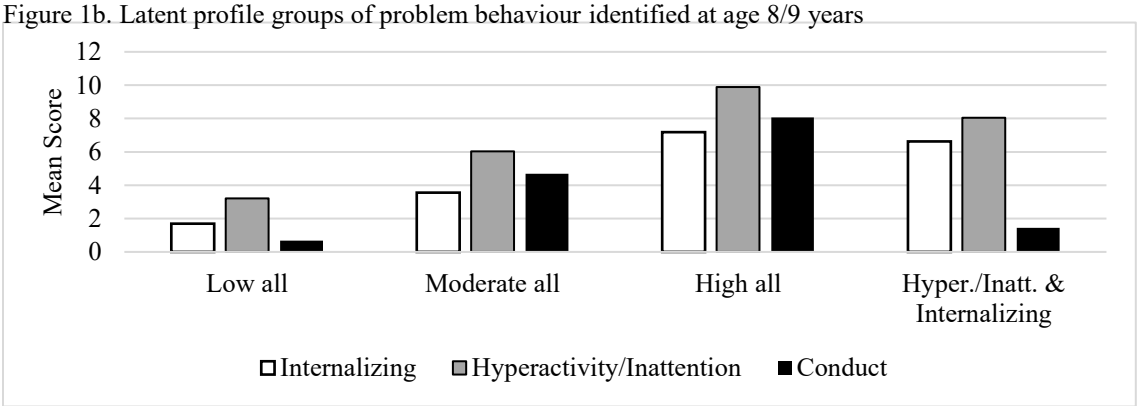
Hyper./Inatt. & Int.: hyperactivity/inattention and internalizing
n: sample size
SD: standard deviation

Figure 1a. Latent profile groups of problem behaviour identified at age 6/7 years



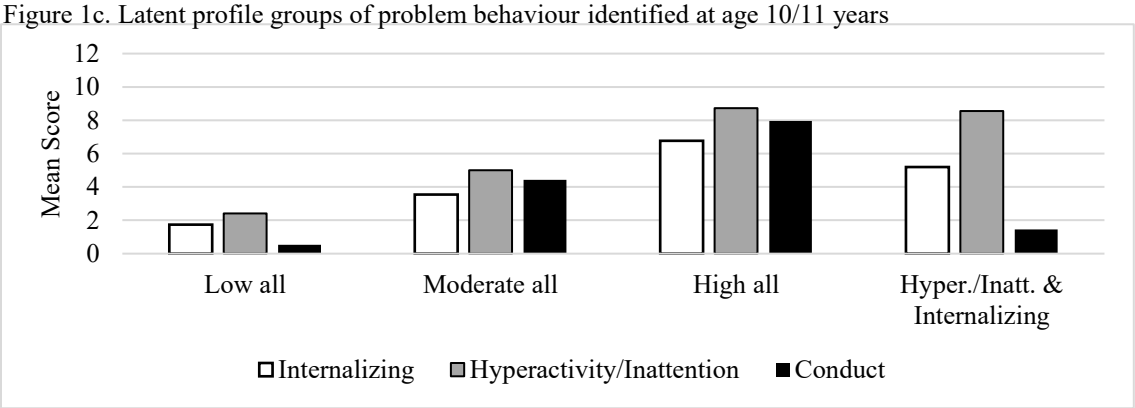
Hyper./Inatt. & Internalizing: Hyperactivity/inattention and internalizing
For comparison to other indicators, conduct has been rescaled to have a maximum score of 14

Figure 1b. Latent profile groups of problem behaviour identified at age 8/9 years



Hyper./Inatt. & Internalizing: Hyperactivity/inattention and internalizing
For comparison to other indicators, conduct has been rescaled to have a maximum score of 14

Figure 1c. Latent profile groups of problem behaviour identified at age 10/11 years



Hyper./Inatt. & Internalizing: Hyperactivity/inattention and internalizing
For comparison to other indicators, conduct has been rescaled to have a maximum score of 14

3.2 Transition Groups

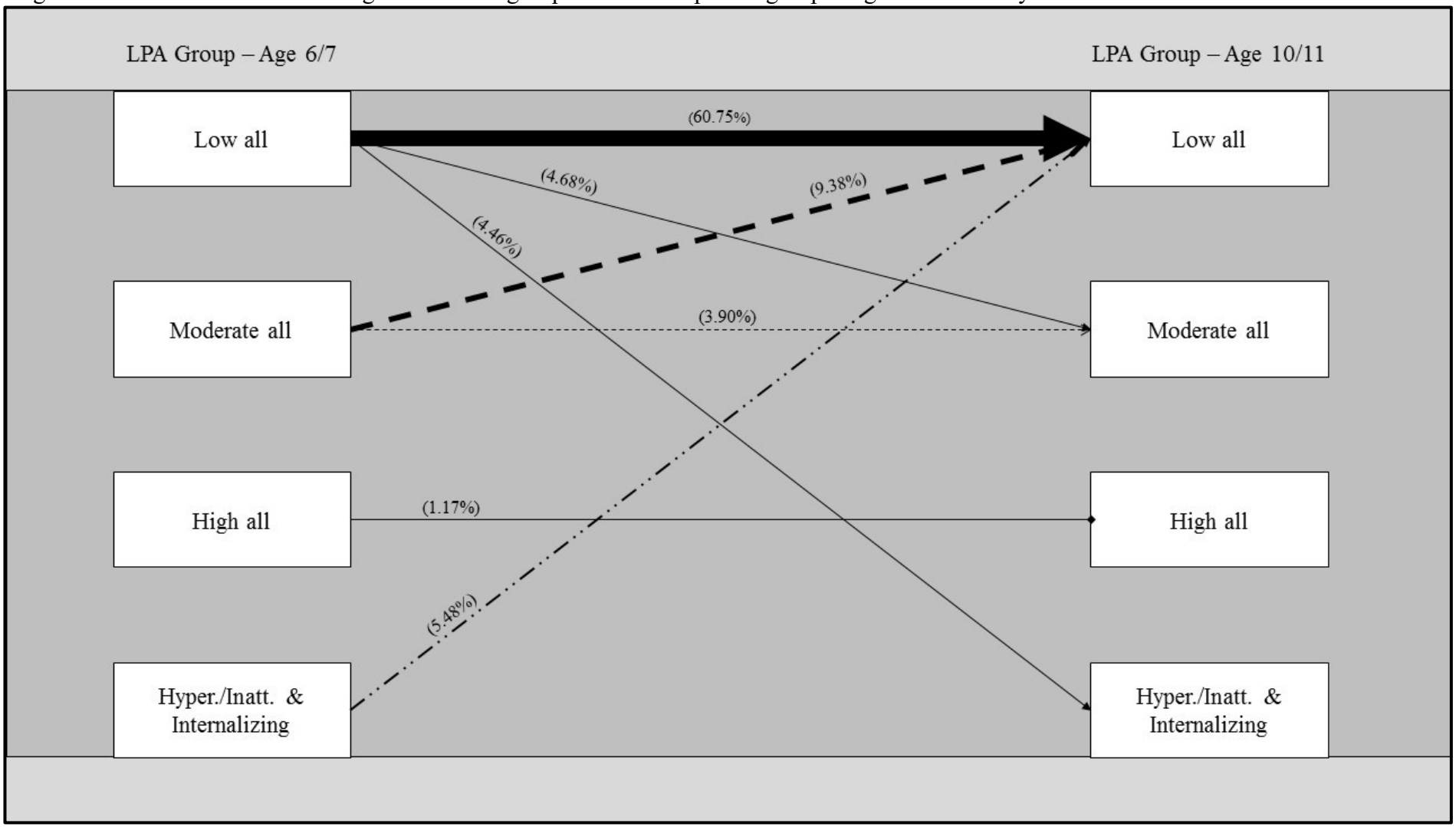
Table 4 shows the transition groups from the latent profile of belongingness at age 6/7 to the latent profile of belongingness at age 10/11. The most prevalent transitions are depicted in Figure 2. A total of 8,266 individuals were assigned a latent profile at each of the two timepoints. This was 80.8% of the individuals in the latent profile analysis at age 6/7 years (non-response analysis found in section 3.7). The majority of individuals stayed in the "Low all" profile across both timepoints (60.8%). A large proportion of individuals who had profiles of either "Moderate all" or "Hyper./Inatt. & Int." at 6/7 transitioned into a "Low all" profile of problem behaviour by age 10/11 (14.9%). A total of 13.7% of individuals were never in the "Low all" profile at either of the timepoints.

Table 4. Frequency of transition groups from latent profile at age 6/7 to age 10/11 years

(*)Transition Group	Weighted %
(A)"Low all" to "Low all"	60.8
(B)"Low all" to "Moderate all"	4.7
(C)"Low all" to "High all"	0.7
(D)"Low all" to "Hyper./Inatt. & Int."	4.5
(E)"Moderate all" to "Low all"	9.4
(F)"Moderate all" to "Moderate all"	3.9
(G)"Moderate all" to "High all"	1.2
(H)"Moderate all" to "Hyper./Inatt. & Int."	1.5
(I)"High all" to "Low all"	0.9
(J)"High all" to "Moderate all"	0.9
(K)"High all" to "High all"	1.2
(L)"High all" to "Hyper./Inatt. & Int."	0.8
(M)"Hyper./Inatt. & Int." to "Low all"	5.5
(N)"Hyper./Inatt. & Int." to "Moderate all"	1.3
(O)"Hyper./Inatt. & Int." to "High all"	0.4
(P)"Hyper./Inatt. & Int." to "Hyper./Inatt. & Int."	2.5

Hyper./Inatt. & Int.: Hyperactivity/inattention and internalizing
*Bracketed letter provided for in-text reference

Figure 2. Prevalence rates of the largest transition groups from latent profile group at age 6/7 to 10/11 years



Hyper./Inatt. & Int.: Hyperactivity/inattention and internalizing
LPA: Latent profile analysis
* Figure lines are weighted based on overall prevalence of each transition group and show the largest transitions from each age 6/7 profile, as well as the remaining transitions >3%

In order to increase statistical power in subsequent logistic regression modelling, transition cluster groups were created and are presented in Table 5. Letter references from Table 4 are provided in brackets. These clusters included [1] Stable low problem behaviour (A); characterized by being in the "low all" profile at both timepoints (60.8%), [2] Decreasing to low problem behaviour (E, I, M); characterized from transitioning from any of the three problem behaviour groups at age 6/7 to the "low all" profile at age 10/11 (15.7%), [3] Increasing from low to problem behaviour (B-D); characterized by individuals who were in the "low all" group at age 6/7 and transitioned into any of the other problem behaviour groups by age 10/11 (9.8%), [4] Homotypic problem behaviour (F, K, P); characterized by staying in the same problem behaviour group across time (7.5%), and [5] Heterotypic problem behaviour (G, H, J, L, N, O); transitioning into a different problem behaviour group across time (6.2%).

Table 6 is a sensitivity analysis of the groupings and is largely the same as in Table 5, with the exception being that the "Moderate all" to "High all" (G) and "High all" to "Moderate all" (J) groups are treated as homotypic transitions instead of heterotypic transitions. This increased the prevalence of the homotypic transition group to 9.6% and decreased the prevalence of the heterotypic transition group to 4.1%. This grouping of the transition group clusters was treated as a distinct independent variable in all subsequent logistic regression modelling.

Table 5. Frequency of transition group clusters

Transition Cluster Groups	Weighted %
Stable low problem behaviour	60.8
Decreasing to low problem behaviour	15.7
Increasing from low to problem behaviour	9.8
Homotypic problem behaviour	7.5
Heterotypic problem behaviour	6.2

Table 6. Frequency of sensitivity grouping of transition clusters

Transition Cluster Groups (Sensitivity)	Weighted %
Stable low problem behaviour	60.8
Decreasing to low problem behaviour	15.7
Increasing from low to problem behaviour	9.8
Homotypic problem behaviour*	9.6
Heterotypic problem behaviour	4.1

* The "Moderate all" to "High all" and "High all" to "Moderate all" transitions are treated as homotypic problem behaviour

3.3 Distribution of Covariates in Latent Profile and Transition Groups

Of those who had complete data on all covariates (n = 7,926; 162 cases removed via listwise deletion [2.7%]), 50.5% of the sample were female, 7.1% were from low SES homes, 17.1% were from single parent homes, 10.5% had a mother with high depressive symptoms, 2.6% had maternal alcohol use, and 32.8% had experienced stressful life events. Table 7 shows the distribution of covariates across the latent profile groups and transition groups, to be used in subsequent logistic regression modelling. At age 6/7 and 10/11, males were significantly more likely to be in any of the three profiles of problem behaviour, relative to females. The highest disparity was observed in the "High all" group, where approximately 70% of the individuals belonging to this group were male. Belonging to a low socioeconomic status family was associated with profile belongingness at both timepoints. A both timepoints, the "High all" group had the highest proportion of low SES, however the profile with the lowest proportion of low SES was the "Moderate all" group at age 6/7 (6.0%) and the "Low all" group at age 10/11 (6.3%). At both timepoints, all problem behaviour groups had significantly higher rates of non-two parent homes relative to the "Low all" group and the highest proportion in the "High all" group. For both age groups high

maternal depressive symptoms was significantly associated with problem behaviour profiles, but the proportion was highest in the “High all” group at age 6/7 years and highest in the “Hyper./Inatt. & Int.” profile at age 10/11 years. High maternal alcohol use was not associated with problem behaviour profiles at age 6/7, but was at age 10/11. The highest proportion was observed in the “Moderate all” group. Lastly, experiencing a stressful life event was associated with problem behaviour profiles at both timepoints and was highest in the “High all” group.

A significant association was observed with transition clusters and all covariates of interest. The “Stable low” group was the only group with a female sex majority (55.4%) and the highest proportion of males was observed in the “Homotypic problems” group (61.3%). The highest proportion of individuals with low SES was observed in the “Heterotypic problems” group (12.9%), followed by the “Low increasing to problems” group (8.7%), and the “Homotypic problems” group (8.6%). Approximately one quarter of individuals in both the “Homotypic problems” group (23.6%) and the “Heterotypic problems” (24.2%) group came from non-two parent homes, with the fewest non-two parent homes being observed in the “Stable low” group (14.2%). High maternal depressive symptoms was highest in the “Heterotypic problems” (22.4%) and “Homotypic problems” (21.9%) groups; stressful life events showed a similar pattern. Lastly, high maternal alcohol use was highest in the “Low to increasing problem” and “Homotypic problem” groups, and lowest in the “Stable low” group.

Table 7. Distribution of covariates in latent profile and transition groups with respective chi-squared tests of independence

Latent Profile at age 6/7 years old					
Covariate	Low all (%)	Moderate all (%)	High all (%)	Hyper./Inatt. & Int. (%)	Chi-Squared (X^2) prob.
Sex (% male)	46.7	60.5	67.6	53.4	<.0001
Low SES	6.4	6.0	13.8	7.9	<.0001
Non-two parent home	14.6	18.6	35.5	21.5	<.0001
High mat. depressive symptoms	8.4	10.3	22.8	21.5	<.0001
High mat. alcohol use	2.3	2.5	3.5	3.5	= 0.1362
Stressful life events	28.8	39.6	48.5	41.6	<.0001
Latent Profile at age 10/11 years old					
Covariate	Low all (%)	Moderate all (%)	High all (%)	Hyper./Inatt. & Int. (%)	Chi-Squared (X^2) prob.
Sex (% male)	47.8	61.1	71.6	53.3	<.0001
Low SES	6.3	10.3	11.6	9.5	<.0001
Non-two parent home	15.9	19.1	31.4	20.5	<.0001
High mat. depressive symptoms	8.0	14.3	20.9	23.3	<.0001
High mat. alcohol use	2.2	4.5	3.3	2.6	= 0.0008
Stressful life events	29.0	38.6	51.9	50.7	<.0001

Transition Cluster Groups (age 6/7 – age 10/11 years old)						
Covariate	Stable Low (%)	Low Increasing to problems (%)	Problems decreasing to low (%)	Homotypic Problems (%)	Heterotypic Problems (%)	Chi-Squared (X^2) prob.
Sex (% male)	44.6	59.4	58.7	61.3	57.6	<.0001
Low SES	6.2	8.9	5.2	8.6	12.9	<.0001
Non-two parent home	14.2	17.6	19.9	23.6	24.2	<.0001
High mat. depressive symptoms	7.5	14.3	10.1	21.9	22.4	<.0001
High mat. alcohol use	2.1	3.8	2.4	3.7	3.4	= 0.0073
Stressful life events	26.8	42.8	36.1	47.2	46.0	<.0001

Hyper./Inatt. & Int.: Hyperactivity/inattention and internalizing
Mat.: Maternal
SES: Socioeconomic status

3.4 Descriptive Statistics for Health-risk Outcomes

3.4.1 Prevalence of Health-risk Outcomes

Table 8 shows the prevalence of all health-risk outcomes that were used in logistic regression modelling. The prevalence of suicidal thoughts at age 14/15 was approximately 3% higher than at age 12/13 years. The combined prevalence of suicidal thoughts from age 12 – 15 years was 13.8%, and was slightly lower in the sensitivity grouping (9.9%). The most prevalent substance use involvement were occasional uses of alcohol, smoking, and cannabis. Regular alcohol use and illicit substance use were the least prevalent substances used, each being endorsed by only about 5.5% of the sample. Of the non-substance use delinquent behaviour, the most prevalent were staying out all night without permission, vandalism, and being questioned by the police for suspected involvement in a crime. The least prevalent non-substance use delinquent behaviours were selling drugs and fighting someone to the point of injury.

Table 8. Weighted prevalence of health-risk outcome behaviours and respective sample sizes

Outcome	Unweighted n	Weighted prevalence (%)
Suicidal Thoughts (age 12/13)	6926	5.2
Suicidal Thoughts (age 14/15)	6521	8.4
Suicide Thoughts (Combined)*	5692	13.8
Suicide Thoughts (Sensitivity)**	7884	9.9
Alcohol		
Occasional	6526	70.1
Regular		5.4
Smoking		
Occasional	6581	24.5
Regular		7.0
Cannabis		
Occasional	6601	19.8
Regular		6.7
Illicit substance use (excluding cannabis)	6541	5.7
Sold drugs	6583	6.1
Stayed out all night	6179	19.8
Ran away from home	6170	8.2
Questioned by police as suspect	6178	18.1
Stealing	6511	6.9
Vandalism	6199	18.3
Fighting to the point of injury	6583	5.8

n = sample size

* Combined prevalence of two timepoints, reported at age 12/13 or age 14/15 years

** For incomplete cases (i.e. at age 12/13 or 14/15), missing is treated as a "No" response

3.4.2 Distribution of Covariates in Primary Outcome – Suicidal Thoughts

Table 9 shows the distribution of covariates among adolescents with and without suicidal thoughts. All covariates except for low SES (p=0.05) and high maternal alcohol use (p=0.20) were significantly associated with suicidal thoughts. Individuals experiencing suicidal thoughts were more likely to be female (65.3%), come from non-two parent homes (20.6%), have a caregiver with high maternal depressive symptoms (15.5%), and to have experienced stressful life events (38.7%).

Table 9. Distribution of covariates among adolescents with and without suicidal thoughts, with respective chi-squared tests of independence

Covariate	Suicidal Thoughts		Chi-Squared (X^2) prob.
	No (%)	Yes (%)	
Sex (% male)	52.4	34.7	<.0001
Low SES	6.6	8.5	0.0531
Non-two parent home	16.3	20.6	0.0036
High maternal depressive symptoms	9.7	15.5	<.0001
High maternal alcohol use	2.6	3.4	0.2032
Stressful life events	32.2	38.7	0.0006

SES: Socioeconomic status

3.4.3 Distribution of Suicidal Thoughts in Independent Variables

Table 10 shows the prevalence of suicidal thoughts throughout the latent profile groups, as well as the transition cluster groups (i.e. non-sensitivity and sensitivity groupings). Individuals in the "High all" profile at age 6/7 years had the highest rates of suicidal thoughts at ages 12-15, followed by those in the "Hyper./Inatt. & Int." profile, "Moderate all" profile, and lastly, the "Low all" profile. Contrasted to this, individuals in the "Moderate all" profile at age 10/11 years had the highest rates of suicidal thoughts in early adolescence (age 12 – 15). Overall, less variability was observed in the rate of suicidal thoughts among the three problem

behaviour profiles at age 10/11 (18.1 – 19.4%) than at age 6/7 years (13.6 – 25.7%). The prevalence of suicidal thoughts among individuals in the homotypic and heterotypic transition cluster groups was highest, each with about one fifth of their members reporting suicidal thoughts. The next largest was the "low increasing to problems" group, with 17.4% reporting suicidal thoughts. Individuals in the "Stable low" group, as well as those whose problem behaviour decreased across time had similar rates of suicidal thoughts, approximately 11%. The sensitivity grouping of the transition clusters elevated the rate of suicidal thoughts in the homotypic group by 0.7% and decreased the rate in the heterotypic group by 3.0%, with the "low increasing to problems" prevalence being slightly higher than the heterotypic group.

Table 11 shows the prevalence of the sensitivity grouping of suicidal thoughts throughout the latent profile groups, as well as the transition cluster groups. While the order of the highest to lowest rates of suicidal thoughts throughout the problem behaviour profiles and transition clusters is near identical to the standard grouping, the magnitude of the prevalence rates was an average of 4.5% lower when using the sensitivity grouping of suicidal thoughts.

Table 10. Distribution of suicidal thoughts throughout latent profile groups and transition cluster groups with respective chi-squared tests of independence

Independent Variable	Suicidal Thoughts		Chi-Squared (X^2) prob.
	No (%)	Yes (%)	
LPA Age 6/7			<.0001
Low all	87.6	12.4	
Moderate all	86.5	13.6	
High all	74.3	25.7	
Hyper./Inatt. & Int.	83.5	16.5	
LPA Age 10/11			<.0001
Low all	88.4	11.6	
Moderate all	80.1	19.9	
High all	81.9	18.1	
Hyper./Inatt. & Int.	80.6	19.4	
Transition Cluster Groups			<.0001
Stable low	88.7	11.3	
Any problems decreasing to low	88.6	11.4	
Low increasing to Problems	82.6	17.4	
Homotypic problems	79.1	20.9	
Heterotypic problems	81.3	18.7	
Transition Cluster Groups (Sensitivity)			<.0001
Stable low	88.7	11.3	
Any problems decreasing to low	88.6	11.4	
Low increasing to Problems	82.6	17.4	
Homotypic problems	78.4	21.6	
Heterotypic problems	84.3	15.7	

Hyper./Inatt. & Int.: Hyperactivity/inattention and internalizing
LPA: Latent profile analysis

Table 11. Distribution of suicidal thoughts (sensitivity grouping) throughout latent profile groups and transition cluster groups with respective chi-squared tests of independence

Independent Variable	Suicidal Thoughts (Sensitivity)		Chi-Squared (X^2) prob.
	No	Yes	
Problem Behaviour Profile Age 6/7			<.0001
Low all	91.1	8.9	
Moderate all	89.9	10.1	
High all	83.1	17.0	
Hyper./Inatt. & Int.	88.5	11.5	
Problem Behaviour Profile Age 10/11			<.0001
Low all	91.6	8.4	
Moderate all	85.1	14.9	
High all	87.6	12.4	
Hyper./Inatt. & Int.	85.5	14.5	
Transition Cluster Groups			<.0001
Stable low	91.8	8.2	
Any problems decreasing to low	91.8	8.2	
Low increasing to Problems	87.1	13.0	
Homotypic problems	84.8	15.2	
Heterotypic problems	86.1	14.0	
Transition Cluster Groups (Sensitivity)			<.0001
Stable low	91.8	8.2	
Any problems decreasing to low	91.8	8.2	
Low increasing to Problems	87.1	13.0	
Homotypic problems	84.1	15.9	
Heterotypic problems	88.4	11.6	

Hyper./Inatt. & Int.: Hyperactivity/inattention and internalizing
LPA: Latent profile analysis

3.5 Logistic Regression Models of Health-risk Outcomes

Tables 12 – 19 show the logistic regression models in the associations between the following exposures: (1) LPA group at age 6/7, (2) LPA group at age 10/11, (3) Transition cluster group, and (4) Transition cluster sensitivity group (Appendix B), with all primary and secondary health-risk outcomes. Tables 13, 14, and 15 are multinomial logistic regression models, where the outcomes are ordinal (>2 categories), while the remaining outcomes are modelled binomially (2 categories). Due to inadequate cell sizes in the association between the 16 transition groups and the outcomes of interest, only the transition cluster groups are presented. Each outcome analysis was modelled to include the maximum amount of complete cases and both an unadjusted and adjusted model are presented for each outcome. Respective sample sizes, odds-ratios, and upper/lower confidence limits are presented in each table.

Table 12 shows the outcomes of suicidal thoughts, as well as its respective sensitivity analysis. Relative to those in the “low all” profile at age 6/7, individuals in the “High all” group showed a two and half fold increase in the odds of suicidal thoughts, while the “Hyper./Inatt. & Int.” group showed a 33% increase in the odds of suicidal thoughts. This association was not observed in the “Moderate all” group. At ages 10/11, belonging to any of the three problem behaviour groups was associated with a 73-83% increase in the odds of suicidal thoughts in adolescence. A similar trend was observed in both the Transition cluster groups and the Transition cluster sensitivity groupings (Appendix B). Relative to those who stayed in the “low all” profile at both timepoints,

heterotypic, homotypic, and increasing problem behaviour transitions were all associated with between 72-94% increased odds of suicidal thoughts in adolescence (83-97% for sensitivity grouping of suicidal thoughts). Individuals who decreased from a problem behaviour group at age 6/7 to the "low all" profile at age 10/11 did not have increased odds of suicidal thoughts, relative to those who stayed low across both timepoints. No evidence supported that there was a difference in the strength of the association with suicidal thoughts based on which significant problem behaviour profile/transition group the individual belonged to.

Tables 13 – 16 show the substance use outcomes of alcohol, cigarettes, cannabis, and illicit substances (not including cannabis). At ages 6/7, belonging to the "Moderate all" and "High all" profiles had the strongest association with substance use. While both profiles showed similar odds of being involved in most substance use, the "Moderate all" profile was associated with the more serious, illicit substance use, while the "High all" profile was not. However, issues of power surrounding the smaller "High all" profile may be an explanation. No significant observations were identified between the "Hyper./Inatt. & Int." profile and any types of substance use. This trend was similar at ages 10/11, in that the "Moderate all" and "High all" profiles were associated with overall higher odds of substance use involvement. The "Hyper./Inatt. & Int." profile was associated with occasional cigarette use at this age, but no profile was associated with increased odds of illicit substance use. When looking at the transition groups, overall, individuals with persisting patterns of problem behaviour (homotypic or heterotypic) had higher odds of substance use, relative to those who showed

stable low problem behaviour or decreased to low problem behaviour across time. Homotypic patterns of problem behaviour were associated with regular alcohol, cigarette, and cannabis use. Heterotypic patterns of problem behaviour were associated with regular alcohol and cigarette use, but only occasional cannabis use. Individuals in the increasing problem behaviour group were at risk for occasional alcohol and cigarette use and, interestingly, the individuals who decreased to levels comparable to the stable low group were at an increased risk for regular cigarette use and illicit substance use.

Tables 16 – 19 show the delinquency outcomes of selling drugs, staying out all night, running away from home, being questioned by police as a suspect, stealing outside of the home, vandalism, and fighting to the point of injury. Similarly to substance use, the heterotypic and homotypic groups were associated with the most different types of delinquency. Heterotypic problem behaviour was associated with running away from home, being questioned by the police, vandalism, and fighting. Homotypic problem behaviour was associated with selling drugs, running away from home, being questioned by the police, and vandalism. The sensitivity analysis showed that when a transition from "Moderate all" to "High all" or vice versa were treated as homotypic, the heterotypic group was no longer associated with running away from home or being questioned by the police. When categorized in this way, homotypic transitions became significantly associated with fighting. Increasing into problem behaviour was associated with running away from home and fighting. Decreasing problem behaviour was only associated with stealing.

An interaction between latent profiles/transition cluster groups and gender was explored in the association with suicidal thoughts. This was done for the latent profiles/transition cluster groups and the sensitivity transition cluster groups for the outcomes of suicidal thoughts, as well as the sensitivity group of suicidal thoughts. None of these analyses yielded a significant interaction between gender and latent profiles/transition cluster groups.

Table 12. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on suicidal thoughts

	Suicidal Thoughts								Suicidal Thoughts (sensitivity)							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	5385				4960				7480				6780			
Moderate all		1.11	0.90	1.37		1.15	0.91	1.45		1.14	0.93	1.41		1.17	0.93	1.47
High all		2.45	1.74	3.45		2.53	1.74	3.68		2.08	1.51	2.88		2.32	1.63	3.31
Hyper./Inatt. & Int.		1.40	1.09	1.79		1.33	1.01	1.75		1.33	1.05	1.70		1.27	0.97	1.65
LPA age 10/11 Reference: "Low all"	5250				5034				7245				6892			
Moderate all		1.89	1.51	2.36		1.83	1.44	2.33		1.92	1.54	2.38		1.85	1.46	2.34
High all		1.67	1.13	2.47		1.81	1.21	2.72		1.55	1.06	2.26		1.73	1.17	2.57
Hyper./Inatt. & Int.		1.83	1.44	2.33		1.73	1.34	2.25		1.86	1.47	2.35		1.81	1.41	2.33
Transition clusters Reference: "Stable low"	5019				4884				6921				6685			
Decreasing to low		1.02	0.79	1.30		1.09	0.84	1.40		1.00	0.79	1.27		1.08	0.84	1.38
Low to problems		1.65	1.28	2.14		1.72	1.32	2.25		1.68	1.30	2.16		1.83	1.41	2.37
Homotypic problems		2.08	1.59	2.72		1.94	1.45	2.60		2.03	1.56	2.63		1.97	1.49	2.61
Heterotypic problems		1.81	1.34	2.45		1.91	1.39	2.63		1.83	1.36	2.45		1.90	1.40	2.59

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 13. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on alcohol use

	Alcohol													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	6264				5743									
Moderate all		1.23	1.04	1.45		1.18	0.99	1.40	1.46	1.06	2.00	1.40	1.00	1.95
High all		1.72	1.18	2.49		1.64	1.12	2.40	2.84	1.60	5.04	2.64	1.45	4.79
Hyper./Inatt. & Int.		0.80	0.66	0.96		0.85	0.69	1.04	1.13	0.78	1.64	1.25	0.84	1.84
LPA age 10/11 Reference: "Low all"	6097				5826									
Moderate all		1.41	1.15	1.73		1.45	1.17	1.80	1.82	1.26	2.61	1.88	1.29	2.74
High all		1.59	1.12	2.26		1.64	1.14	2.36	1.16	0.56	2.42	1.19	0.56	2.50
Hyper./Inatt. & Int.		1.06	0.86	1.29		1.11	0.90	1.38	1.26	0.85	1.86	1.22	0.81	1.85
Transition clusters Reference: "Stable low"	5834				5656									
Decreasing to low		0.98	0.83	1.16		0.97	0.81	1.15	1.18	0.84	1.66	1.18	0.84	1.67
Low to problems		1.26	1.02	1.56		1.27	1.02	1.58	1.04	0.66	1.65	1.03	0.65	1.65
Homotypic problems		1.50	1.17	1.93		1.60	1.23	2.09	2.10	1.36	3.24	2.19	1.40	3.44
Heterotypic problems		1.12	0.87	1.44		1.18	0.91	1.54	1.78	1.13	2.79	1.78	1.12	2.85

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 14. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on cigarette use

	Cigarette													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	6294				5773									
Moderate all		1.10	0.94	1.30		1.13	0.95	1.34	2.09	1.65	2.65	2.26	1.75	2.91
High all		1.73	1.29	2.32		1.63	1.20	2.23	1.96	1.20	3.18	1.48	0.86	2.55
Hyper./Inatt. & Int.		1.17	0.96	1.42		1.19	0.96	1.46	1.59	1.16	2.18	1.37	0.97	1.95
LPA age 10/11 Reference: "Low all"	6127				5858									
Moderate all		1.18	0.97	1.43		1.18	0.96	1.44	1.91	1.45	2.53	1.77	1.31	2.39
High all		1.01	0.72	1.41		1.00	0.71	1.41	2.31	1.52	3.52	1.86	1.18	2.95
Hyper./Inatt. & Int.		1.48	1.22	1.79		1.51	1.24	1.85	1.27	0.90	1.80	1.11	0.77	1.61
Transition clusters Reference: "Stable low"	5862				5687									
Decreasing to low		1.13	0.95	1.34		1.16	0.97	1.38	1.53	1.15	2.03	1.60	1.19	2.14
Low to problems		1.24	1.01	1.52		1.28	1.04	1.58	1.20	0.82	1.73	1.07	0.72	1.59
Homotypic problems		1.27	1.01	1.60		1.31	1.03	1.67	2.54	1.83	3.51	2.36	1.67	3.34
Heterotypic problems		1.49	1.17	1.90		1.50	1.16	1.93	2.40	1.67	3.45	2.06	1.40	3.04

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 15. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on cannabis use

	Cannabis													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	6316				5794									
Moderate all		1.40	1.19	1.65		1.43	1.20	1.70	1.80	1.41	2.30	1.65	1.27	2.14
High all		1.17	0.82	1.65		1.01	0.70	1.48	2.82	1.88	4.22	2.27	1.48	3.47
Hyper./Inatt. & Int.		0.95	0.77	1.18		0.99	0.79	1.25	0.93	0.65	1.35	0.77	0.51	1.15
LPA age 10/11 Reference: "Low all"	6152				5880									
Moderate all		1.50	1.24	1.82		1.46	1.20	1.79	1.31	0.95	1.79	1.24	0.89	1.71
High all		1.59	1.14	2.21		1.54	1.10	2.17	2.82	1.88	4.23	2.17	1.41	3.34
Hyper./Inatt. & Int.		1.25	1.02	1.54		1.21	0.97	1.51	0.68	0.45	1.04	0.65	0.43	1.00
Transition clusters Reference: "Stable low"	5886				5708									
Decreasing to low		1.07	0.88	1.29		1.07	0.89	1.30	1.24	0.93	1.65	1.17	0.87	1.56
Low to problems		1.25	1.00	1.55		1.24	0.99	1.55	0.78	0.52	1.18	0.71	0.47	1.08
Homotypic problems		1.38	1.08	1.76		1.36	1.05	1.75	2.37	1.73	3.25	2.11	1.52	2.92
Heterotypic problems		1.83	1.44	2.33		1.84	1.43	2.36	0.94	0.58	1.54	0.84	0.51	1.38

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 16. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on illicit substances and selling drugs

	Illicit Substance Use (Excluding Cannabis)								Selling Drugs							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	6255				5737				6293				5768			
Moderate all		1.28	0.97	1.69		1.42	1.06	1.90		1.80	1.41	2.32		1.66	1.27	2.16
High all		1.59	0.96	2.62		1.33	0.74	2.37		3.08	2.05	4.61		2.40	1.54	3.73
Hyper./Inatt. & Int.		1.24	0.88	1.76		1.36	0.95	1.96		1.06	0.73	1.53		0.89	0.59	1.34
LPA age 10/11 Reference: "Low all"	6094				5825				6127				5854			
Moderate all		1.06	0.75	1.50		1.07	0.75	1.54		1.15	0.83	1.60		1.14	0.81	1.59
High all		1.36	0.81	2.29		1.46	0.85	2.50		2.41	1.59	3.64		2.06	1.34	3.17
Hyper./Inatt. & Int.		1.02	0.70	1.47		1.02	0.69	1.50		0.77	0.51	1.17		0.69	0.45	1.07
Transition clusters Reference: "Stable low"	5826				5651				5861				5682			
Decreasing to low		1.36	1.02	1.82		1.46	1.08	1.98		1.36	1.02	1.81		1.27	0.95	1.71
Low to problems		1.08	0.74	1.58		1.12	0.76	1.67		0.75	0.48	1.15		0.69	0.45	1.08
Homotypic problems		1.22	0.82	1.84		1.34	0.88	2.04		2.41	1.75	3.31		2.16	1.54	3.01
Heterotypic problems		1.16	0.74	1.82		1.19	0.75	1.90		0.87	0.52	1.44		0.81	0.49	1.36

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 17. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on staying our all night and running away from home

	Staying out all night								Running away from home							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	5907				5424				5900				5415			
Moderate all		1.32	1.12	1.56		1.19	1.00	1.43		1.24	0.97	1.59		1.26	0.97	1.65
High all		1.73	1.25	2.39		1.23	0.87	1.75		2.67	1.81	3.95		2.29	1.48	3.53
Hyper./Inatt. & Int.		0.99	0.79	1.24		0.86	0.68	1.10		0.84	0.59	1.19		0.85	0.58	1.23
LPA age 10/11 Reference: "Low all"	5762				5504				5754				5496			
Moderate all		1.24	1.02	1.52		1.20	0.97	1.48		1.92	1.48	2.50		1.71	1.29	2.26
High all		1.39	1.01	1.92		1.13	0.81	1.59		2.64	1.80	3.88		2.58	1.73	3.84
Hyper./Inatt. & Int.		1.08	0.86	1.34		0.88	0.70	1.12		0.99	0.70	1.40		0.88	0.61	1.27
Transition clusters Reference: "Stable low"	5511				5345				5502				5336			
Decreasing to low		1.17	0.97	1.40		1.12	0.93	1.36		0.97	0.72	1.31		1.02	0.75	1.38
Low to problems		1.23	0.99	1.53		1.12	0.90	1.41		1.47	1.08	2.02		1.46	1.06	2.02
Homotypic problems		1.11	0.86	1.43		0.95	0.73	1.24		1.84	1.33	2.55		1.78	1.27	2.51
Heterotypic problems		1.53	1.19	1.98		1.30	1.00	1.71		1.64	1.14	2.38		1.57	1.07	2.30

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 18. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on being questioned by the police for suspected involvement in a crime and stealing outside of the home

	Questioned by police for suspected involvement in crime								Steal outside of the home							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	5908				5423				6211				5693			
Moderate all		1.38	1.16	1.64		1.33	1.11	1.60		1.65	1.30	2.10		1.51	1.17	1.95
High all		2.59	1.90	3.53		1.99	1.42	2.77		1.74	1.09	2.76		1.19	0.71	2.01
Hyper./Inatt. & Int.		1.11	0.89	1.40		1.00	0.78	1.28		1.12	0.80	1.58		1.02	0.71	1.47
LPA age 10/11 Reference: "Low all"	5760				5504				6121				5847			
Moderate all		1.61	1.32	1.97		1.44	1.17	1.77		1.32	0.99	1.77		1.29	0.96	1.75
High all		2.53	1.87	3.42		2.05	1.50	2.81		1.91	1.25	2.92		1.52	0.97	2.39
Hyper./Inatt. & Int.		1.40	1.12	1.74		1.25	0.99	1.57		0.67	0.45	1.00		0.69	0.46	1.04
Transition clusters Reference: "Stable low"	5510				5344				5856				5677			
Decreasing to low		1.04	0.85	1.27		0.98	0.79	1.20		1.60	1.23	2.09		1.50	1.15	1.97
Low to problems		1.19	0.94	1.50		1.03	0.81	1.32		1.33	0.95	1.86		1.26	0.89	1.77
Homotypic problems		2.22	1.77	2.79		1.94	1.53	2.47		1.39	0.96	2.01		1.30	0.89	1.90
Heterotypic problems		1.77	1.37	2.30		1.51	1.15	1.98		1.17	0.76	1.80		1.09	0.71	1.69

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 19. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on vandalism/destruction of property and fighting to the point of injury

	Vandalism								Fighting to point of injury							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
LPA age 6/7 Reference: "Low all"	5918				5429				6293				5768			
Moderate all		1.56	1.32	1.85		1.42	1.19	1.70		1.38	1.05	1.82		1.19	0.89	1.59
High all		3.21	2.38	4.34		2.50	1.81	3.44		2.56	1.65	3.97		1.59	0.97	2.59
Hyper./Inatt. & Int.		1.06	0.84	1.34		0.97	0.75	1.24		1.09	0.75	1.57		0.93	0.62	1.38
LPA age 10/11 Reference: "Low all"	5762				5504				6127				5855			
Moderate all		1.94	1.60	2.35		1.78	1.46	2.19		2.08	1.56	2.79		1.76	1.29	2.39
High all		2.38	1.76	3.23		1.99	1.45	2.73		2.37	1.51	3.71		1.58	0.98	2.57
Hyper./Inatt. & Int.		1.01	0.79	1.28		0.94	0.73	1.21		1.57	1.12	2.20		1.47	1.03	2.09
Transition clusters Reference: "Stable low"	5511				5344				5861				5682			
Decreasing to low		1.25	1.03	1.52		1.15	0.94	1.41		1.20	0.87	1.67		1.02	0.73	1.42
Low to problems		1.23	0.98	1.55		1.14	0.89	1.45		1.97	1.42	2.74		1.61	1.14	2.26
Homotypic problems		2.14	1.70	2.69		1.90	1.49	2.43		1.77	1.21	2.58		1.42	0.96	2.10
Heterotypic problems		1.85	1.43	2.40		1.68	1.28	2.21		2.14	1.45	3.15		1.78	1.19	2.66

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis – profiles of problem behaviour
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

3.6 Non-Response Analysis

A non-response analysis was conducted to compare latent profile and covariate differences between those assigned a transition group (n = 8,266) who did (31.3%) and did not (68.7%) have missing data on the primary outcomes of suicidal thoughts. Individuals with missing data were more likely to belong to the "Low all", "High all" and "Hyper./Inatt. & Int." groups at age 6/7 years (X^2=29.06; p<0.0001). No significant difference was observed in profile belongingness at age 10/11 years (X^2=2.98; p=0.39). Those with missing data were more likely to be male (X^2=24.12; p<0.0001), from a low SES group (X^2=84.95; p<0.0001), come from a non-two parent home (X^2=60.71; p<0.0001), and report higher rates of maternal depressive symptoms (X^2=23.37; p<0.0001). No significant difference was observed for maternal alcohol use (X^2=0.72; p=0.40), or stressful life events (X^2=0.05; p=0.82). This analysis was replicated based on those who were missing outcome data for the sensitivity grouping of suicidal thoughts (4.6%). The results were nearly identical with the exception that those individuals missing were more likely to belong to the "Low all" profile and less likely to belong to the "Moderate all", but did not differ on the "High all" or "Hyper./Inatt. & Int." profiles, at age 6/7 years (X^2=32.24; p<0.0001).

Chapter 4 – Discussion

An abundance of research has been conducted on the topic of problem behaviour in elementary school children.[4,6,7,25] Research has looked at how patterns of problem behaviour in childhood predict health-risk outcomes in adolescence, such as mental disorder diagnoses, suicidality, substance use, and other types of delinquency.[162,170,177,179,185,191] Despite this, a limited amount of research has been identified that looked at the association between transition patterns of problem behaviour across childhood and health-risk outcomes in adolescence. As such, the present study sought to carry out three primary goals: (1) identify profiles of problem behaviour at multiple time points in elementary school children, (2) examine the transition pathways the problem behaviour takes across time, and (3) investigate the association between these child problem behaviour profiles/transitions and health-risk outcomes in early adolescence.

The initial hypotheses of the present study were that four profiles of problem behaviour would be observed, including a profile low on all symptoms of problem behaviour, a predominantly internalizing group, a predominantly externalizing group, and a comorbid internalizing and externalizing group. While four profiles of problem behaviour were observed in the present findings, the results and the hypotheses deviated in respect to what the patterns of problem behaviour looked like, in that greater patterns of comorbidity between internalizing and externalizing were observed. A profile low on all symptoms was observed (i.e. “Low all” profile), as well as a highly comorbid internalizing and externalizing group (i.e. “High all” profile), but the internalizing-only and

externalizing-only profiles were not observed. Instead, the other two profiles of problem behaviour were varying types of comorbid internalizing and externalizing (i.e. “Moderate all” and “Hyper./Inatt. & Int.” profiles).

The subsequent hypotheses about transition patterns of problem behaviour were developed in relation to what patterns of problem behaviour were predicted to be observed a priori, and therefore, differed from what was observed. From ages 6/7 to 10/11 years, no homotypic pattern of internalizing-only or externalizing-only were observed, nor were pure transitions from externalizing to internalizing observed. However, transition patterns of persistently low problem behaviour, decreasing problem behaviour, and persisting patterns of comorbid internalizing and externalizing, were hypothesized a priori and were observed in the present findings. Another key deviation from what was hypothesized was that the most recent profile of problem behaviour (i.e. at age 10/11 years) predicted health-risk outcomes similarly to the transition groups of problem behaviour. It had been hypothesized that individuals who showed persistence in their problem behaviour, for internalizing-only, externalizing-only, and comorbid internalizing and externalizing, would have the strongest associations to adolescent health-risk outcomes. While patterns of persistent problem behaviour did have a positive association with most health-risk behaviours, these associations did not differ from the most recent profile of problem behaviour at predicting these outcomes.

4.1 Summary of Main Results

4.1.1 Profiles of Problem Behaviour

At all three of the time points in which problem behaviour patterns were explored (i.e. ages 6/7, 8/9, and 10/11 years), the same four groups reappeared and were confirmed to be structurally the same across time, suggesting that unique profiles of problem behaviour remain stable across elementary school. The first, and a commonly identified group in the literature, was labelled as the "low all" profile.[53,149,152,298–300] These individuals were characterized as having low scores of conduct problems, hyperactivity/inattention, and internalizing. Similar research which has looked at patterns of internalizing and externalizing problem behaviours, using mixture modelling techniques (i.e. latent class/profile analyses), often identifies a class/profile characterized by low scores on all indicators.[53,149,152,298–300] Also in line with what has been observed in this research, the majority of children usually belong to this group, a finding which was replicated at each time point of the present study. The "low all" profile of problem behaviour was hypothesized to be observed a priori.

The next two profiles of problem behaviour were similar in structure, but differed in severity. These profiles were defined as the "Moderate all" and "High all" profiles and were characterized by comorbidity/concurrence of conduct problems, hyperactivity/inattention, and internalizing problem behaviours. While a pattern of comorbid internalizing and externalizing (both conduct and hyperactivity/inattention) problem behaviours was hypothesized a priori, it was not expected that there would be two differing severity groups of this profile (i.e.

one in which all three indicators were moderate and one in which all three indicators were high).

A longitudinal study conducted by Althoff et al. (2010) investigated latent classes of problem behaviour in children aged 4 – 16 years using the CBCL.[152] While their analyses featured additional indicators to the variables used in the present design, they did identify a profile which aligns closely with the "High all" group in the present study. In their research, this group was characterized by high scores of internalizing, attention problems, aggression, and rule breaking. Research using the CBCL has defined this profile as the Child Behavior Checklist-Dysregulation Profile (CBCL-DP), which was first described by Biederman (1995).[301] The CBCL-DP has been empirically replicated in numerous population and clinical samples.[150,151,300,302,303] In line with the present findings, it has been suggested that this profile of problem behaviour becomes easier to identifiy during the elementary school period, when self-regulation becomes an increasingly required skill for functioning.[304] Further evidence in support of this being a distinct and valid profile of problem behaviour is that similar findings to this body of research, as well as the present study, have been identified using different measures of childhood problem behaviour such as the Behavior Assessment System for Children (BASC) and the Ohio Scales Problem Severity Scale (OS PS).[305–307]

One study conducted by Mindrila (2016) on a sample of 6-11 year olds found results which do fit with the findings of the present study and other CBCL problem behaviour research.[307] The researcher used a latent profile analysis to

explore typologies of school behaviour using the Behavior Assessment System for Children–Second Edition (BASC-2). The profiles identified in this study are similar to the present study, with the exception that they did not identify the "Hyper./Inatt. & Int." profile of problem behaviour (described below). While their research did integrate items of adaptive skills into their modelling, measures of hyperactivity, conduct problems and anxiety/depression were also included. Interestingly, the researcher identified three profiles – (1) Adequate adjustment, characterized by low/average levels of problem behaviour (akin to the "Low all" group), (2) Mild adjustment difficulties, characterized by moderate levels of disruptive/externalizing and internalizing behaviour (akin to the "Moderate all" group), and (3) Functionally impaired adjustment, characterized by clinical levels of both externalizing and internalizing (akin to the "High all" group). While a larger proportion of their sample fell into the latter two groups, relative to the "Moderate all" and "High all" groups in the present study, the ranking of them was similar, with the adequate adjustment group being the largest, followed by the mild difficulties, and then the functionally impaired group. The traits of the functionally impaired group do seem to share parallels with the CBCL-DP, in that they exhibit high scores across all types of internalizing and externalizing problem behaviours. Results using the BASC-2 were also confirmed in a similar study, using similar methodology.[305] The present study adds to this body of work by providing further evidence for a dysregulation profile of problem behaviour in children, as well as evidence for a less severe profile of internalizing and externalizing concurrence.

The last group identified was labelled as the "Hyper./Inatt. & Int." group and was characterized by comorbid patterns of high internalizing and hyperactivity/inattention scores, with low scores of conduct problems. The comorbidity between internalizing problems and hyperactivity/inattention has been observed in children, especially in clinical presentations where children with ADHD often have comorbid clinical/subthreshold depression, as well as anxiety.[118,126,127,129] One study which used latent profile analyses to investigate comorbid patterns of problem behaviour in children aged 6-11 years identified an "internal inattentive" group, characterized by clinically significant levels of depression with moderate symptoms of inattention and anxiety.[308] These individuals were distinct relative to another group which additionally had high rates of disruptive behaviour (i.e. aggression and conduct problems), likely a more similar construct to the dysregulation profile. Despite evidence in the literature of this comorbid pattern of internalizing and hyperactivity/inattention, it is more commonly reported that children with hyperactivity/inattention problems have comorbidities with other externalizing problems, and therefore, the "Hyper./Inatt. & Int." profile was not expected to be observed a priori.

Overall, the patterns of problem behaviour identified in the present study highlight just how comorbid problem behaviour and mental illness symptoms are in children. Extensive research has been conducted looking at the comorbidity of internalizing and externalizing problems, with extensive overlap of symptoms being observed between these two domains.[95,141–144,146–148,309] Furthermore, research into patterns of dysregulation, whereby subsets of children present with

high syndrome scores of depression, hyperactivity/inattention, conduct problems, and aggression support the notion that problem behaviour in childhood should be viewed dimensionally, across multiple domains.[149–152] Research has shown those scoring high on dysregulation to be at an elevated risk of both internalizing and externalizing psychopathologies, personality disorders, suicidality, and substance use, later in life.[151,298,302,310,311] This suggests that this continuum of dysregulation may serve as a marker for a global risk of mental illness. The present study supports this idea of a continuum of problem behaviour, as three of the key profiles of problem behaviour were merely degrees of overall internalizing and externalizing comorbidity, ranging from the "low all" to "Moderate all" to "High all" patterns of problems. This provides additional support that the field of childhood problem behaviour should consider all-encompassing measures which incorporate symptoms of both internalizing and externalizing problems/symptoms.

4.1.2 Problem Behaviour Transitions

The next step of the research used two timepoints to explore the transitions between profiles of problem behaviour across time. A total of four profiles of problem behaviour were identified at age 6/7 years, as well as age 10/11 years, and therefore a total of 16 unique transitions of problem behaviour were possible.

As was expected, the majority of the individuals stayed in the "Low all" group across time. This is consistent with other bodies of research showing that the majority of children maintain low levels of problem behaviour across development.[53,149,152,298–300,306] The highest proportion of individuals who were in

the “Moderate all” or the “Hyper./Inatt. & Int.” profiles at age 6/7 years also ended up in the “Low all” profile by age 10/11 years, but the highest proportion of individuals in the “High all” profile at the first timepoint were still in this profile at age 10/11 years. Previous research has suggested that, for many children, symptoms of problem behaviour desist across development.[19–24,312] There are however, subsets of children who experience levels of internalizing, externalizing, and the comorbidity of both, that show persistence across time.[4,7,29,235,312–315] This has been shown consistently in research that has looked at persistence in problems for those scoring high on comorbid patterns of internalizing and externalizing, akin to overall dysregulation.[149,152,153,156]

In order to increase the power when investigating health-risk outcomes, the 16 transition groups were clustered into five subgroups – stable low problem behaviour, decreasing to low problem behaviour, increasing from low to problem behaviour, homotypic problem behaviour, and heterotypic problem behaviour. By the time individuals were 10/11 years old, approximately three quarters of them had low patterns of problem behaviour, supporting the notion that the majority of children show minimal problem behaviour, and those who do, tend to desist across development. A small group of individuals who initially showed low problem behaviour, transitioned into a profile of problem behaviour by age 10/11. The majority of these individuals had transitioned into the “Moderate all” or “Hyper./Inatt. & Int.” profiles, with less than one percent transitioning from a low pattern to the “High all” profile. These findings could be pointing towards the progressive nature of problem behaviour. The fact that more individuals shifted

into the “Moderate all” or “Hyper./Inatt. & Int.” profiles and only a small amount shifted directly into the most severe “High all” profile suggests that, for a small minority, a profile of severe dysregulation may develop in a progressive nature across childhood and adolescence. While biological factors may contribute, this suggests that environmental factors during the child’s development may be associated with the onset/exacerbation of these problems. Indeed, research that has investigated factors associated with later onset of internalizing symptoms have found an association with stressful childhoods and hostile parenting.[235,316] Environmental factors have also been shown to be associated with the onset of externalizing problems in late childhood and adolescence, such as deviant peer affiliations.[317] Similar factors are worth exploring in future research to better understand how patterns of dysregulation develop.

Of the individuals in one of the three problemed behaviour groups at age 6/7, approximately one quarter of them displayed a homotypic trend across time, in that they were in the same profile by age 10/11. More specifically, one quarter of individuals who were in the “Moderate all” group at age 6/7 were in the same group at age 10/11, with this same trend being observed in the “Hyper./Inatt. & Int.” group. The highest homotypic stability was observed among those in the “High all” group at age 6/7, with approximately one third of them remaining in this profile of problem behaviour at age 10/11 years.

While no known literature has looked at transitions for these exact profiles of problem behaviour, a plethora of research has established that subsets of children displaying a particular type of problem behaviour will show homotypic

persistence of that same problem behaviour across time.[154,156,159,163,318–321] This research has demonstrated homotypic stability in individuals with internalizing-only problems and externalizing-only problems. There is also evidence suggesting that the strongest stability is observed in individuals with comorbid internalizing and externalizing.[149,153,160]

Lastly, heterotypic trends in which individuals shifted from one problem behaviour profile to a different one, were also explored. Due to the fact that all three of the problem behaviour groups were characterized by varying degrees of both internalizing and externalizing, no heterotypic transitions *between* these two domains were observed. Instead, the present study defined a heterotypic transition as the shifting among different profiles of comorbidity. This was the smallest of the five subgroups of problem behaviour transitions (6.2% of the sample), with one fifth of those who displayed a pattern of problem behaviour at age 6/7 years shifting into a different problem behaviour by age 10/11 years. A sensitivity grouping was also considered, whereby the "Moderate all" and "High all" groups, and the transitions between them, were considered homotypic.

While limited research has investigated the transition from a profile of dysregulation to a less severe dysregulation profile, and vice verse, some evidence for the heterotypic transition between either of these profiles to/from the "Hyper./Inatt. & Int." will be discussed. One study conducted by Weeks et al. (2016) using the CBCL, found that internalizing in childhood increased the risk of physical aggression and conduct problems two years later.[315] Another similar study found a developmental cascade whereby internalizing at age 4 was

associated with an increase in externalizing by age 10.[322] Further evidence from a prospective study of three large cohorts found that individuals with a childhood diagnosis of depression had a four-fold increase in the odds of a diagnosis of conduct disorder in adolescence.[323] This provides some support for a transition from the "Hyper./Inatt. & Int." to either the "Moderate all" or "High all" profiles of problem behaviour, which have similar characteristics of the "Hyper./Inatt. & Int." profile, but also with an elevation in conduct problems. Further support for this transition is that some research has shown ADHD to be associated with subsequent conduct disorder and that ADHD diagnoses tend to predate conduct disorder diagnoses.[324] This provides further evidence for the transition from the "Hyper./Inatt. & Int." profile to the "Moderate all" or "High all" profiles by demonstrating that symptoms of hyperactivity and inattention can also increase the risk of developing conduct problems. An explanation for a transition in the opposite direction (i.e. "Moderate/High all" → "Hyper./Inatt. & Int.") may be that conduct problems may desist across development as children learn how to facilitate their behaviour, while internalizing and hyperactivity/inattention symptoms tend to show more persistence across time.[64,65,312,323]

4.1.3 Health-risk Outcomes

The final step of the research was to investigate the relationship between childhood profiles/transition-groups of problem behaviour and the health-risk outcomes of adolescent suicidal thoughts, substance use, and non-substance use delinquency. At age 6/7 years, the "High all" profile was associated with suicidal thoughts. The "Hyper./Inatt. & Int." profile was also associated with suicidal

thoughts, but this association was not statistically significant when looking at the sensitivity grouping of suicidal thoughts. By age 10/11 years, all three of the problem behaviour profiles ("Moderate all", "High all", and "Hyper./Inatt. & Int.") were associated with suicidal thoughts. While this research cannot confirm how these symptoms are related to suicidality, it does suggest that different patterns of comorbidity (e.g. "High all" vs. "Hyper./Inatt. & Int."), as well as differing severity levels (e.g. "Moderate all" vs. "High all") of the same comorbidity, present similar risks in the development of suicidal thoughts. While synergistic effects cannot be accounted for, the fact that the "Hyper./Inatt. & Int." showed comparable odds of suicidal thoughts relative to the other two problemed behaviour profiles, suggests that symptoms of hyperactivity, inattention, and internalizing may be the symptoms of the "Moderate all" and "High all" profiles which are most strongly associated with suicidal thoughts. It cannot, however, be inferred that the way in which suicidality develops across these profiles is the same without further investigation.

Other research in the field has shown evidence that patterns of comorbid problem behaviours, and specifically, the aforementioned dysregulation profile are associated with suicidal behaviour.[311,325] One specific study, conducted by Holtmann el al. (2011), explored the association between the CBCL-Dysregulation Profile in children and subsequent suicidality in adolescence.[311] A total of 325 children were assessed for the CBCL-Dysregulation Profile between ages 8-10 years old. At age 19, the children were followed up and the authors concluded that high CBCL-DP scores in childhood were associated with both

suicidal thoughts and attempts in late adolescence/early adulthood. Another study conducted by Berona et al. (2017) looked at profiles of mental illness in adolescents hospitalized for suicide attempts also found the dysregulation profile to predict suicide attempts.[325] Other research which has looked at comorbid patterns of internalizing and externalizing, not necessarily defined as dysregulation, have found similar findings, in that high levels of comorbidity are strongly associated with suicidal behaviour in adolescence.[182–184,326–328] The present findings both support this association, as well as add to the field, by identifying a less severe profile of the CBCL-DP and demonstrating that these individuals have similar odds of suicidal thoughts in adolescence and that no dose-response relationship was identified across these two severity groups.

Relative to the "Stable low" transition of problem behaviour, all transitions were associated with suicidal thoughts except for the "Decreasing to low" transition. The fact that those in the "Low to problems" group had similar odds of suicidal thoughts to those in the persistent transitions (heterotypic or homotypic), and that the associations were similar to the profiles at age 10/11 years, suggests that the most recent profile/assessment may be as useful of a predictor of suicidal thoughts as the multi-timepoint transitions. The identification of an association between the age 6/7-year profile and suicidal thoughts may be an indirect one, in that the age 6/7-year profile is associated with the age 10/11-year profile, which in turn, is associated with suicidal thoughts. This suggests that the profile in which an individual is in by late childhood may be more important

than earlier patterns of problems or transition patterns, at predicting adolescent suicidal thoughts.

In relation to substance use, at both age 6/7 and 10/11 years, the "Moderate all" and "High all" profiles of problem behaviour showed the most use, relative to the "Low all" and "Hyper./Inatt. & Int." profiles. Limited evidence supported a dose-response between the "Moderate all" and "High all" groups in relation to the severity of their engagement. Both profiles were at an elevated risk for use of alcohol, cigarettes, and cannabis. The "Moderate all" profile at age 6/7 years was the only profile associated with illicit substance use. It is worth noting, however, that the prevalence of illicit substance use was one of the lowest outcomes and therefore the study may not have been adequately powered to explore this association in the smallest, "High all" profile. The literature does support high patterns of heterotypic comorbidity between internalizing and externalizing to be associated with subsequent substance use/abuse.[329,330] Patterns of dysregulation have also supported this association as the previously mentioned study by Holtmann et al. (2011), also investigated the association between dysregulation and alcohol, nicotine, and cannabis dependence.[311] In line with the present findings, a significant association was found for all three types of substance use.

An explanation for why the "Hyper./Inatt. & Int." profile of problem behaviour did not share an association with most substance use, relative to the other profiles, may be that these individuals do not score high on conduct problems. Indeed, the literature does show that the risk of adolescent substance

use is significantly higher for preadolescent children scoring high on conduct problems, as well as those with comorbid symptoms of conduct problems and hyperactivity/inattention.[330–334] There is some literature which has shown individuals high on hyperactivity/inattention and internalizing to be at an elevated risk for substance use and substance use disorders.[335,336] Despite this, other research supports the present findings.[337] For example, a study conducted by Wilens et al. (2011) showed that the comorbidity of internalizing (either depressive or anxiety disorders) and ADHD, in individuals between ages 6-17 years, did not predict substance use disorders at 10-year follow-up.[337] An additional epidemiologic study of 900 New Zealanders found that childhood (age 8) attention deficit disorders, in absence of conduct problems was not associated with alcohol, tobacco, or illicit substance use in adolescence (age 15).[331] Further research is needed to better understand the factors of these individuals that act against increased substance involvement, but also the exploration of specific elements of internalizing (e.g. depressive vs. anxious symptoms) and their role in differentially increasing or decreasing subsequent substance use risk, particularly in children transitioning into adolescence.

The final set of outcomes that were investigated were patterns of non-substance use delinquency. These results were quite similar to what was identified in the substance use variables, the “Moderate all” and “High all” profiles predicted the most involvement in delinquent behaviours. Both profiles had similar odds of running away from home, being questioned by the police for suspected involvement in a crime, and vandalism. Both age 6/7 profiles were

associated with selling drugs, but by age 10/11, only the "High all" profile predicted this. Both the "Moderate all" and "Hyper./Inatt. & Int." profiles at age 10/11 were associated with getting into fights to the point of injury. Interestingly, this was the only pattern of non-substance delinquency in which the "Hyper./Inatt. & Int." profile was associated with. Being that this outcome had a low prevalence, issues of power may explain why an association wasn't observed in the smaller "High all" profile.

The fact that the "Moderate all" and "High all" profiles were associated with more non-substance delinquency than the "Hyper./Inatt. & Int." profile is unsurprising. The central difference between these profiles is that the latter features low levels of conduct problems, comparable to the "Low all" profile. It is likely that these delinquent behaviours are a progression of the conduct problems observed earlier in the life course. Indeed, the literature does support children high on conduct problems to be at an increased risk for criminal activity later in the life course.[333,338,339] Similar to what was identified in relation to substance use, this risk is compounded further when conduct problems are exhibited in tandem with symptoms of hyperactivity.[338] Interestingly, however, is that patterns of only hyperactivity/inattention problems have also been shown to predict criminality later in the life course, independent of conduct problems.[338] Relative to the present research, this suggests that the internalizing symptoms of the "Hyper./Inatt. & Int." profile may have a protective or inhibitory effect in involvement in delinquent/criminal behaviours. Some support for this comes from

findings that identify internalizing patterns of problem behaviour to be associated with behavioural inhibition and to protect against defiance and aggression.[52,340]

An additional unique finding was that the "Hyper./Inatt. & Int." profile predicted fighting and one possible explanation for this association could stem from victimization. Indeed, some literature supports that the development of this comorbidity is progressive, in that symptoms of hyperactivity and inattention act to alienate children from their peers and, in turn, these feelings of exclusion are internalized[161]. It could be that these individuals are victims of bullying and that their fear and impulsivity (i.e. symptoms of this comorbidity) cause them to fight back against their bully. The role of mediating factors, such as bullying/victimization, warrants further investigation.

Lastly, in relation to the transition patterns of problem behaviour, a similar trend was observed with the substance/non-substance delinquency as was observed with suicidal thoughts. The evidence seemed to suggest that the best predictor of health-risk outcomes in adolescence was the profile that the child ended up in by age 10/11 years, rather than the trajectory of problem behaviour across the four-year period. This was the opposite of what was hypothesized, where it was expected that persistent patterns of problem behaviour would be associated with the highest odds of substance and non-substance delinquency.

4.2 Implications

The profiles and transitions of problem behaviour identified in the present study, as well as their association with health-risk outcomes in adolescence, have important research and public health implications for the field of child and

adolescent mental health. These implications include the identification of high rates and unique patterns of comorbidity in childhood problem behaviour, as well as trends in how these patterns change across development. This research also highlights the prevalence of various health-risk outcomes in adolescence and identifies behavioural antecedents that predict these outcomes.

The first noteworthy implication of the present study is that the profiles of problem behaviour identified emphasize just how comorbid problem behaviours are in children. As such, it is important that domains of both internalizing and externalizing are considered by stakeholders when designing and implementing population level interventions. This could include interventions that target either school or community settings to reduce childhood problem behaviour and indeed, such programs have been evaluated and show efficacy for multiple domains of problem behaviour.[341,342] For example the Generation Parent Management Training – Oregon Model (GenerationPMTO) is a program that teaches effective family management strategies to parents in either group or individual settings.[342] In community samples, the intervention is typically conducted in weekly sessions ranging from 6 – 14 weeks. Studies which have primarily focused on parents of children between ages 4 – 12 have seen reductions in childhood internalizing, externalizing, delinquency, and deviant peer associations.[343–345] Furthermore, the program has been shown to reduce symptoms of mental illness, such as oppositional defiant disorder and aggression, in the parents, themselves.[344] The program has also been evaluated across multiple ethnic groups, as well as two-

and single-parent families, supporting its generalizability across multiple populations.[343,346,347]

While the household environment has important implications for childhood problems, school-based interventions that target symptoms of internalizing and externalizing are also worth consideration. One such program that shows promise is the Incredible Years – Teacher Classroom Management program.[341] This intervention targets children aged 3 – 8 years of age and provides teachers with classroom management skills to foster positive peer, teacher-student, and teacher-parent relationships. This program has been shown to reduce conduct, disruptive, and internalizing behaviours in children, while also improving child self-regulation, cooperation, and interpersonal skills.[348–350] A corresponding Incredible Years – Parent program also exists and is a group-based parenting program that strengthens parent competencies to reduce problem behaviours in children.[341,351] The present study emphasizes the comorbid rates of problem behaviours in childhood. This provides support that intervention programs should target multiple domains of problem symptoms to possibly reduce immediate impairment, as well as subsequent health-risk outcomes in adolescence.

Another implication of the present study was the significant proportion of individuals whose problem behaviour desisted to a low-level across the four-year period. From a public health perspective, and in relation to parents and other stakeholders, it highlights that not all children exhibiting problem behaviours at a younger age will show persistence in those problems across time. From an

alternate perspective, however, the present findings did show that children in the "High all" profile had the greatest amount of homotypic stability, relative to the other two problemed behaviour groups. Further research is needed to better understand which factors contribute to desistance of problem behaviour, as well as which factors are associated with patterns of persistence. Positive and deviant peer-relations, bullying/victimization, and family functioning may mediate these changes of problem behaviour, with some evidence suggesting these factors to have an influence.[235,316,317] Further understanding of how these patterns of problem behaviour desist/persist may provide insights into specific groups of vulnerable children that should be targeted for intervention to potentially reduce health-risk behaviours in early adolescence.

While this research identified specific profiles of problem behaviours in children, it also highlighted that these profiles were associated with suicidal thoughts, substance use, and non-substance delinquency in adolescence. The research identified that approximately 14% of adolescents reported having suicidal thoughts. Research shows that suicidal thoughts can progress into suicide attempts, therefore public health initiatives should be targeting the early adolescent group in an effort to potentially reduce this risk.[191,192] Frequent substance use was less prevalent, but given the younger age of the study population, and the known ramifications of substance use on the developing brain, it too is worth targeting for reducing long-term detriment.[212] Lastly, 1 in 5 early adolescents reported being questioned by the police for suspected involvement in

a crime, as well as for vandalism. In order to deter adolescent involvement in the criminal justice system, these too are worthy targets for intervention.

Research has shown children and adolescents displaying conduct problems to have poorer long-term outcomes into adulthood.[352–354] For example, a study that looked at the forty-year outcomes of adolescents with severe externalizing found that they were at an increased risk for parenthood before age 20, divorce, no educational qualifications, financial difficulties, and global life adversity.[352] A similar risk was observed in individuals with mild externalizing, relative to those with no/low externalizing, which is in line with the risk observed in the "Moderate all" profile of the present study. Similar risks between childhood/adolescent externalizing and long-term negative outcomes have been identified elsewhere, with some research showing that these individuals are also at an increased risk for premature mortality.[353,354] This further supports targeting patterns of problem behaviour early in the life course to deter long-term negative outcomes, which have serious costs to both the individual and society as a whole.

4.3 Limitations

Extensive efforts were made when designing the present study to ensure that bias and error were mitigated, but like all research there were limitations and they are noteworthy when interpreting the findings. The biggest limitations of the study were the use of self-reported outcomes, the use of different raters for the independent and dependent variables, missing data, and measurement error.

4.3.1 Self-reported outcomes

The health-risk outcomes that were selected as dependent variables in the present study were all self-reported by the adolescent and, as a result, could have potentially introduced bias into the study. In particular, social desirability bias, which is a bias introduced when the respondent responds in a way that they feel is socially acceptable, is possible when considering the stigma attached to suicidal thoughts and issues of legality associated with substance use and delinquent behaviours.[355] While it is difficult to ascertain whether social desirability is equally common throughout each profile of problem behaviour, it is possible that the health-risk outcomes were underreported and that the observed association has been biased.

It is important to note that the respondents were informed that their responses would be kept confidential and following the completion of the survey, the respondent sealed their survey in an envelope.[216] This was an additional step taken to potentially mitigate bias introduced from social desirability. Such collection strategies have been suggested as useful precautions to mitigate bias and increase validity.[356] Furthermore, research has been conducted that has looked at the validity of using adolescent self-reported measures for some of the health risk outcomes used in the present study.

In relation to self-reporting substance use, other research has assessed the validity of self-report measures have been contrasted with the results of urinalysis, in adolescents.[357,358] This research has shown that, despite a subset of adolescents showing a tendency towards response bias, a substantial majority consistently

report their substance use, concluding that self-reported measures have fair validity. Research conducted on self-report delinquency measures as a whole (i.e. substance and non-substance), have found that self-reports of criminal activity, in adolescence, can predict future court referrals and that validity of self-reports is high.[359]

Relative to substance use and urinalysis, suicidal thoughts do not have an objective measure to be cross referenced, making the degree of social desirability bias more difficult to ascertain. Much of the research on social desirability and suicidal thoughts has come from comparing items on social desirability scales to those on suicidal thoughts measures, which have yielded significant associations.[360,361] This suggests, that to some extent, responders do provide socially desirable responses on measures of suicidal thoughts. Interestingly, some research has shown the prevalence of suicidal thoughts increases when self-reported using a paper-and-pencil survey, relative to a face-to-face interview, suggesting that confidentiality may mitigate social desirability – a precaution that was taken in the NLSCY.[362,363]

4.3.2 Parent-Report vs. Self-Report Measures

Another limitation related to the way in which the data were collected is the fact that the independent variable data (i.e. problem behaviour) was reported by the caregiver of the children in the study, while the dependent variable data (i.e. health-risk outcomes) was reported by the child/adolescent, themselves. Due to the present methods using secondary data, this was an unavoidable limitation as this is how the data were collected.

A meta-analysis looking at agreement between parent- and self-report measures of problem behaviour and mental illness has found that there is often poor agreement between the two raters.[364] There is, however, evidence that more agreement exists between the two raters when the offspring is younger (<= 12 years old).[365] A review of the literature was conducted and clinical recommendations in the field have suggested that parents are the best informant for both internalizing and externalizing when the child is younger and that older children/adolescents are the better informants of both internalizing and externalizing problems.[366] In relation to substance use, research suggests that if forced to choose, adolescent self-reporting aligns closer to diagnosed cases than does parent/caregiver reporting.[367] While possible misclassification bias is a limitation due to using secondary data, this does provide evidence that the best rater was selected for both the independent and dependent variables.

4.3.3 Missing data

When dealing with waves of survey data across an extensive time period (8 years in the present study), missing data is unavoidable. This was no exception to the present study and must be acknowledged as a limitation when interpreting the findings. When carrying out the latent profile analyses, maximum-likelihood estimation was incorporated for incomplete cases, and only those that were completely missing could not be included. However, it was decided against using maximum-likelihood estimation or imputation methods to impute outcome data, so as to not introduce unknown error in the results. This is supported by research which has shown multiple imputation for outcome data does not help with

reducing attrition bias.[368] Another reason for this decision was that the sample size still remained high after removal of these cases. As such, these cases were removed and compared on the independent variables, as well as other key sociodemographics. Some key differences were identified and are worth noting as potential sources of bias when interpreting the observed findings.

Individuals with missing outcome data were more likely to be male, come from lower SES backgrounds, non-two parent homes, and report higher rates of maternal depression. The same trend was observed whether missing was based on the suicidal thoughts variable or on the sensitivity suicidal thoughts variable. Limited differences were observed in profiles of problem behaviour, with the exception that for those missing on the sensitivity variable of suicidal thoughts, missing cases were more likely to be in the "Low all" profile at age 6/7, but no difference was observed at age 10/11 years. It is known that suicidal thoughts are more common in females than males, so in this respect, the present study may overestimate the true rate of suicidal thoughts.[369] However, it is also known that suicidal thoughts are more prevalent in low SES groups, non-two parent households, and in offspring whose mother displays maternal depression symptoms – across both sexes.[244,258,268,270,283,285] Therefore, it may be more likely that the present study underestimated the true prevalence of suicidal thoughts. Due to the fact that the majority missing on suicidal thoughts were also missing for substance use and delinquent behaviour variables, and that these same sociodemographic factors place individuals at risk for criminality, these rates may too be underestimated in the present study.[244,282,284,286,287,370]

While the dispersal of cases for problem behaviour at age 10/11 years was the same for missing and non-missing, missing cases were more likely to be in the "Low all" profile at age 6/7. Although counterintuitive, other research has found that children with depressive symptoms at age 8/9 years were more likely to stay in the NLSCY.[371] If these cases also had a higher prevalence of suicidal thoughts (as well as substance use and delinquency), than it is potential that this association is biased away from the null and that the magnitude of the association between age 6/7 profile and suicidal thoughts (as well as substance use and delinquency) is weaker than what was observed. In spite of this, the fact that the most recent measure of problem behaviour (age 10/11 years) predicted the outcomes of interest and had even dispersal of missing and non-missing does support the notion of a true association.

4.3.4 Measurement error

In order to investigate the association between profiles of problem behaviour and the health-risk outcomes of interest, cases were grouped based on their most probable profile membership. None of the cases had a perfect posterior probability (p=1.0) of modal assignment (i.e. belonging to their particular profile). The inverse of the posterior probability can be considered the measurement error, or the likelihood of *not* belonging to a given profile. In *Mplus* software this measurement error can be included into the model using logit constraints, however due to aforementioned issues with using complex survey weights in *Mplus*, and a desire for the analyses to be nationally representative, the cases had to be assigned to their most probable profile and exported to SAS software. In

spite of this, the literature does support assigning cases to their most probable profile when entropy scores are high, confirming good class delineation.[290,297] This was the case in the present study, and while some measurement error may bias the results, the appropriate steps were taken to ensure it is reduced.

4.4 Strengths

4.4.1 Sample size

The sample size of the latent profile analyses at each of the three-time points ranged from 8,659 – 10,234 individuals, with a total of 8,266 individuals having a profile of problem behaviour at age 6/7 and age 10/11 years. Relative to other population level research conducted in this field, this was a substantial sample size. The inclusion of maximum likelihood estimation in the latent profile modelling allowed for the inclusion of partially missing cases, maintaining the overall sample size. In relation to the outcome analyses, the present study did have a significant proportion of the initial sample dropout or have missing data across their measures at age 6/7, up until their outcome assessments at ages 12 – 15 years. Despite this, the sample size was still large and adequately powered for the analyses. Overall, this adds strength to the findings, in that the estimates and respective confidence intervals are accurate and that the risk of type II error has been minimized.

4.4.2 Nationally-representative & prospective design

A key strength of this research was the fact that the NLSCY is a nationally representative cohort which includes samples from all Canadian provinces. The study survey weights used in the logistic regression modelling ensure that this

representativeness is accounted for. This means that the associations identified in the present study, as well as the prevalence rates, are generalizable to the majority of Canadian children and adolescents. Furthermore, the fact that the NLSCY collected waves of data prospectively, every two years, reduces the risk of recall bias from both the caregiver and the adolescent.

4.4.3 Multivariate Approach

One final strength that is noteworthy is the extensive number of variables that were included in the entirety of the present study. Three indicators of both internalizing and externalizing problem behaviours, which included depressive/anxiety-, hyperactivity/inattention-, and conduct-symptoms, at three timepoints, across a four-year period, were used to identify distinct patterns of problem behaviour. Furthermore, an extensive set of 12 unique outcome variables, mapping onto three broader categories (i.e. suicidal thoughts, substance use, and non-substance use delinquency) were able to be explored in the association with childhood problem behaviour patterns. Lastly, when exploring these associations, important health and sociodemographic variables were able to be controlled for, including sex, socioeconomic status, two-parent home status, maternal depressive symptoms, maternal alcohol use, and stressful life events. The inclusion of these variables reduces the confounding bias introduced into the findings and increases the support of a true association being observed.

4.5 Future research

The present study offered multiple insights into the patterns of problem behaviour that elementary school aged children experience, as well as how they change

across time. Furthermore, the study highlights how these patterns are associated with health-risk outcome in early adolescence. Future research could be conducted in order to better understand the profiles of problem behaviour identified, how they change across time, and other mediating factors in the development of the health-risk behaviours.

While the present study chose to examine childhood problem behaviour using parent reports, mainly to increase sample size, future research could be replicated using teacher reported measures. Some evidence does suggest that parents tend to report higher rates of comorbid problems in children than teacher reports do.[372] Whether this is a difference in how the child acts at home as opposed to school or a difference in observer opinion has been debated. Interestingly, however, research which has looked at the dysregulation profile in children has been able to identify this profile using both parent and teacher reported methods.[151,373] Nonetheless, future research could integrate reports from multiple observers to better understand how different raters appraise these patterns of problem behaviour.

While the present study did identify a relationship between profiles of problem behaviour in childhood and subsequent health-risk behaviours in early adolescence, it did not focus on social influences that may mediate the development of said behaviours. In particular, future research could investigate the role of peer influence in protecting/risk factors for health-risk behaviours. Evidence does support that positive peer relations can reduce feelings of loneliness and increase wellbeing, while deviant peer relations, as well as social

isolation, can increase some of these risks.[374–380] If it is known that problem behaviours are predictors of these negative outcomes, the next step is to identify targets for intervention that reduce said risk.

Future research should also consider piloting interventions that target the profiles of problem behaviour identified in this body of work. Assessing the efficacy of the aforementioned GenerationPMTO and Incredible Years programs at reducing symptoms of comorbidity and dysregulation may prove useful at reducing immediate detriment, as well as subsequent risk for the negative outcomes identified in adolescence. The fact that these interventions have shown success at ameliorating symptoms of both internalizing and externalizing is promising in reducing the symptoms of the profiles identified in the present findings.

Lastly, while the primary goal of the present study was to see how patterns of problem behaviour across childhood relate to health-risk outcomes in the adolescent period, an additional direction this research could take is to look earlier in the life course at the factors that lead to these highly comorbid patterns. For example, the aforementioned study by Weeks et al. (2014) highlighted stressful childhoods and hostile parenting as early-life factors that associated with the onset of internalizing symptoms.[235] In addition, a meta-analyses conducted on parenting and childhood depression found that parental rejection was strongly related to the subsequent depression in childhood.[381] Additional research has also showed that early patterns of conduct problems at a young age can alienate children from their peers and result in subsequent internalizing symptoms.[161] While the present

research cannot comment on how these patterns of problem behaviour develop, it is possible that early environmental factors and problem behaviours can trigger a cascade leading to overall dysregulation. Identification of such trajectories may offer earlier intervention targets to prevent the development of these comorbid profiles of problems, as well as subsequent health-risk outcomes in adolescence.

4.6 Conclusion

The present study used a longitudinal design to identify profiles of problem behaviour in elementary-school aged children and how these patterns change across time. At ages 6/7, 8/9, and 10/11 years, the same profiles of problem behaviour were identified and included one profile in which individuals were low on internalizing and externalizing (both hyperactivity/inattention and conduct problems), one in which individuals had moderate levels of internalizing and externalizing, one in which individuals had high levels of internalizing and externalizing (akin to the commonly reported 'dysregulation profile'), and one in which individuals had high scores of high internalizing and high hyperactivity/inattention. Transitions between these profiles across time were identified and along with the profiles, were investigated as predictors of suicidal thoughts, substance use, and non-substance use delinquency. Results showed that all three profiles of problemed behaviour were associated with suicidal thoughts, while the "Moderate all" and "High all" profiles were associated with substance use and non-substance use delinquency. This research sheds light on the comorbidity of mental illness symptoms in children, as well as identifies antecedents in the development of health-risk outcomes in adolescence. Further

research should explore why transition patterns of problems did not outperform profile status at age 10/11 years, as well as other mediating factors in the persistence/desistance of problem behaviours, as well as health-risk outcomes, to potentially identify target for intervention.

References

1. Eisenberg N, Cumberland A, Spinrad TL, et al. The Relations of Regulation and Emotionality to Children's Externalizing and Internalizing Problem Behavior. *Child Dev*. 2001;72(4):1112-1134.

2. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime Prevalence and Age-of-Onset Distributions of DSM-IV Disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):593. doi:10.1001/archpsyc.62.6.593.

3. Garber J, Gallerani CM, Frankel S. Depression in Children. In: *Handbook of Depression*. 2nd ed. New York, NY: The Guilford Press; 2009:405-419.

4. Campbell SB, Ewing LJ. Follow-up of Hard-to-Manage Preschoolers: Adjustment at Age 9 and Predictors of Continuing Symptoms. *J Child Psychol Psychiatry*. 1990;31(6):871-889. doi:10.1111/j.1469-7610.1990.tb00831.x.

5. Shaw D, Keenan K, Vondra J. Developmental precursors of externalizing behavior: Ages 1 to 3. *Dev Psychol*. 1994;30(3):355-364.

6. Wittchen HU, Kessler RC, Pfister H, Lieb M. Why do people with anxiety disorders become depressed? A prospective-longitudinal community study. *Acta Psychiatr Scand Suppl*. 2000;(406):14-23. http://www.ncbi.nlm.nih.gov/pubmed/11131466. Accessed July 11, 2016.

7. Hofstra MB, Van der ende J, Verhulst FC. Child and Adolescent Problems Predict DSM-IV Disorders in Adulthood: A 14-Year Follow-up of a Dutch Epidemiological Sample. *J Am Acad Child Adolesc Psychiatry*. 2002;41(2):182-189. doi:10.1097/00004583-200202000-00012.

8. Tanya Navaneelan. Health at a Glance. Statistics Canada Catalogue no. 82-624-X. http://www.statcan.gc.ca/pub/82-624-x/2012001/article/11696-eng.htm. Published 2010. Accessed June 29, 2016.

9. Garrison CZ, Addy CL, Jackson KL, Mckeown RE, Waller JL. A Longitudinal Study of Suicidal Ideation in Young Adolescents. *J Am Acad Child Adolesc Psychiatry*. 1991;30(4):597-603. doi:10.1097/00004583-199107000-00011.

10. Canadian Centre on Substance Abuse. Trends in Drug Use among Youth. Canadian Centre on Substance Abuse.

11. Propel. Tobacco Use in Canada: Patterns and Trends. 2015. www.tobaccoreport.ca. Accessed August 24, 2016.

12. The Chief Public Health Officer's Report on the State of Public Health in Canada, 2015: Alcohol Consumption in Canada. Government of Canada. http://healthycanadians.gc.ca/publications/department-ministere/state-public-health-alcohol-2015-etat-sante-publique-alcool/index-eng.php.

Published 2015.

13. Public Safety Canada. A Statistical Snapshot of Youth at Risk and Youth Offending in Canada. Government of Canada. http://www.publicsafety.gc.ca/cnt/rsrcs/pblctns/ststclsnpsht-yth/index-en.aspx. Published 2016.

14. Achenbach T, Edelbrock C, Howell C. Empirically-based assessment of the behavioral/emotional problems of 2-3 year old children. *J Abnorm Child Psychol*. 1987;15:629-650.

15. Tandon M, Cardeli E, Luby J. Internalizing Disorders in Early Childhood: A Review of Depressive and Anxiety Disorders. *Child Adolesc Psychiatr Clin N Am*. 2009;18(3):593-610. doi:10.1016/j.chc.2009.03.004.

16. Liu J. Childhood externalizing behavior: theory and implications. *J Child Adolesc Psychiatr Nurs*. 2004;17(3):93-103. http://www.ncbi.nlm.nih.gov/pubmed/15535385. Accessed October 9, 2017.

17. Achenbach T. *Achenbach, T. (1991). Manual for the Child Behavior Checklist/4 - 18 and 1991 Profile*. Burlington: University of Vermont Department of Psychiatry; 1991.

18. Achenbach T. *Manual for the Child Behavior Checklist/2-3 and 1992 Profile*. Burlington: University of Vermont Department of Psychiatry; 1992.

19. Luby JL. *Depression*. second. New York: Guilford Press; 2000.

20. Carter AS, Briggs-Gowan MJ, Davis NO. Assessment of young children's social-emotional development and psychopathology: recent advances and recommendations for practice. *Zeanah*. 2001.

21. Campbell SB. *Behavior Problems in Preschool Children: Clinical and Developmental Issues*. 2nd ed. New York: Wiley; 2002.

22. Kessler RC, Avenevoli S, Ries Merikangas K. Mood disorders in children and adolescents: an epidemiologic perspective. *Biol Psychiatry*. 2001;49(12):1002-1014. doi:10.1016/S0006-3223(01)01129-5.

23. Paus T, Keshavan M, Giedd JN. Why do many psychiatric disorders emerge during adolescence? *Nat Rev Neurosci*. 2008;9(12):947-957. doi:10.1038/nrn2513.

24. Offord DR, Boyle MH, Racine YA, et al. Outcome, Prognosis, and Risk in a Longitudinal Follow-up Study. *J Am Acad Child Adolesc Psychiatry*. 1992;31(5):916-923. doi:10.1097/00004583-199209000-00021.

25. Broidy LM, Nagin DS, Tremblay RE, et al. Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six-site, cross-national study. *Dev Psychol*. 2003;39(2):222-245.

http://www.ncbi.nlm.nih.gov/pubmed/12661883. Accessed July 11, 2016.

26. Reiss A, Roth J. *Understanding and Preventing Violence: Volume 3*. Washington D.C.: National Research Council; 1994.

27. Hay DF, Castle J, Davies L. Toddlers' use of force against familiar peers: a precursor of serious aggression? *Child Dev*. 71(2):457-467. http://www.ncbi.nlm.nih.gov/pubmed/10834477. Accessed July 11, 2016.

28. Ezpeleta L, de la Osa N, Doménech JM. Prevalence of DSM-IV disorders, comorbidity and impairment in 3-year-old Spanish preschoolers. *Soc Psychiatry Psychiatr Epidemiol*. 2014;49(1):145-155. doi:10.1007/s00127-013-0683-1.

29. Danzig AP, Bufferd SJ, Dougherty LR, Carlson GA, Olino TM, Klein DN. Longitudinal Associations Between Preschool Psychopathology and School-Age Peer Functioning. *Child Psychiatry Hum Dev*. 2013;44(5):621-632. doi:10.1007/s10578-012-0356-4.

30. Olino TM, Dougherty LR, Bufferd SJ, Carlson GA, Klein DN. Testing Models of Psychopathology in Preschool-aged Children Using a Structured Interview-based Assessment. *J Abnorm Child Psychol*. 2014;42(7):1201-1211. doi:10.1007/s10802-014-9865-x.

31. Lewinsohn PM, Solomon A, Seeley JR, Zeiss A. Clinical implications of subthreshold depressive symptoms. *J Abnorm Psychol*. 2000;109(2):345-351. doi:10.1037/0021-843X.109.2.345.

32. Fergusson DM, Horwood LJ, Ridder EM, Beautrais AL. Subthreshold Depression in Adolescence and Mental Health Outcomes in Adulthood. *Arch Gen Psychiatry*. 2005;62(1):66. doi:10.1001/archpsyc.62.1.66.

33. Keenan K, Hipwell A, Feng X, et al. Subthreshold Symptoms of Depression in Preadolescent Girls Are Stable and Predictive of Depressive Disorders. *J Am Acad Child Adolesc Psychiatry*. 2008;47(12):1433-1442. doi:10.1097/CHI.0b013e3181886eab.

34. Luby JL, Belden AC, Pautsch J, Si X, Spitznagel E. The clinical significance of preschool depression: impairment in functioning and clinical markers of the disorder. *J Affect Disord*. 2009;112(1-3):111-119. doi:10.1016/j.jad.2008.03.026.

35. Seligman M, Walker E. *Abnormal Psychology*. 4th editio. New York: W W Norton & Co Inc; 2000.

36. Davison G. *Abnormal Psychology*. Toronto: Veronica Visentin; 2008.

37. Association AP. *Diagnostic and Statistical Manual of Mental Disorders*. Fifth ed. American Psychiatric Publishing; 2013.

38. Wittchen H-U, Lieb R, Schuster P, Oldehinkel T. When is onset? Investigations into early developmental stages of anxiety and depressive

disorders. In: *Childhood Onset of "adult" Psychopathology. Clinical and Research Advances*. Washington, DC: American Psychiatric Press; 1999:259-302. http://pubman.mpdl.mpg.de/pubman/faces/viewItemFullPage.jsp?itemId=escidoc%3A1646074%3A1. Accessed October 9, 2017.

39. Becker ES, Rinck M, Türke V, et al. Epidemiology of specific phobia subtypes: Findings from the Dresden Mental Health Study. *Eur Psychiatry*. 2007;22(2):69-74. doi:10.1016/j.eurpsy.2006.09.006.

40. Beesdo K, Knappe S, Pine DS. Anxiety and Anxiety Disorders in Children and Adolescents: Developmental Issues and Implications for DSM-V. *Psychiatr Clin North Am*. 2009;32(3):483-524. doi:10.1016/j.psc.2009.06.002.

41. Beesdo K, Pine DS, Lieb R, Wittchen H-U. Incidence and Risk Patterns of Anxiety and Depressive Disorders and Categorization of Generalized Anxiety Disorder. *Arch Gen Psychiatry*. 2010;67(1):47. doi:10.1001/archgenpsychiatry.2009.177.

42. Last CG, Perrin S, Hersen M, Kazdin AE. A Prospective Study of Childhood Anxiety Disorders. *J Am Acad Child Adolesc Psychiatry*. 1996;35(11):1502-1510. doi:10.1097/00004583-199611000-00019.

43. de Graaf R, Bijl R V., Spijker J, Beekman ATF, Vollebergh WAM. Temporal sequencing of lifetime mood disorders in relation to comorbid anxiety and substance use disorders. *Soc Psychiatry Psychiatr Epidemiol*. 2003;38(1):1-11. doi:10.1007/s00127-003-0597-4.

44. Spence SH, Rapee R, Mcdonald C, Ingram M. The structure of anxiety symptoms among preschoolers. *Behav Res Ther*. 2001;39:1293-1316. www.elsevier.com/locate/brat. Accessed July 18, 2016.

45. Eley TC, Bolton D, O'Connor TG, Perrin S, Smith P, Plomin R. A twin study of anxiety-related behaviours in pre-school children. *J Child Psychol Psychiatry*. 2003;44(7):945-960. doi:10.1111/1469-7610.00179.

46. Zahn C, Waxler –, Klimes B, Dougan –, Slattery MJ. Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Dev Psychopathol*. 2000;12:443-466.

47. Masi G, Mucci M, Favilla L, Romano R, Poli P. Symptomatology and comorbidity of generalized anxiety disorder in children and adolescents. *Compr Psychiatry*. 1999;40(3):210-215. doi:10.1016/S0010-440X(99)90005-6.

48. Egger H, Angold A. *Handbook of Preschool Mental Health*. New York: Guilford Press; 2006.

49. Han H. Measuring Anxiety in Children: A Methodological Review of the

Literature. *Asian Nurs Res (Korean Soc Nurs Sci)*. 2009;3(2):49-62. doi:10.1016/S1976-1317(09)60016-5.

50. Rohde P, Lewinsohn PM, Klein DN, Seeley JR, Gau JM. Key Characteristics of Major Depressive Disorder Occurring in Childhood, Adolescence, Emerging Adulthood, and Adulthood. *Clin Psychol Sci*. 2013;1(1):41-53. doi:10.1177/2167702612457599.

51. Eisenberg N, Cumberland A, Spinrad TL, et al. The Relations of Regulation and Emotionality to Children's Externalizing and Internalizing Problem Behavior. *Child Dev*. 2001;72(4):1112-1134. doi:10.1111/1467-8624.00337.

52. Mesman J, Bongers IL, Koot HM. Preschool Developmental Pathways to Preadolescent Internalizing and Externalizing Problems. *J Child Psychol Psychiat Assoc Child Psychol Psychiatry*. 2001;42(5):679-689.

53. Luby JL, Mrakotsky C, Heffelfinger A, Brown K, Martha Hessler B, Edward Spitznagel B. Modification of DSM-IV Criteria for Depressed Preschool Children. *Am J Psychiatry*. 2003;1606.

54. Luby JL, Heffelfinger AK, Mrakotsky C, et al. The Clinical Picture of Depression in Preschool Children. *J Am Acad Child Adolesc Psychiatry*. 2003;42(3). doi:10.1097/01.CHI.0000037035.04952.F8.

55. Boyce WT, Quas J, Alkon A, et al. Autonomic reactivity and psychopathology in middle childhood. *Br J Psychiatry*. 2001;179(2):144-150. doi:10.1192/bjp.179.2.144.

56. Whittle S, Lichter R, Dennison M, et al. Structural brain development and depression onset during adolescence: a prospective longitudinal study. *Am J Psychiatry*. 2014;171(5):564-571. doi:10.1176/appi.ajp.2013.13070920.

57. Birmaher B, Williamson DE, Dahl RE, et al. Clinical Presentation and Course of Depression in Youth: Does Onset in Childhood Differ From Onset in Adolescence? *J Am Acad Child Adolesc Psychiatry*. 2004;43(1):63-70. doi:10.1097/00004583-200401000-00015.

58. Angold A, Costello E. The epidemiology of depression in children and adolescents. In: *The Depressed Child and Adolescent*. Cambridge: Cambridge University Press; 2001:143-178.

59. Egger HL, Angold A. Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. *J Child Psychol Psychiatry*. 2006;47(3-4):313-337. doi:10.1111/j.1469-7610.2006.01618.x.

60. Maughan B, Collishaw S, Stringaris A. Depression in childhood and adolescence. *J Can Acad Child Adolesc Psychiatry*. 2013;22(1):35-40. http://www.ncbi.nlm.nih.gov/pubmed/23390431. Accessed October 9, 2017.

61. Loeber R. Questions and advances in the study of developmental pathways. In: *Rochester Symposium on Developmental Psychopathology, Models and Integrations*. 3rd ed. Rochester, NY: University of Rochester Press; 1991:97-116.

62. Reitz E, Dekovi M, Meijer AM. The Structure and Stability of Externalizing and Internalizing Problem Behavior During Early Adolescence. *J Youth Adolesc*. 2005;34(6):577-588. doi:10.1007/s10964-005-8947-z.

63. Mesman J, Stoel R, Bakermans-Kranenburg MJ, et al. Predicting growth curves of early childhood externalizing problems: Differential susceptibility of children with difficult temperament. *J Abnorm Child Psychol*. 2009;37(5):625-636. doi:10.1007/s10802-009-9298-0.

64. Hartup WW. Aggression in childhood: Developmental perspectives. *Am Psychol*. 1974;29(5):336-341. doi:10.1037/h0037622.

65. Tremblay RE. The development of agressive behaviour during childhood: What have we learned in the past century? *Int J Behav Dev*. 2000;24(2):129-141. doi:10.1080/016502500383232.

66. Fanti K a, Henrich CC. Trajectories of pure and co-occurring internalizing and externalizing problems from age 2 to age 12: findings from the National Institute of Child Health and Human Development Study of Early Child Care. *Dev Psychol*. 2010;46(5):1159-1175. doi:10.1037/a0020659.

67. Gilliom M, Shaw DS. Codevelopment of externalizing and internalizing problems in early childhood. *Dev Psychopathol*. 2004;16(2):313-333. http://www.ncbi.nlm.nih.gov/pubmed/15487598. Accessed October 4, 2017.

68. Campbell SB, Shaw DS, Gilliom M. Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Dev Psychopathol*. 2000;12:467-488.

69. Hill AL, Degnan KA, Calkins SD, Keane SP. Profiles of externalizing behavior problems for boys and girls across preschool: the roles of emotion regulation and inattention. *Dev Psychol*. 2006;42(5):913-928. doi:10.1037/0012-1649.42.5.913.

70. Barkley R. *Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment*. New York: Guilford Press; 1998.

71. Lavigne J V., Gibbons RD, Christoffel KK, et al. Prevalence Rates and Correlates of Psychiatric Disorders among Preschool Children. *J Am Acad Child Adolesc Psychiatry*. 1996;35(2):204-214. doi:10.1097/00004583-199602000-00014.

72. Association AP. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Washington, DC; 2000.

73. Center for Disease Control and Prevention. Attention-Deficit / Hyperactivity Disorder (ADHD). https://www.cdc.gov/ncbddd/adhd/data.html. Published 2013. Accessed October 9, 2017.

74. Mckeown RE, Holbrook JR, Danielson ML, Cuffe SP, Wolraich ML, Visser SN. The Impact of Case Definition on Attention-Deficit/ Hyperactivity Disorder Prevalence Estimates in Community-Based Samples of School-Aged Children. *J Am Acad Child Adolesc Psychiatry*. 2015;54:53-61. doi:10.1016/j.jaac.2014.10.014.

75. Chan E, Fogler JM, Hammerness PG, et al. Treatment of Attention-Deficit/Hyperactivity Disorder in Adolescents. *JAMA*. 2016;315(18):1997. doi:10.1001/jama.2016.5453.

76. Desgranges K, Desgranges L, Karsky K. Attention deficit disorder: Problems with preconceived diagnosis. *Child Adolesc Soc Work J*. 1995;12(1):3-17. doi:10.1007/BF01876136.

77. Reid R, Riccio CA, Kessler RH, et al. Gender and Ethnic Differences in ADHD as Assessed by Behavior Ratings. *J Emot Behav Disord*. 2000;8(1):38-48. doi:10.1177/106342660000800105.

78. Kube DA, Petersen MC, Palmer FB. Attention Deficit Hyperactivity Disorder: Comorbidity and Medication Use. *Clin Pediatr (Phila)*. 2002;41(7):461-469. doi:10.1177/000992280204100702.

79. Busch B, Biederman J, Cohen LG, et al. Correlates of ADHD Among Children in Pediatric and Psychiatric Clinics. *Psychiatr Serv*. 2002;53(9):1103-1111. doi:10.1176/appi.ps.53.9.1103.

80. Cuffe SP, Moore CG, McKeown RE. Prevalence and Correlates of ADHD Symptoms in the National Health Interview Survey. *J Atten Disord*. 2005;9(2):392-401. doi:10.1177/1087054705280413.

81. Sciutto MJ, Eisenberg M. Evaluating the Evidence For and Against the Overdiagnosis of ADHD. *J Atten Disord*. 2007;11(2):106-113. doi:10.1177/1087054707300094.

82. Maughan B, Rowe R, Messer J, Goodman R, Meltzer H. Conduct disorder and oppositional defiant disorder in a national sample: developmental epidemiology. *J Child Psychol Psychiatry*. 2004;45(3):609-621. http://www.ncbi.nlm.nih.gov/pubmed/15055379. Accessed October 3, 2017.

83. Frick PJ, Dickens C. Current perspectives on conduct disorder. *Curr Psychiatry Rep*. 2006;8(1):59-72. http://www.ncbi.nlm.nih.gov/pubmed/16513044. Accessed October 9, 2017.

84. Fergusson D, Boden J, Hayne H. Childhood conduct problems. In:

Improving the Transition Reducing Social and Psychological Morbidity During Adolescence. Auckland, New Zealand: Once of the Prime Minister's Science Advisory Committee; 2011:41-60.

85. Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. Prevalence and Development of Psychiatric Disorders in Childhood and Adolescence. *Arch Gen Psychiatry*. 2003;60(8):837. doi:10.1001/archpsyc.60.8.837.

86. Ford T, Psych MRC, Goodman R, Psych FRC, Meltzer H. The British Child and Adolescent Mental Health Survey 1999: The Prevalence of DSM-IV Disorders. *J Am Acad Child Adolesc Psychiatry*. 2003;42(10):1203-1211. doi:10.1097/01.chi.0000081820.25107.ae.

87. Centre ICE. *Conduct: Disorder in Children and Adolescents*. Paris: Institut national de la santé et de la recherche médicale; 2005. http://www.ncbi.nlm.nih.gov/pubmed/21348167. Accessed November 14, 2017.

88. Boat TF, Wu JT, Disorders C to E the SSIDP for C with M, et al. Prevalence of Oppositional Defiant Disorder and Conduct Disorder. In: *Mental Disorders and Disabilities Among Low-Income Children.* Washington, DC: National Academies Press (US); 2015. https://www.ncbi.nlm.nih.gov/books/NBK332874/. Accessed October 9, 2017.

89. Hirschfeld RMA. The Comorbidity of Major Depression and Anxiety Disorders: Recognition and Management in Primary Care. *Prim Care Companion J Clin Psychiatry*. 2001;3(6):244-254. http://www.ncbi.nlm.nih.gov/pubmed/15014592. Accessed January 30, 2017.

90. Brady EU, Kendall PC. Comorbidity of anxiety and depression in children and adolescents. *Psychol Bull*. 1992;111(2):244-255. http://www.ncbi.nlm.nih.gov/pubmed/1557475. Accessed October 2, 2017.

91. Angold A, Costello EJ, Erkanli A. Comorbidity. *J Child Psychol Psychiatry*. 1999;40(1):57-87. http://www.ncbi.nlm.nih.gov/pubmed/10102726. Accessed October 2, 2017.

92. Avenevoli S, Stolar M, Li J, Dierker L, Ries Merikangas K. Comorbidity of depression in children and adolescents: models and evidence from a prospective high-risk family study. *Biol Psychiatry*. 2001;49(12):1071-1081. http://www.ncbi.nlm.nih.gov/pubmed/11430849. Accessed October 2, 2017.

93. Yorbik O, Birmaher B, Axelson D, Williamson DE, Ryan ND. Clinical characteristics of depressive symptoms in children and adolescents with major depressive disorder. *J Clin Psychiatry*. 2004;65(12):1654-9-1. http://www.ncbi.nlm.nih.gov/pubmed/15641870. Accessed October 2,

2017.

94. Garber J, Weersing VR. Comorbidity of Anxiety and Depression in Youth: Implications for Treatment and Prevention. *Clin Psychol (New York)*. 2010;17(4):293-306. doi:10.1111/j.1468-2850.2010.01221.x.

95. Angold A, Costello EJ, Erkanli A. Comorbidity. *J Child Psychol Psychiat Assoc Child Psychol Psychiatry*. 1999;40(1):57-87.

96. Axelson DA, Birmaher B. Relation between anxiety and depressive disorders in childhood and adolescence. *Depress Anxiety*. 2001;14(2):67-78. http://www.ncbi.nlm.nih.gov/pubmed/11668659. Accessed October 2, 2017.

97. Pine DS, Cohen P, Gurley D, et al. The Risk for Early-Adulthood Anxiety and Depressive Disorders in Adolescents With Anxiety and Depressive Disorders. *Arch Gen Psychiatry*. 1998;55(1):56. doi:10.1001/archpsyc.55.1.56.

98. Cole DA, Peeke LG, Martin JM, Truglio R, Seroczynski AD. A longitudinal look at the relation between depression and anxiety in children and adolescents. *J Consult Clin Psychol*. 1998;66(3):451-460. doi:10.1037/0022-006X.66.3.451.

99. Chorpita BF, Daleiden EL. Tripartite dimensions of emotion in a child clinical sample: measurement strategies and implications for clinical utility. *J Consult Clin Psychol*. 2002;70(5):1150-1160. http://www.ncbi.nlm.nih.gov/pubmed/12362965. Accessed October 2, 2017.

100. Cummings CM, Caporino NE, Kendall PC. Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychol Bull*. 2014;140(3):816-845. doi:10.1037/a0034733.

101. Gallerani CM, Garber J, Martin NC. The temporal relation between depression and comorbid psychopathology in adolescents at varied risk for depression. *J Child Psychol Psychiatry*. 2010;51(3):242-249. doi:10.1111/j.1469-7610.2009.02155.x.

102. Kaufman J, Charney D. Comorbidity of mood and anxiety disorders. *Depress Anxiety*. 2000;12(S1):69-76. doi:10.1002/1520-6394(2000)12:1+<69::AID-DA9>3.0.CO;2-K.

103. Stark KD, Laurent J. Joint Factor Analysis of the Children's Depression Inventory and the Revised Children's Manifest Anxiety Scale. *J Clin Child Adolesc Psychol*. 2001;30(4):552-567. doi:10.1207/S15374424JCCP3004_11.

104. Cole DA, Truglio R, Peeke L. Relation between symptoms of anxiety and depression in children: a multitrait-multimethod-multigroup assessment. *J Consult Clin Psychol*. 1997;65(1):110-119.

http://www.ncbi.nlm.nih.gov/pubmed/9103740. Accessed October 2, 2017.

105. Barlow DH. Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *Am Psychol*. 2000;55(11):1247-1263. http://www.ncbi.nlm.nih.gov/pubmed/11280938. Accessed October 2, 2017.

106. Clark LA, Watson D. Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *J Abnorm Psychol*. 1991;100(3):316-336. http://www.ncbi.nlm.nih.gov/pubmed/1918611. Accessed October 2, 2017.

107. Bienvenu OJ, Ginsburg GS. Prevention of anxiety disorders. *Int Rev Psychiatry*. 2007;19(6):647-654. doi:10.1080/09540260701797837.

108. Kendall PC, Safford S, Flannery-Schroeder E, Webb A. Child Anxiety Treatment: Outcomes in Adolescence and Impact on Substance Use and Depression at 7.4-Year Follow-Up. *J Consult Clin Psychol*. 2004;72(2):276-287. doi:10.1037/0022-006X.72.2.276.

109. Wadsworth ME, Hudziak JJ, Heath AC, Achenbach TM. Latent Class Analysis of Child Behavior Checklist Anxiety/Depression in Children and Adolescents. *J Am Acad Child Adolesc Psychiatry*. 2001;40(1):106-114. doi:10.1097/00004583-200101000-00023.

110. van Lang N, Ferdinand R, Ormel J, Verhulst F. Latent class analysis of anxiety and depressive symptoms of the Youth Self-Report in a general population sample of young adolescents. *Behav Res Ther*. 2006;44(6):849-860. doi:10.1016/J.BRAT.2005.06.004.

111. Biederman J, Mick E, Faraone S V. Depression in attention deficit hyperactivity disorder (ADHD) children: Depression or demoralization? *J Affect Disord*. 1998;47(1-3):113-122. doi:10.1016/S0165-0327(97)00127-4.

112. Power TJ, Costigan TE, Eiraldi RB, Leff SS. Variations in anxiety and depression as a function of ADHD subtypes defined by DSM-IV: do subtype differences exist or not? *J Abnorm Child Psychol*. 2004;32(1):27-37. http://www.ncbi.nlm.nih.gov/pubmed/14998109. Accessed October 3, 2017.

113. Ostrander R, Crystal DS, August G. Attention Deficit-Hyperactivity Disorder, Depression, and Self- and Other-Assessments of Social Competence: A Developmental Study. *J Abnorm Child Psychol*. 2006;34(6):772-786. doi:10.1007/s10802-006-9051-x.

114. Biederman J, Ball SW, Monuteaux MC, et al. New Insights Into the Comorbidity Between ADHD and Major Depression in Adolescent and Young Adult Females. *J Am Acad Child Adolesc Psychiatry*. 2008;47(4):426-434. doi:10.1097/CHI.0b013e31816429d3.

115. Lavigne J V., LeBailly SA, Hopkins J, Gouze KR, Binns HJ. The Prevalence of ADHD, ODD, Depression, and Anxiety in a Community Sample of 4-Year-Olds. *J Clin Child Adolesc Psychol*. 2009;38(3):315-328. doi:10.1080/15374410902851382.

116. Chronis-Tuscano A, Molina BSG, Pelham WE, et al. Very Early Predictors of Adolescent Depression and Suicide Attempts in Children With Attention-Deficit/Hyperactivity Disorder. *Arch Gen Psychiatry*. 2010;67(10):1044. doi:10.1001/archgenpsychiatry.2010.127.

117. Fergusson DM, Boden JM, Horwood LJ. Classification of behavior disorders in adolescence: Scaling methods, predictive validity, and gender differences. *J Abnorm Psychol*. 2010;119(4):699-712. doi:10.1037/a0018610.

118. Meinzer MC, Pettit JW, Viswesvaran C. The co-occurrence of attention-deficit/hyperactivity disorder and unipolar depression in children and adolescents: A meta-analytic review. *Clin Psychol Rev*. 2014;34(8):595-607. doi:10.1016/j.cpr.2014.10.002.

119. Last CG, Hersen M, Kazdin A, Orvaschel H, Perrin S. Anxiety disorders in children and their families. *Arch Gen Psychiatry*. 1991;48(10):928-934. http://www.ncbi.nlm.nih.gov/pubmed/1929763. Accessed October 2, 2017.

120. Jensen PS, Martin D, Cantwell DP. Comorbidity in ADHD: Implications for Research, Practice, and DSM-V. *J Am Acad Child Adolesc Psychiatry*. 1997;36(8):1065-1079. doi:10.1097/00004583-199708000-00014.

121. Pliszka SR. Patterns of psychiatric comorbidity with attention-deficit/hyperactivity disorder. *Child Adolesc Psychiatr Clin N Am*. 2000;9(3):525-40, vii. http://www.ncbi.nlm.nih.gov/pubmed/10944655. Accessed October 2, 2017.

122. Tannock R. Attention-deficit/hyperactivity disorder with anxiety disorders. In: *Attention-Deficit Disorders and Comorbidities in Children, Adolescents, and Adults*. Ed. Washington D.C.: American Psychiatric Press; 2000:235-170.

123. Jensen PS, Hinshaw SP, Kraemer HC, et al. ADHD Comorbidity Findings From the MTA Study: Comparing Comorbid Subgroups. *J Am Acad Child Adolesc Psychiatry*. 2001;40(2):147-158. doi:10.1097/00004583-200102000-00009.

124. Nigg JT, Goldsmith HH, Sachek J. Temperament and attention deficit hyperactivity disorder: the development of a multiple pathway model. *J Clin Child Adolesc Psychol*. 2004;33(1):42-53. doi:10.1207/S15374424JCCP3301_5.

125. Souza I, Pinheiro M, Denardin D, Mattos P, Rohde L. Attention-Deficit/Hyperactivity Disorder and comorbidity in Brazil. *Eur Child Adolesc Psychiatry*. 2004;13(4):243-248. doi:10.1007/s00787-004-0402-2.

126. Biederman J. Attention-Deficit/Hyperactivity Disorder: A Selective Overview. *Biol Psychiatry*. 2005;57(11):1215-1220. doi:10.1016/j.biopsych.2004.10.020.

127. Jarrett MA, Ollendick TH. Treatment of Comorbid Attention-Deficit/Hyperactivity Disorder and Anxiety in Children: A Multiple Baseline Design Analysis. *J Consult Clin Psychol*. 2012;80(2):239-244. doi:10.1037/a0027123.

128. Vance A, Ferrin M, Winther J, Gomez R. Examination of Spatial Working Memory Performance in Children and Adolescents with Attention Deficit Hyperactivity Disorder, Combined Type (ADHD-CT) and Anxiety. *J Abnorm Child Psychol*. 2013;41(6):891-900. doi:10.1007/s10802-013-9721-4.

129. Steinberg EA, Drabick DAG. A Developmental Psychopathology Perspective on ADHD and Comorbid Conditions: The Role of Emotion Regulation. *Child Psychiatry Hum Dev*. 2015;46(6):951-966. doi:10.1007/s10578-015-0534-2.

130. Overgaard K, Aase H, Torgersen S, Zeiner P. Co-Occurrence of ADHD and Anxiety in Preschool Children. *J Atten Disord*. 2016;20(7):573-580. doi:10.1177/1087054712463063.

131. Pliszka SR. Comorbidity of attention-deficit/hyperactivity disorder with psychiatric disorder: an overview. *J Clin Psychiatry*. 1998;59 Suppl 7:50-58. http://www.ncbi.nlm.nih.gov/pubmed/9680053. Accessed October 2, 2017.

132. Schatz DB, Rostain AL. ADHD With Comorbid Anxiety. *J Atten Disord*. 2006;10(2):141-149. doi:10.1177/1087054706286698.

133. Hammerness P, Geller D, Petty C, Lamb A, Bristol E, Biederman J. Does ADHD moderate the manifestation of anxiety disorders in children? *Eur Child Adolesc Psychiatry*. 2010;19(2):107-112. doi:10.1007/s00787-009-0041-8.

134. Jarrett MA, Ollendick TH. A conceptual review of the comorbidity of attention-deficit/hyperactivity disorder and anxiety: Implications for future research and practice. *Clin Psychol Rev*. 2008;28:1266-1280. doi:10.1016/j.cpr.2008.05.004.

135. Kadesjo B, Gillberg C. The Comorbidity of ADHD in the General Population of Swedish School-age Children. *J Child Psychol Psychiat Assoc Child Psychol Psychiatry*. 2001;42(4):487-492. https://journals-scholarsportal-info.proxy.bib.uottawa.ca/pdf/00219630/v42i0004/487_tcoaitgpossc.xml. Accessed October 3, 2017.

136. Wilens TE, Biederman J, Brown S, et al. Psychiatric Comorbidity and Functioning in Clinically Referred Preschool Children and School-Age

Youths With ADHD. *J Am Acad Child Adolesc Psychiatry*. 2002;41(3):262-268. doi:10.1097/00004583-200203000-00005.

137. Spencer T, Biederman J, Wilens T. Attention-deficit/hyperactivity disorder and comorbidity. *Pediatr Clin North Am*. 1999;46(5):915-27, vii. http://www.ncbi.nlm.nih.gov/pubmed/10570696. Accessed October 3, 2017.

138. Steinhausen H-C, Nøvik TS, Baldursson G, et al. Co-existing psychiatric problems in ADHD in the ADORE cohort. *Eur Child Adolesc Psychiatry*. 2006;15(S1):i25-i29. doi:10.1007/s00787-006-1004-y.

139. The MTA Cooperative Group. A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder. *Arch Gen Psychiatry*. 1999;56(12):1073. doi:10.1001/archpsyc.56.12.1073.

140. Connor DF, Doerfler LA. ADHD With Comorbid Oppositional Defiant Disorder or Conduct Disorder. *J Atten Disord*. 2008;12(2):126-134. doi:10.1177/1087054707308486.

141. Lewinsohn PM, Rohde P, Seeley JR. Adolescent psychopathology: III. The clinical consequences of comorbidity. *J Am Acad Child Adolesc Psychiatry*. 1995;34(4):510-519. http://www.ncbi.nlm.nih.gov/pubmed/7751265. Accessed October 3, 2017.

142. Beyers JM, Loeber R. Untangling developmental relations between depressed mood and delinquency in male adolescents. *J Abnorm Child Psychol*. 2003;31(3):247-266. http://www.ncbi.nlm.nih.gov/pubmed/12774859. Accessed October 3, 2017.

143. Wiesner M. A Longitudinal Latent Variable Analysis of Reciprocal Relations Between Depressive Symptoms and Delinquency During Adolescence. *J Abnorm Psychol*. 2003;112(4):633-645. doi:10.1037/0021-843X.112.4.633.

144. Ingoldsby EM, Kohl GO, McMahon RJ, Lengua L, Conduct Problems Prevention Research Group TCPPR. Conduct problems, depressive symptomatology and their co-occurring presentation in childhood as predictors of adjustment in early adolescence. *J Abnorm Child Psychol*. 2006;34(5):603-621. doi:10.1007/s10802-006-9044-9.

145. Moilanen KL, Shaw DS, Maxwell KL. Developmental cascades: externalizing, internalizing, and academic competence from middle childhood to early adolescence. *Dev Psychopathol*. 2010;22(3):635-653. doi:10.1017/S0954579410000337.

146. Obradović J, Hipwell A. Psychopathology and social competence during the transition to adolescence: the role of family adversity and pubertal development. *Dev Psychopathol*. 2010;22(3):621-634. doi:10.1017/S0954579410000325.

147. Gooren EMJC, van Lier PAC, Stegge H, Terwogt MM, Koot HM. The Development of Conduct Problems and Depressive Symptoms in Early Elementary School Children: The Role of Peer Rejection. *J Clin Child Adolesc Psychol*. 2011;40(2):245-253. doi:10.1080/15374416.2011.546045.

148. Brensilver M, Negriff S, Mennen FE, Trickett PK. Longitudinal relations between depressive symptoms and externalizing behavior in adolescence: moderating effects of maltreatment experience and gender. *J Clin Child Adolesc Psychol*. 2011;40(4):607-617. doi:10.1080/15374416.2011.581618.

149. Basten M, Tiemeier H, Althoff RR, et al. The Stability of Problem Behavior Across the Preschool Years: An Empirical Approach in the General Population. *J Abnorm Child Psychol*. 2016;44(2):393-404. doi:10.1007/s10802-015-9993-y.

150. Ayer L, Althoff R, Ivanova M, et al. Child Behavior Checklist Juvenile Bipolar Disorder (CBCL-JBD) and CBCL Posttraumatic Stress Problems (CBCL-PTSP) scales are measures of a single dysregulatory syndrome. *J Child Psychol Psychiatry*. 2009;50(10):1291-1300. doi:10.1111/j.1469-7610.2009.02089.x.

151. Althoff RR, Rettew DC, Ayer LA, Hudziak JJ. Cross-informant agreement of the Dysregulation Profile of the Child Behavior Checklist. *Psychiatry Res*. 2010;178(3):550-555. doi:10.1016/j.psychres.2010.05.002.

152. Althoff RR, Verhulst FC, Rettew DC, Hudziak JJ, van der Ende J. Adult Outcomes of Childhood Dysregulation: A 14-year Follow-up Study. *J Am Acad Child Adolesc Psychiatry*. 2010;49(11):1105-1116.e1. doi:10.1016/j.jaac.2010.08.006.

153. Briggs-Gowan MJ, Carter AS, Bosson-Heenan J, Guyer AE, Horwitz SM. Are Infant-Toddler Social-Emotional and Behavioral Problems Transient? *J Am Acad Child Adolesc Psychiatry*. 2006;45(7):849-858. doi:10.1097/01.chi.0000220849.48650.59.

154. Fischer M, Rolf J, Hasazi J, Cummings L. Follow-up of a preschool epidemiological sample - cross-age continuities and predictions of later adjustment with internalizing and externalizing dimensions of behavior. *Child Dev* . 1984;55(1):137-150.

155. Campbell SB. Behavior Problems in Preschool Children: A Review of Recent Research. *J Child Psychol Psychiatry*. 1995;36(1):113-149. doi:10.1111/j.1469-7610.1995.tb01657.x.

156. Keenan K, Shaw D, Delliquadri E, Giovannelli J, Walsh B. Evidence for the Continuity of Early Problem Behaviors: Application of a Developmental Model. *J Abnorm Child Psychol*. 1998;26(6):441-452. doi:10.1023/A:1022647717926.

157. Mathiesen KS, Sanson A. Dimensions of early childhood behavior problems: stability and predictors of change from 18 to 30 months. *J Abnorm Child Psychol*. 2000;28(1):15-31. http://www.ncbi.nlm.nih.gov/pubmed/10772347. Accessed October 4, 2017.

158. Mian ND, Wainwright L, Briggs-Gowan MJ, Carter AS. An Ecological Risk Model for Early Childhood Anxiety: The Importance of Early Child Symptoms and Temperament. *J Abnorm Child Psychol*. 2011;39(4):501-512. doi:10.1007/s10802-010-9476-0.

159. Vaillancourt T, Brittain HL, McDougall P, Duku E. Longitudinal Links Between Childhood Peer Victimization, Internalizing and Externalizing Problems, and Academic Functioning: Developmental Cascades. *J Abnorm Child Psychol*. 2013;41(8):1203-1215. doi:10.1007/s10802-013-9781-5.

160. Willner CJ, Gatzke-Kopp LM, Bray BC. The dynamics of internalizing and externalizing comorbidity across the early school years. *Dev Psychopathol*. 2016;28(4pt1):1033-1052. doi:10.1017/S0954579416000687.

161. Rudolph KD, Lansford JE, Agoston AM, et al. Peer victimization and social alienation: predicting deviant peer affiliation in middle school. *Child Dev*. 2014;85(1):124-139. doi:10.1111/cdev.12112.

162. Roza SJ, Hofstra MB, Ende J van der, Verhulst FC. Stable Prediction of Mood and Anxiety Disorders Based on Behavioral and Emotional Problems in Childhood: A 14-Year Follow-Up During Childhood, Adolescence, and Young Adulthood. *http://dx.doi.org/101176/appi.ajp160122116*. 2003;160(12):2116-2121.

163. Caspi A, Moffitt TE, Newman DL, et al. Behavioral Observations at Age 3 Years Predict Adult Psychiatric Disorders. *Arch Gen Psychiatry*. 1996;53(11):1033. doi:10.1001/archpsyc.1996.01830110071009.

164. Blair-West GW, Cantor CH, Mellsop GW, Eyeson-Annan ML. *Lifetime Suicide Risk in Major Depression: Sex and Age Determinants*. Vol 55.; 1999. doi:10.1016/S0165-0327(99)00004-X.

165. Angst J, Angst F, Stassen HH. Suicide Risk in Patients With Major Depressive Disorder. *J Clin Psychiatry*. 1999;60(2).

166. Lewinsohn P, Clarke G, Seeley J, Rohde P. Major Depression in Community Adolescents: Age at Onset, Episode Duration, and Time to Recurrence. *J Am Acad Child Adolesc Psychiatry*. 1994;33(6):809-818. doi:10.1097/00004583-199407000-00006.

167. Bostwick JM, Pankratz VS. Affective Disorders and Suicide Risk: A Reexamination. *Am J Psychiatry*. 2000;157(12):1925-1932. doi:10.1176/appi.ajp.157.12.1925.

168. Gilmer WS, Trivedi MH, Rush AJ, et al. Factors associated with chronic

depressive episodes: a preliminary report from the STAR-D project. *Acta Psychiatr Scand*. 2005;112(6):425-433. doi:10.1111/j.1600-0447.2005.00633.x.

169. Nock MK, Hwang I, Sampson NA, Kessler RC. Mental disorders, comorbidity and suicidal behavior: Results from the National Comorbidity Survey Replication. *Mol Psychiatry*. 2010;15(8):868-876. doi:10.1038/mp.2009.29.

170. King SM, Iacono WG, McGue M. Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction*. 2004;99(12):1548-1559. doi:10.1111/j.1360-0443.2004.00893.x.

171. Puleo CM, Conner BT, Benjamin CL, Kendall PC. CBT for childhood anxiety and substance use at 7.4-year follow-up: a reassessment controlling for known predictors. *J Anxiety Disord*. 2011;25(5):690-696. doi:10.1016/j.janxdis.2011.03.005.

172. Merikangas K, Mehta R, Molnar BE, et al. Comorbidity of substance use disorders with mood and anxiety disorders: Results of the international consortium in psychiatric epidemiology. *Addict Behav*. 1998;23:893–907.

173. Kaplow JB, Curran PJ, Angold A, Costello EJ. The Prospective Relation Between Dimensions of Anxiety and the Initiation of Adolescent Alcohol Use. *J Clin Child Psychol*. 2001;30(3):316-326.

174. Hallfors DD, Waller MW, Ford CA, Halpern CT, Brodish PH, Iritani B. Adolescent Depression and Suicide Risk Association with Sex and Drug Behavior. *Am J Prev Med Am J Prev Med*. 2004;27(3):224-230. doi:10.1016/j.amepre.2004.06.001.

175. Swendsen J, Merikangas K. The comorbidity of depression and substance use disorders. *Clin Psychol Rev*. 2000;20(2):173-189. doi:10.1016/S0272-7358(99)00026-4.

176. Cairns RB, Cairns BD, Neckerman HJ, Gest SD, Gariepy L. Social Networks and Aggressive Behavior. *Dev Psychol*. 1988;24(6):815-823.

177. Dodge K. Social-Cognitive Mechanisms in the Development of Conduct Disorder and Depression. *Annu Rev Psychol*. 2003;44:559-584.

178. Jaffee SR, Moffitt TE, Caspi A, et al. Differences in Early Childhood Risk Factors for Juvenile-Onset and Adult-Onset Depression. *Arch Gen Psychiatry*. 2002;59(3):215. doi:10.1001/archpsyc.59.3.215.

179. Apter A, Kotler M, Sevy S, et al. Correlates of risk of suicide in violent and nonviolent psychiatric patients. *http://dx.doi.org/101176/ajp1487883*. 1991;148(7):883-887.

180. Ivanoff A. Background Risk Factors Associated with Parasuicide among Male Prison Inmates. *Crim Justice Behav*. 1992;19(4):426-436.

doi:10.1177/0093854892019004006.

181. Verona E, Sachs-Ericsson N, Thomas E. Joiner J. Suicide Attempts Associated With Externalizing Psychopathology in an Epidemiological Sample. *http://dx.doi.org/101176/appi.ajp1613444*. 2004.

182. Asarnow JR, Carlson G. Suicide Attempts in Preadolescent Child Psychiatry Inpatients. *Suicide Life-Threatening Behav*. 1988;18(2):129-136. doi:10.1111/j.1943-278X.1988.tb00147.x.

183. Capaldi DM. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: II. A 2-year follow-up at Grade 8. *Dev Psychopathol*. 1992;4(1):125. doi:10.1017/S0954579400005605.

184. Nottelmann E, Jensen P. Comorbidity of depressive disorders: Rates, temporal sequencing, course, and outcome. In: *Depressive Disorders in Children and Adolescents: Epidemiology, Risk Factors, and Treatment*. ; 1999:137-191.

185. Dishion TJ. Cross-Setting Consistency in Early Adolescent Psychopathology: Deviant Friendships and Problem Behavior Sequelae. *J Pers*. 2000;68(6):1109-1126. doi:10.1111/1467-6494.00128.

186. Hoyert DL, Heron MP, Murphy SL, Kung H-C. Deaths: final data for 2003. *Natl vital Stat reports*. 2006;54(13):1-120. http://www.ncbi.nlm.nih.gov/pubmed/16689256. Accessed November 13, 2017.

187. Gould MS, Greenberg T, Velting DM, Shaffer D. Youth Suicide Risk and Preventive Interventions: A Review of the Past 10 Years. *J Am Acad Child Adolesc Psychiatry*. 2003;42(4):386-405. doi:10.1097/01.CHI.0000046821.95464.CF.

188. Eaton DK, Kann L, Kinchen S, et al. Youth risk behavior surveillance--United States, 2007. *MMWR Surveill Summ*. 2008;57(4):1-131. http://www.ncbi.nlm.nih.gov/pubmed/18528314. Accessed June 29, 2016.

189. Cash SJ, Bridge JA. Epidemiology of youth suicide and suicidal behavior. *Curr Opin Pediatr*. 2009;21(5):613-619. doi:10.1097/MOP.0b013e32833063e1.

190. Office of Statistics and Programming NC for IP and C. 10 Leading Causes of Death by Age Group, United States - 2010. Centers for Disease Control and Prevention. https://www.cdc.gov/injury/wisqars/pdf/10lcid_all_deaths_by_age_group_2010-a.pdf. Published 2010. Accessed June 29, 2016.

191. Lewinsohn PM, Rohde P, Seeley JR. Psychosocial risk factors for future adolescent suicide attempts. *J Consult Clin Psychol*. 1994;62(2):297-305. doi:10.1037/0022-006X.62.2.297.

192. King C. Suicidal behavior in adolescence. In: Maris R, Silverman M,

Canetto S, eds. *Review of Suicidology*. New York City: Guilford Press; 1997.

193. Stoep A Vander, McCauley E, Flynn C, Stone A. Thoughts of death and suicide in early adolescence. *Suicide Life Threat Behav*. 2009;39(6):599-613. doi:10.1521/suli.2009.39.6.599.

194. Andrews J, Lewinsohn P. Suicidal Attempts among Older Adolescents: Prevalence and Co-occurrence with Psychiatric Disorders. *J Am Acad Child Adolesc Psychiatry*. 1992;31(4):655-662. doi:10.1097/00004583-199207000-00012.

195. Reinherz H, Tanner JL, Berger SR, Beardslee WR, Fitzmaurice GM. Adolescent Suicidal Ideation as Predictive of Psychopathology, Suicidal Behavior, and Compromised Functioning at Age 30. *Am J Psychiatry*. 2006;163(7):1226. doi:10.1176/appi.ajp.163.7.1226.

196. Coryell W, Young EA. Clinical predictors of suicide in primary major depressive disorder. *J Clin Psychiatry*. 2005;66(4):412-417. http://www.ncbi.nlm.nih.gov/pubmed/15816781. Accessed June 29, 2016.

197. Hauenstein E, Boyd M. Suicide: A Major Mental Health Problem. In: *Psychiatric Nursing: Contemporary Practice*. 4th ed. Edwardsville, IL: Wolters Kluwer; 2008:256-258.

198. Fawcett J. *Diagnosis, Traits, States, and Comorbidity in Suicide*. CRC Press/Taylor & Francis; 2012. http://www.ncbi.nlm.nih.gov/pubmed/23035297. Accessed June 29, 2016.

199. Public Health Agency of Canada. *The Cost of Injury in Canada*.; 2010. https://www.canada.ca/en/public-health/services/injury-prevention/cost-injury-canada.html. Accessed October 7, 2017.

200. Reynolds FMT, Cimbolic P. Attitudes toward Suicide Survivors as a Function of Survivors' Relationship to the Victim. *J Death Dying*. 1989;19(2):125-133. doi:10.2190/KR1X-QNG3-2YGM-UDYQ.

201. Range L m. When a loss is due to suicide: Unique aspects of bereavement. *J Pers Interpers Loss*. 1996;1(1):71-81. doi:10.1080/15325029608415460.

202. Stillion J. Survivors of suicide. In: *Living with Grief after Sudden Loss: Suicide, Homicide, Accident, Heart Attack, Stroke*. Ed. Washington D.C.: Hospice Foundation of America; 1996:41-51.

203. Cerel J, Jordan JR, Duberstein PR. The Impact of Suicide on the Family Family Members as Survivors. *Crisis*. 2008;29(1):38-44. doi:10.1027/0227-5910.29.1.38.

204. Cerel J, Roberts TA. Suicidal behavior in the family and adolescent risk behavior. *J Adolesc Heal*. 2005;36(4):352.e8-352.e14. doi:10.1016/j.jadohealth.2004.08.010.

205. Séguin M, Lesage A, Kiely MC. Parental bereavement after suicide and accident: a comparative study. *Suicide Life Threat Behav*. 1995;25(4):489-492. http://www.ncbi.nlm.nih.gov/pubmed/8928203. Accessed October 7, 2017.

206. Higgins L, Range LM. Does Information That a Suicide Victim Was Psychiatrically Disturbed Reduce the Likelihood of Contagion?1. *J Appl Soc Psychol*. 1996;26(9):781-785. doi:10.1111/j.1559-1816.1996.tb01130.x.

207. Peltzer K, Cherian VI, Cherian L. Attitudes toward Suicide among South African Secondary School Pupils. *Psychol Rep*. 1998;83(3_suppl):1259-1265. doi:10.2466/pr0.1998.83.3f.1259.

208. Dyregrov A, Gjestad R, Bie Wikander AM, Vigerust S. Reactions following the sudden death of a classmate. *Scand J Psychol*. 1999;40(3):167-176. doi:10.1111/1467-9450.00114.

209. Poijula S, Wahlberg K, Dyregrov A. Adolescent suicide and suicide contagion in three schools. *Int J Emerg Ment Health*. 2001:163-170. https://scholar.google.ca/scholar?hl=en&as_sdt=0%2C5&as_vis=1&q=Adolescent+suicide+and+suicide+contagion+in+three+secondary+schools&btnG=. Accessed October 7, 2017.

210. Swanson SA, Colman I. Association between exposure to suicide and suicidality outcomes in youth. *CMAJ*. 2013;185(10):870-877. doi:10.1503/cmaj.121377.

211. Randall J, Nickel N, Colman I. Contagion from peer suicidal behavior in a representative sample of American adolescents. *J Affect Disord*. 2015;186:219-225. doi:10.1016/J.JAD.2015.07.001.

212. Squeglia LM, Jacobus J, Tapert SF. The influence of substance use on adolescent brain development. *Clin EEG Neurosci*. 2009;40(1):31-38. doi:10.1177/155005940904000110.

213. Clay S. Risk factors for addiction. *Osteopath Fam Physician*. 2010;2(2):41-45. doi:10.1016/J.OSFP.2009.12.002.

214. Malakieh J. *Youth Correctional Statistics in Canada, 2015/2016*. Statistics Canada; 2016. https://www.statcan.gc.ca/pub/85-002-x/2017001/article/14702-eng.htm. Accessed October 9, 2017.

215. Gabor T. *Costs of Crime and Criminal Justice Responses*. Ottawa; 2015. https://www.publicsafety.gc.ca/cnt/rsrcs/pblctns/2015-r022/2015-r022-en.pdf. Accessed October 9, 2017.

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

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

218. Patten SB, Wilkes TCR, Williams JVA, et al. Retrospective and prospectively assessed childhood adversity in association with major depression, alcohol consumption and painful conditions. *Epidemiol Psychiatr Sci*. 2015;24(2):158-165. doi:10.1017/S2045796014000018.

219. Statistics Canada. *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 4.* Ottawa; 2001.

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

221. Statistics Canada. *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 6.*; 2005.

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

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

224. Statistics Canada. *National Longitudinal Survey of Children and Youth: Microdata User Guide - Cycle 3.* Ottawa; 1999.

225. Baker BL, Blacher J, Crnic KA, Edelbrock C. Behavior Problems and Parenting Stress in Families of Three-Year-Old Children With and Without Developmental Delays. *Am J Ment Retard*. 2002;107(6):433. doi:10.1352/0895-8017(2002)107<0433:BPAPSI>2.0.CO;2.

226. Kerr DCR, Lunkenheimer ES, Olson SL. Assessment of child problem behaviors by multiple informants: a longitudinal study from preschool to school entry. *J Child Psychol Psychiatry*. 2007;48(10):967-975. doi:10.1111/j.1469-7610.2007.01776.x.

227. Combs-Ronto LA, Olson SL, Lunkenheimer ES, Sameroff AJ. Interactions Between Maternal Parenting and Children's Early Disruptive Behavior: Bidirectional Associations across the Transition from Preschool to School Entry. *J Abnorm Child Psychol*. 2009;37(8):1151-1163. doi:10.1007/s10802-009-9332-2.

228. Neece CL, Green SA, Baker BL. Parenting stress and child behavior problems: a transactional relationship across time. *Am J Intellect Dev Disabil*. 2012;117(1):48-66. doi:10.1352/1944-7558-117.1.48.

229. Shain B. Suicide and Suicide Attempts in Adolescents. *Pediatrics*. 2016;138(1). doi:10.1542/peds.2016-1420.

230. Boyle M, Offord D, Hofmann H. Ontario Child Health Study. 1.Methodology. *Arch Gen Psychiatry*. 1987;44(9):826-831.

231. Statistics Canada. *Ontario Child Health Study (OCHS) - 1983 Codebook*. Hamilton, ON; 1983.

232. Boyle MH, Offord DR, Racine Y, Sanford M, Szatmari P, Fleming J. Evaluation of the Original Ontario-Child-Health-Study Scales. *Can J Psychiatry-Revue Can Psychiatr*. 1993;38:397-405.

233. Boyle MH, Offord DR, Racine Y, Fleming JE, Szatmari P, Sanford M. Evaluation of the revised Ontario Child Health Study scales. *J Child Psychol Psychiatry*. 1993;34(2):189-213. http://www.ncbi.nlm.nih.gov/pubmed/8444992. Accessed October 14, 2017.

234. North CR, Wild TC, Zwaigenbaum L, Colman I. Early Neurodevelopment and Self-Reported Adolescent Symptoms of Depression and Anxiety in a National Canadian Cohort Study. Potash JB, ed. *PLoS One*. 2013;8(2):e56804. doi:10.1371/journal.pone.0056804.

235. Weeks M, Cairney J, Wild TC, Ploubidis GB, Naicker K, Colman I. Early-life Predictors of Internalizing Symptom Trajectories in Canadian Children. *Depress Anxiety*. 2014;31:608-616. doi:10.1002/da.22235.

236. Weeks M, Wild TC, Ploubidis GB, et al. Childhood cognitive ability and its relationship with anxiety and depression in adolescence. *J Affect Disord*. 2014;152-154:139-145. doi:10.1016/j.jad.2013.08.019.

237. Kingsbury M, Kirkbride JB, McMartin SE, Wickham ME, Weeks M, Colman I. Trajectories of childhood neighbourhood cohesion and adolescent mental health: evidence from a national Canadian cohort. *Psychol Med*. 2015;45(15):3239-3248. doi:10.1017/S0033291715001245.

238. Statistics Canada. NLSCY - Content and Validation of NLSCY DATA (9.1-9.11). http://people.ucalgary.ca/~libdata/adc/old_file_info/kids/k96-9a.htm. Published 1995.

239. Dupéré V, Leventhal T, Lacourse É. Neighborhood poverty and suicidal thoughts and attempts in late adolescence. *Psychol Med*. 2009;39:1295-1306. doi:10.1017/S003329170800456X.

240. Weeks M, Colman I. Predictors of Suicidal Behaviors in Canadian Adolescents with No Recent History of Depression. *Arch Suicide Res*. 2016;21(2):354-364. doi:10.1080/13811118.2016.1193076.

241. Nock MK, Borges G, Bromet EJ, Cha CB, Kessler RC, Lee S. Suicide and suicidal behavior. *Epidemiol Rev*. 2008;30(1):133-154. doi:10.1093/epirev/mxn002.

242. *National Longitudinal Survey of Youth 1979 Cohort, 1979-2012 (Rounds 1-25)*. Columbus, OH: Center for Human Resource Research; 2014.

243. Zubrick S, Silburn S, Garton A, et al. *Western Australian Child Health Survey: Developing Health and Well-Being in the Nineties.* Perth, Australia; 1995.

244. Wickham ME, Senthilselvan A, Wild TC, Hoglund WLG, Colman I.

Maternal depressive symptoms during childhood and risky adolescent health behaviors. *Pediatrics*. 2015;135(1):59-67. doi:10.1542/peds.2014-0628.

245. Gaub M, Carlson C. Gender Differences in ADHD: A Meta-Analysis and Critical Review. *J Am Acad Child Adolesc Psychiatry*. 1997;36(8):1036-1045. doi:10.1097/00004583-199708000-00011.

246. Carlson C, Tamm L, Gaub M. Gender Differences in Children With ADHD, ODD, and Co-Occurring ADHD/ODD Identified in a School Population. *J Am Acad Child Adolesc Psychiatry*. 1997;36(12):1706-1714. doi:10.1097/00004583-199712000-00019.

247. Hankin BL, Abramson LY. Development of gender differences in depression: An elaborated cognitive vulnerability–transactional stress theory. *Psychol Bull*. 2001;127(6):773-796. doi:10.1037/0033-2909.127.6.773.

248. Wunderlich U, Bronisch T, Wittchen H-U, Carter R. Gender differences in adolescents and young adults with suicidal behaviour. *Acta Psychiatr Scand*. 2008;104(5):332-339. doi:10.1111/j.1600-0447.2001.00432.x.

249. Qin P, Agerbo E, Westergård-Nielsen N, Eriksson T, Mortensen PB. Gender differences in risk factors for suicide in Denmark. *Br J psychiatry*. 2000;177:546-550. http://www.ncbi.nlm.nih.gov/pubmed/11104395. Accessed November 7, 2017.

250. Daigle LE, Cullen FT, Wright JP. Gender Differences in the Predictors of Juvenile Delinquency. *Youth Violence Juv Justice*. 2007;5(3):254-286. doi:10.1177/1541204007301289.

251. Fagan AA, Van Horn ML, Hawkins JD, Arthur MW. Gender Similarities and Differences in the Association Between Risk and Protective Factors and Self-Reported Serious Delinquency. *Prev Sci*. 2007;8(2):115-124. doi:10.1007/s11121-006-0062-1.

252. von Borczyskowski A, Lindblad F, Vinnerljung B, Hjern A. Gender Differences in Risk Factors for Suicide: Findings from a Swedish National Cohort Study. *Can J Psychiatry*. 2010;55(2):108-111. doi:10.1177/070674371005500207.

253. Thompson MP, Light LS. Examining Gender Differences in Risk Factors for Suicide Attempts Made 1 and 7 Years Later in a Nationally Representative Sample. *J Adolesc Heal*. 2011;48(4):391-397. doi:10.1016/j.jadohealth.2010.07.018.

254. Murray J, Irving B, Farrington DP, Colman I, Bloxsom CAJ. Very early predictors of conduct problems and crime: results from a national cohort study. *J Child Psychol Psychiatry*. 2010;51(11):1198-1207. doi:10.1111/j.1469-7610.2010.02287.x.

255. Lorant V, Deliège D, Eaton W, Robert A, Philippot P, Ansseau M. Socioeconomic Inequalities in Depression: A Meta-Analysis. *Am J Epidemiol*. 2003;157(2):98-112. doi:10.1093/aje/kwf182.

256. Morgan P, Farkas G, Hillemeier M, Maczuga S. Risk factors for learning-related behavior problems at 24 months of age: Population-based estimates. *J Abnorm Child Psychol*. 2009;37:401-413.

257. Shonkoff J, Siegel B, Dobbins M, Wood D. Early childhood adversity, toxic stress, and the role of the pediatrician: Translating developmental science into lifelong health. *Pediatrics*. 2012;129(1):224-231.

258. Rehkopf D, Buka S. The association between suicide and the socio-economic characteristics of geographical areas: a systematic review. *Psychol Med*. 2005;36(2):145. doi:10.1017/S003329170500588X.

259. Corak M. Do Poor Children Become Poor Adults? Lessons from a Cross-Country Comparison of Generational Earnings Mobility. In: *Dynamics of Inequality and Poverty (Research on Economic Inequality, Volume 13)*. Emerald Group Publishing Limited; 2006:143-188. doi:10.1016/S1049-2585(06)13006-9.

260. Adams DM, Overholser JC, Spirito A. Stressful Life Events Associated with Adolescent Suicide Attempts. *Can J Psychiatry*. 1994;39(1):43-48. doi:10.1177/070674379403900109.

261. Campbell SB, Pierce EW, Moore G, Marakovitz S, Newby K. Boys' externalizing problems at elementary school age: Pathways from early behavior problems, maternal control, and family stress. *Dev Psychopathol*. 1996;8(4):701. doi:10.1017/S0954579400007379.

262. Sandin B, Chorot P, Santed M, Valiente R, Joiner T. Negative life events and adolescent suicidal behavior: a critical analysis from the stress process perspective. *J Adolesc*. 1998;21(4):415-426. doi:10.1006/JADO.1998.0172.

263. Evans GW, Kim P. Childhood Poverty, Chronic Stress, Self-Regulation, and Coping. *Child Dev Perspect*. 2012;7(1):43-48. doi:10.1111/cdep.12013.

264. Hankin BL, Young JF, Abela JRZ, et al. Depression from childhood into late adolescence: Influence of gender, development, genetic susceptibility, and peer stress. *J Abnorm Psychol*. 2015;124(4):803-816. doi:10.1037/abn0000089.

265. Hanson JL, Nacewicz BM, Sutterer MJ, et al. Behavioral Problems After Early Life Stress: Contributions of the Hippocampus and Amygdala. *Biol Psychiatry*. 2015;77(4):314-323. doi:10.1016/j.biopsych.2014.04.020.

266. Byrne DG, Mazanov J. Sources of adolescent stress, smoking and the use of other drugs. *Stress Med*. 1999;15(4):215-227. doi:10.1002/(SICI)1099-

1700(199910)15:4<215::AID-SMI817>3.0.CO;2-1.

267. Baldwin JA, Brown BG, Wayment HA, Nez RA, Brelsford KM. Culture and Context: Buffering the Relationship Between Stressful Life Events and Risky Behaviors in American Indian Youth. *Subst Use Misuse*. 2011;46(11):1380-1394. doi:10.3109/10826084.2011.592432.

268. Naicker K, Wickham M, Colman I. Timing of First Exposure to Maternal Depression and Adolescent Emotional Disorder in a National Canadian Cohort. Laks J, ed. *PLoS One*. 2012;7(3):e33422. doi:10.1371/journal.pone.0033422.

269. Dykxhoorn J, Hatcher S, Roy-Gagnon M-H, Colman I. Early life predictors of adolescent suicidal thoughts and adverse outcomes in two population-based cohort studies. *PLoS One*. 2017;12(8):e0183182. doi:10.1371/journal.pone.0183182.

270. Goodman SH, Rouse MH, Connell AM, Broth MR, Hall CM, Heyward D. Maternal Depression and Child Psychopathology: A Meta-Analytic Review. *Clin Child Fam Psychol Rev*. 2011;14(1):1-27. doi:10.1007/s10567-010-0080-1.

271. Radloff LS. The CES-D Scale: A Self-Report Depression Scale for Research in the General Population. *Appl Psychol Meas*. 1977;1(3). doi:10.1177/014662167700100306.

272. Roberts R. Reliability of the CES-D scale in different ethnic contexts. *Psychiatry Res*. 1980;2(2):125-134. doi:10.1016/0165-1781(80)90069-4.

273. Myers JK, Weissman MM. Use of a self-report symptom scale to detect depression in a community sample. *Am J Psychiatry*. 1980;137(9):1081-1084. doi:10.1176/ajp.137.9.1081.

274. Kohout FJ, Berkman LF, Evans DA, Cornoni-Huntley J. Two Shorter Forms of the CES-D Depression Symptoms Index. *J Aging Health*. 1993;5(2):179-193. doi:10.1177/089826439300500202.

275. Luthar SS, Sexton CC. Maternal drug abuse versus maternal depression: Vulnerability and resilience among school-age and adolescent offspring. *Dev Psychopathol*. 2007;19(1):205-225. doi:10.1017/S0954579407070113.

276. Rossow I, Lauritzen G. Shattered childhood: a key issue in suicidal behavior among drug addicts? *Addiction*. 2001;96(2):227-240. doi:10.1080/09652140020020955.

277. Lieb R, Merikangas KR, Höfler M, Pfister H, Isensee B, Wittchen HU. Parental alcohol use disorders and alcohol use and disorders in offspring: a community study. *Psychol Med*. 2002;32(1):63-78. http://www.ncbi.nlm.nih.gov/pubmed/11883731. Accessed November 7, 2017.

278. Glowinski AL, Jacob T, Bucholz KK, Scherrer JF, True W, Heath AC.

Paternal alcohol dependence and offspring suicidal behaviors in a children-of-twins study. *Drug Alcohol Depend*. 2004;76:S69-S77. doi:10.1016/j.drugalcdep.2004.08.008.

279. Drinking Levels Defined | National Institute on Alcohol Abuse and Alcoholism (NIAAA). National Institute on Alcohol Abuse and Alcoholism (NIAAA). https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking. Accessed November 7, 2017.

280. Harland P, Reijneveld SA, Brugman E, Verloove-Vanhorick SP, Verhulst FC. Family factors and life events as risk factors for behavioural and emotional problems in children. *Eur Child Adolesc Psychiatry*. 2002;11(4):176-184. doi:10.1007/s00787-002-0277-z.

281. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychol Bull*. 2002;128(2):330-366. doi:10.1037/0033-2909.128.2.330.

282. Amato PR, Keith B. Parental divorce and the well-being of children: a meta-analysis. *Psychol Bull*. 1991;110(1):26-46. http://www.ncbi.nlm.nih.gov/pubmed/1832495. Accessed January 12, 2018.

283. Garnefski N, Diekstra RFW. Adolescents from one parent, stepparent and intact families: emotional problems and suicide attempts. *J Adolesc*. 1997;20(2):201-208. doi:10.1006/jado.1996.0077.

284. Amey CH, Albrecht SL. Race and Ethnic Differences in Adolescent Drug Use: The Impact of Family Structure and the Quantity and Quality of Parental Interaction. *J Drug Issues*. 1998;28(2):283-298. doi:10.1177/002204269802800202.

285. Sauvola A, Räsänen PK, Joukamaa MI, Jokelainen J, Järvelin MR, Isohanni MK. Mortality of young adults in relation to single-parent family background. A prospective study of the northern Finland 1966 birth cohort. *Eur J Public Health*. 2001;11(3):284-286. http://www.ncbi.nlm.nih.gov/pubmed/11582608. Accessed January 12, 2018.

286. Barrett AE, Turner RJ. Family structure and substance use problems in adolescence and early adulthood: examining explanations for the relationship. *Addiction*. 2006;101(1):109-120. doi:10.1111/j.1360-0443.2005.01296.x.

287. Hemovich V, Crano WD. Family structure and adolescent drug use: an exploration of single-parent families. *Subst Use Misuse*. 2009;44(14):2099-2113. doi:10.3109/10826080902858375.

288. Oberski D. Mixture models: Latent profile and latent class analysis. In: *Modern Statistical Methods for HCI: A Modern Look at Data Analysis for*

HCI Research. Springer International Publishing.

289. Muthén L, Muthén B. *Mplus User's Guide*. 6th ed. Los Angeles, CA: Muthén & Muthén

290. Celeux G, Soromenho G. An entropy criterion for assessing the number of clusters in a mixture model. *J Classif*. 1996;13:195-212.

291. Wickrama K, Lee TK, O'Neal CW, Lorenz FO. *Higher-Order Growth Curves and Mixture Modeling with Mplus: A Practical Guide*. New York, NY: Routledge/Taylor & Francis Group; 2016.

292. Muthén B, Muthén LK. Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcohol Clin Exp Res*. 2000;24:882-891.

293. Collins L, Lanza S. *Latent Class and Latent Transition Analysis with Applications in the Social, Behavioral, and Health Sciences*. Hoboken, New Jersey: John Wiley & Sons, Inc; 2010.

294. Bialosiewicz S, Murphy K, Berry T. *An Introduction to Measurement Invariance Testing: Resource Packet for Participants*. http://comm.eval.org/HigherLogic/System/DownloadDocumentFile.ashx?DocumentFileKey=63758fed-a490-43f2-8862-2de0217a08b8. Accessed October 22, 2017.

295. Muthén B, Asparouhov T. *BSEM Measurement Invariance Analysis*.; 2013. www.statmodel.com.

296. SAS Institute Inc. SAS 9.4. 2013.

297. Clark S, Muthén B. *Relating Latent Class Analysis Results to Variables Not Included in the Analysis*.; 2009.

298. Althoff RR, Rettew DC, Faraone S V., Boomsma DI, Hudziak JJ. Latent Class Analysis Shows Strong Heritability of the Child Behavior Checklist–Juvenile Bipolar Phenotype. *Biol Psychiatry*. 2006;60(9):903-911. doi:10.1016/j.biopsych.2006.02.025.

299. Basten MMGJ, Althoff RR, Tiemeier H, et al. The dysregulation profile in young children: empirically defined classes in the Generation R study. *J Am Acad Child Adolesc Psychiatry*. 2013;52(8):841-850.e2. doi:10.1016/j.jaac.2013.05.007.

300. Bianchi V, Brambilla P, Garzitto M, et al. Latent classes of emotional and behavioural problems in epidemiological and referred samples and their relations to DSM-IV diagnoses. *Eur Child Adolesc Psychiatry*. 2017;26(5):549-557. doi:10.1007/s00787-016-0918-2.

301. Biederman J, Wozniak J, Kiely K, et al. CBCL clinical scales discriminate prepubertal children with structured interview-derived diagnosis of mania from those with ADHD. *J Am Acad Child Adolesc Psychiatry*.

1995;34(4):464-471. http://www.ncbi.nlm.nih.gov/pubmed/7751260. Accessed January 3, 2018.

302. De Caluwé E, Decuyper M, De Clercq B. The child behavior checklist dysregulation profile predicts adolescent DSM-5 pathological personality traits 4 years later. *Eur Child Adolesc Psychiatry*. 2013;22(7):401-411. doi:10.1007/s00787-013-0379-9.

303. Masi G, Pisano S, Milone A, Muratori P. Child behavior checklist dysregulation profile in children with disruptive behavior disorders: A longitudinal study. *J Affect Disord*. 2015;186:249-253. doi:10.1016/j.jad.2015.05.069.

304. Blair C. School Readiness Integrating Cognition and Emotion in a Neurobiological Conceptualization of Children's Functioning at School Entry. *Am Psychol*. 2002;57(2):111-127. doi:10.1037//0003-066X.57.2.111.

305. DiStefano C, Kamphaus RW. Investigating Subtypes of Child Development. *Educ Psychol Meas*. 2006;66(5):778-794. doi:10.1177/0013164405284033.

306. Bonadio FT, Dynes M, Lackey J, Tompsett C, Amrhein K. Grouping Youth With Similar Symptoms: A Person-Centered Approach to Transdiagnostic Subgroups. *J Clin Psychol*. 2016;72(7):676-688. doi:10.1002/jclp.22274.

307. Mindrila D. A Typology of Child School Behavior: Investigation using latent profile analysis and cluster analysis. *Psychol Sch*. 2016;53(5). https://journals-scholarsportal-info.proxy.bib.uottawa.ca/pdf/00333085/v53i0005/471_atocsblpaaca.xml. Accessed December 13, 2017.

308. Ostrander R, Herman K, Sikorski J, Mascendaro P, Lambert S. Patterns of Psychopathology in Children with ADHD: A Latent Profile Analysis. *J Clin Child Adolesc Psychol*. 2008;37(4):833-847. doi:10.1080/15374410802359668.

309. Moilanen KL, Shaw DS, Maxwell KL. Developmental cascades: Externalizing, internalizing, and academic competence from middle childhood to early adolescence. *Dev Psychopathol*. 2010;22(3):635-653. doi:10.1017/S0954579410000337.

310. Meyer SE, Carlson GA, Youngstrom E, et al. Long-term outcomes of youth who manifested the CBCL-Pediatric Bipolar Disorder phenotype during childhood and/or adolescence. *J Affect Disord*. 2009;113(3):227-235. doi:10.1016/j.jad.2008.05.024.

311. Holtmann M, Buchmann AF, Esser G, Schmidt MH, Banaschewski T, Laucht M. The Child Behavior Checklist-Dysregulation Profile predicts substance use, suicidality, and functional impairment: a longitudinal

analysis. *J Child Psychol Psychiatry*. 2011;52(2):139-147. doi:10.1111/j.1469-7610.2010.02309.x.

312. Fanti KA, Henrich CC. Trajectories of pure and co-occurring internalizing and externalizing problems from age 2 to age 12: Findings from the National Institute of Child Health and Human Development Study of Early Child Care. *Dev Psychol*. 2010;46(5):1159-1175. doi:10.1037/a0020659.

313. Wittchen HU, Kessler RC, Pfister H, Lieb M. Why do people with anxiety disorders become depressed? A prospective-longitudinal community study. *Acta Psychiatr Scand Suppl*. 2000;(406):14-23. http://www.ncbi.nlm.nih.gov/pubmed/11131466. Accessed July 18, 2016.

314. Mesman J, Bongers IL, Koot HM. Preschool developmental pathways to preadolescent internalizing and externalizing problems. *J Child Psychol Psychiatry*. 2001;42(5):679-689. doi:10.1111/1469-7610.00763.

315. Weeks M, Ploubidis GB, Cairney J, Wild TC, Naicker K, Colman I. Developmental pathways linking childhood and adolescent internalizing, externalizing, academic competence, and adolescent depression. *J Adolesc*. 2016;51:30-40. doi:10.1016/j.adolescence.2016.05.009.

316. Kovacs M, Devlin B. Internalizing disorders in childhood. *J Child Psychol Psychiatry*. 1998;39(1):47-63. http://www.ncbi.nlm.nih.gov/pubmed/9534086. Accessed January 20, 2018.

317. Moffitt TE, Caspi A, Dickson N, Silva P, Stanton W. Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Dev Psychopathol*. 1996;8(2):399-424. doi:10.1017/S0954579400007161.

318. Kim-Cohen J, Arseneault L, Caspi A, Tomás MP, Taylor A, Moffitt TE. Validity of DSM-IV Conduct Disorder in 4½–5-Year-Old Children: A Longitudinal Epidemiological Study. *Am J Psychiatry*. 2005;162(6):1108-1117. doi:10.1176/appi.ajp.162.6.1108.

319. Luby JL, Gaffrey MS, Tillman R, April LM, Belden AC. Trajectories of Preschool Disorders to Full DSM Depression at School Age and Early Adolescence: Continuity of Preschool Depression. *Am J Psychiatry*. 2014;171(7):768-776. doi:10.1176/appi.ajp.2014.13091198.

320. Lahey BB, Lee SS, Sibley MH, Applegate B, Molina BSG, Pelham WE. Predictors of adolescent outcomes among 4–6-year-old children with attention-deficit/hyperactivity disorder. *J Abnorm Psychol*. 2016;125(2):168-181. doi:10.1037/abn0000086.

321. Wichstrøm L, Belsky J, Steinsbekk S. Homotypic and heterotypic continuity of symptoms of psychiatric disorders from age 4 to 10 years: a dynamic panel model. *J Child Psychol Psychiatry*. 2017;58(11):1239-1247. doi:10.1111/jcpp.12754.

322. Bornstein MH, Hahn C-S, Haynes OM. Social competence, externalizing, and internalizing behavioral adjustment from early childhood through early adolescence: Developmental cascades. *Dev Psychopathol*. 2010;22(4):717-735. doi:10.1017/S0954579410000416.

323. Copeland WE, Adair CE, Smetanin P, et al. Diagnostic transitions from childhood to adolescence to early adulthood. *J Child Psychol Psychiatry*. 2013;54(7):791-799. doi:10.1111/jcpp.12062.

324. Mannuzza S, Klein RG, Abikoff H, Moulton III JL. Significance of Childhood Conduct Problems to Later Development of Conduct Disorder Among Children With ADHD: A Prospective Follow-Up Study. *J Abnorm Child Psychol*. 2004;32(5):565-573. doi:10.1023/B:JACP.0000037784.80885.1a.

325. Berona J, Horwitz AG, Czyz EK, King CA. Psychopathology profiles of acutely suicidal adolescents: Associations with post-discharge suicide attempts and rehospitalization. *J Affect Disord*. 2017;209:97-104. doi:10.1016/J.JAD.2016.10.036.

326. Lewinsohn PM, Rohde P, Seeley JR. Adolescent Suicidal Ideation and Attempts: Prevalence, Risk Factors, and Clinical Implications. *Clin Psychol Sci Pract*. 1996;3(1):25-46. doi:10.1111/j.1468-2850.1996.tb00056.x.

327. Shaffer D, Gould MS, Fisher P, et al. Psychiatric diagnosis in child and adolescent suicide. *Arch Gen Psychiatry*. 1996;53(4):339-348. http://www.ncbi.nlm.nih.gov/pubmed/8634012. Accessed January 20, 2018.

328. Brent D, Baugher M, Bridge J, Chen T, Chiappetta L. Age- and Sex-Related Risk Factors for Adolescent Suicide. *J Am Acad Child Adolesc Psychiatry*. 1999;38(12):1497-1505. doi:10.1097/00004583-199912000-00010.

329. Zeitlin H. Psychiatric comorbidity with substance misuse in children and teenagers. *Drug Alcohol Depend*. 1999;55(3):225-234. doi:10.1016/S0376-8716(99)00018-6.

330. Brenner SL, Beauchaine TP. Pre-ejection period reactivity and psychiatric comorbidity prospectively predict substance use initiation among middle-schoolers: A pilot study. *Psychophysiology*. 2011;48:1587-1595. doi:10.1111/j.1469-8986.2011.01230.x.

331. Lynskey MT, Fergusson DM. Childhood conduct problems, attention deficit behaviors, and adolescent alcohol, tobacco, and illicit drug use. *J Abnorm Child Psychol*. 1995;23(3):281-302. doi:10.1007/BF01447558.

332. Clark DB, Vanyukov M, Cornelius J. Childhood Antisocial Behavior and Adolescent Alcohol Use Disorders. *Alcohol Res Heal*. 2002;26:109-113. http://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.493.1399&rep=r

ep1&type=pdf. Accessed January 13, 2018.

333. Fergusson DM, John Horwood L, Ridder EM. Show me the child at seven: the consequences of conduct problems in childhood for psychosocial functioning in adulthood. *J Child Psychol Psychiatry*. 2005;46(8):837-849. doi:10.1111/j.1469-7610.2004.00387.x.

334. Pardini D, White HR, Stouthamer-Loeber M. Early adolescent psychopathology as a predictor of alcohol use disorders by young adulthood. *Drug Alcohol Depend*. 2007;88 Suppl 1(Suppl 1):S38-49. doi:10.1016/j.drugalcdep.2006.12.014.

335. Warden D, Riggs PD, Min S-J, et al. Major depression and treatment response in adolescents with ADHD and substance use disorder. *Drug Alcohol Depend*. 2012;120(1-3):214-219. doi:10.1016/j.drugalcdep.2011.08.001.

336. Zoloto A, Nagoshi CT, Presson C, Chassin L. Attention deficit/hyperactivity disorder symptoms and depression symptoms as mediators in the intergenerational transmission of smoking. *Drug Alcohol Depend*. 2012;126(1-2):147-155. doi:10.1016/j.drugalcdep.2012.05.003.

337. Wilens TE, Martelon M, Fried R, Petty C, Bateman C, Biederman J. Do Executive Function Deficits Predict Later Substance Use Disorders Among Adolescents and Young Adults? *J Am Acad Child Adolesc Psychiatry*. 2011;50(2):141-149. doi:10.1016/j.jaac.2010.11.010.

338. Babinski L, Hartsough C, Lambert N. Childhood Conduct Problems, Hyperactivity-impulsivity, and Inattention as Predictors of Adult Criminal Activity. *J Child Psychol Psychiatry Allied Discip*. 1999;40(3):347-355. https://www.cambridge.org/core/journals/journal-of-child-psychology-and-psychiatry-and-allied-disciplines/article/childhood-conduct-problems-hyperactivity-impulsivity-and-inattention-as-predictors-of-adult-criminal-activity/6AD42DCDBACE2FCFD594ACF18D4CCE1. Accessed January 13, 2018.

339. Frick PJ, Stickle TR, Dandreaux DM, Farrell JM, Kimonis ER. Callous-unemotional traits in predicting the severity and stability of conduct problems and delinquency. *J Abnorm Child Psychol*. 2005;33(4):471-487. http://www.ncbi.nlm.nih.gov/pubmed/16118993. Accessed January 13, 2018.

340. Muris P, Merckelbach H, Schmidt H, Gadet B, Bogie N. Anxiety and depression as correlates of self-reported behavioural inhibition in normal adolescents. *Behav Res Ther*. 2001;39(9):1051-1061. doi:10.1016/S0005-7967(00)00081-4.

341. The Incredible Years Inc. The Incredible Years | Training series for Parents, Teachers, and children. The Incredible Years, Inc. http://www.incredibleyears.com/. Published 2013. Accessed January 21,

2018.

342. Blueprints for Healthy Youth Development. GenerationPMTO parenting skills training | Detailed Evaluation Abstract | Blueprints Programs. Blueprints for Healthy Youth Development. http://www.blueprintsprograms.com/evaluation-abstract/generationpmto. Published 2018. Accessed January 21, 2018.

343. Hagen KA, Ogden T, Bjørnebekk G. Treatment Outcomes and Mediators of Parent Management Training: A One-Year Follow-Up of Children with Conduct Problems. *J Clin Child Adolesc Psychol*. 2011;40(2):165-178. doi:10.1080/15374416.2011.546050.

344. Ollendick TH, Greene RW, Austin KE, et al. Parent Management Training and Collaborative & Proactive Solutions: A Randomized Control Trial for Oppositional Youth. *J Clin Child Adolesc Psychol*. 2016;45(5):591-604. doi:10.1080/15374416.2015.1004681.

345. Rabbit S, Carrubba E, Lecza B, MacWhinney E, Pope J, Kazdin A. Reducing therapist contact in parenting programs: Evaluation of internet-based treatments for child conduct problems. *J Child Fam Stud*. 2016;25:2001-2020.

346. Forgatch MS, DeGarmo DS. Parenting through change: an effective prevention program for single mothers. *J Consult Clin Psychol*. 1999;67(5):711-724. http://www.ncbi.nlm.nih.gov/pubmed/10535238. Accessed January 21, 2018.

347. Martinez CR, Eddy JM. Effects of culturally adapted parent management training on Latino youth behavioral health outcomes. *J Consult Clin Psychol*. 2005;73(5):841-851. doi:10.1037/0022-006X.73.5.841.

348. Webster-Stratton C, Reid MJ, Hammond M. Preventing Conduct Problems, Promoting Social Competence: A Parent and Teacher Training Partnership in Head Start. *J Clin Child Adolesc Psychol*. 2001;30(3):283-302. doi:10.1207/S15374424JCCP3003_2.

349. Hansford L, Sharkey S, Edwards V, et al. Understanding influences on teachers' uptake and use of behaviour management strategies within the STARS trial: process evaluation protocol for a randomised controlled trial. *BMC Public Health*. 2015;15:119. doi:10.1186/s12889-015-1486-y.

350. Kirkhaug B, Drugli MB, Handegård BH, Lydersen S, Åsheim M, Fossum S. Does the Incredible Years Teacher Classroom Management Training programme have positive effects for young children exhibiting severe externalizing problems in school?: a quasi-experimental pre-post study. *BMC Psychiatry*. 2016;16(1):362. doi:10.1186/s12888-016-1077-1.

351. Menting ATA, Orobio de Castro B, Matthys W. Effectiveness of the Incredible Years parent training to modify disruptive and prosocial child behavior: A meta-analytic review. *Clin Psychol Rev*. 2013;33(8):901-913.

doi:10.1016/j.cpr.2013.07.006.

352. Colman I, Murray J, Abbott RA, et al. Outcomes of conduct problems in adolescence: 40 year follow-up of national cohort. *BMJ*. 2009;338:a2981. doi:10.1136/BMJ.A2981.

353. Maughan B, Stafford M, Shah I, Kuh D. Adolescent conduct problems and premature mortality: follow-up to age 65 years in a national birth cohort. *Psychol Med*. 2014;44(5):1077-1086. doi:10.1017/S0033291713001402.

354. Costello EJ, Maughan B. Annual Research Review: Optimal outcomes of child and adolescent mental illness. *J Child Psychol Psychiatry*. 2015;56(3):324-341. doi:10.1111/jcpp.12371.

355. Edwards A. The Social Desirability Variable in Personality Assessment and Research. *Acad Med*. 1957;33(8):610-611. http://journals.lww.com/academicmedicine/citation/1958/08000/the_social_desirability_variable_in_personality.24.aspx. Accessed January 8, 2018.

356. Krumpal I. Determinants of social desirability bias in sensitive surveys: a literature review. *Qual Quant*. 2013;47(4):2025-2047. doi:10.1007/s11135-011-9640-9.

357. Winters KC, Stinchfield RD, Henly GA, Schwartz RH. Validity of Adolescent Self-Report of Alcohol and Other Drug Involvement. *Int J Addict*. 1990;25(sup11):1379-1395. doi:10.3109/10826089009068469.

358. Williams RJ, Nowatzki N. Validity of adolescent self-report of substance use. *Subst Use Misuse*. 2005;40(3):299-311. http://www.ncbi.nlm.nih.gov/pubmed/15776978. Accessed January 8, 2018.

359. Jolliffe D, Farrington DP, Hawkins JD, Catalano RF, Hill KG, Kosterman R. Predictive, concurrent, prospective and retrospective validity of self-reported delinquency. *Crim Behav Ment Heal*. 2003;13(3):179-197. doi:10.1002/cbm.541.

360. Osman A, Barrios FX, Panak WF, Osman JR, Hoffman J, Hammer R. Validation of the multi-attitude suicide tendency scale in adolescent samples. *J Clin Psychol*. 1994;50(6):847-855. doi:10.1002/1097-4679(199411)50:6<847::AID-JCLP2270500606>3.0.CO;2-D.

361. Miotto P, Preti A. Suicide ideation and social desirability among school-aged young people. *J Adolesc*. 2008;31(4):519-533. doi:10.1016/j.adolescence.2007.08.004.

362. Klimes-Dougan B. Screening for suicidal ideation in children and adolescents: methodological considerations. *J Adolesc*. 1998;21(4):435-444. doi:10.1006/jado.1998.0166.

363. Velting DM, Rathus JH, Asnis GM. Asking adolescents to explain discrepancies in self-reported suicidality. *Suicide Life Threat Behav*.

1998;28(2):187-196. http://www.ncbi.nlm.nih.gov/pubmed/9674078. Accessed January 8, 2018.

364. Duhig AM, Renk K, Epstein MK, Phares V. Interparental Agreement on Internalizing, Externalizing, and Total Behavior Problems: A Meta-analysis. *Clin Psychol Sci Pract*. 2006;7(4):435-453. doi:10.1093/clipsy.7.4.435.

365. Grietens H, Onghena P, Prinzie P, et al. Comparison of Mothers', Fathers', and Teachers' Reports on Problem Behavior in 5- to 6-Year-Old Children. *J Psychopathol Behav Assess*. 2004;26(2):137-146. doi:10.1023/B:JOBA.0000013661.14995.59.

366. Smith SR. Making Sense of Multiple Informants in Child and Adolescent Psychopathology A Guide for Clinicians. *J Psychoeduc Assess*. 2007;25(2):139-149. doi:10.1177/0734282906296233.

367. Cantwell D, Lewinsohn P, Rohde P, Seeley J. Correspondence Between Adolescent Report and Parent Report of Psychiatric Diagnostic Data. *J Am Acad Child Adolesc Psychiatry*. 1997;36(5):610-619. doi:10.1097/00004583-199705000-00011.

368. Lewin A, Brondeel R, Benmarhnia T, Thomas F, Chaix B. Attrition Bias Related to Missing Outcome Data. *Epidemiology*. 2018;29(1):87-95. doi:10.1097/EDE.0000000000000755.

369. McKeown R, Garrison C, Cuffe S, Waller J, Jackson K, Addy C. Incidence and Predictors of Suicidal Behaviors in a Longitudinal Sample of Young Adolescents. *J Am Acad Child Adolesc Psychiatry*. 1998;37(6):612-619. doi:10.1097/00004583-199806000-00011.

370. Zimmerman FJ, Katon W. Socioeconomic status, depression disparities, and financial strain: what lies behind the income-depression relationship? *Health Econ*. 2005;14(12):1197-1215. doi:10.1002/hec.1011.

371. McMartin SE, Kingsbury M, Dykxhoorn J, Colman I. Time trends in symptoms of mental illness in children and adolescents in Canada. *Can Med Assoc J*. 2014;186(18):E672-8. doi:10.1503/cmaj.140064.

372. Youngstrom EA, Findling RL, Calabrese JR. Who are the comorbid adolescents? Agreement between psychiatric diagnosis, youth, parent, and teacher report. *J Abnorm Child Psychol*. 2003;31(3):231-245. http://www.ncbi.nlm.nih.gov/pubmed/12774858. Accessed January 16, 2018.

373. Geeraerts SB, Deutz MHF, Deković M, et al. The Child Behavior Checklist Dysregulation Profile in Preschool Children: A Broad Dysregulation Syndrome. *J Am Acad Child Adolesc Psychiatry*. 2015;54(7):595-602.e2. doi:10.1016/j.jaac.2015.04.012.

374. Roberts RE, Roberts CR, Chen YR. Suicidal thinking among adolescents

with a history of attempted suicide. *J Am Acad Child Adolesc Psychiatry*. 1998;37(12):1294-1300. http://www.ncbi.nlm.nih.gov/pubmed/9847502. Accessed January 20, 2018.

375. Johnson JG, Cohen P, Gould MS, Kasen S, Brown J, Brook JS. Childhood adversities, interpersonal difficulties, and risk for suicide attempts during late adolescence and early adulthood. *Arch Gen Psychiatry*. 2002;59(8):741-749. http://www.ncbi.nlm.nih.gov/pubmed/12150651. Accessed January 20, 2018.

376. Ueno K. The effects of friendship networks on adolescent depressive symptoms. *Soc Sci Res*. 2005;34(3):484-510. doi:10.1016/J.SSRESEARCH.2004.03.002.

377. Hughes JN, Dyer N, Luo W, Kwok O-M. Effects of peer academic reputation on achievement in academically at-risk elementary students. *J Appl Dev Psychol*. 2009;30(2):182-194. doi:10.1016/j.appdev.2008.12.008.

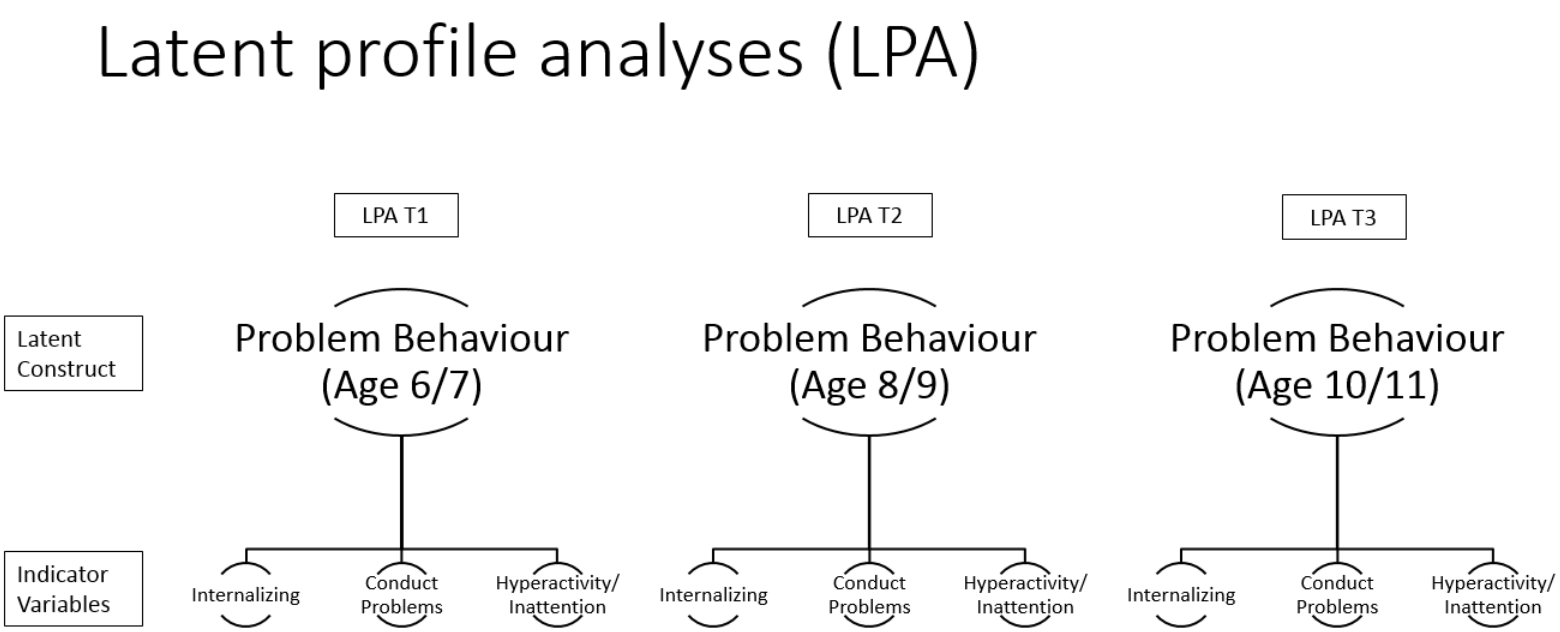
378. Padilla-Walker LM, Bean RA. Negative and positive peer influence: Relations to positive and negative behaviors for African American, European American, and Hispanic adolescents. *J Adolesc*. 2009;32(2):323-337. doi:10.1016/j.adolescence.2008.02.003.

379. Glaser B, Shelton KH, van den Bree MBM. The Moderating Role of Close Friends in the Relationship Between Conduct Problems and Adolescent Substance Use. *J Adolesc Heal*. 2010;47(1):35-42. doi:10.1016/j.jadohealth.2009.12.022.

380. Tomé G, Matos M, Simões C, Diniz JA, Camacho I. How can peer group influence the behavior of adolescents: explanatory model. *Glob J Health Sci*. 2012;4(2):26-35. doi:10.5539/gjhs.v4n2p26.

381. McLeod BD, Weisz JR, Wood JJ. Examining the association between parenting and childhood depression: A meta-analysis. *Clin Psychol Rev*. 2007;27(8):986-1003. doi:10.1016/J.CPR.2007.03.001.

Appendix A


Latent profile analyses (LPA)
LPA T1
LPA T2
LPA T3
Latent Construct
Indicator Variables
Problem Behaviour (Age 6/7)
Problem Behaviour (Age 8/9)
Problem Behaviour (Age 10/11)
Internalizing
Conduct Problems
Hyperactivity/ Inattention
Internalizing
Conduct Problems
Hyperactivity/ Inattention
Internalizing
Conduct Problems
Hyperactivity/ Inattention

Appendix B

Table 12. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on suicidal thoughts

	Suicidal Thoughts								Suicidal Thoughts (sensitivity)							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
Transition clusters (sensitivity)																
Decreasing to low	5019	1.02	0.79	1.30	4884	1.09	0.84	1.40	6921	1.00	0.79	1.27	6685	1.08	0.84	1.38
Low to problems		1.65	1.28	2.14		1.72	1.32	2.25		1.68	1.30	2.16		1.83	1.41	2.37
Homotypic problems		2.16	1.70	2.75		2.10	1.62	2.72		2.13	1.69	2.69		2.12	1.65	2.72
Heterotypic problems		1.47	0.99	2.18		1.52	1.01	2.29		1.48	1.00	2.17		1.52	1.02	2.26

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 13 - Appendix. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on alcohol use

	Alcohol													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
Transition clusters (sensitivity)														
Decreasing to low	5834	0.98	0.83	1.16	5656	0.97	0.81	1.15	1.18	0.84	1.66	1.18	0.84	1.67
Low to problems		1.26	1.02	1.56		1.27	1.02	1.58	1.04	0.66	1.65	1.03	0.65	1.65
Homotypic problems		1.44	1.15	1.80		1.50	1.19	1.90	1.89	1.27	2.82	1.91	1.27	2.89
Heterotypic problems		1.06	0.78	1.44		1.15	0.83	1.59	2.01	1.20	3.36	2.12	1.24	3.63

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 14 - Appendix. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on cigarette use

	Cigarette													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
Transition clusters (sensitivity)														
Decreasing to low		1.13	0.95	1.34		1.16	0.97	1.38	1.53	1.15	2.03	1.60	1.19	2.14
Low to problems	5862	1.24	1.01	1.52	5687	1.28	1.04	1.58	1.20	0.82	1.73	1.07	0.72	1.59
Homotypic problems		1.24	1.01	1.54		1.27	1.02	1.58	2.82	2.12	3.75	2.62	1.92	3.56
Heterotypic problems		1.67	1.25	2.21		1.70	1.27	2.29	1.66	1.00	2.74	1.34	0.78	2.29

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 15 - Appendix. Unadjusted and adjusted multinomial (3-category) logistic regression models showing the association between latent profile(s) and transition clusters on cannabis use

	Cannabis													
	Occasional User								Regular User					
	Unadjusted				Adjusted*				Unadjusted			Adjusted*		
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI	OR	Upper CI	Lower CI
Transition clusters (sensitivity)														
Decreasing to low		1.07	0.88	1.29		1.07	0.89	1.30	1.24	0.93	1.65	1.17	0.87	1.56
Low to problems	5886	1.25	1.00	1.55	5708	1.24	0.99	1.55	0.78	0.52	1.18	0.71	0.47	1.08
Homotypic problems		1.66	1.34	2.04		1.64	1.32	2.04	2.11	1.56	2.85	1.87	1.37	2.56
Heterotypic problems		1.43	1.05	1.94		1.45	1.06	1.99	0.91	0.50	1.63	0.79	0.44	1.44

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 16 - Appendix. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on illicit substances and selling drugs

	Illicit Substance Use (Excluding Cannabis)								Selling Drugs							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
Transition clusters (sensitivity)	5826				5651				5861				5682			
Decreasing to low		1.36	1.02	1.82		1.46	1.08	1.98		1.36	1.02	1.81		1.27	0.95	1.71
Low to problems		1.08	0.74	1.58		1.12	0.76	1.67		0.75	0.48	1.15		0.69	0.45	1.08
Homotypic problems		1.26	0.88	1.81		1.36	0.94	1.98		2.05	1.51	2.78		1.84	1.34	2.53
Heterotypic problems		1.04	0.59	1.85		1.07	0.59	1.93		0.88	0.48	1.63		0.82	0.44	1.53

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 17 - Appendix. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on staying our all night and running away from home

	Staying out all night								Running away from home							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
Transition clusters (sensitivity)	5511				5345				5502				5336			
Decreasing to low		1.17	0.97	1.40		1.12	0.93	1.36		0.97	0.72	1.31		1.02	0.75	1.38
Low to problems		1.23	0.99	1.53		1.12	0.90	1.41		1.47	1.08	2.02		1.46	1.06	2.02
Homotypic problems		1.21	0.97	1.51		1.05	0.83	1.33		1.85	1.38	2.48		1.81	1.33	2.46
Heterotypic problems		1.51	1.10	2.06		1.23	0.88	1.71		1.51	0.95	2.40		1.41	0.87	2.27

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 18 - Appendix. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on being questioned by the police for suspected involvement in a crime and stealing outside of the home

	Questioned by police for suspected involvement in crime								Steal outside of the home							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
Transition clusters (sensitivity)																
Decreasing to low	5510	1.04	0.85	1.27	5344	0.98	0.79	1.20	5856	1.60	1.23	2.09	5677	1.50	1.15	1.97
Low to problems		1.19	0.94	1.50		1.03	0.81	1.32		1.33	0.95	1.86		1.26	0.89	1.77
Homotypic problems		2.20	1.79	2.70		1.93	1.55	2.39		1.39	0.99	1.93		1.30	0.92	1.82
Heterotypic problems		1.60	1.15	2.21		1.32	0.94	1.86		1.06	0.62	1.82		0.99	0.57	1.72

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household

Table 19 - Appendix. Unadjusted and adjusted logistic regression models showing the association between latent profile(s) and transition clusters on vandalism/destruction of property and fighting to the point of injury

	Vandalism								Fighting to point of injury							
	Unadjusted				Adjusted*				Unadjusted				Adjusted*			
	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI	n	OR	Upper CI	Lower CI
Transition clusters (sensitivity)																
Decreasing to low	5511	1.25	1.03	1.52	5344	1.15	0.94	1.41	5861	1.20	0.87	1.67	5682	1.02	0.73	1.42
Low to problems		1.23	0.98	1.55		1.14	0.89	1.45		1.97	1.42	2.74		1.61	1.14	2.26
Homotypic problems		2.13	1.73	2.62		1.90	1.52	2.36		1.86	1.33	2.61		1.51	1.06	2.14
Heterotypic problems		1.72	1.25	2.38		1.58	1.13	2.21		2.10	1.31	3.35		1.74	1.07	2.84

CI: confidence interval
Hyper./Inatt. & Int.: Hyperactivity/inattention & Internalizing
LPA: latent profile analysis
n: sample size
OR: odds ratio
* Adjusted for gender, socioeconomic status, stressful life events, high maternal depressive symptoms, high maternal alcohol use, and non-two parent household