BORDERLINE PERSONALITY DISORDER

BORDERLINE PERSONALITY DISORDER: EXAMINING TRAJECTORIES OF DEVELOPMENT AMONG ADOLESCENTS

By

VALBONA SEMOVSKI, BSc (Hons.)

A Thesis Submitted to the School of Graduate Studies in Partial Fulfillment of the

Requirements for the Degree Master of Science

McMaster University

Master of Science in Health Research Methodology (2016); Department of Clinical Epidemiology; McMaster University, Hamilton, Ontario

TITLE: Borderline personality disorder: examining trajectories of development among adolescents

AUTHOR: Valbona Semovski, BSc (Hons.) (York University)

SUPERVISOR: Dr. K. Boylan

NUMBER OF PAGES: ix, 67

Lay Abstract

Information about the classification and development of borderline personality disorder (BPD) in adolescence is in its early stages. While evidence for similar construct validity to the adult disorder exists for adolescents, major gaps in knowledge regarding the stability in course of BPD symptoms and predictors of clinically significant symptom trajectories in this age group remain. As most clinicians will assess youth already having significant features of the disorder, early detection requires knowledge of the indicators that precede an unfavourable trajectory. This dissertation will help address these gaps by modeling trajectories of BPD symptoms in youth across ages 13-16, whilst examining factors influencing trajectory group membership.

Abstract

Title: Borderline personality disorder: examining trajectories of development among adolescents

Background: Borderline personality disorder (BPD) tends to be highly comorbid with other disorders. In adolescence, information about the classification and development of BPD is in its early stages. There is limited empirical research available that investigates predictors of clinically significant symptom trajectories of the disorder using data collected in childhood. Given the enormous personal and societal costs associated with BPD, early detection and prevention is important. Clinical implications of this research include an improved understanding of risk factors and possible mechanisms for development of BPD symptomatology.

Objectives: To identify trajectories of BPD symptomatology in a Canadian sample of adolescents (N = 703) assessed at ages 13, 14, 15 and 16, while examining predictors of trajectory group membership assessed at age 12.

Methods: Data from the McMaster Teen Study was used to examine trajectories of BPD symptoms using group-based trajectory modeling. The influence of gender, depression, ADHD, family functioning and various sociodemographic variables as predictors of an individual's group membership was tested. Chi-square, analysis of variance and multinomial logistic regression was used to analyze the data.

Results: A four-group trajectory model was most robust at describing BPD symptomatology in this age group. Univariate analyses supported female gender, depression and ADHD at baseline, parental age, marital status, education, and income as significant predictors of group membership. Female gender, depression and ADHD severity at baseline were significant predictors of group membership when adopting a multivariate approach. There is a greater prevalence of girls with higher depression and ADHD scores in the high-increasing features and BPD group.

Conclusion: Findings demonstrate four various developmental trajectories of BPD features. Results further the understanding of the factors associated with development of the disorder across time.

Keywords: adolescence, borderline personality disorder, group-based trajectory modeling

Acknowledgements

I would like to take this opportunity to offer my sincerest gratitude to those who have provided their support and thoughtful feedback in making the presentation of this research possible. First, I would like to acknowledge and thank my supervisor, Dr. Khrista Boylan for her ongoing support, guidance and contribution to this project and my educational experiences. I am grateful for the opportunities you have provided in exercising my potential as a researcher.

Secondly, I would like to express my appreciation for the assistance and support provided by my committee members, Dr. Mark Ferro and Dr. Jennifer Couturier. You have been very knowledgeable advisors. You have provided a wealth of information throughout both the analysis and writing phases of the project. To Dr. Andrea Gonzalez, my external reviewer, many thanks for providing your time and expertise towards the completion of this dissertation.

I would like to acknowledge the youth and their families who participated in the McMaster Teen Study. While I did not have the pleasure of personally meeting you, I am extremely appreciative for your participation. Without your trust, enthusiasm and sustained cooperation, this research would not have been possible. You are all an integral piece in the transformational change surrounding child and adolescent mental health initiatives.

Finally, I would like to acknowledge my family and friends for their love, encouragement and continuous support with my research endeavours and education. To my parents: many thanks for the several sacrifices you have made and for being excellent role models. Without you both, I would not have the work ethic and the outlook on life I possess. For that, I will forever be grateful.

Table of Contents

List of Figures and Tables

List of Abbreviations and Symbols

WHO:	World Health Organization
BPD:	Borderline Personality Disorder
DSM-IV:	Diagnostic and Statistical Manual of Mental Disorders, 4th Edition
MDD:	Major Depressive Disorder
ADHD:	Attention-Deficit Hyperactivity Disorder
DSM-5:	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
SD:	Standard Deviation
Ab-DIB:	Abbreviated Diagnostic Interview for Borderlines
ACC:	Anterior Cingulate Cortex
HPA:	Hypothalamic-Pituitary-Adrenal
SES:	Socioeconomic Status
MacTeen:	McMaster Teen Study
T3:	Time 3
T1:	Time 1
T4:	Time 4
T5:	Time 5
T6:	Time 6
T7:	Time 7
REB:	Research Ethics Board
FIML:	Full Information Maximum Likelihood
ANOVA:	Analysis of Variance
BPFS-C:	Borderline Personality Features Scale for Children
BASC-2:	Behaviour Assessment System for Children, Second Edition
DSM-IV-TR:	Diagnostic Statistical Manual of Mental Disorders, 4th Edition, Text Revision
BCFPI-3:	Brief Child and Family Phone Interview
GBTM:	Group-Based Trajectory Modeling
BIC:	Bayesian Information Criterion
OCC:	Odds of Correct Classification
AvePP:	Average Posterior Probability
GMM:	Growth Mixture Modeling
LCA:	Latent Curve Analysis

Chapter 1 - Introduction

A developmental trajectory describes the course of the child's development along a particular skill or domain of functioning across the life span (Boivin & Hertzman, 2012). For the purposes of this dissertation, trajectories of symptoms consistent with mental disorder are of interest. With vast differences between individuals, it may be difficult deciphering between a healthy trajectory and one deviating from the societal norm. At what point does the presenting symptom or behaviour become worrisome and warrant some form of intervention? Is it possible to prevent or disrupt a risk trajectory or promote a healthy one? With 70% of adults with a mental illness reporting their symptoms as having started in childhood, it is important to identify the influence of factors that could potentially disrupt this pathway ("The Mental Health Strategy For Canada", 2015).

According to the 2004 estimates from the World Health Organization (WHO) World Mental Health Surveys, neuropsychiatric disorders are the leading cause of disability among non-communicable conditions worldwide (Nandi, Beard & Galea, 2009). With children and adolescents comprising approximately a third of the world's population, there have been great strides in research in understanding this age group. In Canada, it is estimated that 1.2 million children are affected by mental illness ("Children and Youth", n.d.), with the number of those experiencing a mental health problem expected to rise (Bor, Dean, Najman & Hayatbakhsh, 2014). Among these mental health problems is borderline personality disorder (BPD).

The majority of existing BPD studies have focused on adults, with more recent efforts directed toward children and adolescents. This followed shortly after the Diagnostic Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) permitted the diagnosis in youth (Sharp et al., 2012). In adolescence, BPD symptoms have been linked to poorer prospective outcomes for up to two decades (Goodman et al., 2011). This personality disorder is highly associated with impairments in psychosocial functioning, high social and economic costs due to the increased health care usage when compared to other disorders and high mortality rates owing to suicide (Vaillancourt et al., 2014; Lieb, Zanarini, Schmanhl, Linehan, & Bohus, 2004).

This dissertation aims to depict developmental trajectories of BPD among adolescents while distinguishing the impact that external covariates have on group membership. To put this in context, the subsequent sections will provide evidence for the validity of adolescent BPD and a review of the phenomenology, epidemiology, reluctance to diagnose BPD in youth, course and stability of the disorder, identified risk factors and comorbidities with an emphasis on major depressive disorder (MDD) and attention-deficit hyperactivity disorder (ADHD). Finally, there will be a brief discussion of what has been omitted in the literature and how attempts to circumvent these gaps are exhibited in the present research project.

1.1- Phenomenology and Epidemiology of Borderline Personality Disorder

BPD is characterized by a pervasive pattern of instability in affect regulation impulse control, interpersonal relationships and self-image. It is considered to be the most complex and impairing personality disorder in clinical practice (Chanen, Jovev,

McCutcheon, Jackson & McGurry, 2008). BPD occurs in approximately 1-3% of the general population, with rates in psychiatric settings reaching roughly 20% (Fossati, 2015; Chang, Sharp & Ha, 2011). Preliminary research has indicated that 1.4% of youth will meet diagnostic criteria for BPD by the age of 16. This prevalence rate is also mirrored in the adult literature. In other cases, higher prevalence rates for adolescents have been cited (Kaess, Brunner & Chanen, 2014). Sharp and Fonagy in 2015, using adolescents in the community, found rates ranging from 11% to 27%. Debate exists as to whether this higher prevalence in adolescence is a reflection of the course of BPD or the weaker validity for the personality disorder in this population (Miller, Muehlenkamp, & Jacobson, 2008).

Studies investigating BPD have mostly considered female participants or have collapsed male and female data together. This practice paired with the composition of clinical samples being predominantly female, has placed undue emphasis on BPD as being a disorder of females (Chang, Sharp, & Ha, 2011). Disagreement remains over this gender inequality, as the same sex differences are not found using population based samples (Paris, 2014; Grant et al., 2008). However, results stemming from studies comparing gender differences have not helped settle the disagreement. Findings have been inconsistent. Differences have been noted in the frequency of treatment utilization. Men have been found to be less likely to seek treatment and are more likely to present with substance abuse and antisocial features. Females on the other hand, present with more identity disturbance, body image issues and anxiety often linked to trauma (Goodman, Patel, Oakes, Matho & Triebwasser, 2013).

Adult BPD criteria have been applied to classify adolescents due to studies citing a similar phenomenology, etiology and rates of adverse childhood experiences in youth and adults with BPD (Chanen & Kaess 2012). For example, identity disturbance, affective instability, and intense anger are suggested to be the most stable symptoms in adolescent BPD (Fossati, 2015). Despite the several similarities between the two populations, there are documented differences. Compared to adults, adolescents are likely to present with more acute symptoms such as impulsive and self-damaging behaviours (Kaess, Brunner & Chanen, 2014). Due to these differences, two minor modifications have been recommended to warrant an adolescent diagnosis of BPD; (i) duration of symptom presentation being reduced from two years to one year with the added condition that (ii) personality traits are required to be pervasive, persistent and not limited to the developmental period of adolescence (APA, 2013; Sharp et al., 2012). There are nine criteria listed in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), of which five need to be met to warrant a diagnosis. A list of all nine criteria is summarized in Table 1.

1.2– To Diagnose or Not To Diagnose in Adolescents? That is Still the Question

BPD has gained an increasing amount of attention from both the scientific and clinical communities. Despite its popularity, it was not until the DSM-IV that the diagnosis in youth was permitted (Sharp et al., 2012). Prior to this point, there had been reluctance to diagnose BPD in adolescence for several reasons. Even at present, there are some mental health professionals hesitant to diagnose an adolescent with BPD (Miller, Muehlenkamp, Jacobson, 2008). Among the reasons for this hesitation are concerns about

stigma, difficulty distinguishing typical features of BPD from normal personality development and the stability of the disorder (Fonagy et al., 2015).

The stigma that may surround the label should not prevent an assessment of BPD when warranted. Several studies have acknowledged that adolescent BPD is as reliable and valid as that in adulthood (Fossati, 2015; Sharp, Mosko, Chang & Ha, 2011). A 2013 study including youth aged 12-18 years concluded that BPD could be reliably and validly diagnosed in those as young as 12-14 years of age (Glenn & Klonksy, 2013). The concept of a personality disorder describing a short-term phenomenon that may be limited to one or two years of adolescence is not acceptable to most clinicians. Increased confidence would be derived from studies demonstrating stability or worsening of symptoms in adolescence to attest to the construct of a personality disorder as beginning in adolescence and showing signs of persistence, a core feature of personality disorders. Further, there is a substantial lack of information in the literature regarding the construct validity, precursors, course, and risk factors related to BPD in children and adolescents (Sharp, Mosko, Chang & Ha, 2011).

Using a dimensional approach in studying personality may be most useful when studying stability or continuity in personality features or disorders. A dimensional approach accounts for the developmental variability found among adolescents as they mature across the lifespan. Chanen et al (2004) studied BPD using both a dichotomous (yes/no diagnosis) and dimensional (trait severity) approach and found that dimensionally measured BPD had higher stability rates. Using a dimensional approach to measure longitudinal stability adjusts for the small variations in symptoms that naturally occur

(Bornovalova, Hicks, Iacono, McGue, 2009).

In summary, the DSM endorses BPD as a valid and important diagnosis to make in adolescents. Studies conducted have alluded to the validity and reliability of such a diagnosis (Fossati, 2015; Kaess, Brunner & Chanen, 2014; Chanen et al., 2008). Adopting a dimensional approach in viewing BPD can further assist in understanding what distinguishes characteristics of BPD versus those of normal development. This dissertation aims to provide evidence on the course and risk factors related to BPD.

1.3- Course of Illness and Stability of BPD

Longitudinal research has suggested an increase in BPD traits following puberty, with a subsequent decline over later years (Kaess, Brunner & Chanen, 2014; Oltmanns and Balsis, 2011). Stepp, Keenan, Hipwell and Krueger (2014) conducted a study with females ages 14-19 from a community sample and found that BPD symptoms measured using the International Personality Disorders Examination appear to peak at age 15 years, decline between 15 and 18 years, and do not change significantly between 18 and 19 years of age. Zanarini, Frankenburg, Khera and Bleichmar (2001) identified the average age of first clinical presentation as 18 years with a standard deviation (*SD)* of 5-6 years.

More recently, Greenfield et al. (2015) conducted a four-year follow-up study on 204 adolescents (M = 14 years of age) that had presented in a Canadian emergency room with suicidal ideation or attempt. Using the Abbreviated Diagnostic Interview for Borderlines (Ab-DIB), adolescents were classified at ages 14 years and 18 years as having BPD that (over 4 years) persisted, remitted, was emergent or not present. Seventy-six percent of adolescents were classified as having persisting BPD, while 13% and 3.4%

met criteria at only one time point and were classified as having remitting and emerging BPD, respectively. Only 7.4% did not meet criteria at either time point and were classified as never having BPD. Adolescents who met BPD criteria at age 14 years were also eight times more likely to meet criteria at age 18 when compared to adolescents that did not meet criteria at baseline. These findings highlight that adolescence is a crucial period for the development of the disorder (Sharp & Fonagy, 2015).

There have been concerns regarding the stability of borderline personality disorder over time. This concern arises because the variability in the course of BPD and the flexibility in the traits exhibited by youth (Lenzenweger & Castro, 2005). The stability of BPD symptoms is difficult to assess from clinical studies, where the majority of children or youth will be exposed to treatment. However, results provide support for a diagnosis of BPD in adolescents as young as 14 years of age and for the stability of a BPD diagnosis over a four-year period (Zanarini et al., 2001; Greenfield et al., 2015) while maintaining that adolescence is a crucial period for the development of the disorder (Sharp & Fonagy, 2015).

These results from both adolescent and adult populations provide preliminary evidence that BPD is a stable disorder for at least a four-year time frame. Acknowledging that BPD symptoms may present early with close attention paid to the evolution of symptomatology over time is important. Due to the difficulty that is associated with establishing stability of the disorder with a clinical sample, this thesis aims to reinforce the previous findings of BPD stability in adolescence within an epidemiological sample. This dissertation provides an added advantage when compared to other epidemiological

studies as both boys and girls are included in the analytical sample.

1.4 - Risk Factors

Conceptualization of the pathways leading to a diagnosis of BPD can be understood by analyzing factors taken from the individual's psychological, sociocultural and biological background while examining the trajectories of impairment from an earlier point in time (Chanen & Kaess, 2012; Cicchetti & Rogosch, 2002). However, little is known about early BPD predictors in adolescence (Goodman et al., 2011). In developing a strategy to understand the influence risk factors have on the development of BPD, a diathesis-stress framework has been proposed (Garber & Rao, 2014). The diathesis for BPD, as many other psychological constructs, has been proposed to include heritable traits which can be measured in younger children in the form of emotional sensitivity/reactivity and various measures of impulsivity. These early dispositional factors are thought to and have been shown to influence the development of emotional regulation skills (Tyrer 2007; Zelkowitz, Paris, Guzder, Feldman, 2001; Stone, 1980). The lived experience of the exposure to stressors such as interpersonal conflict at various or repeated developmental periods may result in some adolescents presenting with some or all features of BPD (Sharp & Fonagy, 2015).

In addition to identifying the potential factors that increase the risk for a diagnosis of BPD, or worsen the trajectory of those who have already obtained a diagnosis, lies another issue – understanding the mechanisms whereby they interact. Several theories have been posited to help disentangle what is known about the development of BPD, all adopting a diathesis-stress framework. Among them are the Biosocial (Crowell,

Beauchaine & Linehan, 2009), Impulsivity-Oriented (Paris, 2005), Mentalization-Based (Fonagy & Luyten, 2009) and Gene-Environment developmental theories (Gunderson & Lyons-Ruth, 2008).

Until recently, there was little information on specific risk factors for BPD in an adolescent population. The Children in the Community Study was the only study publishing prospective risk factors over multiple time points from childhood to adulthood (Cohen, Crawford, Johnson & Kasen, 2005). Among identified risk factors leading to symptoms for the disorder are neuropsychological and biochemical impairment, exposure to traumatizing environments, a deficit of early socialization and challenging family interactions (Leichsenring, Leibing, Kruse, New & Leweke, 2011). Findings from genetic studies indicate heritability estimates for BPD to range from 35-45% (Chanen & Kaess, 2012). Borderline traits are moderately heritable from age 14 to 24 years with a trend for increased heritability between 14 and 18 years (Sharp & Fonagy, 2015). Although, specific genes leading to the disorder have not been definitively identified, Hankin, Jenness, Abela and Smolen (2011) have suggested that carriers of the short allele of the serotonin transporter promoter gene {5=HTTLPR} exhibited the highest level of BPD traits. Neurobiological studies have suggested volume reductions in the frontolimbic network (Leichsenring et al., 2011) including the orbitofrontal cortex and the anterior cingulate cortex (ACC) are associated with several features of BPD (Chanen et al., 2008). In addition, evidence of hypothalamic-pituitary-adrenal (HPA) axis abnormalities have been found in adolescents with BPD (Zimmerman & Choi-Kain, 2009). A more detailed description of the findings explaining the neurobiological studies can be found elsewhere.

Aside from these biological variables, environmental stressors have been identified as correlates and often predictors of BPD symptoms. Studies have indicated that low socioeconomic status (SES) (Cohen et al., 2008), maladaptive parent-child interactions (Macfie & Strimpfel, 2014) and other childhood adversities such as trauma prior to puberty (Zanarini & Wedig, 2014) are predictors of BPD symptomatology. There is emerging literature suggesting that BPD in adolescence may be associated with bullying, peer rejection (Vaillancourt et al., 2014) and teen dating violence (Reuter, Sharp, Temple & Babcock, 2015). Research surrounding the influence of these known risk factors and several others are critical when discussing symptomatology.

Specificity of the risk factors related to BPD compared to other forms of psychopathology, is not strongly validated in the literature (Sharp & Fonagy, 2015). In an effort to add to the existing literature regarding risk factors, the role several of these factors play as predictors of BPD trajectory group in adolescents – specifically gender, depression, ADHD, the interaction between depression and ADHD, family functioning and sociodemographic variables were evaluated. Although the data set used for this research project does not include biological risk factors, we can organize the variables to represent either diathesis or stress risk factors. A theoretical framework to provide context for this research project is illustrated in Appendix A.

1.5- Comorbidities

Adolescents with BPD experience multiple psychiatric comorbidities (Sharp & Fonagy, 2015). Comorbidity is an important correlate of BPD but its relation as a predictor of BPD is still questionable. As BPD consists of symptoms reflecting internalizing and externalizing pathology, the clinical presentation of BPD can lead to confusion with other psychiatric diagnoses. The picture becomes less clear, when BPD presents itself in association with another identifiable mental health disorder (Sharp et al., 2015). BPD has shown to be highly comorbid with depression, anorexia, bulimia, ADHD and substance use.

This high level of comorbidity between BPD and several other disorders has called into question the specificity of BPD during adolescence (Fossati, 2015). However, studies have indicated that higher rates of comorbidity appear in adolescents with BPD when compared to adolescents with either no personality disorder or no disorder at all. Complex comorbidities, especially among adolescents seeking treatment should serve as an indicator of the presence of BPD. For instance, 71% for comorbid mood disorders, 67% for anxiety disorders and 60% for externalizing disorders were reported when studying adolescent inpatients with BPD (Ha, Balderas, Zanarini, Oldham & Sharp, 2014). In addition, rates of comorbidities for adolescents seem to resemble those cited in the adult BPD literature (Fossati, 2015; Eaton et al., 2011; Chanen, Jovev & Jackson, 2007). Studies investigating comorbidities in adolescent samples have cited prevalence rates of 86% in a clinical sample and 50% in the Children in the Community study (Sharp & Fonagy, 2015). Barriers to accurate diagnosis must be considered in the context of the

high rate of comorbidity seen in BPD, especially as comorbid disorders may not improve until BPD is recognized and treated (Skodol, 2015). Literature has supported that MDD and ADHD are precursors to the BPD phenotype and often precede the personality disorder. However, they do not predict its onset with certainty (Chanen & McCutcheon, 2013).

1.5.1- BPD & MDD

MDD is a mental illness that is characterized by a persistent sad mood with feelings of hopelessness and worthlessness. MDD can also be characterized by changes in sleep and appetite, loss of interest in normally pleasurable activities, a difficulty concentrating/making decisions and thoughts of death or suicide (APA, 2013; Bylund & Reed, 2007). Depression as a comorbidity with other psychiatric disorders is very common in adolescent populations, with rates ranging from approximately 42% in community samples to 75% in clinical samples (Garber & Rao, 2014). With BPD in particular, studies have shown that 41-83% of patients with BPD also reported a history of MDD (Lieb et al., 2004). The high comorbidity between depression and BPD calls for a special focus of this association in research (Yoshimatsu & Palmer, 2014). Crick, Murray-Close and Woods (2005) found that symptoms of depression and borderline personality features were highly correlated at each time point of their study. An increase of symptoms of one disorder was linked to increases of symptoms in the other. Longitudinal studies have supported that depressive symptoms in childhood are predictive of a BPD diagnosis in adolescence (Fonagy et al., 2015).

In 2009, a study analyzing trajectories of depression found that increased rates of psychiatric comorbidity were associated with membership in several atypical depressive groups and rates differed by gender (Nandi, Beard & Galea, 2009). Therefore, it is likely that depression will be an important predictor of BPD in young people. This could be because it constitutes part of the diathesis to the disorder – sharing a biological vulnerability to emotional dysregulation or intensity of emotionality – or as a stressor, increasing the risk for BPD symptoms after it begins in adolescence. For the dissertation, depression is considered a "stressor", as the mean age of onset of depression is 14 years. Many youth after that age will continue to experience an increase in severity of depressive symptoms and may experience increased difficulties managing their emotions. BPD symptoms may reflect this increased emotionality in vulnerable individuals.

1.5.2- BPD & ADHD

The key feature of ADHD is a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with development or the individual's functioning. ADHD is a neurodevelopmental disorder which begins in childhood and reflects deficits in frontal lobe functioning which may or may not be persistent with growth and brain maturation (APA, 2013). Epidemiological studies suggest that ADHD occurs in approximately 3-5% of children (Storebo & Simonsen, 2014). The shared features of impulsivity and emotional instability, common to both ADHD and BPD, have lead researchers to propose that the two disorders might be a result of different developmental pathways based on a common origin (Matthies & Phillipsen, 2014). There is indeed evidence of association between these diagnoses within individuals and BPD (Fosssati,

2015; Fischer, Barkley, Smallish & Fletcher, 2002). In a longitudinal study, adolescents with ADHD were more likely than controls to be diagnosed with BPD; 14% versus 1.2% (Matthies & Philipsen, 2014). In another study, ADHD symptoms at age 8 years predicted BPD symptoms, but not depression, at age 14 years (Fossati, 2015).

There are few true longitudinal studies following ADHD youth and looking at BPD features as outcomes. The studies that take this approach do not provide conclusive evidence that ADHD is a robust predictor of risk for BPD at a later time. Retrospective studies of BPD in adults have indicated several cases where ADHD was diagnosed in childhood, suggesting ADHD often precedes the emergence of BPD (Matthies & Phillipsen, 2014). Based on current evidence, it is posited that ADHD and BPD are distinct disorders, however the presence or persistence of ADHD into adolescence or adulthood when comorbid with BPD is an indicator of severity of BPD (Calihol, Gicquel & Raynaud, 2015). Further to this point, is the potential impact of the posited "gender paradox" in reference to ADHD severity in girls and its impact on girls' mental health trajectories. ADHD is five times more prevalent in boys than in girls. However, when ADHD does occur in girls, it is associated with greater severity of symptoms and impairment (Loeber and Keenan, 1994). Therefore, it is hypothesized that ADHD is likely to be an important risk factor for BPD, particularly in girls. ADHD may function as a "diathesis" risk factor as it shares biological vulnerability to impulsivity and poor control of emotions, and manifests early in development. However, it could also be a "stressor" as children with ADHD struggle more socially (Kofler et al., 2011) which occurs most often in middle school.

1.6- Summary

To understand the development of BPD in adolescence, research examining the onset, course, duration and stability of the disorder using longitudinal data is required. Epidemiological sampling with prospectively followed cohorts is critical to broadening this understanding, and having confidence in results. In addition, further research is needed to identify predictors of well-characterized groups with BPD which differentiate these groups from groups with other psychopathological conditions. More specifically, the precursors identified as associated with BPD such as disturbances in attention and emotional regulation should be studied and potentially treated as early as possible (Chanen, 2015). This is both because it is sound on an empirical basis and indicators of disturbances in attention and emotion regulation such as clinical symptoms of ADHD and depression are more easily measured and assessed by clinicians than BPD symptoms. Ideally, data from a community setting collected during a critical time period, between the ages of 12 and 17 years when symptoms are in rapid evolution is required. The majority of the existing adolescent literature is limited by the use of small, clinical samples with relatively short follow up periods, or the use of dichotomous measures for BPD limiting the ability to identify small changes over time (Bornovalova et al., 2009). This research project sets out to provide a stronger foundation for an adolescent BPD construct as well as delineation of its clinical predictors in an epidemiological sample over four years.

1.7-Research Rationale and Objectives

The present study aims to fill existing gaps in the BPD literature as it pertains to a child and adolescent population. Based on the literature review, these gaps include evidence for multi-year dimensional stability, the interactions between the symptomatic youth and their environmental context, as well as high comorbidity. From a clinical perspective, identifying groups of children with different trajectories based on differing courses of symptoms and/or predictors and outcomes is beneficial. Based on our literature review, the specific research objectives for this project are:

(1) To test if there are distinctive trajectories of BPD symptoms over four years where participants are aged 13-16 years (to demonstrate whether a stable and impairing construct is identified that mimics BPD and to understand what comparator BPD symptom groupings might look like).
(2) To investigate the association between these developmental trajectories with gender, depression, ADHD, family functioning and sociodemographic variables as potential predictors of trajectory group membership.
(3) To investigate a possible component (the interaction of ADHD and depression) of the diathesis-stress framework and its influence on the developmental trajectories.

Data from the McMaster Teen (MacTeen) study was used. It is a large epidemiological study that adopts a prospective and longitudinal data collection strategy within the population of interest.

1.8- Research Questions

Our research questions are the following:

(1) Using group-based trajectory modeling what are the specific patterns of development of BPD symptoms across waves 4-7 (age 13-16 years)?

(2) Is gender differentially associated with trajectory group membership?

(3) Is there a group of adolescents who are characterized by high stable or increasing symptoms to support there is a childhood onset and four-year stability of BPD?
Is there a significant association between known predictors of BPD and high stable or increasing trajectory membership, specifically MDD, ADHD, family and sociodemographic variables at time four/age 16 years? Do these predictors differentially associate with the high BPD group as compared to the other trajectory groups identified?

(4) What childhood predictors are associated with decreasing or moderate level trajectory memberships of BPD symptoms?

1.9- Research Hypotheses

No previous study has investigated BPD trajectories using the group-based trajectory model in adolescence. Based on the findings from the literature review, five main hypotheses are proposed. It is hypothesized that three different trajectory groups will be observed such that there will be a high stable/increasing, moderate persistent and decreasing/non-symptomatic group. The high stable/increasing group will correspond to the population prevalence of BPD in adolescents (~10%). The moderate and decreasing group is proposed as reflecting the population of youth who may exhibit clinically or

socially concerning behaviors for a brief period of time, but not for the 4-year duration of the study period. This group may have other (non-BPD) mental health difficulties and manifest some symptoms of BPD only. It is anticipated that approximately 20-30% of the sample will be in this group. The remainder of youth will be classified in the non-symptomatic group (60%).

In several studies, there have been a number of reports of BPD affecting females more often than males. For the second hypothesis, there will be a significantly higher prevalence of girls identified in the moderate persistent and higher level symptom trajectory groups. Due to the comorbidities of depression and ADHD with BPD cited in the literature, it is expected that symptoms of depression at Time 3 (T3) will be significantly associated with membership in the trajectory group characterized by elevated BPD features. Symptoms of ADHD at T3 will also be significantly associated with membership in the trajectory group characterized by elevated BPD features, but perhaps also in the moderate persistent or decreasing groups, as ADHD and relatedly, BPD symptoms in this group would reduce with age and brain maturation. A comorbidity between the two, expressed by an interactive term at T3, will be associated with membership in the trajectory group characterized by elevated BPD features. The group characterized by non-significant features will not be predicted by symptoms of depression or ADHD at baseline.

A more thorough understanding of risk factors and pathways involved in the development of BPD might aid in earlier diagnosis and implementation of intervention programs. This early interference could promote a developmental trajectory that leads to a

more favoured outcome for youth (Newton-Howes, Clark & Chanen, 2015; Chanen & Kaess, 2012). In terms of potential risk factors that influence the manifestation of BPD symptoms in the literature, trajectories characterized by elevated BPD symptoms will include individuals described as those likely to have experienced variables hindering functionality of the family which affects their emotion regulation skill development (ie. problems with communication, support, attachment, general relationships and problem solving within the family). In addition, trajectories characterized by elevated BPD symptoms will include individuals from a lower socioeconomic status.

Chapter 2 - Methods

2.1- Study Data and Participants

Data were obtained from the MacTeen Study, an ongoing prospective study examining the relations among bullying, mental health, and academic achievement using a random sample of 51 schools within the Southern Ontario Public School Board. All grade five classrooms were approached for recruitment. The study began in the spring of 2008 (Time 1 = T1) and has collected data over eight time points, each a year apart. In the spring of each year parents had provided consent for involvement in the study. Students also provided assent for their survey data to be used. Using this recruitment process 1147 participants were recruited, 703 of which agreed to be part of the longitudinal arm of the study.

Seven hundred and three participants were considered as the sample for analysis in the current study. Of particular interest for this research project was Time 4 (T4), 5 (T5), 6 (T6) and 7 (T7). Data collection for T4 through to T7 was completed in the privacy of the child's home. Families were given the option of completing either a paper and pencil or online version of the survey. Parent interviews were completed over the phone with a trained research assistant or by using the paper and pencil version (Vaillancourt, Brittain, McDougall & Duku, 2013).

2.2- Ethical Considerations

The research project was originally approved by McMaster's Research Ethics Board (REB) prior to initiation and annual ethics clearance was requested and approved by both McMaster University and the University of Ottawa (the principal investigator's

primary affiliation) REBs. Parental consent and student assent were obtained with each wave of data collection. Upon completion, student surveys were screened for elevated depressive symptoms. The primary investigator followed up with each student scoring high (and/or their parents) and put individuals in touch with appropriate services and/or referrals.

2.3- Attrition

Attrition is a common problem across epidemiological studies, and this needs to be addressed in the analyses (Nagin & Odgers, 2010). PROC TRAJ, an extension for the SAS software used in this project, can model the trajectories of participants with partial missing data using full information maximum likelihood (FIML). FIML includes participants with partially missing data under the assumption they are missing at random and the pattern at which this data is missing has no significant association with the variables of interest (Nagin, 2005). For the present study, the analytical sample was selected as a function of whether data for the outcome variable of interest was available at one or more waves of data collection and if age was reported. Out of 703 eligible participants, 505 met the inclusion criteria (72%). Two hundred and seventy-one participants (54%) of the analytical sample had completed all four assessments. A total of 198 adolescents were excluded due to missing scores at all four cycles. At T4, 84% of the total analytical sample participated (N = 423), 83% at T5 (N = 419), 78% at T6 (N = 392) and 74% at T7 (N = 375). This rate of attrition is similar to other epidemiological studies (Ferro, Boyle, Avison, 2015). Table 2 summarizes this information.

A Chi-square test was conducted to identify differences in gender between non-participants versus all others. Analysis of variance (ANOVA) tests were conducted to identify differences at baseline between non-participants versus all others on age, family functioning, depression, ADHD scores and sociodemographic variables using SPSS 20.0 (IBM Corp, 2013). Participation at T4 was significantly associated with gender, $X^2(1) = 9.6$, $p = .001$ and with family functioning [$F(1, 385) = 4.1$, $p = .04$] with more girls participating who have higher scores ($M = 22 \pm 2.2$) on measures assessing family functioning. This indicates slightly more issues with communication, support, attachment, general relationships and problem solving within the family than those in the non-participation group. More boys were accounted for in the non-participation group at all four waves with better scores on measures of family functioning ($M = 21 \pm 2.5$). Participation was not significantly associated with age [$F(1, 624) = 3.8$, $p = .052$], depression [$F(1, 479) = 1.8$, $p = .18$] or ADHD [$F(1,455) = 0.3$, $p = .59$] measures. On average, those who never participated were slightly older ($M = 11 \pm .39$), had higher scores on measures of ADHD ($M = 7.0 \pm 6.2$) and had lower scores on measures of depression ($M = 2.7 \pm 3.6$). This is summarized in Table 3.

2.4- Procedures

2.4.1- Measures

Primary Outcome: Symptom Trajectories of BPD Features

The Borderline Personality Features Scale for Children (BPFS-C) was used to examine self-reported features of BPD; the outcome variable, at T4-T8 (Crick, Murray-Close & Woods, 2005). The scale consists of 24 items reported on a likert-type scale with

responses ranging from 0 (not at all true) to 4 (always true) (Vaillancourt et al, 2014). Scores are summed with higher scores indicating greater levels of borderline features. Cronbach's α = .76 has been supported for this measurement tool in previous studies (Crick, Murray-Close & Woods, 2005). For the present study through T4-T7, Cronbach's α = .90 - .86. A copy of the scale is included in Appendix B.

Covariates Distinguishing the Trajectories:

Depressive and ADHD Symptoms

The Behaviour Assessment System for Children Second Edition (BASC-2) was used to assess self-reported symptoms of depression and ADHD. It is a multidimensional measure of behaviour and self-perceptions of people between the ages of 2 and 25 years that includes self, parent and teacher rating scales. It is used to make differential diagnoses of depression and ADHD based on categories outlined in the Diagnostic Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR) (Reynolds & Kamphaus, 2004). The participants answered true/false and likert-type questions with options ranging from 0 (never) to 3 (almost always). The BASC-2 clinical scales have been shown to be psychometrically sound and have good internal consistency with α = .67 - .86 (Vaillancourt et al., 2014; Reynolds & Kamphaus, 2004). In the present study, the internal consistencies for depression over time were at T4 (age 13) α = .89, T5 (age 14) α = .89, T6 (age 15) α = .91 and T7 (age 16) α = .90. The internal consistencies for ADHD over time were at T4 α = .81, T5 α = .81, T6 α = .82 and T7 α = .79.

Outcomes of Family Functioning

The Brief Child and Family Phone Interview (BCFPI-3) is a structured phone interview screening for emotional and behavioural issues in children ages 3-18 years. It was used to assess the parent-reported variability of family functioning for this study. The scale is useful in gathering demographic information, the impacts of the child's mental health problems, risk and protective factors, family readiness for service and potential barriers to service utilization. Reliability coefficients for the subscales have ranged from .77 to .86 in field trials (Boyle et al., 2009). Overall family functioning is assessed on an eight-item subscale. The subscale is located within the risk factors scale of this measurement tool. Parents respond to items using a four-point likert-scale with options ranging from 4 (strongly agree) to 1 (strongly disagree). Higher scores on this subscale reflect problems with communication, support, attachment, general relationships and problem solving within the family. The internal consistency for the subscale across four waves of data was $\alpha = .85$.

2.4.2- Analytic Plan

Longitudinal data allows for the analysis of developmental trajectories. Semi-parametric group based methods were used to identify the number and shape of distinct trajectories of BPD features using four annual prospective waves of data (T4 [Grade 8; age 13 years] – T7 [Grade 11; age 16 years]). Specifically, a group-based trajectory modeling (GBTM) approach was adopted to model developmental trajectories of BPD features. A description of alternative approaches suitable for the depiction of developmental trajectories is summarized in Appendix C. All trajectories were run in

SAS 9.4 software using commands that can be found at the following website: http://www.andrew.cmu.edu/user/ bjones/ (SAS Institute Inc., Cary, NC). GBTM views individual change, as normally distributed within groups that are characterized by their own distinct growth patterns. Those in a particular group are thought to be more similar to each other than to members in alternative trajectory groups (Nagin, 2005).

GBTM allows for the identification of heterogeneity in both the mean of the BPD behaviour at a given age, and in the development of BPD symptomatology over time (Côte et al., 2007). This assumption is useful for conceptualization of clinical problems. The presence of various groups of children characterized with differing patterns of behaviour is expected. This model has been adopted for this research project, as it serves as a good first step in estimating population heterogeneity when depicting trajectories (Jung & Wickrama, 2008). In addition, understanding the development of a disorder over time has utility for description of clinical syndromes, an issue mentioned earlier (Boylan, Vaillancourt & Szatmari, 2012). Given the limited work available to suggest distinct trajectories of BPD features in adolescence, up to a four-group solution was examined.

The trajectory groups were generated using the participant's sum scores on the BPFS-C, for each respective time wave between T4 and T7. Within each group, the intercept and slope terms were assumed to be constant when modeled. Estimation of the trajectories was conducted in two steps. First, the ideal number of trajectory groups was established. A censored normal model was used given the non-parametric distribution of the continuous data. This is appropriate when data points within the dataset can either cluster at the minimum, maximum or both for the BPFS-C measurement scale. GBTM

uses all of the available scores at each time point to determine groups of individuals with similar trajectories. Maximization of the Bayesian Information Criterion (BIC) was used when identifying the maximum number of groups. Ideally, the model with the lowest BIC value would be chosen with strong differences between BIC scores considered in the range of 10 or more units (Raftery, 1995). A complete theoretical discussion of these concepts is presented elsewhere (Nagin, 2005).

In the second step, the slope or shape of the trajectory was tested. When selecting the shape of each group, cubic followed by quadratic, linear and zero growth factor options were considered as possibilities. The best fitting model was chosen where all growth and intercept terms for each trajectory in the model remained statistically significant, a maximization of the BIC score (lowest (-) score) was achieved and a conceptually clear model with a sufficient number of members in each group for comparison were apparent. When multiple alternative models presented with similar BIC scores, the best model was chosen on a combination of other factors assessing reliability of the models. The most parsimonious model was chosen as the one with trajectory groups having the least classification error (posterior probabilities greater than 0.7), the odds of correct classification (OCC) for each trajectory being greater than or equal to five and if a 10-fold difference in Bayes factor was achieved (Haltigan & Vaillancourt, 2016). A higher OCC suggests better classification by the model compared to random assignment to classes. The formula is presented in Appendix D.

The effects of gender differences on trajectory group membership were tested using the chosen model by first comparing the proportion of boys and girls in each

trajectory group. If the effect of gender was significant then gender was included as a variable of interest in a multinomial logistic regression. All other covariates were first assessed for significant associations with group assignment using either ANOVA or Chi-square tests, as appropriate. In testing the comorbidity in the diathesis-stress framework, an interaction variable was created and included in the univariate analysis as well. If statistically significant correlations were found, then those respective covariates were run in a forced entry multinomial logistic regression model with group membership as the dependent variable. Multinomial logistic regression is suitable using forced entry, as we want to predict the association of group assignment (variable with more than 2 outcomes) with our variables of interest, but have no a priori decision as to which variables are more important in predicting group assignment. Researchers believe that the forced entry method in multinomial logistic regression is appropriate when theory testing (Studenmund & Cassidy, 1987). Alternatively, an ordinal logistic regression could be conducted to capture the relationship between the independent variables and group assignment; if group assignment was categorized as varying severities of BPD features. Considering this dissertation is not using a clinical sample and the number of resultant groups is unknown, the group assignments (if any) are nominal in nature. Thus, a multinomial logistic regression is suitable when answering the research questions. Trajectories were modeled using SAS 9.4 (SAS Institute Inc., Cary, NC) and post hoc testing was conducted using SPSS 20.0 (IBM Corp, 2013).

Chapter 3- Results

3.1- Sample Characteristics

At T4, youth included in the research sample (N = 505) were ages 13-16 years (M = 14, SD = .36), 58% identified as female, and 71% belonged to a European-Canadian (white) ethnic background. The mean score of the BPFS-C at T4 was 30 (SD = 14). Mean score for depression and ADHD on the BASC-II were 3.9 (SD = 4.9) and 5.8 (SD = 4.0), respectively. Majority of reporting parents were the child's biological mother (86%) and were over the age of 40 (58%). Reporting parents were mostly married or common law (82%) and employed full time (61%) whilst having an annual household income of greater than or equal to $70, 0000 (Table 4).

3.2- Group-Based Trajectory Modeling: Trajectories of BPD

Each wave in the MacTeen study consisted of participants of various ages. In an effort to control for the affects of different ages nested in each data collection wave, an age-centered variable was created. The mean age at T4 was subtracted from the raw age at each time point and used to plot the trajectories (age-centered). The developmental trajectories of BPD were best characterized by four trajectory groups with the polynomial order of 0, 1, 1, 0 (BIC = -6249.28) (Figure 1). This four-group trajectory model was adopted for analysis as it had the highest probability of being the correct model when compared to other models. An alternative trajectory model was also considered when modeling the trajectories with polynomial order 0, 1, 2 (BIC= -6269.14). Comparison of fit indices between the chosen and the next best fitting models is shown in Table 5. Although the Baye's score is larger for the three-group model, suggesting superiority over

competitors, the other fit indices are very similar to the chosen four-group model. All fit indices were similar to the selected model except for the BIC score that was smaller for the chosen model. The average posterior probability (AvePP) is determined by averaging the estimated probability of being assigned to the group to which the adolescent is actually assigned. The average posterior probability of group membership for the selected model was .89, with actual posterior probabilities ranging from .81 - .93 (Table 6). The data for the chosen models trajectory groups support a high level of assignment accuracy. The first group was best modeled with a zero-order slope (flat pattern), the second and third with a first-order slope (linearly increasing pattern), and the fourth group with a zero-order slope (flat pattern). It is important to note that this study does not test a clinical diagnosis of BPD but instead delineates groups of adolescents with varying BPD features as indicated by BPFS-C scores. Scores of 65 or greater on the scale indicate clinical significance. In terms of BPD symptomatology, the four trajectories represent a non-significant features group, a moderate persistent features group, a high-increasing features group and a group that is depicted by high scores on the BPFS-C that may be comprised of adolescents with BPD coined the BPD group.

3.2.1 Sex Differences in Group Trajectory Membership

The effects of gender were explored by first comparing the proportion of boys and girls within each trajectory group, summarized in Table 7. The fourth trajectory group had the largest difference in representation between girls (N = 6) versus boys (N = 1). A chi-square was conducted to compare the effects of gender on BPD feature trajectories. Gender had a significant effect on group membership $X^2(3) = 22,\ p < .001$. Identifying as

a girl was significantly correlated with membership in groups two, three and four characterized by moderate persistent, high-increasing features and a potential diagnosis of BPD. These three groups were composed of more girls than group one, characterized by non-significant features. As a result, gender was included in the multinomial logistic regression.

3.3- Multinomial Logistic Regression

Prior to conducting the multinomial logistic regression, the responses to measures of depression, ADHD, family functioning and sociodemographic variables such as reporting parents age, marital status, education level, employment status, and household income were analyzed for missing patterns. Table 8 summarizes these results. A missing rate between 15% and 20% is considered common in psychological studies using epidemiological samples (Dong & Peng, 2013). Since values for family functioning is missing at a much higher rate than expected, the pattern of missing data was analyzed for this variable. An expectation maximization technique was implemented to assess if the pattern of missing data is random. If the assumption is met, the results are thought to be unbiased. The chi-square statistic for testing whether values are missing at random is referred to as “Little’s Missing Completely at Random test”. The Little Missing Completely at Random test for this study’s data resulted in a chi-square = 3.1, df= 2, p = .21, indicating the data is missing at random. Considering family functioning is thought to be stable over time, an average composite family functioning variable was created encompassing values at T3, T4, T5, T6 or T7 to mitigate the undue influence of missing data on the outcome variable. This computed variable was then used for all

subsequent analyses.

The impacts these covariates have on group assignment were investigated using Chi-square or ANOVAs, as appropriate. Results of the various statistical tests employed are summarized in Table 9. Results suggested significant independent effects for depression [$F(3, 436) = 27$, $p = < .001$], ADHD [$F(4, 441) = 16$, $p = < .001$] and the interaction between the two disorders [$F(3, 436) = 20$, $p = < .001$] across the four trajectory groups. Posthoc comparisons provided evidence to suggest that adolescents in the fourth trajectory group characterized as having BPD, had higher scores on depression and ADHD measures compared to the non-significant features group. Family functioning [$F(3, 248) = .69$, $p = .56$] was not significantly associated with group membership.

Reporting parents age [$F(3, 463) = 3.5$, $p = .02$], marital status [$F(3, 465) = 3.1$, $p = .03$], education level [$F(3, 465) = 6.7$, $p = < .001$], and household income [$F(3, 465) = 7.6$, $p = < .001$] were significantly associated with group membership. Employment status [$F(3, 466) = 1.6$, $p = .19$] did not influence group membership. Adolescents in the fourth trajectory group had a smaller percentage of married parents, those who had obtained a postsecondary education (college diploma or trades certificate, university undergraduate degree or university graduate degree) and those making more than $70, 000 a year.

Significant predictors of adolescent BPD trajectories in the univariate comparisons were then included in a multinomial logistic regression with trajectory group as the dependent variable and the first (non-significant features) trajectory group as the reference category. The first group was used as the reference point as it is the group

characterized as having negligible symptoms while the others have varying severity and duration of BPD symptom presentation. These variables included gender, depression, ADHD, the interaction between depression and ADHD, reporting parent's age, marital status, education level and household income. Before conducting the statistical test, data was analyzed using six assumptions in assessing suitability for inclusion in a multinomial logistic regression. Data was assessed ensuring the (i) dependent variable (group assignment) was measured on a nominal scale, (ii) one or more independent variables were continuous, ordinal or nominal in nature, (iii) independence of observations, (iv) no multicollinearity, (v) linearity and (vi) no outliers. Thresholds for acceptable values when assessing suitability are described elsewhere (Field, 2009). All results of the analyses for assumptions iv-vi are provided in Appendix E. All of the variables that were found to be statistically associated with group assignment met the six assumptions of suitability for use in a regression model. Thus, these variables were included in a multinomial logistic regression model and run simultaneously.

Calculated odds ratios and associated 95% confidence intervals are presented in Table 10. Beta values in the table illustrate the change in the outcome due to a unit change in the predictor variable (Field, 2009). Summarizing results from the multinomial logistic regression suggested that gender and symptoms of depression and ADHD at T3 were predictive of group membership. More specifically, gender b = -.82, Wald $X^2(1)$ = 12, p = .001 and ADHD b = .12, Wald $X^2(1)$ = 5.9, p = .02 were statistically significant predictors of BPD group membership in the moderate persistent group when compared to the non-significant features group. However, depression was not significantly correlated

with group membership, b = .09, Wald $X^2(1) = 1.5$, p = .23 for this between group comparison. In the high-increasing features group, gender b = -1.2, Wald $X^2(1) = 13$, p = <.001, depression b = .25, Wald $X^2(1) = 10$, p = < .001 and ADHD b = .24, Wald $X^2(1)$ = 17, p = < .001 were also statistically significant predictors of group membership when compared to the reference group. The group that presents with features warranting clinical attention, also known as the BPD group, had gender b = -21, Wald $X^2(1) = 1.5$, p = < .001, depression b = .44, Wald $X^2(1) = 4.0$, p = .05 and ADHD b = .64, Wald $X^2(1)$ = 6.1, p = .01 as significant predictors of group membership. Table 11 presents correlations between the variables included in the model and BPFS-C scores through T4-T7.

Chapter 4 - Discussion

The major objectives of this study were to identify distinctive trajectories of BPD symptoms over a four-year time frame. To our knowledge, this study is the first to examine developmental trajectories in adolescence using a group-based trajectory model. Our results indicated the best fitting model was associated with four developmental trajectories of BPD features characterized as: non-significant features (33.9%), a moderate persistent features (38%), a high-increasing features (26.6%) and a BPD group (1.5%). This finding is not consistent with the first hypothesis regarding the number of resultant trajectory groups. The study hypothesis expected to find three trajectory groups of BPD features (Haltigan & Vaillancourt, 2016; Nakar et al., 2016). Results from this study indicated that a four-trajectory group model is the most robust at describing BPD features among the MacTeen dataset. The prevalence of youth assigned to the high-increasing features group (26.6%) is higher than the estimated rate of ~10%. This finding provides evidence that elevated BPD symptoms in youth require an important focus as they may be a group at risk for BPD. Although the symptoms do not meet clinically relevant levels, they are still considered within a high range and demonstrate stability greater than one year (as per DSM-5) beginning from at least 14 years of age. Studying younger ages will confirm this suspicion. Results also coincide with the notion that higher rates of BPD symptoms appear to be relatively stable across mid to late adolescence (Winsper, Zanarini & Wolke, 2012).

What is also interesting is the moderate persistent features group, composed of 38% of the analytical sample. According to the BPFS-C, they probably are not at risk for

BPD. However, having a linear slope, individuals in this group may find themselves experiencing an increase in symptoms over time. The fourth group (~1.5%) is characterized by clinically high and stable BPD features as demonstrated by their average BPFS-C scores that were greater than 65. This would mean that a high-risk sample of adolescents with BPD features can be identified, consistent with others findings (Stepp, Pilkonis, Hipwell, Loeber & Stouthamer-Loeber, 2010). This group's existence would compliment Sharp et al.'s (2012) finding of BPD prevalence of 0.9-1.4% in an epidemiological sample of adolescents between the ages of 14 and 16 years.

The non-significant features trajectory group was used as the reference group for the multinomial logistic regression. Members of this group were predominantly male and had substantially lower mean scores of depression and ADHD when compared to the other groups. The other objectives aimed to investigate the association between these developmental trajectories with gender, MDD, ADHD and family functioning. This provides an opportunity to learn if the trajectories could be better predicted prior to their onset. Gender had a statistically significant influence on group membership. In line with the second hypothesis, identifying as a female increased the likelihood of being assigned to the BPD, high-increasing and moderate persistent features groups. Although consistent with findings from clinical samples, this gender difference deserves more specific consideration. In epidemiological studies, gender difference for prevalence of BPD in adolescence has not been reported (Paris, 2014; Grant et al., 2008). Further, Haltigan & Vaillancourt (2016) found girls endorsed elevations on interpersonal and intrapersonal factors of the BPFS-C when compared to male respondents. Yet, the scale items do not

show gender bias in terms of respondent patterns. This outcome reinforces the importance of replicating the finding that adolescent girls are more likely to be in a group endorsing persistent elevation in BPD symptoms. This suggests there may be a true gender difference in the prevalence of BPD in adolescence.

With respect to the other risk factors of interest for this research project, symptoms of depression and ADHD at T3 (age 12) were significantly predictive of membership in the second, third and fourth groups characterized by moderate persistent, high-increasing features and a possible diagnosis of BPD, respectively. Members in these three trajectory groups had higher scores on these BASC-2 subscales relative to the first group (non-significant features). The odds ratios indicate that as depression and ADHD responses increase by one unit, the change in the odds of being characterized in the second group rather than the first is 1.1. In regards to the third trajectory group, one unit increases on measures of depression and ADHD corresponds to odds ratios of 1.3, relative to the first trajectory group. Finally, being characterized in the fourth group given one unit increases on measures of depression is 1.6 greater than being identified in group one. As ADHD increases by one unit, the change in the odds of being categorized in group four is 1.9 that of being in group one (Table 10). If an adolescent exhibits depression or ADHD symptoms at T3, they are more likely to present with high-increasing features or features consistent with a diagnosis of BPD instead of no symptoms after four years. These findings are consistent with other studies that found associations between depression (Crowell et al., 2011) and ADHD (Stepp et al., 2012) with BPD features. The results from this study suggest that depression and ADHD are predictors of

subsequent BPD features. These results collaboratively provide evidence that clinicians should screen for BPD in youth who have ADHD or depression prior to age 14.

The diathesis-stress framework asserts that ADHD interacting with depression symptoms increases the risk of exhibiting some or all features of BPD (Figure 2). In this project ADHD was considered a diathesis while depression was viewed as a stressor, as it generally is a stress related phenotype. Using multinomial analyses, the interaction between symptoms of depression and ADHD were not found to be significantly predictive of group assignment. However given the comorbidity, the odds of being characterized in the BPD group rather than the non-significant features group is .98. This suggests that levels of ADHD at baseline are not an important effect modifier of the relationship between depression and trajectory outcome. These results indicate that ADHD and depression are related in ways that require further study. Investigating if ADHD, MDD and BPD persist as comorbid disorders across adolescence is worth testing. In order to accomplish this, future research can implement joint developmental trajectory methods and plot MDD/ADHD development alongside the development of BPD features.

Family functioning was not a statistically significant predictor of group assignment in the multivariate model. This finding is not consistent with the hypothesis that expected to find more issues with family functioning in the second, third and fourth group. Especially when compared to the non-significant features group. Research has indicated that issues with family functioning (Sauer & Baer, 2010) are correlated with BPD features. Our results may not reflect this due in part to the other variables in the regression model having more direct overlap with BPD as an outcome because they are

symptom based measures. There may be a pathway where family functioning measures done earlier in life or measured at a different time may be significant in a model, but measured at T2 were not relevant for the purposes of our study. Family functioning is important, as studies have alluded to family adversity having a direct impact on BPD features in adolescence (Newnham & Janca, 2014). Future research could investigate this relationship using different measures of BPD, specifically items testing abuse and neglect which are important predictors of BPD (Cohen et al., 2005) that the BPFS-C does not capture.

The results of this study have implications for care of adolescents presenting with some BPD features. Chanen et al. (2008) have shown that two or three BPD symptoms are predictive of clinical impairment related to BPD and not all five symptoms have to be met. Specifically, this study is helpful for early recognition. Early recognition provides the opportunity for intervention, reducing the onset of an unfavourable developmental trajectory. The strong link between depression and ADHD as precursors to a trajectory characterized by high BPD features is a good starting point for clinicians. In the context of the current study, early interventions targeting depressive symptoms and symptoms of ADHD would potentially have benefits on the adolescents who may have a vulnerability to developing BPD features at a later time point, especially adolescent females who tend to find themselves in groups characterized by elevated BPD features more often than their male peers.

Decisions to strengthen some aspects of the analytical phase of the project resulted in limitations to the generalizability of our results. Due to our inclusion criteria, the

number of adolescents in the sample was reduced from 703 to 505 participants. The excluded adolescents did not significantly differ from those included in the study other than their gender and family functioning. Those who participated were predominantly female with indication of issues around communication, support, attachment, general relationships and problem solving skills within the family based on their higher scores on the BCFPI-3.

Due to the smaller sample size and the frequency of data collection, questions around reliability of trajectory group membership are important to consider. Reports suggest that trajectories can be reliably reproduced when research follows a consistent approach to identifying the trajectories and when there are greater than 300 individuals in the sample (D'Unger et al., 1998) and four or more waves of data are employed (Nagin & Tremblay, 2005). Although the methods are consistent with these recommendations replication of these findings is necessary for confidence in the results. To confirm the findings of this study, future studies can examine the external validation of the trajectories by comparing the groups on relevant baseline measures or by using the groups to predict outcomes at a later point in time. In addition, due to the lack of diversity (71% Caucasian), the generalizability of our findings are constricted to a population living in Southern Ontario which are primarily white adolescents and their families. Future research with diverse samples (more representation of ethnic minorities) and in a broader catchment area (ie. all of Ontario) would enhance the generalizability of these results.

This study provided evidence for a childhood onset of BPD with a four-year stability. Given the flat pattern of the trajectory, it can be assumed that this stability

would continue in later years. Whether this pattern continues for certainty beyond the seventh wave of data collection for the study can be answered in future studies using the MacTeen dataset. It is important to note this study did not examine a clinical diagnosis of BPD. This remains an important task for future work. Currently underway, is the calculation of the prevalence of the disorder within this population at age 18-21 years. Future studies should investigate whether elevated borderline personality features in mid-adolescence are predictive of a later clinical BPD diagnosis. Our findings however, are consistent with previous findings of BPD symptomatology development in adolescence (Haltigan & Vaillancourt, 2016). For example, biological and genetic markers were not available in the MacTeen dataset but there has been evidence that BPD is heritable (Chanen & Kaess, 2012). The MacTeen dataset in later waves will have diagnostic data for this sample of youth as they reach ages 18-21 years, including genetic analyses. Thus, investigation of genetic markers on BPD features could be analyzed using the analytical sample in the future. In addition, subsequent work can explore joint trajectories of BPD symptoms and other comorbid disorders simultaneously. This approach could allow a test of the specificity of the development of BPD in relation to other emerging (or established) pathologies as the focus on adults has inhibited to an extent, the understanding of the developmental origins of BPD and trajectories over the life course (Bornovalova et al., 2009).

Chapter 5 – Conclusion

This study demonstrated that there is a trajectory of elevated BPD features that would warrant clinical attention, a high-increasing features and a moderate severity trajectory group that is stable across age 13-16. This suggests that many youth endorse BPD symptoms across adolescence and when they do, it is not a 'brief" phenomenon. Thus, this study adds to the validity of BPD in adolescents. Female gender and symptoms of depression and ADHD are predictive of membership in groups characterized by high BPD features using scores on the BPFS-C. This investigation of a typically developing sample provides the opportunity to examine predictors of adjustment problems before they emerge. Overall, the findings from this study contribute to our understanding of factors associated with development of BPD in a Canadian epidemiological sample. Understanding BPD in an adolescent population will aid mental health professionals in the early detection, intervention and evaluation targeting this personality disorder and problematic developmental trajectories. This early intervention with an unfavourable developmental trajectory could reduce impairment of psychosocial functioning, improve the prognosis for adolescents facing BPD while reducing the social and economic burden affiliated with the personality disorder (Fonagy et al., 2015).

References

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*: DSM-5. 5th ed. Arlington, VA: APA, 2013.

Boivin, M., Hertzman, C. (Eds.). (2012). Early Childhood Development: adverse experiences and developmental health. Royal Society of Canada – Canadian Academy of Health Sciences Expert Panel (with Ronald Barr, Thomas Boyce, Alison Fleming, Harriet MacMillan, Candice Odgers, Marla Sokolowski, & Nico Trocmé). Ottawa, ON: Royal Society of Canada.Available from: https://rsc src.ca/sites/default/files/pdf/ECD%20Report_0.pdf

Bor, W., Dean, A. J., Najman, J., & Hayatbakhsh, R. (2014). Are child and adolescent mental health problems increasing in the 21st century? A systematic review. *Aust N Z J Psychiatry, 48*(7) doi: 10.1177/0004867414533834

Bornovalova, M. A., Hicks, B. M., Iacono, W. G., & McGue, M. (2009). Stability, change, and heritability of borderline personality disorder traits from adolescence to adulthood: A longitudinal twin study. *Development and Psychopathology, 21*(4), 1335-1353. doi:http://dx.doi.org/10.1017/S0954579409990186

Boylan, K., Vaillancourt, T., & Szatmari, P. (2012). Linking oppositional behaviour trajectories to the development of depressive symptoms in childhood. *Child Psychiatry and Human Development, 43*(3), 484-497. doi:http://dx.doi.org/10.1007/s10578-011-0277-7

Bowerman, B. L., & O'Connell, R. T. (1990). *Linear statistical models: An applied approach* (2nd ed.). Belmont, CA: Duxbury.

Boyle, M., Cunningham, C. E., Georgiades, K., Cullen, J., Racine, Y., & Pettingill, P. (2009). The Brief Child and Family Phone Interview (BCFPI):2. Usefulness in screening for child and adolescent psychopathology. *Journal of Child Psychology and Psychiatry, 50*(4), 424-431.

Bylund, D. B., & Reed, A. L. (2007). Childhood and Adolescent Depression: Why do Children and Adults Respond Differently to Antidepressant Drugs? *Neurochemistry International*, *51*(5), 246–253. http://doi.org/10.1016/j.neuint.2007.06.025

Calihol, L., Gicquel, L., & Raynaud, J-P. (2015). Borderline Personality Disorder in Adolescents in *IACAPAP e-Textbook of Child and Adolescent Mental Health*. Geneva: International Association for Child and Adolescent Psychiatry and Allied Professions

Chanen, A. M. (2015). Borderline Personality Disorder in Young People: Are We There Yet? *Journal of Clinical Psychology*, *71*(8), 778-791. doi: 10.1002/jclp.22205

Chanen, A.M., Jackson H.J., McGorry P.D., Allott K.A., Clarkson V, Yuen H.P. (2004). Two-year stability of personality disorder in older adolescent outpatients. *Journal of Personality Disorders,*18, 526–41.

Chanen, A. M., Jovev, M., & Jackson, H. J. (2007). Adaptive functioning and psychiatric symptoms in adolescents with borderline personality disorder. Journal of Clinical Psychiatry, 68, 297–306.

Chanen, A. M., Jovev, M., McCutcheon, L. K., Jackson, H. J., & McGorry, P. D. (2008). Borderline personality disorder in young people and the prospects for prevention and early intervention. *Current Psychiatry Reviews, 4*(1), 48-57. doi:http://dx.doi.org/10.2174/157340008783743820

Chanen, A.M., & Kaess, M. (2012). Developmental pathways to borderline personality disorder. *Current Psychiatry Reports*, *14*(1), 45-53. doi: 10.1007/s11920-011-0242-y

Chanen, A. M., & McCutcheon, L. (2013). Prevention and early intervention for borderline personality disorder: Current status and recent evidence. *The British Journal of Psychiatry, 202*, s24-s29.

Chanen A.M., Velakoulis, D., Carison, K., Gaunson, K., Wood, S. J., Yuen, H. P., Yucel, M., Jackson, H. J., McGorry, P. D., Pantelis, C. (2008). Orbitofrontal, amygdala and hippocampal volumes in teenagers with first- presentation borderline personality disorder, *Psychiatry Research,* 163, 116–25.

Children and Youth (n.d.) Mental Health Comission of Canada. Retrieved from: www.mentalhealthcommission.ca/English/focus-areas/children-and-youth

Chang, B., Sharp, C., & Ha, C. (2011). The criterion validity of the borderline personality features scale for children in an adolescent inpatient setting. *Journal of Personality Disorders, 25*(4), 492-503. doi:http://dx.doi.org/10.1521/pedi.2011.25.4.492

Cicchetti, D., & Rogosch, F. A. (2002). A developmental psychopathology perspective on adolescence. *Journal of Consulting and Clinical Psychology*, 70, 6–20.

Cohen, P., Chen, H., Gordon, K., Johnson, J., Brook, J., & Kasen, S. (2008). Socioeconomic background and the developmental course of schizotypal and borderline personality disorder symptoms. Development and Psychopathology, 20, 633–650

Cohen, P., Crawford, T. N., Johnson, J. G., & Kasen, S. (2005). The children in the community study of developmental course of personality disorder. *Journal of Personality Disorders*, *19*(5), 466-486.

Côté, S. M., Vaillancourt, T., Barker, E. D., Nagin, D., & Tremblay, R. E. (2007). The joint development of physical and indirect aggression: Predictors of continuity and change during childhood. *Development and Psychopathology, 19*(1), 37-55. doi:http://dx.doi.org/10.1017/S0954579407070034

Crick, N. R., Murray-Close, D., & Woods, K. (2005). Borderline personality features in childhood: A short-term longitudinal study. *Developmental Psychopathology*. *17*, 1051–1070.

Crowell, S.E., Beauchaine, T.P., & Linehan, M.M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin*, 135, 495–510.

Dong, Y & Peng, C.Y. (2013). Principled missing data methods for researchers. *Springerplus, 2*(1):222.

Eaton, N. R., Krueger, R. F., Keyes, K. M., Skodol, A. E., Markon, K. E., Grant, B. F., & Hasin, D. S. (2011). Borderline personality disorder co-morbidity: relationship to the internalizing-externalizing structure of common mental disorders. *Psychological Medicine, 41*, 1041-1050. doi:10.1017/S0033291710001662

Ellis, L. K., & Rothbart, M. K. (2001). Revision of the Early Adolescent Temperament Questionnaire. Poster presented at the 2001 Biennial Meeting of the Society for Research in Child Development, Minneapolis, Minnesota.

Ferro, M. A., Boyle, M. H., & Avison, W. R. (2015). Association Between Trajectories of Maternal Depression and Subsequent Psychological Functioning in Youth With and Without Chronic Physical Illness. *Health Psychology*, *34*(8): 820-828.

Field, A. P. (2009). Discovering statistics using SPSS: (and sex and drugs and rock 'n' roll). Los Angele, California.: SAGE Publications.

Fischer, M., Barkley, R. A., Smallish, L, Fletcher, K. (2002). Young adult follow-up of hyperactive children: self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD. *Journal of Abnormal Child Psycholo*gy, 30**:** 463–475. doi:10.1023/A:1019864813776

Fonagy, P., & Luyten, P. (2009). A developmental, mentalization-based approach to the understanding and treatment of borderline personality disorder. Development and Psychopathology, 21, 1355–1381.

Fonagy, P., Speranza, M., Luyten, P., Kaess, M., Hessels, C., & Bohus, M. (2015). ESCAP expert article: Borderline personality disorder in adolescence: An expert research review with implications for clinical practice. *European Child & Adolescent Psychiatry, 24*(11), 1307-1320. doi:http://dx.doi.org/10.1007/s00787 015-0751-z

Fossati, A. (2015). Diagnosing Borderline Personality Disorder During Adolescence: A review of the Published Literature. *Scandinavian Journal of Child and Adolescent Psychiatry and Psychology, 3*(1): 5-21.

Garber, J., & Rao, U. (2014). Depression in Children and Adolescents. In Lewis, M., Rudolph K. D. (Eds.), *Handbook of Developmental Psychopathology* (pp. 489-520). New York: Springer Science+ Business Media.

Glenn, C., & Klonsky, D., (2013). Non-Suicidal Self-Injury Disorder: An Empirical Investigation in Adolescent Psychiatric Patients. *Journal of Clinical Child and Adolescent Psychology*, *42*(4): 496-507.

Goodman, M., Hazlett, E. A., Avedon, J. B., Siever, D. R., Chu, K-W., & New, A. S. (2011). Anterior cingulate volume reduction in adolescents with borderline personality disorder and co-morbid major depression. *Journal of Psychiatric Research*, *45*(6), 803–7. doi:10.1016/j.jpsychires.2010.11.011

Goodman, M., Patel, U., Oakes, A., Matho, A., & Triebwasser, J. (2013). Developmental trajectories to male borderline personality disorder. *Journal of Personality Disorders, 27*(6), 764-782. doi:http://dx.doi.org/10.1521/pedi_2013_27_111

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., et al. (2008). Prevalence, correlates, disability, and comorbidity of *DSM-IV* borderline personality disorder: Results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry*, *69*(4), 533–545.

Greenfield, B., Henry, M., Lis, E., Slatkoff, J., Guile, J.M., Doughtery, G., Zhang, X., Raz, A., Arnold, L.E., Daniel, L., Mishara, B.L., Koenekoop, R., & de Catro, F. (2015). Correlates, stability and predictors of borderline personality disorder among previously suicidal youth. *European Child & Adolescent Psychiatry, 24*(4), 397-406. doi: 10.1007/s00787-014-0589-9

Gunderson, J.G., & Lyons-Ruth, K. (2008). BPD's interpersonal hypersensitivity phenotype: A gene-environment- developmental model. *Journal of Personality Disorders,* 22, 22–41.

Ha, C., Balderas J. C., Zanarini, M. C., Oldham, J., & Sharp, C. (2014). Psychiatric

comorbidity in hospitalized adolescents with borderline personality disorder. *Journal of Clinical Psychiatry, 75*(5) e457-464. doi: 10.4088/JCP.13m08696

Haltigan, J. D., & Vaillancourt, T. (2016). Identifying Trajectories of Borderline Personality Features in Adolescence: Antecedent and Interactive Risk Factors. *The Canadian Journal of Psychiatry, 61*(3), 166-175. doi: 10.1177/0706743715625953

Haltigan, J. D., & Vaillancourt, T. (2016). The borderline personality features scale for children (bpfs-c): Factor structure and measurement invariance across time and sex in a community-based sample. *Journal of Psychopathology and Behavioral Assessment,* doi:http://dx.doi.org/10.1007/s10862-016-9550-1

Hankin, B. L., Jenness, J., Abela, J. R., & Smolen, A. (2011). Interaction of 5-HTTLPR and idiographic stressors predicts prospective depressive symptoms specifically among youth in a multiwave design. *Journal of Clinical Child and Adolescent Psychology, 40*(4), 572-585. doi: 10.1080/15374416.2011.581613

Hosmer, D. W., & Lemeshow, S. (1989). *Applied logistic regression*. New York: Wiley.

Jung, T., & Wickrama, K., A., S. (2008). An Introduction to Latent Class Growth Analysis and Growth Mixture Modeling. *Social and Personality Psychology Compass, 2,* 302-317. doi: 10.1111/j.1751-9004.2007.00054.

Kaess, M., Brunner, R.. & Chanen, A. (2014). Borderline Personality Disorder in Adolescence. *Pediatrics*, *134*(782). doi: 10.1542/peds.2013-3677

Kofler, M. J., Rapport, M. D., Bolden, J., Sarver, D. E., Raiker, J. S., & Alderson, R. M. (2011). Working memory deficits and social problems in children with ADHD. *Journal of Abnormal Child Psychology, 39*(6), 805-817.

Leichsenring, F., Leibing, E., Kruse, J., New, A., & Leweke, F. (2011). Borderline personality disorder. *Lancet, 377*(9759): 74-84.

Lenzenweger, M. F., & Castro, D.D. (2005). Predicting change in borderline personality: Using neurobehavioral systems indicators within an individual growth curve framework. *Development and Psychopathology, 17*(4), 1207-1237. doi: 10.1017/S0954579405050571

Lieb, K., Zanarini, M., Schmahl, C., Linehan, M., & Bohus, M. (2004). Borderline personality disorder. *Lancet*, 364.

Loeber, R., & Keenan, K. (1994). Interactions between conduct disorder and its comorbid conditions: effects of age and gender. *Clinical Psychology Review*, 14:497-523.

Macfie, J., & Strimpfel, J.M. (2014). Parenting and the development of borderline personality disorder. In C. Sharp & J.L. Tackett (Eds.), Handbook of borderline personality disorder in children and adolescents (pp. 277– 291). New York: Springer.

Matthies, S. D., & Philipsen, A. (2014). Common ground in Attention Deficit Hyperactivity Disorder (ADHD) and Borderline Personality Disorder (BPD) – review of recent findings. *Borderline Personality Disorder and Emotion Dysregulation, 1*(3). doi: 10.1186/2051-6673-1-3

Menard, S. (1995). *Applied logistic regression analysis*. Sage university paper series on quantitative applications in the social sciences, 07-106. Thousand Oaks, CA: Sage.

Miller, A. L., Muehlenkamp, J. J., & Jacobson, C. M. (2008). Fact or fiction: Diagnosing borderline personality disorder in adolescents. *Clinical Psychology Review, 28*(6), 969-981. doi:http://dx.doi.org/10.1016/j.cpr.2008.02.004

Muthén, B. & Muthén, L.K. (2000). Integrating person-centered and variable-centered analyses: growth mixture modeling with latent trajectory classes. *Alcoholism, Clinical and Experimental Research, 24*(6), 882-891.

Nagin, D. (2005). *Group-Based Modeling of Development*. Cambridge, MA: Harvard Univ. Press

Nagin, D. S., & Odgers, C. L. (2010). Group-Based Trajectory Modeling in Clinical Research. *The Annual Review of Clinical Psychology, 6,* 109-138. doi:10.1146/annurev.clinpsy.121208.131413

Nakar, O., Brunner, R., Schilling, O., Chanen, A., Fischer, G., Parzer, P., . . . Kaess, M. (2016). Developmental trajectories of self-injurious behavior, suicidal behavior and substance misuse and their association with adolescent borderline personality pathology. *Journal of Affective Disorders, 197*, 231-238.

Nandi, A., Beard, J. R., & Galea, S. (2009). Epidemiologic heterogeneity of common mood and anxiety disorders over the life course in the general population: A systematic review. *BMC Psychiatry, 9* doi:http://dx.doi.org/10.1186/1471-244X 9-31

Newnham, E. A., & Janca, A. (2014). Childhood adversity and borderline personality disorder: A focus on adolescence. *Current Opinion in Psychiatry, 27*(1), 68-72.

Newton-Howes, G., Clark, L. A., & Chanen, A. M. (2015). Personality Disorder across the life course. The Lancet, 385(9969), 727–734

Oltmanns, T.F., & Balsis, S. (2011). Personality disorders in later life: Questions about the measurement, course, and impact of disorders. *Annual Review of Clinical Psychology, 7*, 321-349.

Paris, J. (2014). A history of research on borderline personality disorder in childhood and adolescence. *Handbook of borderline personality disorder in children and adolescents.* (pp. 9-16) Springer Science + Business Media, New York, NY. doi:http://dx.doi.org/10.1007/978-1-4939-0591-1_2

Paris, J. (2005). The development of impulsivity and suicidality in borderline personality disorder. Development and Psychopathology, 17, 1091–1104.

Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, *25,* 111-164.

Reuter, T., Sharp, C., Temple, J., & Babcock, J. (2015). The relation between borderline personality disorder and teen dating violence. *Psychology of Violence, 5*(2), 163-173.

Reynolds, C. R., & Kamphaus, R. W. (2004). *Behaviour assessment system for children second edition manual.* Minneapolis: Pearson.

Robins, E., & Guze S. B. (1970). Establishment of diagnostic validity in psychiatric illnesss: its application to schizophrenia*. The American Journal of Psychiatry, 126*(7): 983-987. doi: 11.1176/ajp/126.7.983

Sauer, S. E., & Baer, R. A. (2010). Validation of measures of biosocial precursors to borderline personality disorder: Childhood emotional vulnerability and environmental invalidation. *Assessment, 17*(4), 454-466.

Sharp, C., & Fonagy, P. (2015). Practitioner Review: Borderline personality disorder in adolescence – recent conceptualization, intervention, and implications for clinical practice. *Journal of Child Psychology and Psychiatry*, *56*(12), 1266-1288. doi:10.1111/jcpp.12449

Sharp, C., Green, K. L., Yaroslavsky, I., Venta, A., Zanarini, M. C., & Pettit, J. (2012). Incremental validity of borderline personality disorder relative to major depressive disorder for suicidal ideation and deliberate self-harm in adolescents. *Journal of Personality Disorders, 26*(6), 927-938. doi:http://dx.doi.org/10.1521/pedi.2012.26.6.927

Sharp, C., Mosko, O., Chang, B., & Ha, C. (2011). The cross-informant concordance and concurrent validity of the Borderline Personality Features Scale for Children in a community ample of boys. *Clinical Child Psychology and Psychiatry, 16*(3), 335-

349. doi: 10.1177/1359104510388279

Sharp, C., Wright, A.G., Fowler, J.C., Frueh, B.C., Allen, J.G., Oldham, J., & Clark, L.A. (2015). The structure of personality pathology: Both general ('g') and specific ('s') factors? *Journal of Abnormal Psychology*, *124*, 387–398.

Skodol, A. E. (2015). Personality disorders: a burden in the community, neglected in the clinic? *The Journal of Clinical Psychiatry, 76*(11), 1482-1484. doi: 10.4088/JCP.14com09597.

Stepp, S. D., Keenan, K., Hipwell, A. E., & Krueger, R. F. (2014). The impact of childhood temperament on the development of borderline personality disorder symptoms over the course of adolescence. *Borderline Personality Disorder and Emotion Dysregulation*, *1*, 18. http://doi.org/10.1186/2051-6673-1-18

Stepp, S. D., Pilkonis, P. A., Hipwell, A. E., Loeber, R., & Stouthamer- Loeber, M. (2010). Stability of borderline personality disorder features in girls. *Journal of Personality Disorders, 24*, 460-472.

Stone M. H. (1980). The borderline syndromes: constitution, personality, and adaptation. McGraw-Hill: New York.

Storebø O. J., & Simonsen, E. (2014). Is ADHD an early stage in the development of borderline personality disorder? *Nordic Journal of Psychiatry*, *68*(5), 289-295. doi: 10.3109/08039488.2013.841992

Studenmund, A. H., & Cassidy, H. J. (1987). *Using econometrics: a practical guide*. Boston: Little Brown.

The Mental Health Strategy for Canada: A Youth Perspective. (2015). Mental Health Comission of Canada. Retrieved from: http://www.mentalhealthcomission.ca/sites/default/files/2015-03-1617-MHCC-YouthStrategyReport-E-FINAL_0.pdf

Tyrer, P. (2007). Personality diatheses: A superior explanation than disorder. *Psychological Medicine*, 37, 1521–1525.

Vaillancourt, T., Brittain, H. L., McDougall, P., & Duku, E. (2013). Longitudinal links between childhood peer victimization, internalizing and externalizing problems, and academic functioning: Developmental cascades. *Journal of Abnormal Child Psychology, 41*(8), 1203-1215. doi:http://dx.doi.org/10.1007/s10802-013-9781-5

Vaillancourt, T., Brittain, H. L., McDougall, P., Krygsman, A., Boylan, K., Duku, E., & Hymel, S. (2014). Predicting borderline personality disorder symptoms in adolescents from childhood physical and relational aggression, depression, and

attention-deficit/hyperactivity disorder. *Development and Psychopathology, 26*(3), 817-830. doi:http://dx.doi.org/10.1017/S0954579414000418

Winsper, C., Zanarini, M., & Wolke, D. (2012). Prospective study of family adversity and maladaptive parenting in childhood and borderline personality disorder symptoms in a nonclinical population at 11 years. *Psychological Medicine,* 42, 2405–2420.

Yoshimatsu, K., & Palmer, B. (2014). Depression in Patients with Borderline Personality Disorder. *Harvard Review of Psychiatry, 22*(5). doi: 10.1097/HRP.0000000000000045

Zanarini, M. C., Frankenburg, F. R., Khera, G. S., & Bleichmar, J. (2001). Treatment histories of borderline inpatients. *Comprehensive Psychiatry. 42*(2): 144-150.

Zanarini, M. C., & Wedig, M. (2014). Childhood adversity and the development of borderline personality disorder. In C. Sharp & J.L. Tackett (Eds.), Handbook of borderline personality disorder in children and adolescents (pp. 265–276). New York: Springer.

Zelkowitz, P., Paris, J., Guzder, J., & Feldman, K. (2001). Diathesis and stressors in borderline pathology of child- hood: The role of neuropsychological risk and trauma. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 100–105.

Zimmerman, D. J., & Choi-Kain, L. W. (2009). The hypothalamic-pituitary- adrenal axis in borderline personality disorder: a review. *Harvard Review of Psychiatry,* 17, 167–83.

Figures and Tables

Table 1- DSM-5 criteria for a BPD diagnosis

Symptoms
Efforts to avoid abandonment
Unstable/Intense Relationships
Identity Disturbance
Impulsivity
Recurrent suicidal or self-mutilating behaviour
Affective Instability
Chronic feelings of emptiness
Inappropriate or poorly controlled anger
Stress-related paranoid ideation or dissociative symptoms

Table 2- Missing data distribution of BPFS-C scores: sample available at each time point (N=505)

Data Collection Time Point	Sample Available for Analysis (N)	Sample Available for Analysis (%)
4	423	84
5	419	83
6	392	78
7	375	74

Table 3- Missing data distribution: number of data points per subject and characteristics

Number of Data Points	Sample Available for Analysis (%)	Characteristics				
		Age	% Girls & % Boys	Depression	ADHD	Family Functioning
$4 \geq x \geq 1$	505 (72)	11 (.36)	56 & 44	3.8 (4.9)	5.9 (4.0)	22 (2.2)
0	198 (28)	11 (.39)	41 & 59	2.7 (3.6)	7.0 (6.2)	21 (2.5)

x denotes the number of data points. The values listed under the characteristics columns are reported as means (standard deviations) of raw scores unless otherwise noted.

Table 4- Characteristics of the study sample at time 4

Characteristic	T4 (*N*= 505)
Adolescent Characteristics	
Age (Years)	14 (.36)
Sex (%)	
Female	58
Male	42
Ethnicity	
European-Canadian	71
Middle-Eastern Canadian	1.9
African/West-Indian Canadian	3.3
Asian Canadian	1.7
South-Asian Canadian	3.0
Native- Canadian	1.6
South/Latin American Canadian	1.3
Other	3.5
I don't know	13
BPD	30 (14)
Depression	3.9 (4.9)
ADHD	5.8 (4.0)
Reporting Parent Characteristics	
Relationship to Child (%)	
Biological Mom	86
Biological Dad	10
Other	4.0
Age (%)	
25-30	2.5
31-35	12
36-40	27
> 40	58
Marital Status (%)	
Married or Common Law	82
Single	5.8
Legally Separated	5.5
Divorced	5.8
Widowed	1.4
Employment Status (%)	
Full Time	61
Part Time	19
Unemployed	2.6
Retired	1.1
Student	1.6
Homemaker	12
Other	3.3
Education (%)	
≤ High School	26
College Diploma or Trades Certificate	40
University Undergraduate Degree	25
University Graduate Degree	9.8
Household Income (%)	
< $20, 000	4.7
$20, 000 - $49, 999	21
$50, 000 - $69, 999	15
≥ $70, 000	59

Values represent mean (standard deviation) unless stated otherwise. Frequencies may not sum to 100% due to rounding.

Table 5- Top four model selection results (N= 505)

Number of Groups	Polynomial Order	BIC	Trajectory Group	AvePP	OCC	Bayes Factor ($e^{BIC_1 - BIC_2}$)
3	0, 1, 2	-6269.14	1 2 3	.92 .84 .90	20 7.6 30	1.1
3	0, 1, 1	-6269.19	1 2 3	.91 .84 .89	18 8.5 26	9.0×10^{-9}
4	0, 1, 2, 0	-6250.69	1 2 3 4	.91 .83 .89 .93	20 8.0 23 651	.24
4	0, 1, 1, 0	-6249.28	1 2 3 4	.91 .83 .89 .93	20 8.0 23 651	

Table 6- Parameter estimates for the group-based trajectory model of BPD features

Group	% of Sample	Posterior Probability	OCC	Parameter	Beta (95% CI)	BPFS-C Scores			
						T4	T5	T6	T7
1	33.9	.91	20	Intercept	18 (16, 20)	18 (7.3)	16 (6.5)	18 (7.6)	17 (7.0)
2	38	.83	8.0	Intercept Linear	31 (29, 33) .97 (-.98, 2.9)	31 (10)	32 (7.7)	33 (8.5)	33 (9.2)
3	26.6	.89	23	Intercept Linear	45 (43, 47) 1.3 (-.65, 3.2)	46 (9.2)	47 (9.6)	49 (9.7)	49 (9.4)
4	1.5	.93	651	Intercept	66 (64, 68)	66 (8.3)	71 (4.0)	63 (7.3)	65 (7.1)

BPFS-C scores reported are means (standard deviation). The scores were compared across trajectory groups at each measurement occasion.

Table 7- Relative proportion of boys and girls by trajectory group

Trajectory Group (N)	Boys (N)	Girls (N)
1 (172)	93	79
2 (194)	81	113
3 (132)	38	94
4 (7)	1	6

Table 8- Summary of missing data for covariates of interest

Characteristic	Cases Included	% of Sample Included	Cases Excluded	% of Sample Excluded
Adolescent				
Age	505	100	0	0
Gender	505	100	0	0
Depressive Symptoms at T3	440	87	65	13
ADHD Symptoms at T3	445	88	60	12
Family Functioning	274	54	231	46
Reporting Parent				
Age	467	93	38	7
Marital Status	469	93	36	7
Education	469	93	36	7
Employment Status	470	93	35	7
Household Income	452	90	53	10

Table 9- Comparison of sample characteristics across BPD trajectory groups

Characteristic	Group 1	Group 2	Group 3	Group 4	F/χ^2
Adolescent					
Female, %	**46****	**58****	**71****	**86****	**22**
Depressive Symptoms at T3	**1.9 (2.7)****	**3.4 (4.4)****	**6.6 (6.3)****	**11 (6.5)****	**27**
ADHD Symptoms at T3	**4.3 (2.8)****	**5.9 (3.8)****	**7.0 (4.2)****	**10 (3.9)****	**24**
Depressive x ADHD Symptoms	**9.6 (20)****	**25 (47)****	**51 (69)****	**99 (71)****	**20**
Family Functioning	21 (2.3)	21 (2.2)	21 (2.5)	21 (2.5)	.69
Reporting Parent					
Age Range, years	**36-40 (.71)***	**36-40 (.73)***	**36-40 (.90)***	**36-40 (1.2)***	**3.5**
Married	**1.4 (1.0)****	**1.7 (1.3)****	**1.7 (1.2)****	**2.1 (1.6)****	**3.1**
Postsecondary Education	**3.4 (.98)****	**3.4 (1.0)****	**3.0 (.97)****	**2.4 (.98)****	**6.7**
Full Time Employment	2.2 (1.9)	1.9 (1.7)	2.2 (2.0)	2.7 (2.1)	1.6
Income	**6.8 (2.0)****	**6.3 (2.2)****	**5.9 (2.4)****	**3.5 (2.9)****	**7.6**

Note: Characteristics were measured at baseline when adolescents were 12 or 13 years of age. Values denote mean and (standard deviation) unless specified otherwise. Significant associations are shown in bold. * indicates significance at < .05 and ** indicates significance at < .01.

Table 10- Predictors of adolescent's BPD trajectory groups

Outcome	Group 2				Group 3				Group 4			
	B (SE)	**e^b**	***P***	**95% CI**	**B (SE)**	**e^b**	***P***	**95% CI**	**B (SE)**	**e^b**	***P***	**95% CI**
Gender	**-.83 (.24)**	**.44**	**.001**	**.27-.70**	**-1.1 (.31)**	**.32**	**<.001**	**.17-.59**	**-21(0)**	**5.5x 10^{-10}**	**>.001**	**5.5 x10^{-10} - 5.5x10^{-10}**
Depressive Symptoms	.09 (.07)	1.1	.23	.95-1.3	**.25 (.08)**	**1.3**	**.001**	**1.1-1.5**	**.44 (.22)**	**1.6**	**.05**	**1.0-2.4**
ADHD Symptoms	**.12 (.05)**	**1.1**	**.02**	**1.0-1.2**	**.24 (.06)**	**1.3**	**<.001**	**1.1-1.4**	**.64 (.26)**	**1.9**	**.01**	**1.1-3.1**
Marital Status	.21 (.11)	1.2	.07	.99-1.5	.11 (.14)	1.1	.43	.85-1.5	.55 (.51)	1.7	.28	.64-4.7
Depressive x ADHD Symptoms	.001 (.01)	1.0	.91	.98-1.0	-.007 (.01)	.99	.51	.97-1.0	-.02 (.02)	.98	.34	.04-1.0
Education Level	.13 (.13)	1.1	.34	.88-1.5	-.17 (.17)	.84	.30	.61-1.2	-.77 (.80)	.47	.34	.10-2.2
Parent's Age	-.22 (.18)	.98	.90	.69-1.4	-.27 (.20)	.76	.17	.51-1.1	.19 (.89)	1.2	.82	.22-6.7
Household Income	-.007 (.18)	.98	.92	.87-1.1	-.03 (.08)	.97	.75	.83-1.2	.03 (.31)	1.0	.93	.56-1.9

Predictors were identified using multinomial logistic regression. Significant predictors of group assignment are bold. Group 1 (non-significant features) is the reference group. Note: R^2 =.23 (Cox & Snell), .31 (Nagelkerke).
Model: X^2 (24) = 127, p = < .001.

Table 11- Correlations between significant variables and BPFS-C scores

Characteristic	BPFS-C Scores			
	T4	**T5**	**T6**	**T7**
Gender	.15**	.23**	.25**	.28**
Depressive Symptoms at T3	.42**	.39**	.31**	.31**
ADHD Symptoms at T3	.38**	.35**	.27**	.27**

** denotes Pearson correlations significant at the .01 level using a 2-tailed test.

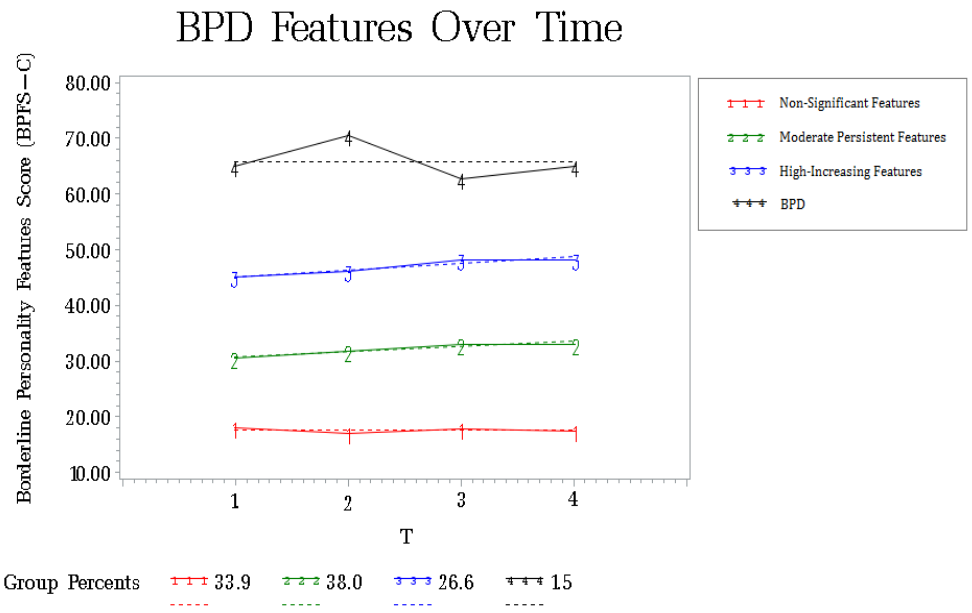

Figure 1: Trajectories of adolescent's BPFS-C scores across four waves of prospective data. Solid lines depict the four observed trajectories and dashed lines predicted trajectories.

Appendices

Appendix A

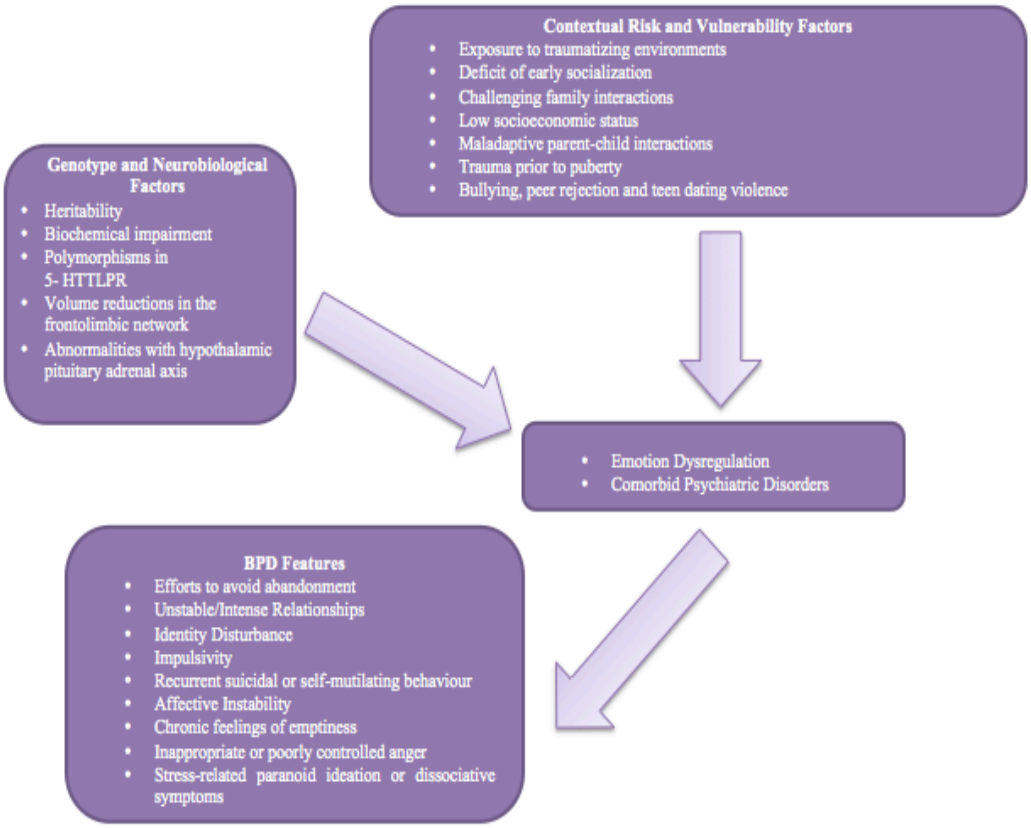

Figure 2: Theoretical framework for the development of adolescent BPD adapted from Sharp and Fonagy 2015 review

Appendix B

1. I'm a pretty happy person.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

2. I feel very lonely.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

3. I get upset when my parents or friends leave town for a few days.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

4. I do things that other people consider wild or out of control.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

5. I feel pretty much the same way all the time. My feelings don't change very often.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

6. I want to let some people know how much they've hurt me.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

7. I do things without thinking.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

8. My feelings are very strong. For instance, when I get mad, I get really really mad. When I get happy, I get really really happy.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

9. I feel that there is something important missing about me, but I don't know what it is.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

10. I've picked friends who have treated me badly.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

11. I'm careless with things that are important to me.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

12. I change my mind almost every day about what I should do when I grow up.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

13. People who were close to me have let me down.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

14. I go back and forth between different feelings, like being mad or sad or happy.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

15. I get into trouble because I do things without thinking.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

16. I worry that people I care about will leave and not come back.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

17. When I'm mad, I can't control what I do.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

18. How I feel about myself changes a lot.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

19. When I get upset, I do things that aren't good for me.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

20. Lots of times, my friends and I are really mean to each other.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

21. I get so mad I can't let all my anger out.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

22. I get bored very easily.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

23. I take good care of things that are mine.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

24. Once someone is my friend, we stay friends.

Not at All True	Hardly Ever True	Sometimes True	Often True	Always True
○	○	○	○	○

Appendix C

Alternative Statistical Approaches

There are several statistical methods that could be implemented when studying the course of development for BPD symptomatology across time. In general, the different approaches can be described as either using a variable-centered approach or a person-centered approach. Variable-centered approaches include regression, growth curve or factor analysis. When using these methods the main focus has been understanding the average pattern of growth of a variable as distributed across the population of interest, and generally assumes homogeneity of the variable of interest (ie. no empirical subpopulations or levels within the variable). Any heterogeneity is modeled by testing the impact of other covariates on the variable of interest. (Muthén & Muthén, 2000). Recently, more person-centered approaches such as growth mixture models, and latent class analysis are being included. A person-centered approach is one that studies individual clusters of characteristics (Nagin & Odgers, 2010). The focus when using these methods is describing the relationships among individuals. In group-based trajectory modeling, a type of latent class analysis, the trajectory group is the unit of analysis (Nagin, 2005). In this section a brief description will be provided of the aforementioned person-centered approaches contrasting them with GBTM, adopted for this research study.

Essentially, all three models: growth mixture, growth curve, and latent curve analysis at their core are similar. They all can be used to illustrate the course of an outcome over age or time (Nagin & Odgers, 2010). However, they have subtle differences

each contributing something different to the interpretation of results. Growth mixture modeling (GMM) relates an observed outcome variable to a time or a time related variable. It assumes that all individuals in the sample come from two or more subpopulations and follow a particular pattern of development (Nagin, 2005; Muthén & Muthén, 2000).

Latent curve analysis (LCA) is used when describing the probabilities of a set of observed categorical variables across groups of individuals where group membership is not observed (Muthén & Muthén, 2000). Population distribution of trajectories using LCA varies continuously across individuals and in a fashion that can be explained using population parameters consistent with a normal distribution. GBTM, like other person centered approaches does not make these assumptions about the population. Instead, it assumes that the population is composed of distinct groups each characterized by traits exclusive to the resultant trajectory group they belong to (Nagin & Odgers, 2010). Under these assumptions GBTM has the capacity to identify qualitatively distinct developmental progressions that are not identifiable using ad hoc classification rules and that are not uniform and exclusive to one particular pattern of development (Nagin, 2005).

In summary, GBTM methods are often used to compliment theories that predict differing developmental trajectories within the population. They are also very suitable when the object of the study is not represented best around a population mean (Nagin, 2005). Disorders imply a different group of individuals who vary across multiple indicators of problematic symptoms as well as course of disorder (Robins & Guze, 1970), but there is a threshold that distinguishes a group as disordered. This method seems to be

the most suitable when attempting to answer the research questions framed in terms of identification of trajectory groups and the factors distinguishing group membership over time.

Appendix D

$$\mathrm{OCC}_j = \frac{\mathrm{AvePP}_j \,/\, (1 - \mathrm{AvePP}_j)}{\widehat{\pi}_j \,/\, (1 - \widehat{\pi}_j)}.$$

Appendix E

Assumption IV: Multicollinearity

Multicollinearity is an issue as it influences the *b* values obtained. As collinearity increases so do the standard errors of the *b* coefficients. If the largest VIF is greater than 10 then there is cause for concern. If the average VIF is greater then 1 then the regression may be biased (Bowerman & O'Connell, 1990). Tolerance values below 0.1 indicate serious problems and tolerance values below 0.2 indicate a potential problem (Menard, 1995). For this current model, the VIF values are below 10. In conjunction with the tolerance values that are well above 0.2, provides confidence that results are not biased by collinearity.

Table 12: Multicollinearity results

Characteristic	Variance Inflation Factor (VIF)	Tolerance
Gender	1.0	.98
Depression	1.2	.87
ADHD	1.1	.91
Interaction	1.6	.65
Parents Age	1.1	.98
Marital Status	1.3	.79
Education	1.2	.84
Employment	1.1	.94
Income	1.5	.66

Assumption V: Linearity

To test linearity before running a multinomial logistic regression it has been suggested a logistic regression be run including the continuous predictors of interest and the logit of the outcome variable. The assumption is tested by investigating the significance between the interaction term and the log transformation (Hosmer & Lemeshow, 1989). None of the associations between the interaction terms and the log

transformation were significant, indicating that the assumption of linearity has been sustained.

Assumption VI: Outliers

Outliers were investigated by calculating Cook's distance. Cook's distance is a measure of the overall influence of a case on the regression model. Values greater than 1, are considered to bias the results from the model (Field, 2009). For this model, Cook's distance was $M = .006$ ($SD = .01$), with values ranging from .000 - .06. Thus, all values were substantially smaller than 1, indicating the outliers that do exist in this sample do not warrant exclusion. To be confident that these outliers do not bias the results, trimmed means versus calculated means were consulted. Trimmed means for each variable in the regression model would illustrate the average based on distribution of scores that have been removed from each extreme of the distribution (Field, 2009). Since the values of the trimmed means are not vastly different than the actual means that include values of the outliers, all data points were kept in the analysis.

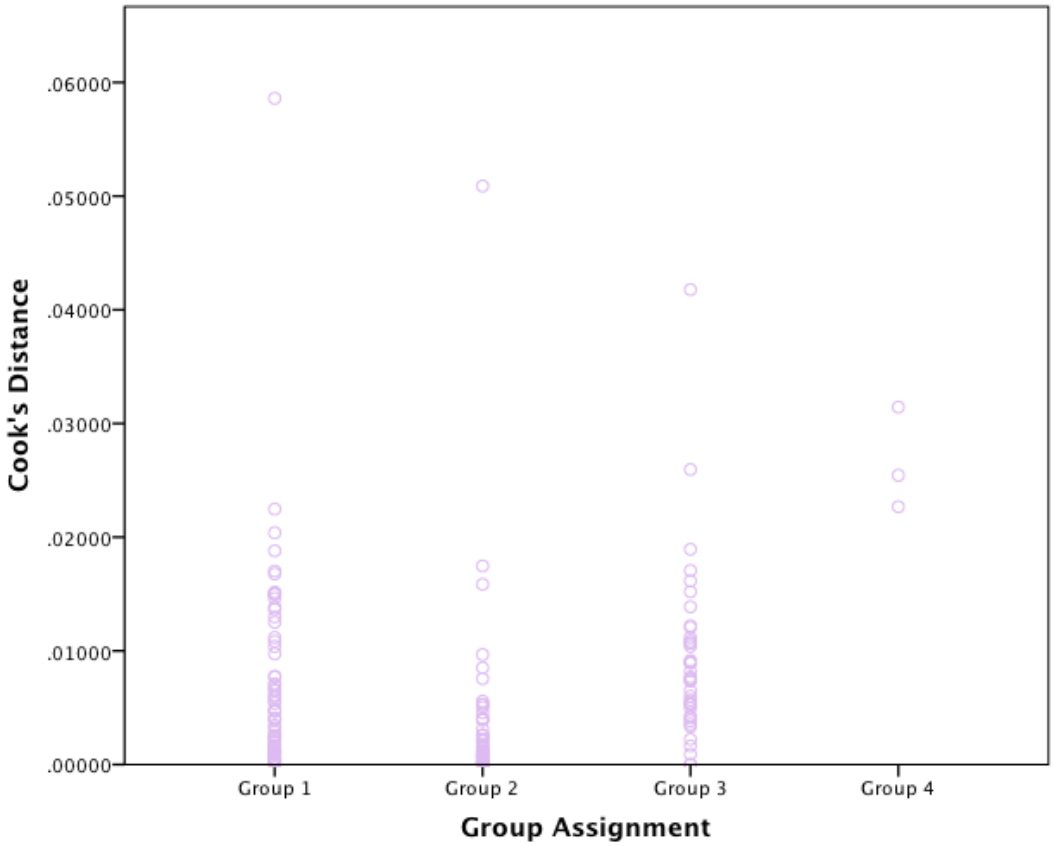

Figure 3: Depiction of the outliers in the analytical sample categorized by trajectory group assignment.

Table 13: Actual mean value versus trimmed mean values for the variables included in the multinomial logistic regression model for each trajectory group.

Characteristic	Group							
	1		2		3		4	
	Mean	Trimmed Mean	Mean	Trimmed Mean	Mean	Trimmed Mean	Mean	Trimmed Mean
Gender	.46	.45	.58	.59	.71	.74	.86	.89
Depression	1.9	1.5	3.4	2.8	6.6	6.0	11	11
ADHD	4.3	4.1	5.9	5.7	7.0	6.8	10	9.9
Interaction	9.6	6.6	25	18	51	41	99	96
Parents Age	4.5	4.6	4.5	4.6	4.3	4.3	4.1	4.2
Marital Status	1.4	1.2	1.7	1.6	1.7	1.5	2.1	2.1
Education	3.4	3.4	3.4	3.4	3.0	3.0	2.4	2.4
Income	6.8	7.0	6.3	6.5	5.9	6.0	3.5	3.4