# A Taxonomy of Antisocial Behaviors:

# the subtypes and their associated features

LAW, Yuen Wah Sonya

A Thesis Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

in

Clinical Psychology

The Chinese University of Hong Kong

April 2011





ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 - 1346

Thesis / Assessment Committee

Professor Helene Hoi-lam Fung (Chair)

Professor Patrick Wing-leung Leung (Thesis Supervisor)

Professor Winne Wing-sze Mak (Committee Member)

Professor Alma May-lan Au (External Examiner)

# Abstract of thesis entitled:

**BACKGROUND**: Adolescent antisocial behaviors are versatile in terms of their onset, severity, pervasiveness, continuity, and developmental outcomes. A substantial body of literature on developmental pathway of antisocial behaviors indicates that meaningful subtypes exist within these heterogeneous antisocial behaviors, rendering important implications to their etiology, causal mechanism and intervention. This study tests a taxonomy of antisocial behavior by examining whether different offending groups can be distinguished by their different group features including background risks and external correlates. First, two broad offending groups, i.e., the early-onset group and the adolescent-onset group were identified in a clinical sample of 118 adjudicated male adolescents based on age of onset of symptoms of Conduct Disorder. Further, two distinct subtypes, i.e. antisocial behavior associated with symptoms of Attention Deficit Hyperactivity Disorder (ADHD) and antisocial behavior associated with callous-unemotional traits (CU traits), a defining feature of psychopathy, were hypothesized to coexist within the broad early-onset offending group, based on two lines of recent studies indicating ADHD and CU traits as important correlates of antisocial behaviors. These two subgroups were identified within the sample in this current study.

**METHOD**: Data were collected from 118 adjudicated male adolescents from a

centralized probation facility in Hong Kong and 63 non-delinquent male control subjects from mainstream secondary schools, all aged between 12 and 17. Group comparisons and multinominal logistic regression were performed to test whether these offending groups could be distinguished by different background risks and deficits including variables pertaining to cognitive processes, family, parenting, and deviant peers, etc.

**RESULTS**: The early-onset offending group could be differentiated from the adolescent-onset offending group by their association with adolescent adjustment difficulties, more background risks, ADHD diagnosis, and callous unemotional traits. The two early-onset subgroups, early-onset ADHD and early-onset CU traits group, shared similarities of having severer delinquency and poorer adolescent adjustment, but demonstrated differences in terms of disinhibitory processes.

**CONCLUSION**: Different offending groups could be discerned by their distinctive associated group risks and deficits, giving evidence to different developmental pathways to antisocial behaviors. Implications to understanding and intervention of antisocial behaviors were discussed.

Submitted by LAW Yuen Wah, Sonya
for the degree of Doctor of Philosophy in Clinical Psychology
at The Chinese University of Hong Kong in April 2011

# 摘要

**背景**：青少年的違規和反社會行為（antisocial behaviors）十分多樣化；根據有關青少年反社會行為的發展的研究，這些多樣化的行為可根據其發展型態作出識別和分類；這些分類有助於我們了解青少年違規和反社會行為的成因、發展機制、處理和治療。本研究旨在就青少年違規和反社會行為提出一個系統分類，並比較不同的犯事類型的特有背景因素及特徵。本研究首先從一百一十八位在囚青少年中，根據他們品行疾患(Conduct Disorder)的病徵的最早發病年齡，識別出兩種犯事行為的類型:包括(一)兒童期犯事類型及(二)青少年期犯事類型；再根據有關研究，進一步在兒童期犯事類型中，識別兩個組別，其中一個犯事組別有專注力不足過動症的特徵（Attention Deficit Hyperactivity Disorder），另一組有情感冷漠的性格特徵(callous-unemotional traits)。

**方法**：本研究的資料從一百一十八位十二至十七歲的香港在囚青少年及六十三位就讀於一般中學的青少年（對照組）中搜集；然後就不同的犯事類型，作出組別比較及多元回歸分析，以測試這些犯事類型的獨有背景危機包括認知能力、家庭、管教、同儕影響和其他相關特徵。

**結果**：兒童期犯事類型有別於青少年期犯事類型；他們有青少年適應

困難、較多的背景危機、患專注力不足過動症的比例較多、及有較高情感冷漠的性格特徵。另外，在兒童期犯事類型中，具專注力不足過動症病徵的組別和具情感冷漠性格的組別二者都有明顯的青少年適應困難和嚴重的犯事行為；他們雖同屬於兒童期犯事類型，但卻有不同的背景危機因素和組別特徵。

**結論**：根據分析結果，不同的犯事行為類型顯示特有的背景危機因素和特徵，反映出違規及反社會行為的不同的發展機制；本文將就本研究的結果探討反社會行為的發展理論及處理。

# Acknowledgement

I wish to express my gratitude to Professor Patrick Leung, my thesis supervisor. Prof. Leung has given me timely guidance throughout my long years of study. Importantly, he has shown me what a "scientist" is. I can see his interest in knowledge, his seriousness in handling data and his non-dogmatic attitude in scientific inquiry, which I still need to learn. His spirit inadvertently upkeeps my interest and standard in the research process.

I must also thank the personnel in the Social Welfare Department, including Mr. Lau Kar Cho, Chief Clinical Psychologist, who supported my proposal in collecting data in Tuen Mun Children and Juvenile Home, Mr. Cheung Hing Wah, now Deputy Director, who approved the data collection, and all my social work colleagues in Tuen Mun Children and Juvenile Home, who helped out in the data collection. I am thankful to the personnel of Lee Kau Yan Memorial School and Fukieu Secondary School, especially Mr. Jonathan Lai Ping Wah (Principal of Lee Kau Yan Memorial School) and Mr. Cheung Man Fung (teacher of Lee Kau Yan Memorial School), Mr. Wong Kwan Yu (Principal of Fukien Secondary School) and Miss Lee Shui Fong (teacher of Fukien Secondary School), who had supported my data collection in community setting.

I am grateful to all the adolescents and their parents who participated in my research. I would like to contribute what I have learnt from them to many other adolescents and parents who strive for a good development and a good life. I wish to thank also my clinical psychologist colleagues, particularly, Dr. Samuel Lo, who provided timely help in response to many of my questions regarding statistics that urgently popped up, Dr. Maggie Poon, who proofread a significant part of my thesis, and Miss Delphine Yau, who gave me warm support during the period I intensely did the writing up. I am also fortunate to have the long-lasting encouragement from my women friends (R, T, L and Miu).

Last but not the least; I am grateful to Elvin, my husband, who took good care of our twin sons, Yat Hou and Yat Lok, during my absence from home in many weekends and never made grumble to me. Like Frodo, heading his way to Mount Doom (*Lord of the Ring*), doubtful about how one can accomplish the task, I also wondered, for many times, how my task could be done. With all the help mentioned above, I am glad that I could climb up to Mount Doom, not to destroy the evil ring, but to have derived some understanding about our human world. The task has been done yet this is not the end of my journey.

# Table of Contents

# List of tables

# Chapter I

# Introduction

## Definition of terms

The focus of this study is on juvenile delinquency, and broadly speaking, antisocial behaviours of adolescents. Juvenile refers to adolescents under age of 18. Delinquency is a legal term, referring to adolescents who commit unlawful behaviours that do harm to others and violate societal rules (Johnson, McCaskill IV, & Werba, 2001). Delinquency could vary from minor law-breaking behaviour (such as not paying traffic fee) to serious crime (such as homicide). The legal consequences of delinquency involve prosecution or finally conviction and sentencing. Juvenile crime constitutes a significant portion of total crime (Johnson, McCaskill IV, & Werba, 2001; Lahey, 2008). The most common offences committed by adolescents are theft-related offences such as shoplifting, violent offences such as assault, status violation offences which include curfew violations, running away from home, and offences involving drug use (Rutter, Giller & Hagell, 1998a; OJJDP, Department of Justice, U.S., 2009 ). In local context, the number of youth (age 10-20) arrested is 9008 in 2008, making up about 21% of the total number of persons arrested, mostly

for theft-related offence (32.9%), violent offences (15.2%), and drug-related offences (12.8%) (HK Police Force, 2009). Antisocial behaviours also refer to acts that involve violating societal norms or basic right of others (Vermeiren, 2003; Hiatt & Dishion; 2008). But the term broadly encompasses both criminal and non-criminal acts (such as irresponsibility, lying) which do not necessarily enter the realm of law or result in any legal consequences. Children and adolescents who commit illegal acts might not be brought up to the attention of legal system or end up in conviction for a variety of reasons. For example, their acts might go undetected; and those who commit illegal acts but are below the age of criminal responsibility will not be prosecuted (the age of criminal responsibility varies worldwide from 6 to 18 and in Hong Kong, it is 10 years). But it is important not to miss out antisocial acts committed by adolescents in a broader sense if we are to understand the development of delinquency (Rutter, Giller & Hagell, 1998b).

From the point of view of psychopathology, the concept of delinquency overlaps with two diagnoses, classified as disruptive behaviour disorders of antisocial in nature in the Diagnostic and Statistical Manual of Mental Disorder (DSM-IV-TR, APA, 2000), namely conduct disorder (CD) and Oppositional Defiant Disorder (ODD). These disorders are characterized by a persistent pattern of behaviours that violate the

rights of others and age-appropriate societal norms. The diagnosis of CD involves within 12 months, the manifestation of at least 3 from a list of 15 antisocial behaviours grouped under four areas including (1) aggression to people and animals (e.g. often bullies others, initiates physical fights, has forced someone into sexual activity), (2) destruction of property (e.g deliberate fire setting), (3) deceitfulness or theft (broken into someone else's house, building or car, shoplifting, often lies to obtain goods or favors or avoid obligations) and (4) serious violations of rules (e.g. often stays out at night despite parental prohibition, often truant from school) (DSM-IV-TR, APA, 2000). The manifestation of symptoms causes significant impairment in daily functioning of children and adolescents and are often seen as unmanageable by significant others. ODD is characterized by a pattern of negativistic, hostile, and defiant behaviour displayed by children or adolescence that causes significant impairment in daily functioning (DSM-IV-TR, APA, 2000). The diagnosis is made when there is recurrent engagement in at least four disruptive behaviours in a list of eight symptoms such as arguing with adults, deliberately annoying people, defying adults' request or rules, and often being spiteful and revengeful, often loosing temper, etc., within six months. ODD symptoms often bring severe impairment of social relationship for children and adolescent. ODD is closely linked to CD and is often conceptualized as a precursor to CD (Lahey, 2008;

Nock, Kazdin, Hiripi, & Kessler, 2007; Rowe, Maughan, Pickles, Costello, & Angold, 2002). Many adolescents with CD diagnosis also meet diagnosis of ODD. In a nationally representative sample of 10438 5-15-year olds children in UK, 56% of girl and 62% of boys who met DSM CD diagnosis also met the diagnosis of ODD (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). In the DSM system, there is a hierarchical organization between CD and ODD diagnosis, such that ODD is not diagnosed in the presence of CD. Prevalence of CD in community samples ranges from 1.8% to 16% for boys and 0.8 % to 9.2% for girls, with gender difference remaining stable across ages (Loeber, Burke, Lahey, Winters, & Zera, 2000; Loeber, Burke, & Pardini, 2009). The prevalence of ODD ranges from 2.6 % to 15.6% (Boylan, Vaillancourt, Boyle, Szatmari, 2007; Loeber, et al., 2009). Sex difference in prevalence of ODD appears to be different by age; with higher prevalence in boys in childhood but no gender difference in adolescence (Boylan et al., 2007).

Conceptually the overlap between delinquency and CD is obvious, for various CD symptoms are delinquent acts which are unlawful, such as shoplifting, forcing someone into sexual activity, stolen while confronting a victim (like armed robbery), fire setting and destruction of property (vandalism), etc. However, some of the diagnostic criteria of CD do not necessarily infringe on the law, such as lying to

obtain goods or avoid obligations. The characteristic symptoms of ODD do not necessarily infringe on law, but for children and adolescents, recurrent violation of adults' or parental rules and expectation through defiance and disobedience also falls into the realm of antisocial behaviours (Hiatt & Dishion, 2008).

Empirical studies with major focus on juvenile delinquency found that many delinquent adolescents meet the criteria for the diagnosis of CD or ODD. Vermeiren (2003) reviewed 15 studies published from 1984 to 2003. The percentage of male adolescent offenders with CD diagnosis ranged from 53% to 100% as found by most studies. Amongst the 15 studies, only two of them found a percentage of the delinquents having the diagnosis of CD lower than 40% (but higher than 30%). Also amongst these 15 studies reviewed, five reported on the percentage of diagnosis of ODD in their samples, which ranged from 9% to 55%. Another more recent review (Fazel, Doll, & Langstrom, 2008) that included 25 studies deriving clinical diagnoses for samples based on clinical examination or clinical interview using structured diagnostic instruments in juvenile detention and correctional setting published between 1996 to 2006 found that an average of 52.8% (40.9% - 64.7%) of adolescent male offenders (n = 13,778; age range: 10 – 19, mean age: 15.6 years) had diagnosis of CD. Recent studies similarly found that CD and ODD were prevalent in

adolescents who displayed delinquent behaviours (Abrantes, Hoffmann, & Anton, 2005; Drerup, Croysdale, Hoffmann, 2008). In the studies of CD/ODD, a similar picture has been found. Many children and adolescents diagnosed with CD and ODD were found to involve or subsequently involve in delinquent acts. In the Christchurch longitudinal study which tracked the development of a birth cohort of 1265 children born in 1977 in Christchurch of New Zealand, Fergusson and his associates found that 90% of children with three or more CD symptoms at age 15 reported to be frequent offenders at age 16, as compared to only 17% of children with no CD symptoms (Fergusson & Horwood, 1995). It was also later reported that in the Christchurch study conduct problems of boys at ages 7-9 predicted their offending at ages 21-25 (Fergusson, Horwood, & Ridder, 2005). In another longitudinal study tracking the outcome of childhood psychiatric disorders, Copeland, Miller-Johson, keeler, Angold and Costello (2007) reported that violent crime at a later stage (16-21) was predicted by CD under age 16, in a representative sample of 1420 children followed from ages 9 to 21 in North Carolina of the United States.

Delinquency and disruptive behaviour disorder (CD and ODD) thus have significant conceptual and the empirical overlap. They are likely to share similar risks factors and could be ameliorated or prevented by similar interventions

(Farrington, 2009). For the purpose of this study, studies on psychopathological conduct disorders would be drawn on when they are relevant to adolescent delinquent and antisocial behaviours. Gender differences are found for both delinquency and disruptive behaviour disorders (such as in prevalence). The present review and study will focus on male juvenile offending. Gender issue and findings regarding juvenile offending and related psychopathological disorders in females are beyond the scope of the current study and will only be highlighted when necessary.

## Heterogeneity of adolescent delinquent and antisocial behaviours

It is common that adolescents at some points involve in behaviours that are not legally sanctioned (such as underage drinking) or illegal (Rutter, Giller, & Hagell, 1998a). This could be found in numerous studies using both self report data and official crime data; despite that the prevalence of delinquency according to self-reports is higher than those using official records (Kirk 2006; Farrington, 2009). The International Study of Self-Reported Delinquency in 13 western countries showed that only between 3% and 34% of adolescents denied to have engaged in any forms of antisocial behaviour. Across countries a majority of adolescents admitted having committed delinquent acts at some points, indicating that antisocial behaviour

by youths seems to be a "normative phenomenon" (Junger-Tas, 1994). Total abstainer who is not involved in any antisocial behavior during childhood and adolescence is few. In the Dunedin study (which will be reviewed in detail below), abstainers were less than 10% of the cohort (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt, 2006). In local context, the Fight Crime Committee of Hong Kong launched a large-scale study on juvenile delinquency in Hong Kong, which included more than 2000 adolescents and nearly 400 young offenders. This study found that three quarters of all males and about 40% of female had committed one or twice delinquent acts during their adolescence (Vagg, Bacon-Shone, Gray, & Lam, 1995). A more recent local survey in Hong Kong found that 51.8% of 912 subjects who were secondary school students aged 11 to 16 reported having committed at least one antisocial behaviour out of a list of 14 (e.g. property destruction, shoplifting, paying less traffic fee than required, etc.) in the past one year (New Century Forum, 2006). Thus, in local context too antisocial behaviours are frequent behaviours in adolescents. Even when indicated by official records, such as convictions or number of person arrested, the proportion of juvenile delinquency is substantial. This can be shown by the cumulative prevalence (also termed as life-time prevalence or ever prevalence) of juvenile delinquency found in the Cambridge Study of Delinquent Development which followed 411 South London males from age 8 (in 1961) to their age of 50, that

20% of this sample were convicted before age 17 (Farrington, 1995; Farrington, Coid, Harnett, Jolliffe, Soteriou, & Turner, et al., 2006). In local context, the Official statistics indicate that number of juveniles (age 10-15) and young person (16-20) arrested was 4173 and 4830 respectively, a total of 9008, making up 21.9% of the total number of persons arrested in Hong Kong in 2008 (HK Police Force, 2009).

While many adolescents similarly involve in crime, there is a great variety in their antisocial behaviours in terms of the rate of offending over the course of development, the type of offence committed, and pervasiveness and the persistence. The "age-crime curve" found by many official records and self-reports indicates a developmental pattern in which the prevalence of offending is usually low in late childhood and early adolescence, but increases sharply with age and peak in mid to late adolescence and declines afterwards (Farrington, 2009; Kirk, 2006; Loeber, Farrington, & Stouthamer-lober, & White, 2008). In the US National Youth Survey which sampled and followed up 1725 adolescents (national probability sample) aged 11-17 (in 1976) to their age of 27 – 33 (in 1993), age 17 is the peak age for violent offending (aggravated assaults, robberies and rapes that involved inquiry or a weapon), with over 25% of all male subjects reporting at least one or more serious violent offences (Elliott, 1994). Similarly, in the Cambridge Study, data searched from

official criminal records showed that the number of offences and offenders peaked at age 17 (69 offences and 46 offenders) and closely followed by age 18 (67 offences and 44 offenders) (Farrington, Coid, Harnett, Jolliffe, Soteriou, &Turner et al., 2006). The Pittsburgh Youth Study which followed 1517 boys from ages 7 - 13 to adulthood got similar findings, that the offending of boys for both violent and non-violent crime peaked in mid to late adolescent years according to data from official records, and report by self or other informants (mothers and teachers) (Loeber et al., 2008). While there is a major group of young people who follow the typical age crime curve in their offending trajectory, a group of youngsters, though much fewer in number, starts to display antisocial behaviour in childhood years and continue offending in adulthood (Moffitt and Caspi, 2002, Odgers & Moffitt, 2008).

Though the base rate of adolescent antisocial behaviours is high, a significant portion of antisocial behaviours is committed by a small minority of adolescents, whose delinquent behaviour are of great pervasiveness. In the Cambridge study, a proportion of 7% of the study male accounted for about half of all crimes in the study (Farrington, Ohlin, & Wilson, 1986; Farrington, 1995; Farrington et al., 2006). In the US National Youth Survey (Elliott, 1994), a small group of offenders, approximately 5% of the NYS sample in the year of 1980 (aged 15 to 21) were

serious violent offenders (reported a minimum of 3 serious violent offences) and they accounted for 83% of index offences and half of all crime reported. These studies thus show that a small group of adolescent offenders are pervasive offenders who commit offences more frequently than the other.

When the type of offence committed is concerned, adolescents are found to be generalized and versatile rather than being "specialized" in their offending, as for example, shown by studies regarding violent offending. In the Cambridge study (Farrington, 1995), 86% of violent offenders also committed nonviolent offences. Similarly, in the US National Youth Survey (Elliott, 1994), more than 50% of the serious violent offenders (approximately 5% of the NYS sample) were found to have involved in various types of other offences such as theft, vandalism, public disorder, drug use, etc.), apart from violent offences. It has once been thought that frequent violent offender might be characterized by distinct childhood features. But both violent and nonviolent frequent offenders were found to be indistinguishable in terms of childhood background features. In the Oregon Youth Study which followed 206 boys of age 9 to 10 to adulthood in Eugene/Springfield area, U. S., violent arrestees and non-violent arrestees were not different on their family background variables such as socioeconomic status, number of parental transitions, parental antisocial behavior,

family management variables including discipline and supervision, and childhood behavioral variables such as peer relations, deviant peer association, academic skills measured at grade 4 (Capaldi & Patterson, 1996). In the Philadelphia Collaborative Perinatal project, which followed 987 subjects from birth to age 18, frequent violent offenders also showed to be similar to frequent non-violent offenders in terms of childhood family background variables such as family structure, maternal education, socioeconomic class and child development variables such as low birth rate, neurological abnormalities and school disciplinary problem (Piquero, 2000). Delinquency is also found to be associated with various other types of antisocial behavior. As found in the Cambridge study, boys convicted before the age of 18 had significantly more drinking, smoking, gambling, and drug use (Farrington, 1995).

Two adolescents similarly committed an offence of theft could be both labeled as delinquent. But the above reviewed varieties of delinquency suggest that a same label by no means informs uniform pattern of antisocial behavior and outcomes, not to mention their associated background or causal mechanisms. In their 1998's review of antisocial behaviours of young people, Rutter et al. (1998c) cogently pointed out that adolescent antisocial behaviour could no longer be treated as a homogeneous construct, and that delinquent adolescents refer to a heterogeneous group. An

important issue remains as to whether the surface varieties of antisocial behaviours represents meaningful heterogeneity and thus meaningful subtypes could be discerned and identified. This task has become a major focus of many studies in the past two decades. By the comprehensive review of Rutter et al. (1998c) on this issue, it was found that empirical evidence supported at least two well-validated differentiators associated with delinquency, one being the age of onset and the other, the presence or absence of hyperactivity. What this means is: delinquency associated with different age of onset and delinquency associated with hyperactivity has distinct developmental pattern and features, which differentiate them from other antisocial behaviours. It is noted that there are both significant convergence and differences among these studies, as regards how to subdivide delinquent / antisocial activities and also regarding the validating characteristics of those classificatory subdivisions. The relevant findings will be reviewed in next section.

**Subtyping based on developmental paths of antisocial behavior : life-course-persistent versus adolescence-limited delinquency**

The review of Rutter et al. (1998c) pointed out that one well-validated differentiator associated with delinquency is age of onset. The importance of the age of onset for differentiating delinquency is stressed by the developmental theories of

antisocial behaviour advanced by both Moffitt (Moffitt, 1993; Moffitt, Caspi, Dickson, Siva, & Stanton, 1996) and Patterson (Patterson, 1996; Patterson, DeBaryshe, & Ramsey, 1989; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998).

Moffitt proposed and named the theory as developmental taxonomy of antisocial behaviour (Moffitt, 2003, 2006, 2009). The term "taxonomy" denotes some explanation. In her proposal of the theory in 1993, it was noted that "a classification becomes taxonomy if it engenders assertions about origins and outcomes by weaving a nomological net of relationships between the taxa and their correlates" and "taxon carries a network of meaning over and above a behavioural description" (p.674, Moffitt, 1993) and "includes implication for etiology, course, prognosis, treatment, and relations with other taxa (p.674, Moffitt, 1993). In simpler terms, Moffitt proposes a classification of antisocial and delinquent behaviour with a developmental perspective in which the origins, development and outcomes of different types of antisocial behaviours are specified, and that the classification has implications in etiology, course, prognosis and treatment of antisocial behaviour.

Moffitt proposed two prototypes of delinquency in the developmental taxonomy: life-course-persistent (LCP) versus adolescent-limited (AL) delinquency. The

antisocial behaviours of the LCP offenders originate in neuro-developmental processes, start early in childhood and continue persistently into adulthood whereas the AL antisocial behaviours originate in social processes, begin in adolescence and desist in young adulthood. The LCP delinquents are few, persistent and pathological whereas the AL antisocial individuals are common, relatively temporary and near normative (Moffitt, 1997; Moffitt, 2003; Moffitt, 2006).

The theory proposed did comprise many theoretical statements about the LCP and the AL antisocial behaviour including the aetiology and the developmental course including the onset, the continuity and desistance of antisocial behaviours, which could be subjected to empirical tests. The life-course-persistent group (LCP) is characterized by an early onset of antisocial behaviours in childhood and a persistence of these behaviours into adult life through a transaction process. The early antisocial behaviours occur when a high-risk environment exacerbates the difficult behaviours of a "high-risk" young child, who inherits or acquires subtle neuropsychological deficits, manifested early as subtle cognitive deficits, difficult temperament or hyperactivity. The environment risks comprise a list of family risks such as inadequate parenting, disrupted family processes and poverty and extend outside the family as the child grows up to include social risks such as affiliation with delinquent

peer. Overtime a sequence of transactions occurs between the child and the environment in a way to reinforce and exacerbate antisocial behaviours, resulting in a stable and persistent pattern of offending. A disordered personality marked by features of physical aggression and persistent antisocial behaviour into adulthood is gradually constructed.

In contrast, the adolescence-limited (AL) group has no notable history of early antisocial behaviours. They suffer low individual and environmental risks and thus have low "potentials" for future persistent offending. Their antisocial behaviours start in puberty and desist when the adolescents enter into adulthood. The aetiology of the AL antisocial behaviours comes from the adolescents' desire to accelerate social maturity and personal independence by mimicking the antisocial behaviours of the delinquent peers after they enter in a relatively role-less period of adolescence and experience the "maturation gap" before adulthood. The maturation gap develops because adolescents experience biological maturation much earlier than the social maturation and privileges that the society permits. Such maturation gap is particularly pronounced in modern society where the age of biological maturation arrives earlier because of improved health care and nutrition but participation in work force and taking corresponding responsibilities are delayed to later years (Moffitt,

1993), prolonging the duration of adolescence. Adolescents in such maturation gap are fuelled by the dissatisfaction with their still dependent status and find the delinquent style appealing for demonstrating autonomy and accelerating social maturation. The pre-delinquent development of the AL group is normal and most of the AL delinquents are predicted to be able to desist from crime after they enter into real adult roles, in which social acceptance of their adult status and privileges are realized. The theory about the AL path thus regards the adolescent-onset antisocial behaviour as an adaptation response to the adolescent's developmental context but not a product of cumulative pathological development. The recovery of the AL individual will be delayed if they encounter factors called "snares" (such as criminal record, addiction, incarceration, truncated education without credentials, hampering career and social relationship of the adolescents) that hinder successful transition to adulthood (Moffitt & Caspi, 2001; Moffit, 2003).

According to the developmental taxonomy, the following major hypotheses could be generated regarding the LCP and AL path of delinquency: 1. The LCP is characterized by a high risk childhood background including the child's risks, the high risk environment such as familial-parental risks, and subsequent social risks , whereas the AL group has background similar to normative peers 2. Despite manifestation of

antisocial and delinquent behaviours during adolescence for both groups, the antisocial behaviours of the LCP group persist into adulthood whereas those of the AL group desist. 3. Accompanied by the persistent antisocial pattern into adulthood, the LCP group has worse adult outcomes whereas the AL could transit into adulthood as normative peers in the absence of poor adult outcomes.

The theory is tested in the Dunedin longitudinal study that follows a cohort of 1037 children born between April 1972 and March 1973 from age 3 for over 30 years in New Zealand (Moffit, Caspi, Dickson, Silva & Stanton, 1996; Moffitt & Caspi, 2001, Moffit, Caspi, Harrington & Milne, 2002; Moffit, 2003). The families of the cohort represented the full range of socio-economic status in the general population of New Zealand. Two taxonomic groups had been derived according to the theory using clinical cut off and later, modelling technique (Odgers, Moffitt, Broadbent, Dickson, Hancox, & Harrington, et al., 2008). Data on the relevant childhood risks was collected. Adult outcomes at age 26 (a total of 79 measures grouped under criminal offending, personality, psychopathology, personal life and economic life) and age 32 (violence at 32, mental health, physical health, and economic problems) were reported in 2002 and 2008 (Moffitt, Caspi, Harrington, & Milne, 2002; Odgers et al., 2008). It is important to note that it has been over more than 10 years since the

theory was proposed in 1993 and since the cohort was followed. Some hypotheses generated by the theory have been supported by the subsequent empirical testing but some aspects of the theory have been challenged. These changes will be highlighted subsequently.

In the Dunedin study, children were designated antisocial if they displayed antisocial behaviours that were stable across time (at least three of the assessment point at ages 5, 7, 9, and 11) and persistent across situations (reported by parents and teachers on the Rutter Child Scales). Adolescent members were defined as antisocial if they self-reported extreme delinquency at the interview of age 15 or 18 (the Self-Reported Delinquency interview administered at ages 15 and 18). To derive the two taxonomic groups, study members who met criteria for antisocial across both childhood and adolescence were designated into the LCP group, whereas study members who met criteria for antisocial status only as adolescents but not in children were designated into AL group. By using these criteria, 10% of males (47 males) and 1% of females (6 females) were found on the LCP path and 26% of males (122 males) and 18% of females (78 males) were designated on the AL path.

*Do the two groups have differential childhood risks in the Dunedin sample?* The

theory asserts that the LCP is characterized by a high risk childhood background including child's risks and a high risk environment whereas the AL group has background similar to normative peers. Two major findings derived from Moffitt & Caspi (2001) are: 1. as predicted, the two delinquent groups were different in terms of childhood risk background, that the LCP group had significant worse risks than the AL group 2. as predicted, the AL group had average childhood background, that there was no significant difference in almost all risk variables between the AL group and the cohort norm. But it is important to note that when the data was reanalysed with new modelling techniques (General Growth Mixed Modelling, Muthen, 2004), and when the grouping was revised accordingly (Odgers & Moffitt et al., 2008), the finding that AL group has normative childhood background becomes less conclusive and questionable.

Specifically, according to Moffitt and Caspi (2001), a list of 26 childhood predictors initially grouped under 1. family adversity and inadequate parenting, 2. child neuro-cognitive risk, and 3. child temperament and behaviour were collected at different assessment points (ages 3, 5, 7, 9, 11, 13, 15 and 18) and compared amongst groups. It is noted that one cannot easily distinguish between the LCP adolescents and the AL adolescents by only referring to their participation in delinquency in the

adolescent period. Both LCP and AL males offended more than the unclassified members but they were found to exhibit similar mean levels of delinquent offending at the age of 15 and 18 and did not differ on police arrests or court convictions. Because there was no gender difference found on background risk factors across different trajectory groups, the data was combined for subsequent analyses to test the group differences amongst the group of LCP, AL and unclassified cohort members. The LCP and the AL group differed significantly on 21 out of 26 risks factors including that of undesirable parenting and family processes (harsh discipline, inconsistent discipline, parental report of higher family conflict, rejecting mother-child interaction, longer period of single parenting, more changes in child giver, etc), child neuro-cognitive risk (e.g. neurological abnormality at age three, lower IQ, reading deficit, lower heart rate, reading problem, poorer neuropsychological memory, etc.), and child temperament-behaviour risks (early difficult-to-manage behaviour, parent- and teacher-observed hyperactivity and aggressive behaviour, parent and teacher-reported peer rejection, etc.), indicating that the LCP members experienced significantly worse risk than the members on the AL path. The AL members had less dysfunctional family background, less neuro-cognitive deficits and less childhood temperamental-behavioral problem such as impulsivity and aggressive behaviours. The AL-path group did not differ

significantly from the unclassified members in the study in most of the childhood risk factors, indicating an average childhood background as predicted. The only significant difference found was in the domain of peer relationship. The children on the AL path were less likely than the unclassified group to be rejected by the peers in primary school. Also the AL group had more affiliation with delinquent peers than the unclassified group at age of 13 and 18.

The data set of Dunedin was reanalysed using the general growth mixture modelling (GGMM) to test whether the hypothesized subgroups of LCP, AL and normative group (named as low antisocial group) existed within the longitudinal Dunedin cohort. The GGMM is the latest generation of trajectory-based modelling techniques (Muthen, 2004) and an extension of traditional growth curve modelling (McArdle, Nesselroade, Schinka, & Velicer, 2003). It estimates latent variables based on multiple indicators of the construct observed across time. The method can test for the existence of the various trajectories within a population of individuals without a priori assumptions about the taxonomic grouping. It derives a best fitting model, and can show whether the relative goodness of fit of alternative models of having one, two, three, or more groupings. With this analytic method, 10.5% of the male members of the cohort were in the LCP group (10% of males using clinically

defined method), 19.6% in the AL group (as compared to 25% using the previous method) and 45.6% in the low antisocial group (Low group, characterized by low level of antisocial problems). The childhood risk factors were regrouped into 12 variables under three domains including family characteristics and context (low SES, child maltreatment, family conflict, inconsistent parenting), parental features (mother's mental health, mother's IQ, parent criminal conviction) and child factors (child IQ, undercontrolled temperament, low resting heart rate, reading achievement, ADHD) and peer delinquency at ages 15 and 18. As predicted, the LCP group has significantly worst childhood risks. Specifically, as compared with the Low antisocial group, the LCP group suffered significantly worst on all 12 childhood risks. The LCP group is different from the AL on a majority of the childhood risk factors, again, indicating that the LCP group also had significant worse risks than the AL group. Also as predicted, both the LCP and the AL group showed higher level of peer delinquency at age 15 as compared to the Low group. However, amongst the 12 childhood risks, the AL group was significantly different from the Low group on half of them, indicating that the group had a greater extent of childhood risks than the Low group (1. lower SES, 2. more child maltreatment, 3. more family conflict, 4. more childhood undercontrolled temperament, 5. higher rate of low IQ, 6. lower reading achievement). In this case, the assertion that the AL group has an average

background similar to the normative peers is questionable.

*Are the two groups different in terms of the persistence of their antisocial behaviours into adulthoods?* The theory predicts that antisocial behaviours of AL individuals will desist in adulthood when the adult role and privileges becomes realized whereas the antisocial behaviours of the LCP group, marked by more severe aggression and violence, will persist into adulthood. The empirical finding (Moffitt et al., 2002; Odgers & Moffitt, et al., 2008) renders partial support to the theory. Moffitt et al. (2002) reported on the age 26 adulthood outcomes of different trajectory groups. The LCP group did continue antisocial behaviour in adulthood, but not as predicted, the antisocial behaviours of the AL group had not clearly desisted.

At the age of 26 years, not only the LCP but also AL members offended more than the cohort males according to their self-report. Both the LCP and the AL group reported having committed a similar variety of different offence types (including property crime, rule violations, drug crimes and violence). Overall however, the LCP group reported a significantly higher mean number of offences. The LCP group did not differ from the AL group in self-reported property crimes or rule violations, but the LCP group reported higher drug-related offences and violent offences. In

terms of cumulative court conviction, again, not only the LCP but also AL group were convicted more than the other cohort males. But the conviction of the LCP group tended to be more frequent and serious than that of the AL group. Particularly, the LCP males had worse conviction records than AL men for property crimes, court-order violations, drug trafficking and violent crimes.

The findings are similar when the age 32 violence outcomes were looked at. Consistent with the theory's prediction, the LCP's antisocial behaviour marked by physical violence persisted, that they were different from the low antisocial group on four of the six violence outcomes (including controlling abuse towards partner, self-reported violence, informant-reported violence, and official violence convictions). Overall 59% of them engaged in one of the six of the violent outcomes and 33% of them had a conviction for violence between ages 26 to 32. When followed up to age 32, the antisocial behaviour of the AL group had not desisted as the theory hypothesized. The group was different from the Low group on five of the six violence outcomes with large effect size. But they were not the worst group. As compared to the LCP group, they were significantly lower in court conviction for violence. The LCP men were four times more likely to have a conviction than the AL men between ages 26 to 32.

*Are the two groups different in terms of other adult outcomes*? As the theory predicted, the LCP group has worst adult outcomes. But the AL group had considerable difficulties in leading their adult life, as shown by various adult outcomes at age 26 and age 32. Moffit et al. (2002) reported adult outcomes of the Dunedin study members of in terms of personality, psychopathology, personal life and economic life up to their age of 26 apart from the persistence of antisocial behaviour. In terms of personality and psychopathology, the LCP males scored significantly higher than the AL males on negative emotionality (neuroticism) and callousness (a major trait in psychopathy marked by lack of guilt and empathy, shallow affect, and being manipulative interpersonally), and had more psychopathology including antisocial personality disorder. On the whole, the LCP group fared poorly than the AL group at the age of 26 in terms of different adult outcomes, but the AL men at 26 years fared poorly relative to the unclassified normative members. The term 'adolescent-limited' is indeed challenged by the finding of the persistence of the antisocial behaviours into adulthood of the AL group. As can be seen from the outcomes, the proposed short-term delinquent career of the AL group is not so short term and the consequences are not benign.

The picture regarding age 32 outcome is similar. The LCP group had the worst

outcomes in terms of mental health (psychiatric disorder, suicide attempt, informant reports of internalizing symptoms and substance abuse and informant reports of substance use problems), physical health (such as cardiovascular disease, sexual health at age 32, injuries between ages 26 and 32, respiratory function, etc.) and economic problem (e.g. household income, unemployed, informant-rate financial problem, no money for food or other necessities, etc). The AL men, as compared to the low antisocial group, had higher prevalence rates of drug problem in terms of mental health, had more problems in physical health (significant differences found for 6 out of 11 physical health outcomes) and economic problems (significant differences found for 5 out of 7 economic outcomes). But AL group is not the worst group, that they did not experience the same extent of problems in mental health, physical health and economic status as compared to the LCP group.

The Dunedin's findings reviewed above are consistent with some, but not all predictions generated by Moffitt's developmental taxonomy of antisocial behaviour. Support for the theoretical statements about the life-course-persistent path is overall strong, that this group was shown to suffer significantly worst risk background as compared to the average cohort and AL group, that their antisocial behaviour persisted into adulthood when followed up to ages 26 and 32, and that they had worst outcomes

in terms of mental and physical health and economic status. Particularly important, they scored significantly higher in callousness and had higher rate in antisocial personality disorder as compared to the other groups, supporting the theory's assertion that overtime an antisocial personality had been constructed. However, the theory's predictions about the adolescent-limited group have received significant challenges. The AL group had less background risk than the LCP group but the assertion that their background was no different from that of the normative peers was questionable. The delinquent and antisocial behaviour of AL group clearly had not desisted up to ages 26 and 32 as predicted, and this group also had poorer outcomes in terms of mental, physical health and financial status, albeit they were not as worse as the LCP group. The research team of Moffitt renamed the group "adolescent-limited" to "adolescent-onset" as the group's participation in antisocial behaviour is beyond adolescence (p.689, Odgers & Moffitt, 2008). But what accounts for the unexpected findings?

That the AL group shows background risks more than their normative peers, continues their delinquent and antisocial act beyond adolescence, and suffers undesirable adult outcomes are contrary to the taxonomy's predictions, challenging the aetiology hypothesized. Odgers and Moffitt (2008) attributed these unexpected

findings to some possible methodological problems of the study. They explained that in the Dunedin study, some adolescent-limited members might have been missed out and misclassified to the low-antisocial group. Thus those assigned to the AL group were thus more persistent than expected. This was due to two reasons, according to Odgers & Moffitt (2008). Firstly, the antisocial behaviours of the AL are transient and intermittent. To timely and rightly capture the AL member one would need a more intensive measurement schedule. The measurement frame of the Dunedin study (which is at ages 11, 13, 15, and 18 to assess past-year antisocial behaviours) might not be able to capture the transient AL. Members who offended between the ages of 11 and 12, 13 and 14, and 15 and 17 would not end up in the AL group but could be assigned in the Low group. Secondly, individuals who engaged in antisocial behaviour during only one of the three assessment periods were assigned into the Low group or childhood-limited group in the solution despite theoretically they fit the criteria for adolescent-limited pathway. It is possible that the classification rule adopted in the study is not sensitive enough to detect the AL members. While these methodological problems could possibly lead to unexpected findings regarding the adolescent-limited group, it is also possible that alternative theories better explain the AL path of offending, such as Patterson's theory, which will be described later in this review.

## Convergent and discrepant findings regarding the two-trajectory groups of offending proposed by Moffitt

The subtyping of delinquency based on developmental trajectories is not only tested in the Dunedin study but various other studies in the recent two decades. Many of these studies are longitudinal studies that followed their cohort over an extended period of time and reported the findings at different follow-up points that serve to test different aspects of the theoretical predictions about the specific offending subtypes. Evidence accumulates for the differentiation between the early onset life course persistent group and the adolescent onset group in terms of differential risks and outcomes; despite that differences exist regarding theory of aetiologies and some important group features. There are also studies with cross-sectional design that map the differential correlates distinct to different delinquent subtypes. Consensus about the existence of the life-course-persistent path of offending is strong. The following reviews the convergent and the discrepant findings on the two-trajectory subtypes of adolescent antisocial behaviour.

*Convergent findings on the differentiation between the early- onset*

*life-course-persistent offending versus the adolescent-limited offending.* The Christchurch longitudinal study, also a New Zealand study, followed an unselected birth cohort of over 1265 children born in the Christchurch, New Zealand urban region during mid 1977 from birth to adulthood (Fergusson, Horwood, & Nagin, 2000; Fergusson, Horwood, & Ridder, 2005; Fergusson, Lynskey, & Horwood, 1996). Fergusson et al. (2000) reported the offending trajectories of the cohort up to the follow up at age 18. The cohort was studied at birth, four months, followed up annually from ages 1 to 16 and again on 18 years. Data about the sample' offending behaviours from ages 12 to 18 were collected. Using a latent class modelling approach, Fergusson et al. (2000) identified four trajectory groups of offending, including the chronic offender group (6.8% of the sample demonstrating high risk of offending through the whole period of follow-up since birth until the age of 18) and the adolescent-onset offender group (7% of the sample having a small risk of offending before age 14 and a marked increase in offending thereafter), which are similar to the life-course-persistent offending group and the adolescent-limited offending group proposed by Moffitt. Also consistent with Dunedin findings, the chronic offender group suffered the most adverse childhood background measured before the age of 12 including socio-demographic background (e.g. below average family living standards), family functioning (high marital conflicts of parents, parental

history of criminality and illicit drug use, etc.), and individual factors (e.g. early attention problem, lower IQ score, etc.)

With a large Swedish cohort, Kratzer & Hodgins (1999) tested Moffitt's two trajectory of offending by comparing the patterns of offending of each type of offender and comparing the hypothesized determinants and correlates of each type of offending. The cohort was consisted of 7101 males and 6751 females born in Stockholm in 1953, Sweden and residing there in 1963. The cohort was followed from childhood up to age 30 years. Data regarding the subjects' intelligence at age 13, academic performance, teacher ratings of the behaviours, and reports of conduct problem, and individual and family problems requiring social, psychological and/or psychiatric service were available for study. Among the four groups of offenders identified, the stable early-starters (6.2% of the cohort men / 441 males) and the adolescent-limited (9.9% of the cohort men / 703 males) resemble the life-course-persistent and the adolescent-limited offending type proposed by Moffitt. Consistent with Moffitt's theory, the study found that the early-starter group involved in crime more frequently and diversely than the other offending groups. This group accounted for 70% of all offense committed by male in the cohort and they committed on an average of 4.33 types of offences (SD = 1.48), which contrasted with the

adolescent-limited group that accounted for 9% of all offences and committed an average of 1.42 types of offences (SD = 0.73). The stable early starters could be differentiated from non-offenders by childhood background risks noted above such as academic performance and low global intelligence, whereas the adolescent-limited group could not be distinguished from non-offenders by these background variables.

Using also a high risk US sample, Aguilar, Sroufe, Egeland, and Carlson (2000) tested the differentiation of the early-onset / persistent and the adolescence-onset antisocial group. Subjects were drawn from a 20-year longitudinal study of normative and non-normative development in a high-risk urban population of firstborn children (original n = 267). Four offending groups were identified based on the reported level of externalising behaviour at six assessment points from kindergarten through 16 years (using externalising scales of CBCL, TRF and YSR of Achenbach). It was found that the early-onset / persistent group could be differentiated from the adolescent-onset group and the never-antisocial group in early psychosocial risk variables (such as low quality of parental responsiveness and caregiving, higher stress of the mother, neglectful and physically abusive parenting, etc). The results are convergent with the Dunedin findings about the differential background risks associated with the LCP versus the AL offending group. In this

study, there was no significant difference between the adolescent onset group and the never antisocial group in terms of early and later psychosocial risks.

Apart from longitudinal studies, there are also cross-sectional studies that test the differentiation of the two offending groups by looking at their differential correlates. Carroll, Hemingway, Bower, Ashman, Houghton, and Durkin (2006) tested the impulsivity among early-onset, late-onset and non-offending group in a sample of 129 adolescents including 86 institutionalized adolescents and 43 regular school students. Mean age of the total sample was about 15 years. The early-onset and late-onset group were derived according to the Moffitt's taxonomy. Institutionalized adolescents with history of conviction prior to 12 years old were assigned to early-onset group and those without offending history until age 13 were in the late-onset group. Impulsivity was measured by both performance-based tests (including stroop colour and word test, time perception, accuracy game and risk-taking game) and questionnaire (the Eysenck Impulsiveness questionnaire). It was hypothesized that the offender groups will have higher level of the impulsivity than control, and early-onset group will have highest level of impulsivity than the late-onset group. Result rendered certain support to the differentiation of the early-onset group from the late-onset group in terms of the level of delinquency, that

the early-onset group had significantly higher involvement than the late-onset group in self-reported delinquency (stealing, school misconduct, vehicle offences, physical aggression and hard drug use). Results regarding impulsivity as external correlate to differentiate the different groups were partial and mixed. The two offender groups did differ from the non-offenders on certain measures (on test of time perception and the Eysenck Impulsiveness questionnaire), indicating a higher level of impulsivity, but the early-onset group and the late-onset group could not be distinguished by these measures of impulsivity in this study.

Dandreau & Frick (2009) also tested differences between the childhood-onset group and adolescent-onset group of antisocial behavior with 78 pre-adjudicated adolescent boys of age ranging from 11 to 18 collected in two short-term detention facilities and an outpatient treatment program for boys at risk for delinquent and behavior problem. Age of onset of conduct problem was used to decide the group membership, which was collected from the subjects' self report and parent. Age 11 was chosen as cutoff that demarcated the two offender groups (based on the previous finding that chances for diagnosis of antisocial personality disorder in adult was over two times more likely for youth with onset of serious conduct problem on or before age 11). The two groups were compared on impulsivity, dysfunctional parenting practices, callous-unemotional traits (similar to the concept of psychopathy that refers

to specific affective and interpersonal styles including fearlessness, lack of remorse and guilt, lack of empathy, etc), affiliation with delinquent peers, rebelliousness (perceptions that parents allow too little autonomy), and traditionalism (tendency to endorse conventional attitude towards authority and traditional institutions vs. non-conformity), which were measured by self-reported questionnaires and rating. Results showed that the childhood-onset group had greater levels of dysfunctional parenting, callous-unemotional traits, and affiliation with delinquent, adding support to the differentiation between the two groups.

It must be pointed out that apart from the life-course-persistent and the adolescent-limited offending trajectory; other trajectory group had been identified. For example, in Dunedin study, apart from the two prototypic offending groups, a small group of males was found to display offending pattern that closely resembled a low-level chronic offender. This group was found to have displayed high level and pervasive antisocial behaviour in childhood but then only engaged in low to moderate delinquency during adolescence. They were labelled as "recovery" group by Moffitt initially but subsequent follow-up found that this small group re-offended intermittently (Moffitt, 2003). In Aguilar et al.'s study (2000) already mentioned above, with the US high risk sample of 180 adolescents followed up from kindergarten through 16 years, a total of four groups were identified, which were

early-onset / persistent, adolescent-onset, childhood-limited (antisocial in childhood but not in adolescence) and never antisocial group, based on the teacher and parent's report of externalizing behaviour at six assessment points. In the Sweden cohort study (Kratzer & Hodgins, 1999) reviewed above, apart from a no-crime group, a total of four offender groups were identified, which included the stable early-starters and the adolescents-limited group ( resembled the LCP and the AL offending groups), and also an adult-starter group and a discontinuous offender group.

Also important to note is, the two trajectory model proposed by Moffitt is linked to a priori theory and the two offending groups were defined by a priori classification criteria (Moffitt, 1993; Moffitt et al., 1996). Subsequent studies using new modelling techniques (Muthen & Shedden 1999; Muthen, 2004; Nagin, 1999) to distinguish offending groups based on the heterogeneity in developmental courses of delinquent behaviour often identified offending paths not originally predicted by the two-trajectory taxonomy of Moffitt. These new modelling methods ascertain whether distinct trajectories exist in a population whose offending behaviours have been measured repeatedly in the course of development and this bottom-up approach makes the results relatively free from the investigator's bias (Nagin, 1999; Nagin & Tremblay, 2001). For the Dunedin's data, using modelling technique of GGMM,

Odgers and Moffitt, et al. (2008) reported a group named as childhood-limited, which was similar to the LCP group in terms of childhood risk background and level of childhood antisocial behaviour, but with no elevated violence at age 32. So together with the LCP, AL and the Low group already mentioned, the new modelling came up with a four-class solution that best fits the longitudinal data (LCP: 10.5% of the cohort, AL : 19.6% of the cohort, Childhood-limited: 24.3% of the cohort, and the low group: 45.5% of the cohort). The childhood-limited group was not originally hypothesized in Moffitt's two-trajectory model.

In the Christchurch longitudinal study, Fergusson et al. (2000) found that a four-class latent model best fit the offending pattern of the sample when the follow-up was up to the age 18. Apart from the chronic offender group and the adolescent-onset offender group already reviewed above, the other two groups identified were the desister group, whose members displayed antisocial behaviours in childhood but desisted in adolescence (resemble childhood-limited group of Dunedin study), and also a no-problem group.

Using data from the Oregon Youth Study which followed 204 at-risk boys annually from ages 9 to 10 and consequently to ages 23 to 24 (final $n = 204$), Wiesner & Capaldi (2003) reported to have identify six trajectory classes, which included: 1.

*chronic high-level* (n = 32, started with high levels of delinquent behaviour, increased markedly through the middle adolescence and dropped toward the initial high level of offending); 2. *decreasing high level* (n =57; started with high level of delinquent behaviour and then declined toward zero), 3. *chronic low-level* (n=38, started with moderate level of offending, increased with a relatively late peak around ages 19 to 20 and then decreased slightly), 4. *decreasing low-level* (n=44; started with moderate level of delinquent behaviours but gradually decreased toward zero thereafter), 5. *rare offender*s (n = 23; less than one offense each year), and 6. *non-offenders* (n = 10, reported no offending behaviour). In this study, the chronic high level group is similar to the life-course-persistent offending. No clear adolescent-limited group was identified but the authors suspected that the low-level chronic group could be conceptualised as offenders with adolescent onset.

One study (Van Lier, Wanner, & Vitaro, 2007) using also general mixture model (Muthen & Shedden, 1999; Nagin, 1999) identified three groups that resembled the two-trajectory model of Moffitt, including a childhood-onset, adolescent-onset, and a low antisocial behaviour groups in a community sample of 165 boys and 151 girls from in Quebec, Canada followed yearly from age 6 to age 15.

There is by far no conclusive finding across studies as to how many offending trajectories best categorize delinquent behaviour. The variations of findings will partly depend on the characteristics of sample such as unselected sample versus at risk or offender sample, follow-up period, and measurement used, and statistical methods, etc. Piquero (2008) reviewed major trajectory studies using modelling techniques with offender samples (nine studies) and general population samples (over 30 studies) covering the period of childhood, adolescence or childhood/adolescent/adulthood. It was found that on average between three to five groups tend to be identified by studies using the modelling technique. Across these trajectory-based empirical studies, an adolescent-peaked pattern and a chronic offender pattern are repeatedly evident (Piquero, 2008), which are similar to the LCP and the AL group in terms of offending pattern over development, providing support for the two-trajectory model proposed by Moffitt.

*Differences in the aetiology of the life-course-persistent versus the adolescent-limited delinquency and mixed findings regarding the associated features of the adolescent-limited group.* Patterson also differentiated two groups of delinquency, the early-onset and the adolescent-onset (named as late-onset group) but the proposed aetiologies of these two offending paths, particularly for the

adolescent-onset group, are different from that proposed by Moffitt. Similar to Moffitt's account, Patterson proposed that individuals on the early-onset path suffered highest risks and had more chance to develop persistent delinquency beyond adolescence. This was found in the Oregon Youth Study which followed 204 at-risk boys annually from ages 9 to 10 up to adulthood. Specifically, early-onset boys (arrested before the age of 14, n = 43) could be discerned from those late-onset boys (arrested between 14 to 18, n = 52) in terms of difference in social risks of the family, the level of parental skills, the levels of child deviancy and social skills (Patterson, 1996; Patterson & Yoerger, 1997). As compared with the no-arrest group, the early-onset group displayed more antisocial behaviour in mid-childhood and was found to suffer more family risks including poor parental discipline and parental antisocial behaviour.

Patterson's coercion model stressed the importance of poor parental monitoring of child activities; disruptive family transitions and inconsistent parental discipline are the major aetiological factors of delinquent behaviour, and it is true for both the early-onset and the late-onset offending, (Dishion, & Patterson, 2006; Granic & Patterson, 2006; Patterson, 1996; Patterson, Reid & Dishion, 1992; Patterson & Yoerger, 1993; Patterson & Yoerger, 1997) The coercion model assumes that all social behaviours, including antisocial and delinquent behaviours, exist because they

are functional in a particular way and the reactions of the others are key to decide whether the children and adolescents will over select certain behaviours (such as delinquent behaviours) over the others (such as prosocial behaviours). For the early-onset group, the "training" on deviant behaviour starts very early at home. In these families, the child's coercive behaviours (e.g. temper tantrums, hitting, whining) are functional that they are the major effective means for terminating conflicts or to escape aversive parental discipline whereas the parents (or siblings) interact with the child in a way to reinforce the coercive behaviour, for example, to pacify the coercive child by yielding to his / her demands. Thus inept parenting here is key to decide whether the coercive acts of the child will be functional. Over numerous practices the child learns coercive, antisocial behaviours over prosocial behaviour and coercion becomes the child's primary interpersonal / coping strategy. The child receives further "training" in school where he/she selects deviant peers for support and association. The child's antisocial behaviours further undermine the chances for the development of prosocial behaviours and the effect is to produce an adolescent who is both socially unskilled and deviant (antisocial), posing the path to persistent offending. When the parental monitoring continues to break down, deviant peers serve a proximal risk to facilitate coercive behaviour into delinquent behaviours. The adolescent selects new forms of antisocial behaviour in forms of delinquent acts and

begins the path of early arrest and chronic offending.

For the late-onset path, it is hypothesized that these families demonstrate moderate risks and the parents have limited if not inept parenting skills. As compared to the families of early-onset boys, coercive and antisocial behaviours in the late-onset families do not work so well and the relative payoffs for prosocial behaviours (e.g. doing household chores, paying effort in school learning, etc.) are better than the payoffs found in the early-onset families (but not as high as in families that foster non-delinquent boys). Late-onset boys tend to acquire moderately developed social skills. Their adjustment is better than the early-onset boys but not so well as the non-delinquent boys. The growth in antisocial and delinquent behaviors occurs when adolescents start to form affiliation with deviant peers in which antisocial behaviours are rewarded and sustained. Coupled with the breakdown of parenting, the adolescents start offending (Patterson & Yoerger, 1997); Thus for the early-onset group, the deviancy training in the context of poor parenting starts in family as early as in preschool year and then continues in a later stage by deviant peers. For the late-onset group, the deviant peers come as a major source for deviancy training and with the breakdown of parental monitoring and practices in the family then the adolescents starts their delinquent career. Regardless of whether it is

early onset or late onset, the growth of antisocial behaviour is associated with breakdown in parenting such as monitoring, discipline and family problem solving.

Thus in the coercion model of Patterson, inept parenting is key to generate the early-onset antisocial behaviours whereas in Moffitt's theory, the emphasis is also on the high-risk characteristics, particularly neuropsychological deficit of the child as a primary force in the transactions with the high-risk environment. In Moffitt's terms, the primary force of the adolescent-onset delinquency comes from the adolescent's developmental process of building autonomy when facing the maturation gap and parenting is a less important contributing force. Moffitt (2006) argued that negative correlation between parental monitoring and adolescent-onset delinquency might exist; but this correlation arises because adolescents evade their parents' supervision to obtain autonomy. In Patterson's theory, decrease and breakdown in parenting when children enter into adolescence are major factors key to the onset of delinquency, apart from deviant peer training.

Moffitt and Patterson's theory predict different associated features of the offending group, particularly on the adolescent-onset group. In Moffitt's theory, the pre-delinquent development and the background of the AL group including family and

parenting are normal. Patterson's theory predicts that the late-onset group has intermediate family and parenting risks between the early-onset group and the non-delinquent group. The pre-delinquent development (such as pro-social skills) of the late onset adolescents is marginal, as compared to the non-delinquent peers. Patterson specified that there would be parenting breakdown associated with the adolescent-onset offending whereas Moffitt was not explicit about the role of parenting for the adolescent-onset offending. The different aetiologies and group features proposed render different implications for intervention. If Moffitt's theory is correct, intervention for adolescence-limited offending could target on counteracting peer influence during adolescence (Dishion, McCord, & Poulin, 1999; Howell & Hawkins, 1998). If Patterson's theory is correct, the intervention or prevention should include measures addressing the background risks and the marginal development of the adolescents, apart from focusing on deviant peer influence and parenting during adolescence.

As noted above, in the Dunedin study, Moffitt et al. (2001) did find that the AL group had a normative background not different from the unclassified members in terms of parenting and family adversities, child neuro-cognitive deficits, and childhood temperament and behavioral problems, though the later report (Odgers &

Moffitt, et al., 2008) is less conclusive.

In the study of Aguilar et al. (2000) which compared four offending groups (including never antisocial, 2. childhood-limited, 3. adolescent-onset, 4. early-onset / persistent delinquent groups), the adolescent-onset group did not experience higher levels of psychosocial adversity in early and late childhood (including family and parenting adversities such as abusive parenting and maladaptive family functioning). They also showed no difference from the never antisocial group on the early and later neuropsychological variables (such as intellectual development measured by Bayley Scales of Infant Development at 9 months, IQ measured by WISC-R in the third grade). These findings are consistent with the proposal of Moffitt's proposal that AL group has a normative background including parenting, which could not be differentiated from the non-delinquents.

On the other hand, some other studies found that the antisocial behaviors with adolescent-onset were associated with considerable background risks. Fergusson, Horwood, & Nagin (2000) compared the psychosocial profiles of the offending groups including the non-offenders, moderate offenders, adolescent-onset offenders and chronic offenders using the dataset in the Christchurch longitudinal study. The

chronic offenders and the adolescent- onset offenders are similar to Moffitt's LCP and AL groups. The psychosocial profiles compared included socio-demographic variables (including mother's young age at birth of child, mother lacked formal education, lower SES of family, single parenthood, poor living standard), family functioning (including higher marital conflict, higher adverse family event, parental criminality, parental problems with alcohol and use of illicit drug), and individual factors (including early childhood conduct, early attention problem and lower IQ). It was found that for all comparisons except on the measure of adverse family life event, the non-offender group had the most favorable profile characteristics, the chronic offenders had the least favorable, and the remaining group including the adolescent-onset group lay between two extremes. For the measure of adverse family life event, all the three offending groups had similar score, which was different from the non-offender group that experienced lowest rate of adverse family life event. The results thus indicate that the adolescent-onset offender group is characterized by intermediate background risks, consistent with that postulated by Patterson.

Brennan, Hall, Bor, Najman, & Williams (2003) tested the developmental model of antisocial behaviour of Moffit with an Australian high-risk sample of 370 adolescents. Accordingly they hypothesized that the adolescent-onset aggressive

boys and nonaggressive boys would not differ in terms of associated biological and social risks measured at four assessment points (3-4 days after birth of the child, at the child's 6 months, age 5 and age 15). Contrary to this prediction, they found that the adolescent-onset aggression group was significantly different from the non-aggressive group in having higher level of age 15 cumulative social risk count including variables pertaining to parenting risks such as inadequate parental monitoring and youth perception of paternal and maternal acceptance and youth perception of maternal hostility. The results are more in line with Patterson's prediction.

The above findings reviewed find mixed results regarding the associated group features of the AL group. Whether the AL group has a normative risk background including parenting, and whether they have acquired development (e.g., prosocial skills) similar to their non-delinquent counterparts will be tested in the current study.

**Identifying homogeneous subgroup within the early-onset offending group**

As mentioned before, the review by Rutter, et al. (1998c) on the heterogeneity of adolescent antisocial behaviours found two well-validated differentiators of antisocial behaviours, one being the age of onset, which has already been reviewed above, and

the other is antisocial behaviours associated with hyperactivity. Comorbid hyperactivity is found to be important in terms of its effect on the development, course, and severity and outcomes of adolescent antisocial and offending behaviour (Rutter, et al, 1998c; Rutter, Maughan, Meyer, Pickles, Silberg, Simonoff, & Taylor, 1997). From the point of view of child and adolescent psychopathology, understanding the comorbidity (co-occurrence) between antisocial / conduct problem and hyperactivity has also been regarded as important for understanding the etiology and treatment of conduct disorder (CD) and Attention Deficit Hyperactivity Disorder (ADHD), two commonly found childhood disruptive disorders that frequently occur together (Martin & Cantwell, 1997; Russell & Rosermary, 1995). There is consideration whether CD-ADHD can be considered as a distinct taxonomic category different from its pure condition (Waschbusch, 2002) and the distinctiveness of this comorbid condition has been under considerable research investigation. The following will review relevant research findings, which have implications to the subtyping of adolescent antisocial behaviours.

*Overview of antisocial behaviours associated with ADHD.* As summarized already in this review, CD is characterized by a pervasive pattern of violation against the basic rights of others or major age-appropriate societal norms or rules

(DSM-IV-TR, American Psychiatric Association, 1994). For ADHD, the symptoms could be subsumed into two major areas. The first, being related to inattention which comprises of a list of nine symptoms (e.g., make careless mistakes in schoolwork or activities, have difficulty sustaining attention in activities, not seem to listen when spoken to, fail to follow through instruction and finish schoolwork or duties, etc.), and the other related to hyperactivity-impulsivity which comprises a list of nine symptoms, (e.g. always fidget with hands or feet, often talk excessively, difficulty awaiting turn, often blurt out answers before questions have been completed, etc.) according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; APA, 1994). Having six or more symptoms persisted for over 6 months in either one area and having some symptoms onset before age 7 years are required to make the diagnosis. CD and ADHD are both prevalent childhood disorders under the category of disruptive behaviour disorder in the Diagnostic and Statistical Manual of Mental Disorder (DSM-IV-TR, APA, 2000). The prevalence of CD in community samples ranges from 1.8% to 16% for boys and 0.8 % to 9.2% for girls, with gender difference remaining stable across ages (Loeber, Burke, Lahey, Winters, & Zera, 2000; Loeber, Burke, & Pardini, 2009). Prevalence of ADHD is estimated to at 3% - 7% of school-age children among general population, also more frequent in males than in females (DSM-IV-TR, APA, 2000; Hinshaw, 1994; Szatmari,

1992; Szatmari, Offord, & Boyle, 1989). A recent US study that included a large representative sample of 9380 subjects aged 7 to 29 years ascertained an overall prevalence of 9.2% of the disorder for the full sample with a male to female ratio of 2.28 to 1 based on DSM-IV ADHD symptom criteria, and specifically 15.69% and 13.75 % of prevalence respectively for male children and adolescents, 7.49% and 5.38% of prevalence respectively for female children and adolescents (Ramtekkar, Reiersen, Todorov, Todd, 2010).

The rate of CD and ADHD is higher in delinquent sample as compared to that found in general population stated above. In the review of Vermeiren (2003), a prevalence of CD over 50% up to 90% in delinquent adolescents was found by 13 out of 15 studies published between 1984 and 2003. The rate of ADHD was found to be diverse in delinquent samples; with seven studies reported a prevalence rate of 14% to 27% and a rate of 50% to 75% were reported by two studies. Another more recent review (Fazel, Doll, & Langstrom, 2008) that included 25 studies published between 1996 to 2006 (total numbers of boys = 13,778; age range: 10 – 19, mean age: 15.6 years) found an average prevalence of 52.8% (95% confidence interval: 40.9% - 64.7%) for CD diagnosis in adolescent male offenders based on 15 studies; and an average prevalence of 11.7% (4.1% - 19.2%) for ADHD diagnosis based on 11 studies,

which are both higher than the average prevalence of 3.3% (3.0% - 3.6%) for psychotic illness and the average prevalence of 10.6% (7.3% - 13.9%) for major depression in male delinquent adolescents.

A high rate of occurrence (comorbidity) between Conduct Disorder (CD) and Attention Deficit Hyperactivity Disorder (ADHD) is found by both earlier studies (comorbidity ranging from 30% to 50% in the study of Biederman, Faraone, & Lapey, 1992; 36% to58.5% in the study of Szatmari, Boyle, & Offord, 1989) and later reviews (Jensen, Martin & Cantwell, 1997; Angold, Costello & Erkanli, 1999; Pliszka, 2000). Jensen et al. (1997) reviewed studies from 1987 up to 1994 and found a range of 42.7% to 93% of comorbidity between ADHD and CD/ODD, as compared to lower rate of comorbidity between ADHD and internalizing disorders (13.0% to 50.8%). Angold et al. (1999) reviewed major epidemiological studies (spanning from 1987 to 1998) using samples drawn from general population regarding the comorbidity between CD and ADHD and found that a median odd ratio (OR, an estimate of the probabilities of co-occurrence between disorder) of 10.7 for the comorbidity between CD and ADHD, which is the highest OR among the rates of comorbidity between pairs of disorders (OR = 5.5 for ADHD and depression, OR= 6.6 for CD and depression, OR = 8.2 for depression and anxiety disorder, OR = 3.2 for CD and anxiety disorder, OR = 3.0 for ADHD and anxiety disorder). More recent

epidemiological studies similarly found high comorbidity of the two disorders (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004; Nock, Kazdin, Hiripi, & Kessler, 2007). For example, In a nationally representative sample of 10438 5-15-year olds children in UK, odd ratio for the comorbidity between ADHD and CD for boy was 21.2, and for ODD and ADHD was 19.1, which were the highest odds among the rates of comorbidity between CD / ODD and other disorders (anxiety and depression) (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004).

The validity of CD and ADHD as two separable disorders was shown by their distinct clinical correlates and developmental outcomes (Leung, Ho, Luk, Taylor, Bacon-Shone, & Lieh Mak, 1996; Szatmari, Boyle, & Offord, 1989; Taylor, Schachar, Thorley & Wieselberg, 1986 a & b; Taylor, Chadwick, Heptinstall, & Danckaerts, 1996; Waschbusch, 2002). For example, childhood CD is found to be more related to antisocial parents, dysfunctional family processes and parenting, low socioeconomic status, whereas childhood ADHD is more associated with neuro-developmental problem (such as motor and language delay), learning difficulties and cognitive deficits (Hinshaw, 1987; Leung et al., 1996; Taylor, 1999; Waschbusch, 2002). There are several hypotheses postulated to account for the frequently occurred comorbid condition of these two disorders (Caron & Rutter; 1991;

Neale & kindler, 1995; Rutter, 1997). For example, (1) the ADHD+CD could be conceptualized as a hybrid or additive combination of pure ADHD and CD, having the difficulties associated with each disorder (in this hypothesis the risk factors for one disorder increases the probability of the risks for the second disorder), or (2) ADHD, CD and ADHD+CD all come from one single underlying disorder with similar risk factors but differ in manifestation such as developmental course, or (3) the comorbid ADHD and CD is a distinct condition differing from pure CD and ADHD, and (4) one disorder produces a symptomatic phenocopy of the other disorder but does not carry the underlying deficit of the other disorder (Caron & Rutter, 1991). There is no conclusive agreement regarding the causes of comorbid condition of CD and ADHD (Rhee, Willcutt, Hartman, Pennington, & DeFries, 2008) but evidence accumulates to suggest that CD-ADHD could be conceptualized as an additive combination or a hybrid of both disorders (Schachar & Tannock, 1995; Oosterlaan, Logan, & Sergeant, 1998; Waschbusch, 2002; King et al., 2005), having the unique difficulties of each disorder and resulting in a more severe condition as compared to the pure condition of CD (Vermeiren, 2003) and ADHD (Connor, Steeber, & McBurnett, 2010).

It was found that when CD is accompanied also by ADHD, it becomes more

severe and persistent (Abikoff & Klein, 1992; Cantwell & Baker, 1992; Loeber, Green, Keenan, & Lahey, 1995, Loeber et al, 2000), and its onset is also earlier than the onset of pure CD alone (Moffitt, 1990; Loeber et al., 2000 and Vermeiren, 2003). The higher severity and persistence of antisocial behaviour of the comorbid group are clearly indicated by a comprehensive meta-analytic review that included 82 studies spanning from 1982 to 1998 on the issue of comorbidity between CD and ADHD (Waschbusch, 2002). The CD-ADHD group had significantly higher parent or teacher reports of conduct problems, and significantly higher score in ADHD symptoms than that of the pure CD group and pure ADHD group. The conduct problems of the CD-ADHD group (manifested at base-line age ranging from 4-16) were more likely to persist at follow-up (at age ranging from 7 to 31) than the ADHD group or the CD only group. Moreover, the average age of onset was youngest for the comorbid group (mean = 2.8 years), followed by the ADHD group (mean = 3.3 years) and then the CD group (mean = 5.2 years). In every study reviewed, the comorbid group and the ADHD group had significantly lower age of onset as compared to the CD group. A recent report on age of onset of delinquency and delinquent outcomes on the Pittsburgh ADHD Longitudinal study data set that followed 288 males with childhood ADHD and 209 comparison males from age 5-12 up to their age 11-28 indicates that the comorbid ADHD and CD group had

significantly worse delinquency outcomes in terms of committing a greater variety of delinquency and more severe delinquency as compared to control, ADHD only group, and ADHD+ODD group. The ADHD+CD group also initiated severe delinquency at a significantly earlier age than the ADHD only group and ADHD+ODD group (Sibley, Pelham, Molina, Gnagy, Waschbusch, & Biswas, et al., 2010).

Apart from a higher severity and persistence of antisocial problem and an early onset, CD-ADHD comorbid group was found to associate with multiple correlates that characterize both pure ADHD and CD. For example, Rutter et al. (1998c) reviewed that adolescent antisocial behaviours, when accompanied by hyperactivity or inattention, were marked by below-average cognitive abilities, poor scholastic performance, a strong association with social malfunction and poor peer relationships, and a beneficial response to stimulant medication (e.g.. methylphenidate), which are correlates associated with ADHD. Meta-analytic study of Waschbusch (2002) also found that the CD-ADHD group had significantly lower performance IQ relative to the control, which resembled the ADHD only group but not the CD group. Overall, the CD-ADHD group had significantly lower verbal IQ than all the groups (the ADHD, CD and control group).

Adverse family context and parenting that found for CD were also found in antisocial behaviors comorbid with ADHD. For example, Jensen et al., (1997) reviewed studies that reported relationship between family environmental factors and the comorbid condition of ADHD and CD (Barkley et al, 1989; Barkley et al., 1990, Barkely et al., 1991). It was found that ADHD children with aggression displayed more impaired family situations than non-aggressive ADHD children. Specifically, children with comorbid ADHD and aggression displayed more negative interactions with their mothers and their mothers were found to be more commanding and negative in interacting with their children. CD comorbid with ADHD was also significantly associated with more maternal depression and marital discord. Somewhat similarly, Pfiffner, McBurnett, Rathouz & Judice (2005) also found that ADHD comorbid with CD associated with more symptoms of parent psychopathology (paternal antisocial personality disorder and paternal depression) and problem of parenting practices including less positive maternal involvement, more maternal and paternal negative / ineffective discipline. Apart from dysfunctional parent-child processes and parenting, Waschbusch's review (2002) found that similar to the CD group, CD-ADHD group had a higher rate of parents with antisocial problem, relative to the control and the ADHD group. The comorbid group had significantly lower SES scores than the CD and the ADHD group; and similar to the studies reported

above, the CD-ADHD group was associated with more negative parent-child relationships as compared to the pure group of CD and ADHD.

Apart from the association with the adverse family and parenting background reviewed above, conduct problem comorbid with ADHD were found to be associated with nervousness and anxiety (Frick, Lillenfeld, Ellis, Loney & Silverthorn, 1999; Kuhne, Schachar, & Tannock, 1997; Pardini , Lochman, & Frick, 2003). Kuhne et al. (1997) found that in a sample of 91 children participated for treatment of ADHD, the ADHD-CD group (n=12) had significantly higher rates of parent-reported anxiety assessed by a semi-structured interview based on DSM anxiety symptoms or child-reported anxiety measured on a rating scale (the Revised Children's Manifest Anxiety Scale, Reynolds and Richmond, 1985), as compared to pure ADHD group (n=33). In a sample of 169 adjudicated adolescents with mean age of 15.81, Pardini et al. (2003) found that impulsive / conduct problem (I/CP) measured by the factor one of the Psychopathy Screening Device (renamed now as Antisocial Processing Screening Device) was positively related to behavioral dysregulation (such as easily fidget) and also positively related to measures of affective distress in stressful situation (as measured by Interpersonal Reactivity Index, Davis, 1983). Frick et al. (1999) found that also impulsive / conduct problem was significantly and positively

correlated with all the measures of anxiety in the study (significant r ranging from .17 to .35), including CBCL anxiety/depression scale, DSM-III-R symptoms of anxiety measured by structured interview in a sample of 143 children of age 6-13 referred to outpatient mental health clinic.

There are at least two ways that the above-reviewed findings of CD comorbid with ADHD are relevant to the subtyping of delinquency. Firstly, as antisocial behavior comorbid with ADHD is characterized by an early onset, a higher rate of comorbid ADHD thus could be expected to exist in the early-onset offending group but not in the adolescent-onset group. For example, Shaw, Lacourse, & Nagin (2005) traced the developmental trajectories of conduct problems and hyperactivity from ages 2 to 10 in a community sample of 310 boys from the low-income families recruited in the Pittsburgh metropolitan area and found that 55% of children following a chronic conduct problem trajectory also have persistent hyperactivity.

Secondly, many of the above reviewed associated characteristics of the comorbid group of CD and ADHD were similarly found in the life-course-persistent (early-onset) delinquent group reviewed above in this paper. As reported for the Dunedin study (Moffitt et al., 2001), both teacher-reported and parent-reported hyperactivity at the

ages of 5, 7, 9, and 11 significantly distinguished the early-onset life course persistent group and the adolescent-onset group, that the Dunedin members with early-onset antisocial behaviors also showed significantly more symptoms of hyperactivity and inattention in their childhood (hyperactivity measured with Rutter Child Scales supplemented with items about inattention, impulsivity, and hyperactivity from the DSM-III). They are also marked by significantly lower cognitive abilities (age 5 Binet IQ, ages 7, 9, and 11 WISC-R VIQ) and reading achievement (ages 7, 9, and 11), and they have exposed to more family adversities and inadequate parenting, obviously consistent with the characteristics hallmarking children with CD comorbid with ADHD reviewed above. In a Canadian sample of 316 children being followed from ages 6 to 15 and assessed on their antisocial behavior, attention-deficit-hyperactivity symptoms, and affiliation with deviant peers, a childhood-onset, an adolescent-onset and a low antisocial behavior trajectory were identified (Van Lier, Wanner, & Vitaro, 2007). It was found that the child-onset trajectory was predicted by the prior membership in the high attention-deficit- hyperactivity trajectory in childhood (ADHD symptoms measured yearly between age 6 to 10 by teacher's rating), whereas the adolescent-onset class was associated with increases in peer's antisocial behavior (assessed yearly from ages 10 to 13) but not attention deficit and hyperactivity problems.

It seems that these two groups, i.e., CD comorbid with ADHD group and the early onset life-course-persistent group, being named differently and researched with different interest could actually highly overlap. Rutter (2003) raised the possibility that these two groups are synonymous. It is thus plausible that the delinquent group associated with inattention / hyperactivity will form a homogenous subgroup in the early-onset life course persistent group that share similar features and risks with the comorbid group of CD-ADHD reported in the literature of child and adolescent disruptive behavior disorders.

It is noted that the distinguishing feature of ADHD is a primary deficit in response inhibition, an executive function largely belonging to function of the prefrontal cortex (Barkley, 1997; Nigg, 2001). It was proposed that deficit response inhibition (such as inhibition of ongoing response or a prepotent response) led to impairment in many important executive functions that depend on effective inhibition, including working memory, internalization of speech, self-regulation of affective-motivational arousal and reconstitution (reorganizing and generating new behavior) (Barkley, 1997). Impaired response inhibition of ADHD is shown by studies that used various performance-based laboratory tasks including basic go/no go

task (e.g. Borger & Van der Meere, 2000), stroop task (e.g. Semrud-Clikeman et al., 2000), directed forgetting task (e.g. Gaultney, Kipp, Weinstein, & NcNeil, 1999 ), the stop task (Kuntsi, Oosterlaan & Stevenson, 2001), the change paradigm (related to the stop task; e.g., Schachar, Tannock, Marriott, & Logan, 1995), negative priming (e.g. Gaultney et al. 1999,), and the delayed response tasks (Schweitzer & Sulzer-Azaroff, 1995), etc. According to the review of Rapport, Chung, Shore, Denney, and Isaacs (2000), amongst all the performance-based tasks, the stop task can best differentiate the performance between children with ADHD and normal children (with the largest average effect size = 1.03 among several performance-based tasks reviewed), as compared to other less reliable performance tasks such as finger tapping, WISC-R Mazes, trail Making test, etc. Also, an earlier meta-analysis of 8 studies (Oosterlaan, Logan, & Sergeant, 1998) and a more recent meta-analysis of 29 studies (Lijffijt, Kenemans, Verbaten, & Engeland, 2005) consistently revealed a significant difference between the ADHD subjects and the normal control on response inhibition as indicated by a longer stop signal reaction time (SSRT) on the Stop Task, rendering strong evidence to the hypothesis of response inhibition as core deficit of ADHD. Moreover, the review of Oosterlaan et al. (1998) found that subjects with comorbid ADHD and CD did not differ from the ADHD only subjects on inhibition indicated by the SSRT, rendering certain support to the hybrid hypothesis on the comorbid

condition mentioned above. Thus in this study the early-onset delinquent group comorbid with high ADHD symptoms is expected to show deficit in response inhibition on the stop-task.

*Overview of antisocial behaviours associated with high callous-unemotional traits.* Rutter et al. (1998c) reviewed that psychopathy, which is marked by a lack of socio-emotional responsiveness and behavioral deviancy, is another possible valid differentiator that matters in subtyping antisocial behaviors. By the time of Rutter et al.'s review in 1998, almost all the research on psychopathy was conducted with incarcerated adults. However, it is suspected that the emotional detachment that hallmarks adult psychopathy constitutes a meaningful differentiating feature in child and adolescent antisocial behaviours, as in adult antisocial problems. Since the review of Rutter et al. (1998c), advancement had been made with regard to study of a differentiator closely linked to psychopathy, i.e. callous-unemotional traits (CU traits) in child and adolescent antisocial behaviour. Many subsequent studies in this area directly focus on child and adolescent samples. Apart from the antisocial group accompanied by hyperactivity, recent research advances that children and adolescent with callous unemotional trait (CU trait) form a distinct group in children with conduct problems

CU traits refer to deficient affective / interpersonal responses such as lack of guilt and empathy, being uncaring to others, callous use of the others and showing shallow affects (Frick, Barry, & Bodin, 2000; Frick, 2006). Conceptually, CU trait is comparable to the first factor of Hare's two factor model of adult psychopathy (Hare, 1991). Specifically, in Hare's two factor model, factor one refers to callous and remorseless use of others whereas factor two refers to chronically antisocial lifestyle and impulsive behavioral style (Hare, 1970; Harpur, Hakstian, & Hare, 1988; Harpur, Hare, & Hakstian, 1989). Adult psychopathy had been proven to associate with particularly severe and violent antisocial behaviors (Hemphill, 2007; Porter & Woodworth, 2006). Based on these findings in adults, Frick and Hare firstly developed an instrument, the Psychopathy Screening Device (Frick, O'Brien, Wootton & McBurnett, 1994, now named as Antisocial Process Screening Device, APSD, Frick & Hare, 2001), based on the Psychopathy Checklist-Revised (PCL-R, Hare, 1991), a widely used instrument in adult, to explicitly focus on early form of psychopathy in children and adolescents. Like a downward extension from adulthood to childhood, it has been shown that similar distinct emotional and interpersonal style characteristic of adult psychopathy could be identified in children and adolescent. Consistent with the adult literature, Frick and colleagues (Frick et

al., 1994; Frick, Bodin, & Barry, 2000) identified two separable psychological dimensions with the APSD in clinically referred children and community sample: 1. callous-unemotional trait: similar to the description of factor one of Hare's model, CU refers to specific affective (such as lack of guilt, shallow affect) and interpersonal characteristics (such as no empathy, use of others for one's own gain) 2. impulsivity / conduct problem (ICP)– refers to poor impulse control and antisocial behavior. In the literature of adult psychopathy, impulsive and antisocial behavior characterized by the factor two of Hare's psychopathy was elevated in most adults with criminal history or antisocial personality disorder but not only specific to adult with high psychopathy (Hare, 1985). The unemotional and deficient affective responses (captured by factor one) are specific hallmark of psychopathy. Likewise, it is the high level of CU traits but not impulsive conduct problem (factor 2) that designates a distinct subgroup of adolescents who show severe antisocial behavior and characteristics of psychopathy. In a large German community sample of 1443 adolescents of age 13 to 18 (774 boys and 669 girls), a newly developed measurement of CU traits (Inventory of Callous-unemotional traits) was subjected to exploratory and confirmatory factor analyses, yielding a best fit for a high order single factor model nested with three lower order factors (callousness, uncaring and unemotional) held for both genders (Essau, Sasagawa, & Frick, 2006). In the same study, CU

traits were found to be significantly associated with aggressive and antisocial behavior in childhood. Increasing levels of CU traits were also associated with increased psychosocial impairment including school performance, peer relationship and self care / home duties.

Frick and Dickens (2006) reviewed 24 studies (published between 1996 and 2006) using child or adolescent sample, both community and adjudicated, and found that CU traits / psychopathic traits in general were associated with severe conduct problems, delinquency or aggression. Studies also showed that CU traits predicted severe and persistent antisocial behavior. For example, Frick, Stickle, Dandreaux, Farrell, & Kimonis (2005) identified four groups equal in size (n=25 for each group) from an initially 1136 community children for a four-year longitudinal follow up. The four groups with mean age from 12.20-12.68 at initial assessment included two high conduct problem groups with and without CU trait, a low conduct problem and high CU trait group, and a last group low on both conduct problem and CU trait serving as control. At each yearly follow-up, the high CU trait and conduct problem group showed the highest rates of conduct problems, self-reported delinquency, and parent-reported police contacts. This double high group accounted for more than half of all the police contacts reported across the last three points of data collections.

The high conduct problem low CU trait group was found to maintain a high rate of conduct problem but was not different from the control in terms of self-reported delinquency, whereas the second highest rate of self-reported delinquency was found for the high CU trait low conduct problem group in the initial assessment. In a recent study that recruited a large and nationally representative sample in the UK, 5326 children (age 5 to 16) participated for a 3-year follow-up. Of the two percent of the sample who diagnosed with DSM-IV Conduct Disorder, 46.1% of them were on high CU trait. As compared to the control, CD group without CU trait, and CU group without CD problem, this group showed most severe behavioral disturbance (more conduct problem, impairment and lower prosocial behavior) as measured by Strength and Difficulties Questionnaire (Goodman, 2001). At a 3-year follow up, this group showed highest risk of psychiatric outcome, particularly, CD diagnoses and police contact (Rowe, Maughan, Moran, Ford, Briskman, & Goodman, 2010). On the whole, evidence accumulates for the importance for CU traits in designating a more severe and persistent pattern of antisocial behavior within children and adolescents with antisocial behaviors (Frick & White, 2008).

It was also found that antisocial behavior marked by CU traits tended to have an early onset (Frick & Dickens, 2006). In the study of Silverthorn, Frick, & Reynolds

(2001), among 72 adolescents of age 13 to 18 being detained in a detention facility, 11 boys were defined into the early-onset group (at age 9 or younger) and 13 into adolescent offending group (at age 12 or older), based on the onset age of the first DSM-IV CD symptom or the first police contact (whichever age was earlier). It was found that the age of these early-onset boys (mean age: 13.91) was significantly younger than that of the adolescent-onset boys (mean age: 16.00), indicating that these childhood-onset boys engaged in antisocial behaviour serious enough to enter into the forensic setting much earlier than the adolescent-onset boys. In the study of Rowe et al. (2010), the CD + CU trait group (mean age = 11.9, n = 70) identified from the representative sample was significantly younger than the CD group low on CU trait (mean age = 13, n = 82). As far as subtyping of antisocial behaviour is concerned, these findings suggest the possibility that there is an overlapping between a subtype of antisocial behaviour marked by CU trait and the broad category of the early-onset antisocial behaviour, apart from the overlap between early-onset antisocial behaviour and a subtype marked by ADHD discussed so far.

Studies by Frick and others also found that the group marked by high CU trait demonstrates distinct temperamental and cognitive styles, which are postulated to be important correlates of severe antisocial behaviours and indicative of a distinct causal

pathway to antisocial behavior (Frick & Ellis, 1999; Frick, 2001, Frick, Cornell, Bodin, Barry & Loney, 2003; Frick & Dickens, 2006). Firstly, studies consistently found that children with high CU trait showed several emotional deficits, i.e., decreased reactivity to threatening and emotionally distressing stimuli, and deficits in cognitive and emotional empathy as compared to other antisocial adolescents (Blair, 1999; Kimonis, Frick, Fazekas., & Loney, 2006; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Pardini, Lochman, Frick, 2003). For example, in the study of Loney et al. (2003), emotional processing was signified by the subjects' speed of recognition for emotional (e.g. scare) versus non-emotional words (cup) in a lexical decision tasks. With an sample of 65 adolescents (mean age = 16.01) referred for diversion program for delinquent behavior, subjects with high level of CU traits showed reduced emotional reactivity as indicated by slower reaction times to negative emotional words as compared to impulsive-antisocial group and control, controlling relevant confounds such as IQ and word knowledge . In a non-referred sample of 50 6 to13-year-old children, children with conduct problem and high CU traits showed reduced reactivity to pictures involving distressing (e.g. crying child) and threatening content (e.g. vicious dog attacking), whereas children with high conduct problem but low CU trait showed a heightened reactivity to these provocative stimuli (Kimonis, et al.,2006).

Secondly, apart from the above emotional deficit, children with antisocial behaviors marked by CU traits also show more thrill and adventure seeking and absence of anxiety (Frick, Lilienfeld, Ellis, Loney & Silverthorn, 1999; Frick et al., 2003), as shown for example by the significant positive correlation between the CU trait and the preference for sensation-seeking behavior measured by Sensation Seeking Scale for Children (Zuckerman, kolin, Price & Zoob, 1964) in a study that recruited a referred sample of 143 6- to 13-year-old children (Frick & Lillienfeld, et al., 1999). Also in this study, CU trait was not significantly associated with any measures of anxiety (including CBCL anxiety/depression, overanxious symptoms and anxiety symptoms measured by the Diagnostic Interview Schedule for Children – 2.3, Shaffer, et al, 1996), and was found to be negatively correlated with anxiety after controlling for conduct problems .

A third distinct correlate of children with CU traits is poor passive avoidance learning, i.e., not inhibiting behavior that would result in punishment. Poor passive avoidance learning is often evident when a reward-oriented response set is primed, known as reward dominance (Barry, Frick, DeShazo, McCoy, Ellis, & Loney, 2000; Frick et al., 2003; Fisher & Blair, 1998; O'Brien & Frick, 1996). That children with high CU trait had a reward-oriented response set and poor response to punishment are

consistently replicated in studies with laboratory / performance-based task (Barry, et al., 2000; Frick et al., 2003; Fisher & Blair, 1998; O'Brien & Frick, 1996). One of the performance-based tasks, i.e., the card-perseveration task (Newman, Widom, and Nathan, 1985) is frequently used to test the reward dominance. The paradigm of the task involves presentation of 10 blocks of card games on which the respondents could choose to continue to view the cards or quit. Each block contains 10 trials thus resulting in 100 trials in total. The reward ratios over playing 100 trials of the cards decrease from 90% in the first block to 0% in the $10^{th}$ block. Thus in the card task the reward for viewing cards at the beginning is very high and so a reward-dominant response set is primed, but more and more punishment is in place as more cards are viewed until in the last block, the punishment is 100%. Studies showed that adults with psychopathic traits were more likely to continue card viewing despite the increasing of punishment to rewards over 10 blocks of 100 trials (Newman, Patterson, & Kosson, 1987).

Newman's paradigm has been extended to children and adolescents with CD, and more recently to children with CU trait, known as reward dominance of children with high CU trait highlighted above (Barry, Frick, DeShazo, McCoy, Ellis, & Loney, 2000; Frick et al., 2003; Fisher & Blair, 1998; O'Brien & Frick, 1996). In the study of

Barry et al. (2000), only children of ADHD and CD marked also by high CU trait played significantly more trials on the tasks than other groups including ADHD + CD group but with low CU trait, ADHD group and clinical control group. In a study which recruited 98 non-referred children with mean age of 12.36 (Frick & Cornell et al., 2003), high CU and conduct problem group, high CU only group, high conduct problem only group and control group were tested on reward dominance task. Children high on CU traits played significantly more trials than children low on CU traits, regardless of level of conduct problem. Frick, et al. (1999) found differential correlation between anxiety and the two dimensions of psychopathy. The second factor of psychopathy, impulsive/conduct problem (the second factor), which strongly associated with CD and ADHD symptoms, was positively correlated with anxiety whereas callous unemotional traits (the first factor) were not significantly correlated with anxiety. O'Brien and Frick (1996) found that only non-anxious conduct-problem children played significantly more trials in a reward dominance task than the other groups including anxious conduct-problem group, ADHD group and normal control.

The above review shows that the high CU group shares some similarities with the features reported for the early-onset group: their antisocial behaviours start early

in childhood (Rowe et al, 2010; Silverthorn, Frick, & Reynolds, 2001), and the antisocial behaviours are persistent ( Frick, Stickle, Dandreau, Farrell & Kimonis, 2005) and show to be more severe and aggressive (Caputo, Frick, & Brodsky, 1999; Kruh, Frick, & Clements, 2005, Rowe et al., 2010). There is as yet no longitudinal study that tracks the adult outcomes of the high CU trait group. However, Lynam, Caspi, Moffitt, Loeber and Stouthamer-Loeber (2007) using the longitudinal data of Pittsburgh Youth Study, showed that measure of CU traits at age 13 predicted adult measures of psychopathy at age 24, having controlled for childhood antisocial behaviour and other psychosocial risk factors. From the literature of adult psychopathy, it is known that psychopathy is associated with various poor outcomes such as chronic offending in violent and non-violent offences (Hemphill, Hare, & Wong, 1998; Salekin, Rogers, & Sewell, 1996) and higher recidivism (Hemphill, 2007), which resembles the poor adult outcomes of the early-onset life persistent group shown in the longitudinal studies by Moffit and others. It is also noted that one of the adult outcome of the males in the early-onset life-course-persistent group found by the Dunedin study is their significantly higher level of callousness in adulthood (significant across self-report and informant's report) (Moffit, Caspi, Harrington & Milne, 2002). This adds support to the possibility that the early-onset group in fact comprises those with high on CU traits in childhood. A recent finding

(Dandreaux & Frick, 2009) does show that adjudicated adolescents of the childhood-onset delinquent group showed significantly higher level of CU traits as compared with adolescent-onset delinquent group.

However, previous studies showed that antisocial behaviour marked by CU traits were different from the early-onset antisocial behaviors with respective to the following two features. First, antisocial behaviour with high CU traits was found to be less associated with the background of dysfunctional parenting practices, but noted that studies that looked at the relation between CU traits and social context such as family adversities and parenting were relatively few. One such early study was that of Wootton, Shelton, and Siverthorn (1997) which tested the relationship between conduct problem, CU traits and parenting. They hypothesized that children with high CU traits would be relatively unresponsive to typical socialization practices because of their unique cognitive and temperamental style and that their antisocial behaviour would develop independent of parenting practices. On the contrary, children without CU traits would be susceptible to inadequate child rearing and socialization processes and their conduct problem would be more strongly associated with ineffective parenting. One hundred thirty-six children of ages 6 to 13 referred for mental health service and 30 community control subjects matched in age, gender

and ethnicity were recruited. It was found that for children with high CU traits (categorical cut off defined at a score of 10 or above on the CU factor of Antisocial Process Screening Device which is the upper quartile of the total sample), association between their conduct problems and ineffective parenting was not significant. Children with high CU traits showed high rates of conduct problems, regardless of parenting, whereas increased conduct problems was significantly associated with ineffective parenting only for children without significant level of CU traits. In another word, CU trait had moderated the relationship between antisocial behaviour and poor parenting. This moderating role of CU trait was further tested and partially supported by another study which included a larger sample of children (n=243, mean age = 8.24) (Oxford, Cavell, & Hughes, 2003). Using CU trait as continuous score for analyses, it was found that teacher-rated externalizing problems and peer-rated aggression were significantly related to ineffective parenting only for children with relatively low CU traits but not for children with high CU traits, though this pattern was not found for parent-rated externalizing problems and when CU trait was used as a categorical variable. That children of high CU traits exhibited great levels of externalizing behavior that were unrelated to social context such as ineffective parenting links to the suggestion for a more genetic base for antisocial behaviors tied to this traits, with support from recent genetic studies (Nathalie, Rijsdijk, Fruhling,

McCrory, &Viding, 2010; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Frick, & Plomin, 2007; Viding, Jones, Frick, Moffitt, & Plomin, 2008).

Apart from a lack of association with ineffective parenting, there is also a lack of low intelligence in children who have conduct problems and high CU traits, whereas low IQ was commonly found in the early-onset antisocial behavior (Barry, et al., 2000; Loney, Frick, Ellis, & McCoy, 1998). In a sample of 117 clinic-referred children of age 6 to 13, three groups including conduct problem CU trait group, conduct problem group and control group were compared on verbal IQ, performance IQ and full scale IQ measured by Wechsler Intelligence Scale for Children-Revised (Wechsler, 1974), controlling for the effect of age (Loney et al., 1998). It was found that the conduct problem only group had a significantly lower Full Scale IQ than the control (mean IQ = 89.71 vs. mean IQ = 97.22) whereas the Full Scale IQ of the conduct problem CU trait group was intermediate (mean IQ = 92.63, higher than CP only group but lower than the control). Similar trend was also found for verbal IQ and performance IQ. In the study of Barry et al. (2000), the full scale IQ of the ADHD-CD-low CU trait group was significantly lower than that of the clinic control (mean full scale IQ = 84.42 vs. 95.22), whereas the ADHD-CD-high CU trait group (mean full scale IQ = 93.38) was not different from the clinic control. The lack of association with

ineffective parenting and lack of intellectual deficit contrasts with the background risks reported for the early-onset antisocial behaviors, and also and the early-onset antisocial behaviors marked by ADHD. That the conduct problem marked by high CU trait was not associated with anxiety and emotional distress also contrasted with CD comorbid with ADHD, which showed positive correlation with measures of nervousness and anxiety (Frick, et al., 1999; Loney, et al., 2003).

*The importance of both ADHD and CU traits in subtyping early-onset antisocial behaviour.* Literature reviewed above points to the importance of ADHD in differentiating a subtype of antisocial behaviour. As noted already, conduct problem comorbid with ADHD has distinct features closely resembling the early-onset delinquent group, including early onset and high severity of antisocial behavior, lower IQ and increased family adversities and disrupted parenting. Another line of literature reviewed above also points to the importance of CU traits in isolating a homogenous group within children and adolescents with antisocial behaviour. Similar to conduct problem comorbid with ADHD, antisocial behaviours marked by CU traits were characterized by a high severity and persistence and an early-onset, but were associated with distinct temperamental and cognitive profiles (such as reward dominance, no association with anxiety and a lack of intellectual deficit) and

parenting risks (less associated with ineffective parenting). However, very few studies have examined on the role of ADHD and CU trait simultaneously with regard to antisocial behaviours. A recent study of Waschusch and Willoughby (2008) tested whether ADHD and CU simultaneously moderated the association between conduct problem and impairment (such as academic impairment, student-child relationship) and aggression such that high score on both ADHD and CU would associate with more aggressive and impairment with a sample of 214 children (mean age = 8.38). It was found that CU trait and ADHD did not act synergistically but was independent moderator of conduct problem and impairment and aggression. For example, for the association between conduct problem and teacher-child relationship, ADHD was an independent moderator that only at higher level of ADHD, increased conduct problem was associated with increased problem of teacher-child relationship whereas CU was not a moderator. On the other hand, for conduct problem and proactive aggression, CU trait is an independent moderator where at higher level of CU traits the magnitude of association between conduct problem and proactive aggression was stronger. These findings indicate that both ADHD and CU traits are important but independent moderators of antisocial behaviours and its correlates or impairment, suggestive of antisocial behaviours developed through different pathways.

The differential roles of ADHD and CU traits on antisocial behaviours are also

suggested by the findings on different cognitive processes that possibly underlie antisocial behaviours comorbid with ADHD versus antisocial behaviours marked by CU traits. As already reviewed, there is evidence that similar to the deficit characterizing ADHD, antisocial behaviors comorbid with ADHD are also associated with inhibition deficit shown on the Stop Task (Oosterlaan et al., 1998; Lijffijt, et al., 2005), whereas antisocial children with CU traits show perseverated responses (continued viewing of cards) in the face of increasing punishment on the Card Playing Task, known as reward dominance (Barry, Frick, DeShazo, McCoy, Ellis, & Loney, 2000; Frick et al., 2003; Fisher & Blair, 1998; O'Brien & Frick, 1996). Conceptually, inhibition deficits (inhibition of a pre-potent motor response) shown by the Stop Task and perseverated responding shown by the Card Playing Task can be regarded as similar in terms of deficient control over behaviour. Both can be conceptualized as "impulsivity" or "disinhibition", a key construct central to the conception of a broad, multi-faceted category of disinhibitory psychopathology including childhood disruptive behaviour disorders (Beauchaine & Neuhaus, 2009; Zaparniuk & Taylor, 1997). Based on a review of cognitive and temperament/personality literatures pertaining to inhibition/disinhibition, Nigg (2000, 2006) differentiated two different types of disinhibition presumably involving different brain systems, of which one was executive disinhibition and the other was motivational inhibition. The deficit inhibition shown in the Stop Task hypothesized

to characterize the offending group with ADHD belonged to executive inhibition, which was defined as intentional control or suppression of response in the service of higher order or longer term goals and involved more heavily on prefrontal structures and associated cortical-cortical connections in the brain (Nigg, 2000, 2001). The perseverated responses shown in the Card Playing task indicated deficit of motivational inhibition, which was a bottom-up cessation of behaviour driven by anxiety/fear in the context of a novel situation or cues for punishment and involved more heavily on limbic structures and subcortical-subcortical connections in the brain (Nigg, 2000, 2001). As shown in the Card Playing task, motivational inhibition involves explicit motivational contingencies (reward and punishment) and taps on the individual's processing of reward and punishment (i.e., consequences of behaviours). It is hypothesized in this study that the inhibition deficit shown in the Stop task is uniquely related to the ADHD offending subgroup, while the reward dominance shown in the Card Playing task is uniquely associated with the CU offending subgroup. It is further conjectured that antisocial behaviours marked by ADHD and CU traits are respectively underlain by deficiencies of different inhibitory processes, despite superficial similarities on overt "disinhibitory", or "impulsively" behaviours. They are distinguishable by experimental tasks, namely, the Stop Task and Card Playing task that tap on underlying cognitive processes.

## Focus of the current study: the hypothesized subtypes of adolescent antisocial behaviours and their associated group features

The present study intends to test a taxonomy of antisocial behaviour with an adjudicated sample and non-delinquent control in Hong Kong by examining whether the hypothesized delinquent subtypes could be distinguished by differential group features. Based on the studies of Moffitt and colleagues, Patterson and colleagues, and many other studies reviewed in this paper, there is evidence for an early-onset group of antisocial behaviour as differentiated from the adolescent-onset / limited group. Accordingly, these two groups are different in onset, persistence and adult outcome of antisocial behaviour. They are also characterized by different background risks of delinquency. Within the limitation of a cross-sectional design, the current study does not aim at distinguishing these two groups with regards to the future persistence and adult outcomes of their antisocial behaviour. Focus will be put on the differential background risks that hypothesized to be associated with these two groups. According to both Moffitt and Patterson, the early-onset group is a severe group characterized by the worst background risks. Up to the stage of adolescence, their development (particularly prosocial behavior) and adolescent adjustment are predicted to be the worst. Moreover, two important clinical features

hypothesized to mark two distinct subgroups within the early-onset group, namely, inattention/hyperactivity and callous-unemotional traits, are expected to be prominently found in the early-onset group but not the adolescent-onset group. In sum, the following hypotheses are generated regarding the early-onset group:

Early-onset offending group (EO):

(1) showing worst background risks of delinquency including cognitive risk, family adversities and deficient parenting (including poor parental monitoring)

(2) showing worst adolescent development in terms of prosocial behaviour and adolescent adjustment

(3) showing a higher rate of ADHD and CU traits.

The early-onset group will be compared to the non-delinquent control and the adolescent-onset group to test the above hypotheses.

Another major task of this study is to test the group nature of the adolescent-onset group, noted that Moffitt and Patterson proposed different etiology of the adolescent-onset offending and thus different group features could be hypothesized. Briefly, Moffitt hypothesized that the adolescent-onset offending arises as when adolescents facing the maturational gap, seek to accelerate adult's role and privileges. These adolescent-onset youngsters have normal pre-delinquent development and are not different from the normative peers in terms of background. Patterson hypothesized that similar to the early-onset group, the adolescent-onset group also suffers from disrupted family processes and parenting risks, but in an intermediate

degree. Their prosocial behaviour is marginally developed and their adjustment is also marginal. Both Moffitt and Patterson similarly hypothesized the key role of deviant peer as risk of adolescent-onset delinquency. This study will test whether the adolescent-onset group is characterized by group features hypothesized by Moffitt's developmental taxonomy or by Patterson's coercion model as follows:

Based on Moffitt's theory, the adolescent-onset group will have the following features:

1. no difference from the non-delinquent control group in terms of background risks including family adversities and inadequate parenting and cognitive risks
2. normative pre-delinquent development as indicated by prosocial behaviour and adolescent adjustment
3. affiliation with deviant peers as compared to non-delinquent control group

According to the Patterson's theory however, this group will have the following features:

1. intermediate between the early-onset group and the non-delinquent control in terms of background risks including family adversities and inadequate parenting, and particularly poor parental monitoring

2. better pre-delinquent development (as indicated by prosocial behaviour) as compared to the early-onset delinquent group but less well-developed as compared to non-delinquent group
3. affiliation with deviant peers as compared to the non-delinquent group

Integrated two lines of studies separately on conduct problem with ADHD and antisocial behaviors marked by CU traits, a subgroup associated with ADHD and a subgroup marked by CU traits are hypothesized to coexist within the broad category of early-onset antisocial behaviour. Previous studies have successfully showed the distinct features of antisocial behaviour associated with hyperactivity, and with high CU traits respectively in separate groups of child and adolescent sample. No study has identified these two groups simultaneously within the same sample. The current study tries to achieve this task. Two laboratory tasks, that is, the Stop Task and Card Playing Task will be adopted to test whether these two groups show specific deficits, i.e., inhibition deficit that hallmarks ADHD, and poor passive avoidance learning / reward dominance that hallmarks CU traits. Based on the previous findings reviewed in this paper, the following group features of the two early-onset subtypes are hypothesized as follows:

Early-onset group 1: early-onset offending associated with ADHD (EADHD)

1. showing severe delinquent behaviour on indexes of delinquency
2. association with cognitive impairment (such as indicated by IQ)
3. association with family adversities and parenting risks
4. association with anxiety
5. association with deficient behavioral inhibition as shown on the Stop Task

Early-onset group 2: early-onset offending associated with high callous unemotional traits (ECU)

1. showing severe delinquent behaviour on indexes of delinquency
2. lack of association with deficient parenting
3. lack of cognitive impairment as indicated by low IQ
4. lack of association with comorbid anxiety
5. association with reward dominance as shown on the card-playing task using Newman's paradigm (Newman, Widom, and Nathan, 1985).

**End of chapter I**

# Chapter II Method

## Participants

It is planned that male adolescent subjects of ages 12 to 17 will be recruited from two kinds of settings, with the offender subjects being recruited from Tuen Mun Children and Juvenile Home (TMCJH), which is a centralized detention and correctional / probation facility under the management of the Social Welfare Department, HKSAR Government, to form the clinical group, and the non-offending subjects being recruited from two mainstream secondary schools in community to form the comparison group. Juveniles placed in the custody of the TMCJH age from 10 to 16 years (by their admission) and are all sentenced by court from 6 months to 30 months because of having committed criminal offences. Common types of offences committed are violent offences (e.g. common assault), and property offences (e.g. theft and burglary), while other offences include status / rules violation (e.g. breach of probation order, infringed copyright), drug-related offences and sex offences (e.g. indecent assault). For the current study, subjects with intelligence lower than 70 (assessed by the short-form of HK-WISC or Chinese version of WAIS-III) and comparison subjects with a criminal history will be excluded from the study.

The current study will compare different hypothesized offending groups and control on a list of risk variables and external correlates by planned contrast following an analysis of variance. The offending groups would be identified according to set criteria and the group size would likely be unequal. Moffitt & Caspi (2001) reported that the effect sizes of the group difference between the two offending groups (i.e., the LCP vs. the AL) as indicated by Cohen's *d* on 26 risk factors ranged from small to large (small effect size for 15 risk factors, medium for 3 risk factors and large for 6 risk factors). A preliminary analysis indicated that with the power set at 0.8 and alpha set at .05, to detect two-group differences with unequal group size on variables with large effect size will require a sample size below 50. But for those with medium and small effect size, it will require a sample size of over 100. For example, to detect the group difference on "hyperactivity reported by parent" of which Cohen's *d* reported is 1.11 between the LCP and the AL group (Moffitt & Caspi, 2001), the total sample size required for the two groups is 39, whereas to detect group difference on reading difficulties with Cohen's $d = .57$ (Moffitt & Caspi, 2001) will require a sample size of 112 for the two groups. Considering the need to maintain reasonable statistical power and balancing the feasibility of data collection, the current study set the target sample size for the offender sample as 120 and normal control as 60, a total sample of 180 for this study.

As planned, data from a total of 188 male adolescents had been collected. Of them, 123 are offender subjects from TMCJH and 65 controls from two mainstream secondary schools (Lee Kau Yan Memorial School and Fukieu Secondary School, Siu Sai Wan). Five subjects from the TMCJH had IQ below 70 and two subjects from the mainstream secondary schools had history of criminal offence. These seven subjects were excluded from the data analysis, resulting in 118 offender subjects and 63 controls, a total of 181 subjects for the full sample. It was also planned that the parent / major-care taker of the subjects would be recruited to participate in this study so that multi-sources data could be obtained. However, the response rate of the parents was low. Many parents could not be contacted or refused to make themselves available to attend a data collection procedure estimated to last for two hours. However, considering that parent's report is a more valid source of information to derive ADHD diagnosis (Shaffer, Fisher, Lucas, Dulcan, & Schwabstone, 2000, also see measure), a second round recruitment for parent's participation was implemented, limiting data collection to only the part related to ADHD in the Chinese Parent version of the Diagnostic Interview Schedule for Children 4.0 (P-DISC-IV). The time required was estimated to be less than one hour. Consequently, P-DISC-IV ADHD data could be obtained from 78 parents of 118

offender subjects and 33 parents of 63 control subjects. Thus a total of 111 P-DISC-IV ADHD data could be combined with those from DISC-IV administered to youths (Y-DISC-IV) to yield an ADHD diagnosis basing on the combined scoring algorithm of DISC-IV in this study.

For the 118 offender subjects in the current study, their mean age is 14.21 (*SD* = 0.96, *range* = 12 – 16). The index offences committed leading to the subjects' adjudication are listed in Table one. The most common offences committed are theft (27.1%), breach of probation order (19.5%) and common assault (17.8%). Up to the time of data collection, average number of months receiving training in the TMCJH is 3.42 (*SD* = 3.93 months, *range* = < 1 month - 29 months). For the 63 control subjects, their mean age is 14.29 (*SD* = 1.02, *range* = 12 – 17). There is no significant difference between the offender group and the control group in terms of their age (see Table 2). The mean education level of the offender group is 7.53 which is significantly lower that of the control group (mean education level = 8.86, $t$ = 10.82, $df$ = 160, $p < .001$) (see table 2).

Table 1
Index offense committed by the offender sample ($n$ = 118)

| Offense | Frequency | Percentage |
|---|---|---|
| 1. Theft | 32 | 27.1 |
| 2. Burglary | 10 | 8.5 |
| 3. Common assault | 21 | 17.8 |
| 4. AOABH | 8 | 6.8 |
| 5. criminal damage | 4 | 3.4 |
| 6. Robbery | 5 | 4.2 |
| 7. Breach of probation order | 23 | 19.5 |
| 8. possession of dangerous drug | 4 | 3.4 |
| 9. trafficking dangerous drug | 3 | 2.5 |
| 10. indecent assault | 2 | 1.7 |
| 11. unlawful sexual intercourse | 2 | 1.7 |
| 12. other | 4 | 3.4 |
| Total | 118 | 100 |

Note: AOABH = Assault Occasioning Actual Bodily Harm

Table 2

Descriptive and comparison of age and education level between the offender group ($n$ = 118) and the control group ($n$ = 63)

| variables | Offender group<br>N = 118<br>Mean / frequency<br>(SD / %)<br>Range | Control<br>N = 63<br>Mean / frequency<br>(SD / %)<br>Range | t test / p |
|---|---|---|---|
| Age (year) | 14.21 (0.96) | 14.29 (1.02) | 0.47 (ns) |
| | 12 – 16 | 12 - 17 | |
| Education level | | | |
| Primary 4 | 1 (0.8%) | / | |
| Primary 5 | / | / | |
| Primary 6 | 9 (7.6%) | / | |
| Form 1 | 50 (42.4%) | 2 (3.2%) | |
| Form 2 | 45 (38.1%) | 14 (22.2%) | |
| Form 3 | 9 (7.6%) | 38 (60.3%) | |
| Form 4 | 4 (3.4%) | 9 (14.3%) | |
| Number of years of education | 7.53 (0.93) | 8.86 (0.69) | [a]10.82*** |

Note:
*p ≤ .05, ** p ≤ .01, *** p ≤ .001, ns = non-significant
a = equal variances not assumed, adjusted $df$ = 160.3

The percentages of having the DISC past-year diagnoses of the three disruptive behaviour disorders, i.e., Conduct Disorder (CD), Oppositional Defiant Disorder (ODD), and Attention Deficit Hyperactivity Disorder (ADHD) of the offender and the

control group are listed in table 3. Proportion of having the diagnosis of CD, ADHD and ODD was significantly higher in offender group as compared to the control group as indicated by the significant chi squares. Strength of the association between the diagnosis and the group ranged from small to strong (significant Φ ranges from 0.18 to 0.69), with the correlation between CD and the group being a very strong one (Φ = 0.69).

Table 3

Offender group and the control compared on the percentage of DISC diagnoses of CD, ODD, and ADHD

| DISC diagnosis | Offender group $n = 116$ Frequency (%) | Control $n = 63$ Frequency (%) | $\chi^2$ / p | Phi (Φ) / p |
|---|---|---|---|---|
| CD | 88 (75.9%) | 2 (3.2%) | 86.29*** | 0.69*** |
| ODD | 30 (25.9%) | 2 (3.3%) | 13.49*** | 0.28*** |
| ADHD[a] | 14 (12.1%) | 1 (1.6%) | 5.6* | 0.18* |

Note:
DISC = NIMH Diagnostic Interview Schedule for Children-Version 4.0 (DISC-IV)
CD = DISC diagnosis of Conduct Disorder
ODD = DISC diagnosis of Oppositional Defiant Disorder
ADHD = DISC diagnosis of Attention Deficit Hyperactivity Disorder
[a] = diagnosis based on parent and youth version of DISC-IV
*p ≤ .05, ** p ≤ .01, *** p ≤ .001

## Procedure

Graduate research assistants were trained and assisted in the data collection. Data was collected through one-to-one administration of three kinds of measures: 1. structured interviews 2 questionnaires 3 performance-based tasks: computerized task and short-form IQ test. Data collection for each adolescent subject required about 180 minutes, which was divided into 2 separate sessions. In the first session, structured interviews and IQ assessment would be conducted, which required approximately 100 minutes, and in the second session, questionnaires and computerized tasks would be administered, which required about 80 minutes (see appendix 1). For most of the subjects, the two sessions of data collection were carried out on two separate days within a period of seven days. For subjects who completed data collection on the same day, a break of at least half an hour would be arranged between the two sessions so as to avoid the effect of fatigue. The entire data collection was carried out in a standardized manner in which standardized instructions and steps were followed (see appendix 1). In both TMCJH and school setting, the venue for data collection was an enclosed quiet room equipped for the subjects to be interviewed, to fill questionnaires and to do the performance-based tasks. For the parents who participated in this study, the part related to ADHD of

the P- DISC-IV was administered in the venue of TMCJH and schools, following a standardized procedure, which requires about 45 minutes.

Informed consent of all subjects had been obtained. The theme of the study, the procedure, use of data and confidentiality were stated in the consent letter and explained to all participants. In the TMCJH particularly, the adolescents and the parents were told that their willingness to participate in the current study would not affect the clinical services or the legal proceeding that the adolescents would receive. And also the data obtained in the study would not be used for other purposes (such as for sentencing). All the adolescent residents in the probation home and in the reformatory school of the TMCJH were target subjects and were invited to participate in the study. A briefing was conducted by the researcher to all residents collectively to introduce the study, before having their consent obtained. In the two secondary schools, teachers followed a randomized procedure to select students and invited their participation. The study and the data collection were introduced firstly by the teachers to the students based on the standard information provided by the researcher. The theme of the study, procedure of data collection, use of data and confidentially were explained again to the participating students before they signed the consent form. The whole data collection lasted for six months, from

September 2009 to March 2010.

Data collection in the TMCJH for the current research purpose had been approved by the Social Welfare Department of the HKSAR Government. The current study was also approved by the Survey and Behavioural Research Ethics Committee of the Chinese University of Hong Kong.

## Measures

*DSM diagnosis of CD, ODD and ADHD.* DSM-IV CD, ODD and ADHD were derived by administration of the youth version of the NIMH Diagnostic Interview Schedule for Children-Version 4.0 (DISC-IV). Symptoms related to CD and ADHD would be used for group formation, which will be detailed later. The DISC-IV (Shaffer, Fisher, Lucas, Dulcan, & Schwabstone, 2000) assesses DSM-IV psychiatric symptoms and disorders in children and adolescent aged 6 to 17 years. It was released in 1997 for field use where its first version (DISC-I) was developed in 1979 in response to the need for an instrument to be used in surveying the prevalence of child and adolescent mental disorders. It covers over 30 DSM-IV clinical diagnoses grouped under six diagnostic sections: the Anxiety Disorders, Mood Disorders,

Substance-Use Disorders, Schizophrenia, and Miscellaneous disorders. The DISC-IV is a highly structured and respondent-based interview that could be used by non-clinicians (Shaffer, Fisher, Lucas, Dulcan, & Schwabstone, 2000). The interviewer reads the exact wording of the questions to the respondent and codes the responses accordingly. The interview begins with the introductory module to elicit demographic data and teach the respondent about the answer format and time frame covered by the questions of the subsequent diagnostic modules. To help the respondent to understand different specific time period to be focused on (e.g. one month ago, half year ago, one year ago, since you are five year old, etc.) and recall relevant information, the interviewer goes through a time line exercise (whole life time line and past year chart) with the respondent in which the respondent relates past salient events (such as when to start current schooling) with reference to particular time frame. These salient events help to mark different time periods covered in the subsequent interview and will be frequently referred to. In the subsequent diagnostic modules, the response options for most questions that focus on the major symptoms of the disorders are "yes" or "no" (module of CD: *Have you ever run away from home overnight?*). On some follow-up / contingent questions there are some other responses such as "sometimes" or "somewhat" option or close-end frequency choice (e.g. *Did you do this more than five times in the last year?*). In the current study, the

DISC-IV was administered by the trained research assistant using the computer programme of the DISC-IV.

Shaffer, Fisher, Lucas, Dulcan, & Schwabstone (2000) reviewed the reliability and validity of the DISC. In a clinical sample of 82 children and adolescents aged 9 to 17, the test-retest reliability Kappa (mean interval between tests = 6.6 days) for child version of past-year DISC-IV ADHD, ODD and CD are .42, .51, and .65 respectively, while for parent-version DISC-IV ADHD, ODD and CD, the test-retest reliability is .79, .54, and .43 respectively (Shaffer, Fisher, Lucas, Dulcan, & Schwabstone, 2000). The test-retest reliability for parent-report ADHD (kappa = .79) is noted to be higher than that of the child report (kappa =.42), while for the diagnosis of CD, Kappa (.65) is higher for youth version than that of parent version (kappa = .43). On the other hand, when the test-retest reliability of DISC- symptom counts are examined (instead of the categorical diagnosis), for most diagnoses including ADHD, the reliability indicated by the intra-class correlation coefficients (ICC) becomes better. ICCs for the youth report of DISC-IV ADHD, ODD and CD are .65, .64 and .83 respectively, whereas ICCs for the parent report of DISC-IV ADHD, ODD and CD are .84, .85, .63 respectively. It is noted that again the ICC for the youth report of CD (.83) is higher than that of the parent's report (.63), where ICC

for youth report of ADHD (.65) is lower than that of the parent's report (.84).

Validity indicated by the agreement between the DISC diagnoses and clinician rating was reported to be moderate to very good for parent and youth version, both separately and combined (Shaffer, Fisher, Lucas, Dulcan, & Schwabstone, 2000). Amongst the diagnosis of ADHD, CD and ODD, agreement between youth report of ADHD and clinician rating has a low kappa (Kappa = .27) whereas Kappa is .72 for the agreement between parent reported ADHD and clinician rating. For the diagnoses of ODD and CD, the agreements between the youth report and clinician rating (Kappa = .54 and .77 respectively) were comparable to the agreements between the parent report and the clinician rating (Kappa = .59 and .74 respectively).

As regards the Chinese DISC-IV, Ho, Leung, Lee, Tang, Hung & Kwong, et al. (2005) reported comparable test-retest reliability of the Chinese version as compared to the English version. Similarly, the test-retest reliability kappa for youth report of ADHD is low (kappa =.25) as compared to that of the parent report (Kappa = .81). In the current study, the DISC diagnoses of CD, ODD, and ADHD and their symptoms count are derived by the youth version. But for ADHD, parents' information would be combined to derive the diagnosis, considering the above reported findings on the reliability and validity of the ADHD diagnosis.

*Callous Unemotional Traits (CU traits).* CU traits were measured by the inventory of Callous Unemotional Traits (ICU, Essau, Sasagawa, & Frick, 2006). The ICU is a recently developed instrument to assess callous unemotional traits in children and adolescent. It is designed to overcome some potential psychometric limitations of the CU scale of the APSD (Antisocial Process Screening Device, Frick & Hare, 2001), which is a widely used questionnaire to assess CU trait but has only low to moderate internal consistency (e.g. coefficient $\alpha$= 0.49 in Loney, Frick, Clements, Ellis., & Kerlin, 2003; coefficient $\alpha$ = 0.52 in Pardini, Lochman, & Frick, 2003), probably due to small number of items (6 items) and the 3-point rating scale (Essau, Sasagawa, & Frick, 2006; Dandreaux & Frick 2009). Using some original items of the APSD CU scale, the ICU was developed with 24 items on a four-point Likert scale from 0 (not at all true) to 3 (definitely). The ICU measures the deficient affective experience hallmarking psychopathy such as lack of empathy, lack of guilt, being uncaring, showing shallow affects, etc. The scale showed adequate internal consistency with alphas equal to .77 in a large community sample ($n$ = 1443) of 13- to 18-year-old non-referred German adolescents (Essau, et al., 2006), and .81 in an American sample of juvenile offenders ($n$ = 248) of age between 12 and 20 (Kimonis, Frick, Skeem, Marsee, Cruise, & Munoz, et al.,2008). In terms of factor structure and construct validity, exploratory and confirmatory factor analysis yielded a best fit

for a high order single factor model nested with three lower order factors (callousness, uncaring and unemotional) held for both genders in a large German community sample of 774 boys and 669 girls (Essau et al., 2006). The ICU showed expected associations with aggression, delinquency, conduct disorder, and personality traits (such as sensation seeking) in several studies (Essau et al. 2006; Kimonis et al. 2008). Since the ICU was developed based on the items of the CU subscale of the APSD and both similarly measures the callous unemotional traits of children and adolescents, they should be correlated. This was shown by the study of Kimonis et al. 2008, that the total score of the ICU was found to be moderately correlated with the CU subscale of the APSD with $r = .45$ in the detained sample.

In the current study, the self-report version of the ICU are translated into Chinese and approved for use by the author (Frick, P., the University of New Orleans, personal communications, 2008). Cronbach's $\alpha$ of the ICU total is .78 in the current sample, indicating adequate internal consistency. The ICU total would be used to form a subgroup of high CU trait in the broad early-onset offending group.

*Self delinquency.* Self delinquency was measured by the Self-reported Delinquency Scale (SRD) (Elliott, Huizina & Ageton, 1985), a standardized

instrument developed by Elliott and Huizinga (1989) for the National Youth Survey, which is a longitudinal study of delinquent and antisocial behaviour in the American youth population. It had been adopted in other large-scale studies that focus on antisocial behaviour and delinquency across samples, such as New Zealand sample in the Dunedin Study (Moffitt & Caspi, 2001) and US sample in the Pittsburgh Youth Study (Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi,1998; Loeber, Farrington, Stouthamer-Loeber, Moffitt, Caspi, & Lynam, 2001). The SRD includes a general delinquency scale, an index offence scale and a minor delinquency scale (Elliot, Ageton, & Huizinga, 1985). Respondents were asked to report on the frequency of the delinquent acts in the past one year. The test-retest reliabilities of the SRD measures in the National Youth Survey are in the range from .70 to .95 (Elliott, Huizinga, & Morse, 1987). Elliot et al. (1987) reviewed the validity of the SRD and found that the self-reported scores on the SRD could discriminate well between groups with different levels of involvement in the justice system and also the majority of those with official records of delinquency reported most of their known offences on the SRD. Review on the self-reported measures of delinquency suggested that amongst various administration methods (such as face-to-face interview, telephone interview, self-administered questionnaire) no one method is consistently better than the other but self-administered questionnaires resulted in

higher levels of missing data (Elliott & Huizinga, 1989). In the current study, the General Delinquency Scale of the SRD, which is consisted of 24 items and the scale on Drug Use (5 items) of the SRD were administered in the interview with the subjects. The 24 delinquent acts include offences involving aggression, property crime, sex crime, and drug. In the interview, subjects were asked to report on whether they had, and if they had how many times they had committed the specific delinquent behaviour in the past one year before they had been adjudicated. Two kinds of score which describe different aspects of juvenile delinquency would be derived: 1. variety score: amongst various delinquent acts how many were committed at least once during the past one year (range 0-34). 2. frequency score: the frequency of the sampled delinquent acts. It is noted that inquiry on the use of two more illicit drugs is added (Ecstasy and cough medicine) on the Drug Use Scale, apart from the original 5 items (Hallucinogens, amphetamines, barbiturates, Heroin, Cocaine) so as to include illicit drug commonly used in HK (Narcotics Division, Security Bureau of Hong Kong, 2008). In this study, the variety score has a high correlation with the frequency score with correlation = .91 ($p$ = .001, $n$ = 178), indicating those committed higher frequency of delinquent acts also committed more variety of delinquency.

*Criminal attitude.* Criminal attitude was measured by the Attitude toward Delinquency Scale (ATD) (Vitaro, Brendgen, & Tremblay, 2000) and administered in the interview with the subjects. The ATD measures the degree of the subject's favourable attitude towards deviant behaviours (including stealing at home and outside home, drug use, skipping school, runaway from home and property damage). The scale is consisted of six items on which the subject indicates their attitude towards each item by rating from 0 (no problem at all) to 3 (very problematic). The score derived ranges from 0 to 18, with higher score indicating a more favourable attitude delinquency. It was found in a longitudinal study that adolescents' favourable delinquent attitude was related to subsequent higher levels of delinquent behaviour (Vitaro, et al., 2000). Alphas of this scale found by Vitaro, et al. (2000) were .80 and .81 with male adolescent subjects of age 11 and 12 years. In the current sample, alpha for the scale is .82.

*Family risks.* Subjects were administered a structured interview to obtain information on the family risk variables including 1. socio-economic status of the family as indicated by parents' education, income, unemployment in the last year and history of receiving social security assistance in the last year 2. parents' criminality 3. parents' mental health problem 4. number of family transitions from birth to age

11 5. number of year of single parenting from birth to age 11 6. child abuse of parents. It is noted that the subjects were inquired on their childcare history since birth in details according to the interview protocol to assist in deriving data on family transitions and year of single parenting before age 11.

*Parenting risks.* Parenting risks were measured by the Alabama Parenting Questionnaire (APQ: Shelton, Frick, & Wootton, 1996). The questionnaire includes 42 items that assess parenting practices by parent report and child report format. Respondents give rating to each specific parenting behaviour on a scale ranging from 0 (never) to 5 (always). Five domains of parenting could be derived from 35 items of the APQ, which include: 1. parental involvement 2. positive parenting 3. poor monitoring and supervision 4. inconsistent discipline, and 5. corporal punishment. Apart from these five scales, 7 other items were included to ask about other specific discipline practices other than corporal punishment (scale 5), so that corporal punishment items were not asked in isolation from other forms of discipline method. Among the five domains of parenting derived, two of them including parental involvement and positive parenting signify positive side of parenting and lower score on these scales indexed less positive parenting behaviours. The other three indicate poor parenting practices including poor monitoring and

supervision, inconsistent discipline, and corporal punishment. Higher score on these scales indicate higher level of poor parenting practices. Coefficient alphas across a 2- to 4-week time period range from .70 to .87 for self report, indicating that the scales provide a stable estimate of the parenting domains (Shelton, Frick, & Wotton, 1996). A number of studies also showed that the APQ scales are associated with conduct problems with different samples including youth in community (Dadds, Maujean, & Fraser, 2003), clinic-referred sample (e.g. Hawes & Dadds, 2006), and inpatient samples (Blader 2004).

The Chinese-translated version of the APQ was approved for use by the author (Frick, P., the University of New Orleans, personal communications, 2008). Internal consistency of the APQ subscales was found to be satisfactory in the current study, except for the subscale of inconsistent parenting. Cronbach's alpha was .85 for parental involvement (mother), .88 for parental involvement (father), .86 for positive parenting, .70 for poor parental monitoring , and .88 for corporal punishment, whereas $\alpha$ was .39 for inconsistent parenting. The subscale of inconsistent parenting was thus not used for subsequent analysis. Intercorrelations among the other four subscales ranged from $r = -.16$ ($p = .03$, $n = 174$) between poor parental monitoring and mother's parental involvement, to $r = .65$, ($p < .001$, $n = 174$) between mother's

parental involvement and parent's positive parenting, all in expected directions (see Chapter III results).

*Cognitive risks.* Cognitive risks in terms of intelligence was assessed by administering the short-form of the Hong Kong Wechsler Intelligence Scale for Children (HK-WISC, Wechsler, 1981) to subject with age below or equal to 15. Four subtests, namely, Similarities, Vocabulary, Block Design and Object Assembly were administered. Local norm on the short form of HK-WISC is available for use (Education Bureau, HKSAR, 1984, internal documents). For subjects with age equal to or above 16, four subtests including Information, Vocabulary, Block Design, Matrix Reasoning that compose the short form of the Wechsler Adult Intelligence Scale – third edition, Chinese version (WAIS-III, Chinese version, Wechsler, 2002) were administered. This four-test combination has good reliability and validity, with both reliability and validity coefficient above 0.90 (Sattler & Ryan, 2001). In an unpublished local study, the IQ derived by this 4-subtest short form has no significant difference from the overall IQ derived with the full version of WAIS-III (Chinese version) in a sample of 189 adults with both normal and sub-normal IQ (Wan, 2006). Subjects with a full-scale IQ below 70 (defined as mental retardation) would be excluded from the data analysis. Cognitive risks in terms of history of repeating a

class and history of receiving special training were obtained in the structured interview with the subjects.

*Affiliation with deviant peers.* Affiliation with deviant peers was measured by the following two questionnaires administered in the interview with the subjects:

*Peer Delinquency Scale (PDS, Keenan, Loeber, Zhang, Stouthamer-Loeber, & Van Kammen, 1995)* The PDS assesses the level of deviant peer group affiliation by having the subjects reported their friends' engagement in various disruptive and delinquent behaviour. It consists of 12 items on each the subjects rated how many of their friends (all, most, half, few or none) engaged in the specific deviant behaviour in the past 12 months. The PDS was firstly developed for use in the Pittsburgh Youth study. The scale ranges from 0, which means none of his/her friends has done the deviant behavior, to 4, which means all of his / her friends have done so. Any rating above none (0) was considered as indicating some level of delinquent peer association. The level of peer delinquency on each deviant behavior was summed. The coefficient alpha for the scale was .84 (Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi, 1998). Concurrent association between peer deviant behaviours and the subjects' disruptive behaviour was reported. Boys' exposure to peer deviant

behaviour also significantly predicted boys' later initiation of disruptive behaviours of all kinds including covert, overt and authority conflict (Keenan, Loeber, Zhang, Stouthamer-Loeber, & Van Kammen,1995; Farrington, Stouthamer-Loeber, Moffitt, Caspi, & Lynam, 2001). Cronbach's alpha of the scale is .89 ($n$ = 177) in the current study.

*Delinquency of My Best Friend Scale (DBF, self-constructed).* This self-constructed scale indicates the level of deviant behaviour of the study members' best friend. Study members first identified his best friend and reported on the name (optional), gender, schooling status and class level of his best friend. He then rated how often his best friend had committed on each of the 23 norm-violating and delinquent behaviours in the past one year on a 4-likert scale (no, yes but very few, sometimes, and frequent). Score can range from 0 to 69. Higher score indicates higher level of deviancy of the best friend. Cronbach's alpha of this scale is .93 ($n$ = 169) in the current study. As found by the study of Vitaro, et al. (2000), the deviancy of the best friend (measured in classroom setting by Pupil Evaluation Inventory; Pekarik, Prinz, Liebert, Weintraub, & Neale, 1976) significantly predicted adolescents' subsequent delinquent behaviour, with the adolescents' previous level of delinquent behaviour being controlled.

*Adolescent adjustment difficulties and prosocial behaviours.* Adolescent adjustment difficulties and prosocial behaviours were measured by the Strengths and Difficulties Questionnaire (SDQ, Goodman, 1997). The SDQ comprises 25 items on which respondents rate "true", "somewhat true" and "not true". The 25 items forms five subscales. Four are clinical scales that measure respectively peer problem, emotional symptoms, conduct problem, and hyperactivity. The remaining one scale measures prosocial behaviour. The four clinical scales sum to a total difficulties score ranging from 0 to 40 (SDQ total score). The SDQ also generates an impact score (range 0 – 10) which indicates distress and social impairment because of the SDQ difficulties (a total impact score of 2 or more could be classified as abnormal, and 1 as borderline). In this study, the SDQ total difficulties and SDQ impact score would be used as index of adolescent adjustment difficulties. Prosocial behaviors of adolescents would be indexed the SDQ prosocial behaviour scale. Reliability and validity of the SDQ were tested with a large and representative community sample of 10438 British children and youths of age 5 to 15 (Goodman, 2001). The five-factor structure of the SDQ was supported. The internal consistency of the subscales was generally satisfactory, with Cronbach's $\alpha = .80$ for the SDQ total difficulties, .66 for emotional symptoms, .60 for conduct problems, .67 for hyperactivity scale, .66 for prosocial behaviour, and .81 for the impact sore. Cronbach's $\alpha = 0.41$ was notably

low for peer problem scale. Average test-retest reliability was at least .62 after 4 to 6 months. In terms of validity, the SDQ subscales showed expected agreement and correlations with independent psychiatric disorders. The odd ratios for having a psychiatric disorder in high- versus low-risk group (as defined by the Youth SDQ score) was 6.2, average across all scales, noted that the prosocial behaviour scale had a weak association with psychiatric diagnosis (with the odd ratio being 1.7). In the current sample, the internal consistency of the SDQ subscales and total score are as follows: emotion symptom $\alpha$ = .58, conduct problem $\alpha$ = .56, hyperactivity $\alpha$ = .68, peer problem $\alpha$ = .27, prosocial behaviour $\alpha$= .65, impact score $\alpha$ = .55, SDQ total difficulties $\alpha$ = .72. Similar to that reported by Goodman (2001), the internal consistency for peer problem scale is particularly low, as it is found in the current study with an alpha of .27 for this scale. The impact score also had a comparatively low alpha than that reported by Goodman (2001), while alpha for other scales are comparable.

*Anxiety symptoms.* Anxiety symptoms were measured by the Anxious / Depressed subscale of the Youth Self Report (YSR, Achenbach, 1991). YSR is a parallel instrument of the Child Behaviour Checklist which is an instrument widely used across cultures to assess on emotional, behavioural and social problems of child

and adolescents of age from 6 to 18 (Ivanova, Achenbach, Dumenci, Rescorla, Almqvist, & Weintraub, et al., 2007). Two broadband syndromes, i.e., externalizing problem and internalizing problem could be derived from its 8 syndromes on the YSR. The Anxious/Depressed Subscale is one of the 8 syndromes and is also one of the three syndromes that forms the internalizing problems (the other two being: Withdrawn and Somatic Complaints). The scale comprises 16 items that cover anxious (such as nervous, high-strung) and dysphoric behaviours (such as unhappy, sad, cries a lot). Respondents give rating on a 3-point scale (not true, somewhat true and very true) on each specific behaviour for the past 6 months. The Chinese version of the YSR and the data of local Hong Kong norm are available for use (Department of Psychology, CUHK, 2004). The Chinese YSR has satisfactory test-retest reliability, with intra-class correlation = .81 for the internalizing problem, (Leung, Kwong, Tang, Ho, Hung, Lee, & Hong, et al., 2006). In terms of validity, the YSR internalizing problem predicted well the overall clinical status and the corresponding anxiety/mood disorders. Youth appears to be valid informant reporting on symptoms relating to internal emotional states (Leung, Kwong, Tang, Ho, Hung, Lee, & Hong, et al., 2006). Internal consistency of the YSR is satisfactory, with $\alpha = .87$ in the current sample.

*Reward dominance.* Reward dominance was measured with the card playing

task (based on Newman & Kosson, 1986.) The card playing task is a laboratory paradigm developed by Newman and colleagues (Newman & Kosson, 1986; Newman, Patterson, & Kosson, 1987), who observed that antisocial individuals demonstrated insensitivity to cues for punishment when a competing and dominant reward set was established. The basic paradigm of the card playing is that: for the 10 sets of cards played (10 cards for each set, with a total of 100 cards contained in the game), there is the presence of initially high rate of rewards (90%) in the first block, decreasing by 10% for each block, to a low rate of reward (0%) and a high rate of punishment (100 %) for the last block of 10 cards. Newman et al. (1987) found that prison psychopaths played significantly more cards and lost more money than non-psychopaths, demonstrating what they named as "response perseveration", a maladaptive pattern characterized psychopaths. This paradigm has been extended to children and adolescent samples and across various studies, children and adolescents with conduct disorder played significant more cards as compared to normal control (Frick & Loney, 2000).

The card playing task adopted in the current study follows the above paradigm. The task consists of 100 cards. The subject would win 1 score when the card viewed was a face card (Jack, Queen, King) and would loose 1 score if the card viewed was a

number card (1-9). Each card was presented together with a question appearing on the same screen: "are you going to view the card?" The subject then decided to view the card or quit the game by pressing on the relevant box on the screen using the mouse. A remark of "You win!" or "You loose!" would appear after each card was viewed, presented together with a cumulative mark on the same screen. The subjects were instructed to play as many cards as they want and to quit the game when they wish. The subjects were told that the cards in the game was not a standard deck which had 52 cards and there was no need for them to count the number of trials when they played the game. The subjects were shown that they would win a large prize (a large pack of chocolate) if they obtained a score of 24 or above in the game, a medium prize (a pack of chocolate of medium size) if they obtained a score of 20 or above (20-23), and a small prize (a pack of chocolate of small size) if they obtained a score of 16 or above (16-19). The game was displayed on a notebook computer with a screen of 9 inches x 11 inches. The subject will do two practice trials, with one showing the face card and the other showing the number card before the start of the game.

The order of the loosing cards in each block of 10 cards is arranged with a randomized procedure generated by computer program. In 500 computer-simulated

card playing tasks, the maximum score ranges from 20 to 24. If the subjects do not stop after around 75$^{th}$ trials, they will start to have a minus mark and if all 100 cards are viewed they will obtain a mark of -10. The dependent measures are the total number of cards viewed before quitting the game and the final score gained.

*Response inhibition.* Response inhibition was measured by the Stop task (Verburggen, Logan, & Stevens, 2008).) The Stop task is widely used in investigation of child and adolescent psychopathology involving response inhibition deficit such as Attention Deficit Hyperactivity Disorder (Lijffit, Kenemans, Verbaten, & Engeland, 2005, Verbruggen & Logan, 2008). The basic paradigm of the stop task requires the subject to respond (to press the corresponding key) as quickly as possible in response to the go stimulus but to withhold the response when the go stimulus is followed by a "stop" signal (an auditory tone). The interval between the go stimulus and stop signal (known as stop signal delay, SSD) can be varied so as to alter the difficulty of response inhibition. The probability of successful inhibiting the go response will be higher if the delay of the stop signal is brief but lower when the delay of the stop signal increases. In another word, it will be easier to inhibit the go response if the stop signal is presented soon after the presence of the go signal but as the delay increases, it will be harder or even impossible to inhibit the ongoing go

response. Logan (1994) used a race model to describe the performance of the response inhibition, in which if the go process is completed before the stop process, the response is emitted and could not be stopped, but if the stop process finishes before the go process, the response is successfully inhibited. The inhibition function indicated by the "stop-signal reaction time" (SSRT) describes the speed of the stop process, which is covert and could be estimated by subtracting the mean delay of the stop signal (stop–signal delay, SSD) from the mean reaction time (MRT) (Boucher, Palmeri, Logan, & Schall, 2007; Verbruggen & Logan, 2008). Numerous studies have found deficient response inhibition in clinical samples of ADHD (clinical groups have significantly longer SSRT) as compared to normal control sample with large effect size larger (Lijffit, et al., 2005; Oosterlaan, Logan, &Sergeant, 1998; Rapport, Chung, Shore, Denney, Isaacs, 2000).

The stop-task used in the current study is a free software called STOP-IT provided by Verbruggen, Logan, Stevens (2008) and the key dependent measures including the SSRT, SSD, MRT are derived by the analyzing program called ANALYZE-IT, also provided by these authors. The stop-task is run with a notebook computer with a screen of 9 inches x 11 inches. The task consists of a block of 60 practice trials, followed by 3 blocks of experimental trials (60 trials for each block).

Thus altogether there are 180 experimental trials. The subject will be instructed to press the "Z" key with their left index finger whether they see the presence of "□" on the screen, and to press "/" with their right index finger when they see the presence of "O" on the screen, both as quick and as accurate as possible. The subject is also instructed "not" to press any key when they hear a tone "Doo" even when the stimulus "□" or "O"appears but the subject is instructed not to wait for the presence of the stop signal to respond.

## Group assignment

*Overview*. The major interest of this study is to differentiate subtypes of adolescent antisocial behaviour. Major hypotheses had been derived regarding the differential features of different offending groups. The first important task is thus to assign members into different groups according to a priori criteria. Specifically, members of the offender group will be first assigned into two broad offending groups, namely, the early-onset offending group (EO) and the adolescent-onset offending group (AO). They were then compared with the control group on a list of hypothesized risks and external correlates. It is also hypothesized that within the early-onset group there exist two distinct subgroups, i.e., those with high comorbid

ADHD symptoms (EADHD) and those with high callous unemotional traits (ECU). Members of the early-onset offending group were further assigned to these two groups according to the set criteria. These two groups, together with the adolescent-onset group and the control were compared on the hypothesized risks and external correlates.

*Formation of the early onset offending group (EO) and adolescent-onset offending group (AO).* All the subjects collected from the TMCJH had been convicted of a criminal offense and all belonged to the offending group. Relevant to note, individuals on the early-onset offending path could not be distinguished from those on the adolescent-limited offending path in terms of their level of delinquency committed in the period of adolescence (Moffitt & Caspi, 2001). But the EO group has early onset of conduct problem in childhood whereas the conduct problem and delinquency of the AO arise later in adolescence. Information on conduct problem prior to offending and the age of onset of such conduct problem are thus crucial for the group assignment. In this study, the offending members were assigned based on the presence of symptoms of DSM-IV Conduct Disorder obtained through the DISC-IV. The earliest age of onset of the CD symptom reported was counted for group assignment. Specifically, In the DISC-IV interview, subjects went through each CD

symptom and for those symptoms being endorsed, the age of onset would be further inquired for each symptom (e.g. how old were you the first time you got into trouble for staying out too late? How old were you the first time you ran away overnight? etc.). It was observed in the data collection that the subjects had no difficulties reporting the age of onset as once suspected. They could report the age or the school grade which they studied when they started to have a specific symptom. Age of onset might be different for different CD symptoms and the earliest age of onset reported for a particular CD symptom amongst all would be counted to signify the onset of conduct problem in childhood. In this current study, the age of onset ranged from the earliest age of three, reported for theft, to age 15, reported for various CD symptoms (including theft, staying out overnight, runway, lie, truant, broken into house, destroyed property, bullying / threatening other, use weapon to harm other, initiate fighting). The average of the earliest onset age of CD symptom is 9.11 years (SD = 2.28 years).

The second important step for group formation is to decide on the cut off age that demarcates the early-onset versus adolescent-onset conduct problems. It is noted that in the literature there is a lack of consistency as to how to define these two groups in terms of age of onset. The cut off age could range from 10 to 14. For example,

onset of CD symptoms prior to the age of 10 is defined as the childhood-onset type in DSM-IV (APA, 2000). Validity study of the CD subtypes based on the age of onset of 10 found a steep decline in aggression occurring around that age but the onset age was not related to non-aggressive conduct symptoms, family income, rating of child global impairment, school expulsions and suspensions (Lahey, Loeber, Quay, Applegate, Shaffer, Waldman, et al., 1998). In the Dunedin study, children were designated antisocial if they displayed antisocial behaviours in at least three of the assessment point at ages 5, 7, 9, 11 and persistent across situations (reported by parents and teachers on the Rutter Child Scales). In this case, age 11 is the latest age that childhood antisocial behaviour is counted to define the LCP group (Moffitt et al, 2001). In the Oregon Youth study, boys with first arrest prior to age 14 were defined as early-onset offender (Patterson, 1996; Patterson & Yoerger, 1997). Carroll et al. (2006) defined institutionalized adolescents with history of conviction prior to 12 in early-onset delinquent group and those who with no offending history until the age of 13 into the late-onset group in a sample of 129 Australian adolescents with mean age around 15. Dandreaux and Frick (2009) used the cut off age of 11 to differentiate the early-onset versus the adolescent-onset group (inclusive of 11) in a group of adjudicated adolescent boys with age ranging from 11-18 (mean age: 15.15). Dandreaux and Frick (2009) also tested the groupings using different cut off (both age

10 and 12, and those with first onset of severe conduct problem in the questionable age range 11-13 being eliminated), apart from grouping based on selected cut off age of 11. They found that the results obtained were consistent across groupings based on different cut off age. There is by far no one specific age that could be concluded as superior than the other cut off age in terms of predictive validity in differentiating early-onset versus adolescent-onset antisocial behaviour.

As indicated by the developmental model reviewed so far (Moffitt & Patterson), the early-onset antisocial behaviours arise from the transaction between a high risk child and a high risk environment and thus their conduct problems mostly manifest before adolescence. On the other hand, the presence of risk factors in the adolescent stage (parenting breakdown according to Patterson, and deviant peers according to both Moffitt and Patterson) is hypothesized to fuel the start of adolescent-onset antisocial behaviour. The start of adolescence is thus an important reference point. Adolescence could be marked by biological, psychological and social change (Coleman & Hendry, 1999). Particularly in Hong Kong, when children complete elementary education and enter into secondary school, they are allowed more freedom, autonomy and responsibilities, typically signifying the start of adolescence as differentiated from childhood where more parental involvement is required. In the

local scenario, children of age 11 have mostly completed their primary education and by age 12 most of them enter into secondary school. This is indeed the case in the present sample, where 50.9% of the total sample were age 12 when admitted to Form one (24.1% were age 13 or older and 25% were age 11). Taking into these considerations, age 11/12 is adopted as a cut off. This means that subjects with the onset of CD symptoms at or before age 11 would be assigned into the early-onset offending group where those displayed CD symptoms at or after age 12 will be assigned into adolescent-onset group. With this criterion, 95 subjects (82% of the offender group) were assigned in the EO group versus 21 subjects (18% of the offender group) in the AO group. It is noted that two subjects in the offender group has missing data on the age of onset and thus could not be classified.

*Further formation of the two distinct groups (EAHDH and ECU) wtihin the early-onset offending group.* The EADHD members are those who have both conduct problems and also a high level of inattention and hyperactivity symptoms in childhood. In this study, inattention and/or hyperactivity symptoms were that of DSM-IV ADHD symptoms assessed through the DISC-IV. The DISC-IV derives both categorical diagnosis (past-year diagnosis of ADHD) and a count of symptoms as a dimensional measure (ADHD Symptom score). Higher symptom score indicates

higher level of ADHD symptoms. For the categorical diagnosis of ADHD, the age of onset (some symptoms present before age 7) and pervasiveness of the symptoms (some symptoms present in two or more setting such as school and home) are also criteria to meet, apart from presence of the symptoms. Both ADHD diagnosis and ADHD symptom score could be used to identify EO members with high ADHD. However, in this study, only 13 subjects in the EO group had the ADHD diagnosis (one in the AO group). To balance the need of retaining a reasonable number of subjects in the group and thus power for statistical analysis, the symptom score would be used to define the group of high ADHD. ADHD symptom score ranges 0 to 23. The mean score of the early-onset group is 9.08 (*SD* = 4.82, *range* = 0 -21). Subjects with the symptom score of 13 (or above) were above 75$^{th}$ percentile of the EO group and were assigned to the EADHD group, resulting in a group with 24 subjects.

The ECU members are those early-onset group members with high CU traits. The total score of the Inventory of Callous Unemotional Traits (ICU, Essau, Sasagawa, & Frick, 2006) was used as a cut off criterion. The ICU total score ranges from 0 to 72. In the validation study of the ICU (Kimonis & Frick, et al., 2008), the mean total score obtained by 98 incarcerated male adolescents is 26.07 (*SD* = 8.05). In the

present sample, the mean total score of the EO group is 29.88 (*SD* = 7.95, range = 10-47, *n* = 89). Subjects with the ICU total score of 37 were above 75$^{th}$ percentile of the group and were assigned into the ECU group, which consequently has 17 subjects.

It is important to note that an overlap of 6 subjects was found for these two groups, i.e., these six subjects had two attributes, high CU trait and high ADHD and were in both two groups. These 6 subjects were removed from the groups, resulted in two independent groups of EADHD group with *n* = 17, and ECU group with *n* = 11 for comparison.

## Data analysis

Comparison among groups were done through a series of one-way analysis of variance ( ANOVA) with group as a between subject variable. Difference between specific pair of groups was examined by planned contrast to test the hypothesized difference on the group features. Bonferroni correction was performed to keep the family-wise error level at .05 on two measures indexing self delinquency (SRD total score and SRD variety score, .05/2 = .025) and two measures indexing peer delinquency (peer delinquency total and delinquency of best friend, .05/2 = .025).

All the statistical analysis was conducted with SPSS 16.0 version for windows. As the size of the groups derived in this study is sharply unequal (*n* of EO = 95, AO = 21 and control = 63) and some of the group has a small group size (*n* of EADHD = 17 and ECU =11), and if the groups also have unequal variance and so the assumption of homogenous variances being violated (as showed by the significant finding of the homogeneity of variance test), ordinary *F* ratio will not be a robust measure of group difference. When large sample variances are associated with small group sizes, *F* statistic is liberal, i.e., the null hypothesis is rejected falsely too often (Stevens, 2009). In such case, the Welch adjusted *F* will be referred to instead of the ordinary *F*, where the degree of freedom is adjusted (Cohen, 2001). The Welch adjusted F test is an available option in the SPSS. Apart from ANOVA, multinominal logistic regression were performed to predict class membership of different offending groups using selected background risk variables and external correlates as predictors. The details will be described in the results.

**End of chapter II**

# Chapter III

# Results

## Screening out invalid data and data transformation

As stated before, five subjects with IQ lower than 70 in the offender sample and two subjects having a criminal history in the control had been excluded from analysis, resulting in 118 offender subjects and 63 controls, a total of 181 subjects for the full sample. Response frequency of this data set had been scrutinized. Items regarding the education level of both parents and their respective income were not used for data analysis because of high percentage of missing / "don't know" responses (percentages of "don't know" responses / missing on mother's income, mother's education, father's income, and father's education level are 45.3%, 29%, 42%, and 28% respectively). All subjects were administered the stop task (Verburggen, Logan, & Stevens, 2008) as planned. Examination of data found that the percentage of missed responses for 17 subjects are greater than 10%, indicating that these subjects were not responding to a significant proportion of the trials (Verbruggen, 2009, personal communications). The STOP-IT program uses a tracking procedure to adjust the stop-signal delay (SSD: the time interval of the stop signal following the go

stimulus). The SSD will be increased by 50 milliseconds when inhibition is successful and decreased by 50 milliseconds when inhibition is unsuccessful, so that subjects can stop half of their responses, resulting in a probability of 0.5 in successful inhibition used for a reliable SSRT estimation. For 15 subjects, the probability of inhibition is significantly different from 0.5, which violates the assumption of the estimation method used by the program of the STOP-IT (Verbruggen & Stevens, & Logan, 2008). These data are considered to be invalid and could mess up the estimate of the stop signal reaction time (Verbruggen, 2009, personal communications). As a result, data of 29 subjects on the stop task were excluded from analysis (7 subjects had both missed responses more than 10% and the probability of successful inhibition significantly different from 0.5). Data from 13 subjects on the card playing task were also considered invalid as these subjects (all from TMCJH) obtained knowledge regarding the paradigm of the card-playing task from other already involved participants and pre-selected a strategy to maximize the chance to obtain the reward. Their tendency of reward dominance could not be truly reflected in the card task. Their data was thus excluded from analysis. The percentage of missing for any items used for analysis is less 10%, except for the three items that tap on criminality of the father: whether the father had been imprisoned (percentage of missing = 11.5%), whether the father had history of being arrested

(percentage of missing = 16%), whether the father had criminal offense record (percentage of missing = 17.5%). It is so designed that response to one of these three items will give information on the father's criminality (endorsement to any one of these three items will be assigned one score to form a composite index on parents' criminality). Examination of the data found that no one subject had missing data on all the three items. Retaining the data of these three items despite of their relatively high percentage of missing is still considered to be useful to capture the information on the parent's criminality.

A series of ANOVA will be carried out to compare different groups on the purported background risks and various external correlates. Though F test is robust against the violation of the assumption of normality with respect to the protection of type I error (false rejection of null hypothesis) (Stevens, 2009), data with serious skewness were transformed. Skewness within the range from 1 to -1 indicates an approximately normal distribution of the variable (Leech, Barrett, & Morgan, 2005). Variables with skewness exceeding this range were transformed by taking its square root in this study. As a result, square root transformation had been done on the following 9 variables that had skewness coefficient larger than 1 (all positively skewed) before group analysis: 1. Self-reported delinquency, 2. SDQ impact score,

3. parents' criminality, 4. SES, 5. number of family transition before age 11, 6. single parenting before age 11, 7. child abuse of parents, 8. mental health of parents, 9. anxiety measured by Youth Self Report.

## Correlations among risk variables and external correlates

Correlations (Pearson *r)* among risk variables being grouped in a same class and other external correlates for group comparison are reported below and listed in the lower part of the diagonal in the tables.

*Delinquency.* The three variables indicating the subjects' delinquency (SRD total frequency, SRD variety of delinquency, and the criminal attitude) were all positively correlated with each other (see table 4). Correlation between the total frequency of delinquency and the variety of delinquency was high ($r = .91, p \leq .001$), indicating subjects who committed higher level of delinquency also committed more variety of those acts. Higher score on the ATD indicates more favorable attitude towards delinquent act, which was also correlated positively with both variety ($r = .50$, $p \leq .001$) and frequency of delinquency ($r = .47, p \leq .001$). Thus, for this sample, the more favorable the delinquent attitude, the higher frequency and more variety of the delinquent acts committed.

Table 4

Correlations among variables indexing "delinquency"

| Variables | 1 | 2 | 3 |
|---|---|---|---|
| 1. Frequency of delinquency in the past 12 months (SRD) | | . | |
| 2. variety of delinquency in the past 12 months (SRD) | .91*** ($n$ = 178) | | |
| 3. criminal attitude (ATD) | .47*** ($n$ = 177) | .50*** ($n$ = 177) | |

Note:
SRD = Self-reported Delinquency Scale
ATD = Attitude Towards Delinquency Scale
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Adjustment and prosocial behavior.* The two variables pertaining to the adjustment in adolescence (SDQ total difficulties and SDQ impact score) was correlated with $r = .45$, $p < .001$. Thus higher level of SDQ total difficulties was associated with more perceived distress and impairment. Prosocial behavior was not correlated with distress and impairment but negatively related to the SDQ total difficulties with a relatively small $r = -.18$, $p \leq .01$, indicating higher level of prosocial behavior only tended to have less SDQ total difficulties in this sample (see table 5).

Table 5
Correlations among variables pertaining to "adjustment difficulties and prosocial Behavior

| Variables | 1 | 2 | 3 |
|---|---|---|---|
| 1. SDQ total difficulties | | | |
| 2. SDQ impact (distress/impairment) | .45*** ($n = 180$) | | |
| 3. .SDQ prosocial behavior | -.18** ($n = 180$) | -.12 ($n = 181$) | |

Note:
SDQ = Strength and Difficulties Questionnaire
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Cognitive risks.* Among three variables pertaining to cognitive risks, IQ was negatively correlated with times of repeating a class ($r = -.30, p \leq .01$), indicating that those with higher IQ had fewer class repeating. History of receiving follow-up and training was not significantly correlated with the other two variables (see table 6).

Table 6
Correlations among variables pertaining to "cognitive risks"

| Variables | 1 | 2 | 3 |
|---|---|---|---|
| 1. Over IQ (HK-WISC/WAIS-III) | | | |
| 2. Times of repeating a class | -.30*** ($n = 180$) | | |
| 3. History of special follow-up / training | .05[a] ($n = 171$) | .11[a] ($n = 170$) | |

Note:
[a] $r$ known as point biserial correlation between dichotomous variable and continuous variable
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Family adversities.* Correlations among the six variables grouped in the family adversities (see table 7) ranged from $r = .66$ (between years of single parenting before 11 and number of family transition before 11) to $r = .16$ (between parents' criminality and parents' child abuse).

Table 7
Correlations among variables indexing "family adversities"

| Variables | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| 1. years of single parenting before 11 | | | | | | |
| 2. number of family Transition before 11 | .66*** ($n$ = 175) | | | | | |
| 3. low SES | .20** ($n$ = 175) | .23* ($n$ = 174) | | | | |
| 4. Parents' criminality | .26** ($n$ = 175) | .30*** ($n$ = 173) | .21** ($n$ = 175) | | | |
| 5. parents' child abuse | .18* ($n$ = 165) | .19* ($n$ = 175) | .27** ($n$ = 166) | .16* ($n$ = 165) | | |
| 6. Parent's mental health problem | .09 ($n$ = 174) | -.02 ($n$ = 173) | .20** ($n$ = 175) | .06 ($n$ = 175) | .04 ($n$ = 165) | |

Note:
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Parenting risks.* Correlations among parenting risk variables indicated by different dimensions of the APQ are listed in Table 8. Parental involvement, both for the father and mother, was positively correlated with parents' positive parenting with $r = .65$ and $r = .60$ respectively, noted that these two sets of variables signified positive

side of parenting and higher score indicates higher level of these positive parental behaviors. The other three parenting subscales (poor parental monitoring, inconsistent parenting, and corporal punishment) captured poor parenting practices. Higher score indicates higher level of these poor parenting. As can be seen, these parenting behaviors had low correlation or negative correlation with the three variables indicating positive parenting practices, for example, mother's involvement was negatively correlated with poor parental monitoring with $r = -.16, p \leq .05$.

Table 8
Correlations among variables pertaining to "parenting risks"

| Variables | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1. APQ mother's involvement | | | | | |
| 2. APQ father's involvement | .46*** (n = 160) | | | | |
| 3. APQ positive parenting | .65*** (*n* = 174) | .60*** (*n* = 165) | | | |
| 4. APQ poor parental monitoring | -.16* (*n* = 174) | -.08 (*n* = 165) | -.07 (*n* = 179) | | |
| 5. APQ inconsistent parenting | .14 (*n* = 172) | .04 (*n* = 162) | .17* (*n* = 176) | .45*** (*n* = 175) | |
| 6. APQ corporal punishment | .07 (*n* = 174) | -.04 (*n* = 165) | -.04 (*n* = 179) | .20** (*n* = 179) | .24** (*n* = 175) |

Note:
APQ = Alabama Parenting Questionnaire
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Family adversities and parenting risks.* Correlations between the variables in the class of family adversities (year of single parenting before 11, number of family transition before 11, low SES, parents' criminality, parents' child abuse and parents' mental health problem) and the six variables of parenting risks measured by the APQ were computed. There was no significant correlation between these two sets of variables, except one, i.e., APQ corporal punishment was significantly correlated with parents' child abuse, with $r = .32, p \leq .001$.

*Deviant peer affiliation.* Two variables indicating deviant peer affiliation and influence was highly correlated with $r = .74, p \leq .001, n = 171$. Subjects who knew more delinquent peers tended to have a best friend with higher delinquency.

*Computerized tasks: reward dominance and response inhibition.* Reward dominance indexed by the Card Playing Task and response inhibition measured by the Stop Task was not significantly correlated. The two outcome variables of the Card Task (number of card rounds played and the final score) highly overlapped with $r = -.96, p \leq .001$ (see table 9). It is noted that in the Card Playing Task, the score obtained will start to become a minus one after around the 75$^{th}$ trial. The score will be -10 if all cards are viewed. For the full sample, the average number viewed is 79

(*SD* = 27.8). On the whole, this sample played a high number of rounds in the card task and obtained a low score, and thus a negative high correlation was found. In the subsequent analysis, group comparison was done on the card rounds played, considering the high overlap between the two variables and the key interest here is know the extent to which the subjects would perseverate in viewing the cards in the face of punishment (loosing mark).

Table 9
Correlation between reward dominance (Card Playing Task) and response inhibition (Stop Task)

| Variables | 1 | 2 | 3 |
|---|---|---|---|
| 1. Card Task - round | | | |
| 2. Card Task - score | -.96*** ($n$ = 162) | | |
| 3 Stop Task – SSRT | -.04 ($n$ = 139) | -.04 ($n$ = 139) | |

Note:
SSRT = stop signal reaction time
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*CU trait and ADHD diagnosis / symptoms.* The offending groups will be compared on the dimensional measure of DISC ADHD (ADHD symptom score), DISC ADHD diagnosis and callous unemotional traits. Table 10 lists the

correlations among these three variables. The past year DISC ADHD symptom score was moderately correlated with the past year DISC ADHD diagnosis with $r = .33, p \leq .001$. These ADHD measures were also positively correlated with callous-unemotional trait, with $r = .33, p \leq .001$ for CU trait and DISC ADHD symptom score, and $r_{pb} = .17, p \leq .05$ for CU trait and DISC ADHD diagnosis.

Table 10
Correlations among CU traits and ADHD diagnosis / symptoms

| Variables | 1 | 2 | 3 |
|---|---|---|---|
| 1. CU traits (ICU total) | | | |
| 2. DISC ADHD diagnosis (past year) | .17* $n = 169$ | | |
| 3 DISC ADHD symptom score (past year) | .33*** $n = 168$ | .33*** $n = 174$ | |

Note:
$r_{pb}$ = point-biserial correlation
ICU = inventory of Callous unemotional trait
DISC = Diagnostic interview schedule for children
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

## Correlations between delinquency (offending status) and purported risk factors of delinquency and external correlates

Correlations between delinquency and the variables hypothesized as risk factors and correlates of delinquency were examined. Background risk variables were selected based on prior research (e.g. Moffitt, et al., 2002; Odgers et al., 2008, Patterson et al., 1998) that supported their association with juvenile delinquency. The correlation between these purported risk factors and offending status of the subjects was examined and was listed in the following tables. The offending status is an objective delinquent status (whether the subject is offender or non-offender) which does not reply on self-report of the subjects, thus did not share the same informant variance with various risk factors and correlates that were derived from various measurements using self-report as source of information. The offending status is a dichotomous variable (offender coded as 1 vs. non-offender coded as 0). When the risk factor counted is also a dichotomous variable (such as clinical diagnosis), phi correlation ($\Phi$) will be computed, whereas point-biserial correlation ($r_{pb}$) will be referred to for correlations between offending status and continuous risk variables.

*Offending status and cognitive risks.* All the three variables of the cognitive risk were significantly correlated with offending status (Table 11). Offending status was negatively correlated with IQ ($r_{pb} = -.25, p < .001$), indicating that being offender was associated with lower IQ. Being offender was positively correlated with more class repeating ($r_{pb} = .30, p \leq .001$) and receiving follow up service / training ($\Phi = .21$, $p = .005$)

Table 11.
Offending status and cognitive risks

| | Overall IQ [b] | Number of times repeating a class [b] | Follow-up service / training [a] |
|---|---|---|---|
| Offending status (offender vs non-offender) | -.25*** ($p = .001$) $n = 180$ | .30*** ($p \leq .001$) $n = 181$ | .21** ($p = .005$) $n = 172$ |

Note:

**a**: $r$ = phi correlation($\Phi$)

**b**: $r$ = point-biserial correlation ($r_{pb}$)

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and family adversities.* Most of the variables of family adversities, except parents' mental health problem were significantly correlated with offender status, despite with relatively small correlations ( $r_{pb}$ ranging from .18 to .34), indicating that being offender was associated with more family adversities (see table 12).

Table 12.
Offending status and family adversities

| | Parents' criminality | Parents' mental health | Parents' child abuse | Low SES | Years of single parenting before age 11 | Number of family transition before age 11 |
|---|---|---|---|---|---|---|
| Offending status (offender vs non-offender) | .22** ($p$ = .003) $n$ = 176 | .12 ($p$ = .13) $n$ = 176 | .18* ($p$ = .02) $n$ = 167 | .19* ($p$ = .014) $n$ = 177 | .26*** ($p$ < .001) $n$ = 178 | .34*** ($p$ < .001) $n$ = 178 |

Note:
All $r$ = point-biserial correlation ( $r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and parenting variables.* Among the various parenting variables, offender status was significantly correlated with more poor parental monitoring with correlation being moderately strong ($r_{pb} = .47$, $p \leq .001$), and with less mother's involvement, with correlation being small ($r_{pb} = -.15$, $p < .05$) (see table 13).

Table 13.
Offending status and parenting risks

| | APQ Mother's involvement | APQ Father's involvement | APQ positive parenting | APQ poor parental monitoring | APQ corporal punishment |
|---|---|---|---|---|---|
| Offending status (offender vs non-offender) | -.15* ($p$ = .05) $n$ = 175 | -.08 ($p$ = .32) $n$ = 166 | .005 ($p$ = .95) $n$ = 180 | .47*** ($p \leq .001$) $n$ = 180 | .08 ($p$ = .32) $n$ = 180 |

Note:
All $r$ = point-biserial correlation ( $r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and deviant peer affiliation.* Offender status was strongly and positively correlated with the two variables of deviant peer affliliation ( $r_{pb}$ = .62 and .73, $p < .001$), indicating that being offender associated with peers with higher delinquency and had best friend with higher delinquency (see table 14).

Table 14.
Offending status and deviant peer affiliation

| | Peer delinquency (total) | Delinquency of best friend |
|---|---|---|
| Offending status (offender vs non-offender) | .62*** ($p \leq .001$) $n$ = 177 | .73*** ($p \leq .001$) $n$ = 175 |

Note: All $r$ = point-biserial correlation ( $r_{pb}$); *$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and adolescent adjustment and prosocial behavior.* Table 15 shows that being offender was significantly and positively correlated with more adolescent adjustment difficulties ($r_{pb} = .18, p < .05$ for offending status and SDQ difficulties and $r_{pb} = .21, p < .05$ for offending status and SDQ impact score) and less prosocial behavior ($r_{pb} = -.30, p < .001$).

Table 15.
Offending status and adolescent adjustment and prosocial behavior

| | SDQ total difficulties | SDQ impact score | SDQ prosocial behavior |
|---|---|---|---|
| Offending status (offender vs non-offender) | .18* ($p = .014$) $n = 180$ | .21** ($p = .005$) $n = 181$ | -.30*** ($p < .001$) $n = 181$ |

Note:
All $r$ = point-biserial correlation ($r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and clinical diagnoses.* As shown in table 16, offender status was strongly and positively related to conduct disorder, regardless of whether it was in form of diagnosis or dimensional measure ($r_{pb} = .69$ and $r = .77$, all $p < .001$). Offender status was also positively correlated with DISC ODD ($r_{pb} = .28, p < .001$) and its symptom score ($r_{pb} = .50, p < .001$). Correlation between offender status and

DISC diagnosis of ADHD was non-significant, whereas being offender was positively correlated with the dimensional measure of ADHD ($r_{pb} = 0.38$, $p < .001$), indicating that being offender associated with higher level of ADHD symptoms.

Table 16.
Offending status and DISC diagnoses and dimensional measure of CD, ODD, ADHD

| | DISC CD (past year)[a] | DISC ODD (past year) [a] | DISC ADHD (past year) [a] | DISC CD Symptom count (past year)[b] | DISC ODD symptom count (past year) [b] | DISC ADHD symptom count (past year) [b] |
|---|---|---|---|---|---|---|
| Offending status (offender vs. non-offender) | .69*** ($p < .001$) $n = 179$ | .28*** ($p < .001$) $n = 176$ | .18* ($p = .02$) $n = 177$ | .77*** ($p < .001$) $n = 180$ | .50*** ($p < .001$) $n = 176$ | .38*** ($p < .001$) $n = 176$ |

Note:
a correlation = phi correlation (Φ)
b correlation = point-biserial correlation ( $r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and callous-unemotional traits and anxiety.* Table 17 shows that being offender was positively correlated with callous-unemotional traits ($r_{pb} = .29$, $p < .001$) whereas offending status was not significantly correlated with anxiety

Table 17.
Offending status and callous-unemotional traits and anxiety

| | CU trait (ICU total) | Anxiety (YSR) |
|---|---|---|
| Offending status (offender vs non-offender) | .29*** ($p < .001$) $n = 173$ | .14 ($p = .07$) $n = 180$ |

Note:
All $r$ = point-biserial correlation ( $r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

*Offending status and reward dominance measured by the card task and inhibition deficit measured by the stop task.* Offending status was not significantly related to the outcomes of the two computerized tasks, namely, the card round played in the card playing task, indicating reward dominance, and the stop signal reaction time of the stop task, indicating inhibition deficit (table 18).

Table 18.

Offending status and reward dominance measured by the card task and inhibition deficit measured by the stop task

| | Card round (card task) | SSRT (stop task) |
|---|---|---|
| Offending status (offender vs non-offender) | .02 ($p$ = .81) $n$ = 162 | -.04 ($p$ = .62) $n$ = 152 |

Note:

SSRT = stop-signal reaction time

All $r$ = point-biserial correlation ( $r_{pb}$)

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

## Comparison among the two broad offending groups and control on delinquency, risk factors of delinquency and external correlates

Based on the earliest age of CD symptom reported and the cut off age of 11, two broad offending groups, i.e., the early-onset offending group (EO) which has 95 members and the adolescent-onset offending group (AO) which has 21 members were formed. Table 19 lists the age and class level of the three groups. There was no significant difference among the three groups in terms of their age. Significant difference was found for their class level. Post-hoc multi-comparison (by Dunnett's C test as group variance not homogenous) showed that both the class level of the EO and AO was significantly lower than that of the control respectively. Further correlation analyses indicated that correlations between the class level and all study variables were either non-significant or low for both offender and normal control, thus it was not controlled as a covariate in the subsequent group comparison. In fact, the class level of the participants in this study is more than a demographic variable to be controlled. Poor educational attainment is well known to be linked with anti-social behavior (Loeber & Stouthamer-Loeber, 1998; Loeber, Pardini, Homish, Wei, Crawford, Farrington, et al., 2005) so that it is more like an independent variable in this current study.

Table 19
Age and class level of the EO, AO and control

| Variables | 1. EO group<br>*n* = 95<br>Mean / frequency<br>(SD / %)<br>Range | 2. AO group<br>*n* = 21<br>Mean / frequency<br>(SD / %)<br>Range | 3. Control<br>*n* = 63<br>Mean / frequency<br>(SD / %)<br>Range | F ratio / *p* /<br>Post-hoc comparisons |
|---|---|---|---|---|
| Age (year) | 14.16 (1.01)<br>12 – 16 | 14.33 (0.80)<br>12 – 16 | 14.29 (1.02)<br>12 - 17 | 0.46 |
| Education level | | | | |
| Primary 4 | 1 (1.1%) | | / | |
| Primary 5 | / | | / | |
| Primary 6 | 9 (9.5%) | | / | |
| Form 1 | 41 (43.2%) | 9 (42.9%) | 2 (3.2%) | |
| Form 2 | 35 (36.8%) | 10 (47.6%) | 14 (22.2%) | |
| Form 3 | 6 (6.3%) | 2 (9.5%) | 38 (60.3%) | |
| Form 4 | 3 (3.2%) | | 9 (14.3%) | |
| Mean class level | 7.46 (0.94) | 7.67 (0.66) | 8.86 (0.69) | 63.00*** /<br>1, 2 < 3*** |

Note: EO = early onset offending group, AO = adolescent-onset offending group
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 20 lists the results of the comparison among the two broad offending groups and the control on a total of 28 variables pertaining to their delinquency, the purported risk factors of delinquency, and other external correlates. Overall group differences were tested by separate one way ANOVAs where planned contrasts were carried out to test the hypothesized group difference. On variables including stop

task, card task and anxiety where there is no a prior hypothesized group difference among the three groups, post-hoc multiple comparisons where error level is duly adjusted to prevent inflation of type I error (false rejection of null hypothesis) would be conducted instead of planned contrast where alpha is set at .05. Bonferroni correction was performed to keep the family-wise alpha level to .05 on two measures of delinquency (self-reported delinquency total and variety score, .05 / 2 = .025), and two measures of deviant peer affiliation (peer delinquency total and delinquency of my best friend, .05 / 2 = .025). Effect size of the group difference was indicated by Cohen's delta ( *d* ), where *d* of 0.2 to 0.49 is regarded as small, 0.5 to 0.79 is medium and above 0.8 is regarded as large (Cohen, 1988). No analysis had been done on the APQ subscale of inconsistent parenting as this subscale had a low alpha (0.39) in this sample.

According to Moffitt's two-trajectory proposal, while the early-onset group and the adolescent-onset group showed to be delinquent on a same level in adolescence, it was the early-onset group that suffered the worst background whereas the adolescent-onset group had background similar to that of the normal peers. On the other hand, Patterson proposed that the adolescent-onset delinquents suffered background risks of intermediate level. The adolescent-onset group suffered more

risks than control but less than the early-onset group. Thus, the key interest of the three-group comparison here is to test the difference between the individual offending groups versus the control, and also the difference between the two offending groups.

Overall significant group differences were found for 20 variables amongst 28, respectively for all three variables pertaining to adolescent delinquency (Bonferroni-adjusted for delinquency total and variety score), all three variables pertaining to adolescent adjustment and prosocial behavior, all three variables of cognitive risks, four out of six variables of family adversities, two out of five parenting variables, all two variables of peer delinquency which were Bonferroni-adjusted , both dimensional and categorical measure of ADHD; and callous unemotional traits. There was no significant group difference on parents' mental health, card playing task, stop task, and YSR anxiety; and three variables of parenting risks. For parent's child abuse, the test for overall group difference could not be done as both the control group and the AO group had zero child abuse and zero group variance.

On all the three variables indicating delinquency, both the EO and AO group were significantly different from control with all effect sizes being very large ($d =$

1.06 to 3.44). The EO and AO groups exhibited significantly much higher level of delinquency, both in terms of variety (variety score) and frequency (total frequency score), as compared to control. The two offending groups also had much more favorable attitude towards delinquency (criminal attitude). The EO group was not different from the AO group on both variety and level of delinquent acts committed. Thus the EO and the AO could not be distinguished only by the level or variety of delinquency shown in the adolescent period. But significant difference of a medium effect size ($d = 0.5$) was found between the two offending groups on criminal attitude, that the EO had significantly more favorable attitude towards delinquency than the AO.

The EO group was found significantly worse than the control on all variables pertaining to adjustment and prosocial behavior of adolescent, with small to medium effect sizes. Specifically, as compared to the control, the EO group had more SDQ difficulties ($d = 0.42$) and rated themselves to be significantly more impaired and distressed by their difficulties (higher SDQ impact score, $d = 0.50$). They had less prosocial behaviors, when compared respectively with the control ($d = 0.74$) and also the AO group ($d = 0.48$). The other offending group, the AO, was not significantly different from the control on these three variables, but all the means obtained were

found to be intermediate between that of the EO and control.

Four classes of variables purported to be the background risks of adolescent delinquency include cognitive risks, family adversities, parenting risks, and deviant peer influence during adolescence. On all the three variables indicating cognitive risks, both the two offending groups were significantly worse than the control. Each offending group had lower IQ and more times of repeating class than the control, with the differences being large between the AO and the control ($d$= 1.87 on IQ $d$ =1.02 on times of repeating class), and the difference between the EO and the control being moderate ($d$ = 0.58 on IQ and $d$ = 0.62 on times of repeating class). Significantly more EO and AO members had attended special follow-up or training than the control. No significant difference between the two offending groups was found on all these three variables.

In the realm of family adversities, the EO group was significantly worse than the control on five variables out of six (with 3 effect sizes being medium and two being small). Specifically, the EO group had more years of single parenting in childhood before age 11 ($d$ = 0.58), more family transition before their age 11 ($d$ = 0.71), lower SES ($d$ = 0.43), more parents' criminality ($d$ = 0.56) and more child abuse of parents

($d$ = 0.46). Out of the six variables, the AO was significantly worse than the control on two variables with effect sizes being large. They had significantly more years of single parenting ($d$ = 0.96) and more family transitions before age 11 ($d$ = 1.57). It was noted that on parents' criminality the AO was higher than that of the control group with an effect size of 0.51 but the alpha being .16. The non-significant finding could be due to relatively small sample size of the group ($n$ = 21). The EO group was different from the AO group in having significantly more parents' child abuse ($d$ = 0.39, effect size small), but on the other hand, the AO had significantly more family transitions before age 11 as compared to the EO ($d$ = 0.62, effect size medium).

On the five parenting variables, the EO group was significantly different from the control on two variables, that this group had significantly lesser mother's involvement ($d$ = 0.4, small effect size) and much more poor parental monitoring ($d$ = 1.19, large effect size). Similarly the AO had significantly more poor parental monitoring as compared to control ($d$ = 0.79, medium effect size). No significant difference between EO and AO was found in all the parenting variables.

On the two variables pertaining to deviant peer influence, similarly, both EO and AO group showed marked difference from the control, that each group had much

more peers being delinquent, and their best friend being more delinquent also. All differences between the offending group and the control had large effect sizes with *d* ranging from 1.75 to 2.55. No significant difference between the EO and AO was found, indicating that both groups were not different in terms of having deviant peer affiliation during adolescence.

As adolescents, the EO and the AO group were significantly different on adjustment difficulties and prosocial behavior, with the EO group being worst and the AO group being intermediate, though not significantly different from the control. As regards the purported background risks, on the whole, the EO group had worst background risks and was different from control across all risk areas. A total of 12 significant different findings out of 16 risk variables were found from the planned paired contrasts, with three effect sizes being large, five being medium, and four being small. The AO was significantly worse than the control on eight risk variables spreading across all risk areas. Six significant differences between the AO group and the control had a large effect size. There are more significant findings of the EO group compared to the control, versus that of the AO group compared to the control. The EO-control versus the AO-control difference mainly comes from the family adversities and parenting risks, but not on cognitive risks. Both the EO and the AO

group were similarly worse than the control on all variables indicating cognitive risks. There were few significant differences between the EO and the AO on the background risks of delinquency (on prosocial behavior with medium effect size, child abuse of parents with effect size small and number of family transition before age 11 with medium effect size).

*Group comparison on other external correlates.* The three groups were compared on callous-unemotional traits and symptoms of attention and hyperactivity characteristics of ADHD, both of which are attributes hypothesized to be more prominent in the early-onset offending group. Overall significant group difference was found for the CU traits (indicated by total score of the ICU). Results of the paired contrast indicated that the EO group had significantly higher level of CU traits as compared to the control ($d = 0.67$, medium effect size) whereas the difference between the AO and the control group was not significant. In terms of the symptoms of inattention and hyperactivity, two variables were counted, the first being the DISC ADHD symptom score, which is a dimensional measure of the ADHD symptom, and the other, the DISC ADHD diagnosis (combined sources). One of the key differences between these two measures of ADHD is about the age of onset of symptom. To make the diagnosis, some of the ADHD symptoms will need to be

present before age 7. The symptom score is a count of past year ADHD symptoms regardless of the onset. On the DISC ADHD symptom score, both of the EO and the AO group had significantly higher ADHD symptoms as compared to control respectively, with the difference between the EO and control being large ($d = 0.89$) and the difference between the AO and control being medium ($d = 0.73$). No significant difference was found between the EO and the AO group on the ADHD symptom count. On the other hand, for the ADHD diagnosis, the distribution was significantly higher in the EO group versus the other two groups, as shown by the results of the partition of the overall $G^2$ (overall $G^2 = 8.64$, $df = 2$, $p \leq .01$). Specifically, the distribution of the diagnosis in the AO group versus that in the control was not significantly different ($G^2 = 0.56$, $df = 1$, $p > .05$) whereas $G^2$ is 8.08 ($df = 1$, $p \leq .01$) for the EO versus the other two groups. As can be seen, 13.7% of the EO members had ADHD diagnosis versus 4.8% of the AO group and 1.6% of the control group. There is a significant correlation between ADHD diagnosis and group with $\Phi = .20$, $p \leqq .01$.

Different level of anxiety and different outcomes on the stop task and the card task are hypothesized to characterize two distinct subgroups within the early-onset group, which will be looked at in the subsequent analysis. No a priori differences

had been hypothesized on the two broad offending groups and the control on anxiety and these two performance tasks. Thus post-hoc multiple comparisons instead of planned contrasts would be done when a significant overall group difference was found. For all the three variables, no significant overall group difference was obtained and thus no post-hoc analysis was done.

Table 20

Early-onset offending group (EO), Adolescent-onset offending group (AO) and the control group compared on self-reported delinquency, risk factors of delinquency and external correlates:

| | 1.**Early-onset** *n* = 95 Mean (*SD*) / Frequency (%) | **2. Adolescent-onset** *n* = 21 Mean (*SD*) / Frequency (%) | **3. Control** *n* = 63 Mean (*SD*) / Frequency (%) | F-ratio/ Welch-adjusted *F* / $\chi^2$ | EO- C differences *t* test / Cohen's *d* | AO – C differences *t* test / Cohen's *d* | EO-AO differences *t* test / Cohen's *d* |
|---|---|---|---|---|---|---|---|
| **Delinquency** | | | | | | | |
| 1. *variety of delinquency in past 12 months* | 8.95 (4.38) *n* = 93 | 7.43 (4.32) | 0.48 (0.76) *n* = 62 | 188.61*** | 18.29*** /2.5 | 7.33*** / 3.13 | 1.45 / 0.35 |
| 2. *frequency of delinquency** *In past 12 months* | 6.65 (2.62) *n* = 93 | 5.86 (2.59) | 0.67 (0.95) *n* = 62 | 227.11*** | 20.17*** /2.84 | 9.00***/ 3.44 | 1.25 / 0.30 |
| 3. *Criminal attitude* | 9.21 (3.86) | 7.29 (3.82) | 4.35 (2.38) *n* = 62 | 47.77*** | 9.75*** /1.46 | 3.30** / 1.06 | 2.09* / 0.50 |
| **Adjustment difficulties And prosocial behaviour** | | | | | | | |
| 4. SDQ total difficulties | 13.86 (4.82) | 13.40 (4.96) n = 20 | 11.83 (4.93) | 3.35* | 2.50** / 0.42 | 1.56 / 0.32 | 0.39 / 0.1 |
| 5. *SDQ impact (distress impairment)** | 0.72 (0.80) | 0.48 (0.67) | 0.36 (0.60) | 5.37** | 3.43*** / 0.50 | 0.7 / 0.2 | 1.67 / 0.31 |
| 6. *SDQ prosocial behaviour* | 5.24 (2.11) | 6.19 (1.47) | 6.65 (1.61) | 11.34*** | -4.75*** / 0.74 | -1.21 / 0.3 | -2.45* / 0.48 |

Table 20 (cont'd)

| | **1. Early-onset** $n = 95$ Mean (*SD*) / Frequency (%) | **2. Adolescent-onset** $n = 95$ Mean (*SD*) / Frequency (%) | **3. Control** $n = 63$ Mean (*SD*) / Frequency (%) | *F*-ratio/ Welch-adjusted *F* / $\chi^2$ | EO- C differences *t* test / Cohen's d | AO – C differences *t* test / Cohen's *d* | EO-AO differences *t* test / Cohen's *d* |
|---|---|---|---|---|---|---|---|
| **Background risks of delinquency** | | | | | | | |
| **Cognitive risks** | | | | | | | |
| 7. Overall IQ (HK-WISC / WAIS-III) | 99.36 (14.67) n = 94 | 95.02 (13.23) | 105.93 (11.89) | 6.85*** | -2.97** / 0.58 | -3.19** /1.87 | 1.32 / 0.3 |
| 8. *average times of repeating a class* | 0.78 (0.84) | 0.91 (0.62) | 0.32 (0.56) | 100.26*** | 4.13***/0.62 | 3.82***/ 1.02 | -0.78 / 0.16 |
| 9. history of special follow-up / training [a] | 19 (21.8%) | 5 (25%) | 7 (6.3%) | 8.50** *df* = 2 | 3.81* /Φ= 0.15* *df* = 1 | 4.68* /Φ= 0.26* *df* = 1 | / |
| **Family adversities** | | | | | | | |
| 10. *years of single Parenting before age 11** | 0.93 (1.29) *n* = 93 | 1.19 (1.33) *n* = 20 | 0.29 (0.79) | 9.76*** | 3.87***/0.58 | 2.86** / 0.96 | -0.78 / 0.13 |
| 11. *Number of family Transition before age 11** | 0.52 (0.56) | 0.87 (0.60) | 0.17 (0.39) | 19.00*** | 4.66*** / 0.71 | 5.03***/1.57 | -2.44* / 0.62 |
| 12. *Low SES** | 0.67 (0.67) *n* = 92 | 0.53 (0.64) | 0.40 (0.56) *n* = 62 | 3.66* | 2.72** / 0.43 | 0.83 / 0.23 | 0.91/ 0.21 |

Table 20 (cont'd)

| | **1. Early-onset** *n* = 95 Mean (*SD*) / Frequency (%) | **2. Adolescent-onset** *n* = 21 Mean (*SD*) / Frequency (%) | **3. Control** *n* = 63 Mean (*SD*) / Frequency (%) | *F*-ratio/ Welch-adjusted *F* / $\chi^2$ | EO- C differences *t* test / Cohen's *d* | AO – C differences *t* test / Cohen's *d* | EO-AO differences *t* test / Cohen's *d* |
|---|---|---|---|---|---|---|---|
| 13. *Parent's criminality** | 0.36 (0.55) *n* = 92 | 0.30 (0.57) *n* = 20 | .10 (0.32) *n* = 62 | 6.81** | 3.62***/ 0.56 | 1.45 / 0.51 *p* = .16 | 0.42 / 0.11 |
| 14. *child abuse of parent** [b] | 0.14 (0.40) *n* = 85 | 0 *n* = 19 | 0 *n* = 62 | / | 3.3*** /0.46 | / | 3.30*** / 0.39 |
| 15. *mental health Problem of parents** | 0.18 (0.49) *n* = 91 | 0.08 (0.38) | 0.05 (0.28) *n* = 62 | 2.01 | | | |
| **Parenting risks** | | | | | | | |
| 16. *APQ mother's Involvement* | 23.43 (6.76) *n* = 91 | 25.76 (10.53) *n* = 21 | 26.21 (6.47) *n* = 61 | 3.33* | - 2.56* / 0.42 | -0.19 / 0.06 | 0.97 / 0.31 |
| 17. APQ father's Involvement | 21.75 (8.02) *n* = 87 | 24.50 (8.08) *n* = 21 | 23.47 (7.18) *n* = 59 | | | | |
| 18. APQ positive Parenting | 16.56 (4.91) | 18.71 (5.40) | 16.94 (4.84) *n* = 62 | | | | |
| 19. APQ parents' Poor monitoring | 32.76 (5.66) *n* = 95 | 30.62 (6.11) *n* = 21 | 25.82 (6.16) *n* = 62 | 26.08*** | 7.20*** / 1.19 | 3.22** / 0.79 | 1.50 / 0.38 |
| 20. APQ corporal Punishment | 5.89 (3.01) *n* = 94 | 4.81 (1.60) | 5.27 (2.59) | | | | |

Table 20 (cont'd)

| | **1. Early-onset** $n = 95$ Mean (*SD*) / Frequency (%) | **2. Adolescent-onset** $n = 21$ Mean (*SD*) / Frequency (%) | **3. Control** $n = 63$ Mean (*SD*) / Frequency (%) | *F*-ratio/ Welch-adjusted *F* / $\chi^2$ / $G^2$ | EO- C differences *t* test / Cohen's *d* | AO – C differences *t* test / Cohen's *d* | EO-AO differences *t* test / Cohen's *d* |
|---|---|---|---|---|---|---|---|
| **Deviant peer affiliation in adolescence** | | | | | | | |
| 21. *delinquency of Peer group* | 25.46 (6.98) $n = 92$ | 23.25 (7.11) $n = 20$ | 15.65 (2.75) | 79.33*** | 12.18*** / 1.75 | 4.47*** / 1.83 | 1.26 / 0.32 |
| 22. *delinquency of Best friend* | 22.43 (9.30) $n = 90$ | 20.95 (13.93) | 3.48 (2.75) $n = 62$ | 164.53*** | 17.66*** /2.55 | 5.69*** / 2.35 | 0.46 / 0.14 |
| **Inattention and hyperactivities** | | | | | | | |
| 23. DISC-ADHD total Symptom count | 9.08 (4.82) | 8.14 (4.65) | 4.97 (4.33) | 14.70*** | 5.39*** / 0.89 | 2.7** / 0.73 | 0.84 / 0.2 |
| 24. DISC-ADHD Diagnosis [c] | 13 (13.7%) | 1 (4.8%) | 1 (1.6%) | 8.64** $df = 2$ | 8.08** /Φ=.20** $df = 1$ (EO vs. C & AO) | 0.56 /Φ= 0.09 $df = 1$ (AO vs. C) | / |
| **Callous unemotional traits** | | | | | | | |
| 25. ICU total score | 29.88 (7.95) $n = 89$ | 27.71 (7.48) $n = 19$ | 25.04 (6.12) | 8.88*** | 4.24*** / 0.67 | 1.14 / 0.42 $p = 0.17$ | 1.14 / 0.28 |

Table 20 (cont'd)

| | **1. Early-onset** *n* = 95 Mean (*SD*) / Frequency (%) | **2. Adolescent-onset** *n* =21 Mean (*SD*) / Frequency (%) | **3. Control** *n* = 63 Mean (*SD*) / Frequency (%) | *F*-ratio/ Welch-adjusted *F* / $\chi^2$ | EO- C differences *t* test Cohen's *d* | AO – C differences *t* test / Cohen's *d* | EO-AO differences *t* test / Cohen's *d* |
|---|---|---|---|---|---|---|---|
| 26. card round played / Card playing task mark obtained / | 80.39 (27.53) *n* = 79 0.42 (13.65) | 74.37 (32.23) *n* = 19 1.95 (14.03) | 78.33 (27.05) 2.08 (13.59) | 0.38 | | | |
| 27. SSRT / Stop task | 256.39 ms (47.8 ms) | 253.73 ms (45.39 ms) | 260.83 ms (42.38 ms) | 0.23 | | | |
| 28. YSR anxiety* | 2.1 (1.02) | 2.08 (1.36) | 1.73 (1.09) | 2.26 | | | |

**Note:**

*Variables* in italic form: Welch-adjusted *F* instead of F ratio is referred to when the variance among groups is unequal as indicated by the significant findings (Levene statistic) of the test of homogeneity of variances in ANOVA.

Variables* : square root transformation of the data was done because of skewness coefficient > 1.

a. overall $\chi^2$ reported is Likihood Ratio Chi Square ($G^2$ ), which is further partitioned to investigate the source of group difference. Number of partitions made is bounded by df = (row -1) X (column -1) = (2-1) X (3-1) = 2 partitions. The following partitions of $G^2$ were made: AO vs. control with $G^2$ = 4.68* and EO vs. (AO and control) = 3.81*. Strength of association between group and ADHD diagnosis is indicated by phi correlation, showed in the column of effect size.

b. Child abuse of parent: F test cannot be performed for child abuse of parents because at least one group has 0 variance.

c. overall $\chi^2$ reported is Likihood Ratio Chi Square ($G^2$ ), which is further partitioned to investigate the source of group difference. Number of partitions made is bounded by df = (row -1) X (column -1) = (2-1) X (3-1) = 2 partitions. The following partitions of $G^2$ are made: AO vs. control with $G^2$ = 0.56 and EO vs. (AO and control) = 8.08**. Strength of association between group and ADHD diagnosis is indicated by phi correlation, showed in the column of effect size.

SDQ = Strength and Difficulties Questionnaire, APQ – Alabama Parenting Questionnaire, ICU = Inventory of Callous Unemotional Traits

YSR = Youth Self Report, SSRT = stop signal reaction time

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

## Two early-onset offending subgroups compared with Adolescent-onset group and control on delinquency, risk factors of delinquency and external correlates

Two distinct subgroups within the early-onset offending group, namely, the early onset high ADHD group (EADHD) and the early-onset high CU traits group (ECU) were formed respectively based on high callous-unemotional trait (score above 13 = > 75$^{th}$ percentile on the Inventory of Callous Unemotional Traits) and high ADHD symptoms (score of 37 = > 75$^{th}$ percentile on DISC ADHD Symptom Count). There was an overlap of six subjects between these two groups and they were removed, resulting in $n$ = 17 for EADHD and $n$ = 11 for ECU. These two independent offending groups, despite both belonging to the early-onset group, are hypothesized to have some distinct features. These hypothesized differences were tested by the following four-group comparison which compared the EADHD, ECU, AO and control on delinquency, adolescent adjustment and prosocial behaviors, background risks, two computerized tasks (Card Task and Stop Task), and level of anxiety. Similar to that in the previous three-group comparison, planned contrast would be adopted to test specific group difference. Here the key interest is to examine how the EADHD is different from the AO and control versus how the ECU is different

from the AO and control. It is also relevant to examine the direct difference between these two early-onset groups. The difference between the AO group and the control had been tested in the previous three-group analysis but will still be shown in this set of analysis for clarity. Table 21 lists the age and class level of the three groups. Table 22 shows the results of the four-group comparison and table 23 lists the details of the planned contrast and the effect sizes for specific group differences. As the group size was notably small in the two early-onset groups and the four groups were also very unequal in size, power to detect group difference (particularly of small effect size) is admittedly lower than that of the previous three-group analysis.

Table 21

Age and class level of the EADHD, ECU, AO and control

| variables | 1. EADHD group $n = 17$ Mean / frequency (*SD* / %) Range | 2. ECU group $n = 11$ Mean / frequency (*SD* / %) Range | 3. AO group $n = 21$ Mean / frequency (*SD* / %) Range | 4. Control $n = 63$ Mean / frequency (*SD* / %) Range | *F* ratio / *p* / Post-hoc Comparison |
|---|---|---|---|---|---|
| Age (year) | 13.65 (0.10) | 14.18 (0.75) | 14.33 (0.80) | 14.29 (1.02) | 2.19 |
| | 12 – 15 | 13 - 15 | 12 – 16 | 12 - 17 | |
| Education level | | | | | |
| Primary 6 | 4 (23.5%) | 1 (9.1%) | / | / | |
| Form 1 | 10 (58.8%) | 4 (36.4%) | 9 (42.9%) | 2 (3.2%) | |
| Form 2 | 2 (11.8%) | 5 (45.5%) | 10 (47.6%) | 14 (22.2%) | |
| Form 3 | 1 (5.9%) | 1 (9.1%) | 2 (9.5%) | 38 (60.3%) | |
| Form 4 | / | | | 9 (14.3%) | |
| Mean class level | 7.0 (0.79) | 7.55 (0.82) | 7.67 (0.66) | 8.86 (0.69) | 40.71*** 1, 2, 3 < 4*** 1 < 3* |

Note:
EADHD = early-onset high ADHD group, ECU = early-onset high CU trait group, AO = adolescent-onset offending group
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

There was no significant difference among the four groups in terms of their age. Significant difference was found for their class level. Post-hoc multiple comparisons by the Tukey 's Honestly Significantly Difference Test indicated that the class level of both the two early-onset groups and the adolescent-onset offending was all significantly lower than that of the control respectively. The class level of the EADHD group was also significantly lower than that of the AO group. Since correlation

analyses indicated either non-significant or low correlations between the class level and all other study variables for the offender and control group, class level was not controlled as covariate in the group comparison.

Table 22

Early-onset high ADHD group (EADHD), Early-onset high CU trait group (ECU), Adolescent-onset group (AO) and control (C) compared on delinquency, risks factor for delinquency and other external correlates

| | **1. Early-onset high ADHD (EADHD)** *n* = 17 mean (*SD*) / Frequency (%) | **2. Early-onset high CU trait (ECU)** *n* = 11 mean (*SD*) / Frequency (%) | **3. Adolescent-Onset (AO)** *n* = 21 mean (*SD*) / Frequency (%) | **4 Control (C)** *n* = 63 mean (*SD*) /Frequency (%) | ***F*-ratio / Welch-adjusted *F*** $/\chi^2$ | **Planned pairwise contrast by *t*-test** |
|---|---|---|---|---|---|---|
| **Delinquency** | | | | | | |
| 1. *variety of delinquency in past 12 months* | 11.12 (4.37) | 7.40 (3.27) *n* = 10 | 7.43 (4.32) | 0.48 (0.76) *n* = 62 | 61.21*** | 1 > 2, 3 > 4 |
| 2. *frequency of delinquency in past 12 months** | 7.36 (2.65) | 5.84 (1.33) *n* = 10 | 5.86 (2.59) | 0.67 (0.95) *n* = 62 | 94.08*** | 1, 2, 3 > 4 |
| 3. criminal attitude | 8.70 (3.41) | 12.18 (3.76) | 7.29 (3.82) | 4.35 (2.38) *n* = 62 | 21.84*** | 2 > 1, 3 > 4 |
| **Adjustment difficulties /prosocial behaviour** | | | | | | |
| 4. SDQ total difficulties | 17.59 (4.30) | 16.82 (5.0) | 13.40 (4.96) | 11.83 (4.93) | 8.24*** | 1 > 3, 4<br>2 > 4 |
| 5. *SDQ impact (distress impairment)* * | 0.81 (0.86) | 1.15 (0.87) | 0.48 (0.67) | 0.36 (0.60) | 3.71* | 1 > 4 (p = .053),<br>2 > 3, 4 |
| 6. SDQ prosocial behavior | 6.06 (1.89) | 3.09 (2.02) | 6.19 (1.47) | 6.65 (1.61) | 14.20*** | 2 < 1, 3, 4 |

Table 22 (cont'd)

| | **1. Early-onset high ADHD (EADHD)** $n = 17$ mean (*SD*) / Frequency (%) | **2. Early-onset high CU trait (ECU)** $n = 11$ mean (*SD*) / Frequency (%) | **3. Adolescent-Onset (AO)** $n = 21$ mean (*SD*) / Frequency (%) | **4 Control (C)** $n = 63$ mean (*SD*) /Frequency (%) | ***F*-ratio / Welch-adjusted *F*** $/\chi^2$ | **Planned pairwise contrast by *t*-test** |
|---|---|---|---|---|---|---|
| **Background risk of delinquency: cognitive risks** | | | | | | |
| 7. IQ (HK-WISC / WAIS-III) | 98.47 (10.55) | 103.45 (15.62) | 95.02 (13.23) | 105.93 (11.89) | 4.81** | 1 < 4<br>3 < 4 |
| 8. *average time of repeating a class* | 0.76 (0.97) | 0.64 (0.67) | 0.91 (0.62) | 0.32 (0.56) | 5.42** | 3 > 4 |
| 9. history of special follow-up / training | 3 (21.4%) $n = 14$ | 3 (27.3%) | 5 (25%) $n = 20$ | 4 (6.3%) | 7.37 ($p$=.061) | |
| **Family adversities** | | | | | | |
| *10. Parents' criminality** | 0.43 (0.58) $n = 16$ | 0.27 (0.47) | 0.30 (0.57) $n = 20$ | 0.10 (0.32) $n$= 62 | 2.24 | |
| 11. Low SES* | 0.87 (0.67) $n = 16$ | 0.71 (0.74) | 0.53 (0.64) $n$= 62 | 0.40 (0.56) | 2.97* | 1 > 4 |
| 12. Number of family transition before age 11* | 0.55 (0.55) | 0.40 (0.57) | 0.87 (0.60) | 0.17 (0.39) | 9.67*** | 1 > 4<br>3 > 2, 4 |
| 13. years of single parenting before age 11* | 0.78 (1.31) $n = 15$ | 0.78 (1.11) | 1.19 (1.33) | 0.29 (0.77) | 2.38* ($p = 0.54$) | 3 > 4 |

Table 22 (cont'd)

| | **1. Early-onset high ADHD (EADHD)** *n* = 17 mean (*SD*) / Frequency (%) | **2. Early-onset high CU trait (ECU)** *n* = 11 mean (*SD*) / Frequency (%) | **3. Adolescent-Onset (AO)** *n* = 21 mean (*SD*) / Frequency (%) | **4. Control (C)** *n* = 63 mean (*SD*) /Frequency (%) | ***F*-ratio / Welch-adjusted *F*** **/ $\chi^2$** | **Planned pairwise contrast by *t*-test** |
|---|---|---|---|---|---|---|
| 14. child abuse of parents [a]* | 0.53 (0.83) | 0.10 (0.32) | 0 | 0 | / | 1 > 3, 4 |
| 15. mental health problem of parents* | 0.42 (0.62) *n* = 16 | 0.10 (0.43) | 0.08 (0.38) | 0.05 (0.28) *n* = 62 | 0.44 | |
| **Parenting risks** | | | | | | |
| 16. *APQ mother's involvement* | 22.82 (6.05) | 19.55 (5.03) | 25.76 (10.53) | 26.21 (6.47) *n* = 61 | 5.28** | 2 < 3, 4 |
| 17. APQ father's involvement | 18.13 (6.37) *n* = 15 | 17.73 (4.90) | 24.50 (8.08) | 23.47 (7.18) *n* = 59 | 4.43** | 1,2 < 3, 4 |
| 18. APQ positive parenting | 16.12 (5.36) | 12.18 (3.54) | 18.71 (5.40) | 16.94 (4.84) *n* = 62 | 4.38** | 2 < 1, 3, 4 |
| 19. APQ parents' poor monitoring | 33.47 (4.77) | 30.00 (5.87) | 30.62 (6.11) | 25.82 (6.16) *n* = 62 | 9.27*** | 1, 2, 3 > 4 |
| 20. *APQ corporal punishment* | 7.35 ( 3.79) | 4.60 (1.84) *n* = 10 | 4.81 (1.60) | 5.27 (2.59) | 2.46 | |

Table 22 (cont'd)

| | **1. Early-onset high ADHD (EADHD)** $n = 17$ mean (*SD*) / Frequency (%) | **2. Early-onset high CU trait (ECU)** $n = 11$ mean (*SD*) / Frequency (%) | **3. Adolescent Onset (AO)** $n = 21$ mean (*SD*) / Frequency (%) | **4. Control (C)** $n = 63$ mean (*SD*) /Frequency (%) | ***F*-ratio / Welch-adjusted *F* / $\chi^2$** | **Planned pairwise contrast by *t*-test** |
|---|---|---|---|---|---|---|
| **Deviant peer affiliation** | | | | | | |
| 21. *delinquency of peer group* | 28.24 (6.65) | 25.18 (5.55) | 23.25 (7.11) $n = 20$ | 15.65 (2.75) | 32.55*** | 1 > 3 > 4 1 > 4 |
| 22. *delinquency of best friend* | 24.12 (10.16) | 24.70 (7.63) | 20.95 (13.93) | 3.84 (2.75) $n = 62$ | 52.50*** | 1, 2. 3 > 4 |
| **Computerized tasks** | | | | | | |
| 23. *card round played Card task* | 72.29 (31.44) (mark = 4.18) | 93.67 (12.86) (mark = -5.00) | 74.37 (32.23) $n = 19$ (mark = 1.95) | 78.33 (27.05) (mark = 2.08) | 3.60* | 2 > 1, 3, 4 |
| 24. SSRT / Stop Task | 254.02 ms (68.23 ms) | 252.49 ms (53.89 ms) | 253.73 ms (45.39 ms) | 260.83 ms (42.38 ms) | 0.18 | / |
| 25. anxiety* | 2.59 (1.12) | 2.27 (1.50) | 2.09 (1.36) | 1.73 (1.10) | 2.62* ($p = 0.54$) | 1 > 4 |

**Note:**

**a** Child abuse of parent: F test cannot be performed for child abuse of parents because at least one group has 0 variance

*Variables in italic form*: Welch test of equality of mean (a test robust against violation of equality of group variance) is referred to instead of F ratio because results of test of homogeneity of variances are significant, indicating that equality of variances of the three groups is not assumed.

Variables*: square root transformation of the data was done because of skewness coefficient > 1.

SDQ = Strength and Difficulties Questionnaire, APQ – Alabama Parenting Questionnaire , ICU = Inventory of Callous Unemotional Traits

SSRT = stop signal reaction time, YSR anxiety = anxious/depressed scale of Youth Self Report; *$p \leq .05$, ** $p \leq .01$, *** $p \leq .00$

Table 23.

Results of the planned contrasts and effect size of group differences among the three offending groups and control

| | Contrast 1 | Contrast 2 | Contrast 3 | Contrast 4 | Contrast 5 | Contrast 6 |
|---|---|---|---|---|---|---|
| | EADHD vs C | ECU vs. C | AO vs. C | EADHD vs ECU | EAHDH vs AO | ECU vs AO |
| | *t* / *p* | *t* *p* | *t* *p* | *t* *p* | *t* *p* | *t* *p* |
| | (effect size ) | (effect size ) | (effect size ) | (effect size ) | (effect size ) | (effect size ) |
| **Delinquency** | | | | | | |
| 1. variety of delinquency in past 12 months | EADHD > C 9.99*** (5.12) | ECU > C 6.65*** (4.96) | AO > C 7.33*** (3.13) | EADHD > ECU 2.51* (0.96) | EADHD > AO 2.6* (0.87) | |
| 2. frequency of delinquency in past 12 months | EADHD > C 10.25*** (4.60) | ECU > C 11.84*** (5.21) | AO > C 9.00*** (3.44) | | | |
| 3. criminal attitude | EADHD > C 5.23*** (1.68) | ECU > C 7.96***(3.03) | AO > C 3.86***(1.06) | ECU > EADHD 2.99** (1.02) | | ECU > AO 4.38***(1.33) |
| **Adjustment difficulties / prosocial behaviour** | | | | | | |
| 4. SDQ total difficulties | EADHD > C 4.35*** (1.21) | ECU > C 3.15* (1.02) | | | EADHD > AO 2.62** (0.92) | |
| 5. SDQ impact] (distress / impairment) | EADHD > C 2.06* (0.69) | ECU > C 2.91* (0.71) | | | | ECU > AO 2.21* (0.46) |
| 6 SDQ prosocial behavior | | ECU < C 6.52*** (2.16) | | ECU < EADHD 4.59*** (1.59) | | ECU < AO 4.99*** (1.91) |

Table 23 (cont'd)

| | Contrast 1 | Contrast 2 | Contrast 3 | Contrast 4 | Contrast 5 | Contrast 6 |
|---|---|---|---|---|---|---|
| | EADHD vs. C<br>*t p*<br>(effect size ) | ECU vs. C<br>*t p*<br>(effect size ) | AO vs. C<br>*t p*<br>(effect size ) | EADHD vs. ECU<br>*t p*<br>(effect size ) | EAHDH vs. AO<br>*t p*<br>(effect size ) | ECU vs. AO<br>*t p*<br>(effect size ) |
| **cognitive risks** | | | | | | |
| 7. IQ (HK-WISC / WAIS-III) | EADHD < C<br>-2.21* (0.65) | | AO < C<br>-3.50** (0.92) | | | |
| 8. average time of repeating a class | | | AO > C<br>3.82** (1.04) | | | |
| 9. history of special follow-up / training | | | | | | |
| **Family adversities** | | | | | | |
| 10. Parents' criminality | | | | | | |
| 11. Low SES | EADHD > C<br>2.78** (0.81) | | | | | |
| 12. Number of family transition before age 11 | EADHD > C<br>2.74**(0.90) | | AO > C<br>5.03*** (1.57) | | | ECU < AO<br>-.2.16* (0.82) |
| 13. years of single parenting before age 11 | | | AO > C<br>2.86** (0.98) | | | |
| 14. child abuse of parents | EADHD > C<br>2.60* (1.43) | | | | EADHD > AO<br>2.60* (0.98) | |
| 15. mental health problem of parents | | | | | | |

Table 23 (cont'd)

| | Contrast 1 | Contrast 2 | Contrast 3 | Contrast 4 | Contrast 5 | Contrast 6 |
|---|---|---|---|---|---|---|
| | EADHD vs. C<br>*t p*<br>(effect size ) | ECU vs. C<br>*t p*<br>(effect size ) | AO vs. C<br>*t p*<br>(effect size ) | EADHD vs. ECU<br>*t p*<br>(effect size ) | EAHDH vs. AO<br>*t p*<br>(effect size ) | ECU vs. AO<br>*t p*<br>(effect size ) |
| **Parenting risks** | | | | | | |
| 16. APQ mother's involvement | | ECU < C<br>-3.86*** (1.07) | | | | ECU < AO<br>-2.26* (0.71) |
| 17. APQ father's involvement | EADHD < C<br>-2.62* (0.77) | ECU < C<br>-2.49* (0.84) | | | EADHD < AO<br>-2.59 (0.88) | ECU < AO<br>-2.51 (0.97) |
| 18. APQ positive parenting | | ECU < C<br>-2.94** (1.03) | | ECU < EADHD<br>-2.06* (0.86) | | ECU < AO<br>-3.56*** (1.39) |
| 19. APQ parents' poor monitoring | EADHD > C<br>4.71*** (1.31) | ECU > C<br>2.15* (0.69) | AO > C<br>43.2** (0.79) | | | |
| 20. APQ corporal punishment | | | | | | |
| **Deviant peer affiliation** | | | | | | |
| 21. delinquency of peer group | EADHD > C<br>7.63*** (3.31) | ECU > C<br>5.58*** (2.97) | AO > C<br>4.67*** (1.85) | | EADHD > AO<br>2.20* (0.74) | |
| 22. delinquency of best friend | EADHD > C<br>8.25*** (3.92) | ECU > C<br>8.56*** (5.52) | AO > C<br>5.69*** (2.37) | | | |

Table 23 (cont'd)

| | Contrast 1 | Contrast 2 | Contrast 3 | Contrast 4 | Contrast 5 | Contrast 6 |
|---|---|---|---|---|---|---|
| | EADHD vs. C | ECU vs. C | AO vs. C | EADHD vs. ECU | EAHDH vs. AO | ECU vs. AO |
| | *t p* | *t p* | *t p* | *t p* | *t p* | *t p* |
| | (effect size ) | (effect size ) | (effect size ) | (effect size ) | (effect size ) | (effect size ) |
| **Computerized tasks** | | | | | | |
| 23. card round played / | | ECU > C | | ECU > EADHD | | ECU > AO |
| Card Playing task | | 2.80** (0.60) | | 2.44* (0.83) | | 2.56* (0.72) |
| 24. SSRT / Stop task | | | | | | |
| 25. anxiety (YSR) | EADHD > C | | | | | |
| | 2.62** (0.79) | | | | | |

Note:
EADHD = early-onset high ADHD group, ECU = early-onset high CU trait group, AO = adolescent-onset group, C = control
SDQ = Strength and Difficulties Questionnaire, APQ – Alabama Parenting Questionnaire , ICU = Inventory of Callous Unemotional Traits
SSRT = stop signal reaction time, YSR anxiety = anxious/depressed scale of Youth Self Report
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Overall significant group difference was obtained for 19 variables among all the 25 variables being tested, with Bonferroni correction performed for two measures on self delinquency (.05 / 2 = .025) and two measures on peer delinquency (.05 / 2 = .025). On all three variables indicating delinquency, all the three offending groups showed significantly higher level of delinquency, both in terms of frequency and variety and much more favorable delinquent attitude as compared to the control. All the differences between individual offending group and the control have large to huge effect sizes, ranging from 1.06 to 5.12. While the three offending groups had similar high participation in delinquency, the EADHD group reported significantly higher variety of delinquency as compared to the ECU ($d$ = 0.96) and AO group ($d$ = 0.87), whereas the ECU group had the highest mean of criminal attitude and was significantly different from that of the EADHD ($d$ = 1.02) and AO ($d$ = 1.33) respectively, with all the effect sizes being large (Table 23). Thus amongst the offending groups, the EADHD committed a more variety of delinquency, while the ECU had significantly much more favorable delinquent attitude.

On the two variables pertaining to adjustment of adolescence (SDQ difficulties and impact score), the two early-onset groups showed significantly higher difficulties and rated to be more impaired as compared to control, with effect sizes being medium

to large (ranging from $d$ =0 .69 to 1.21). But amongst the three offending groups, the EADHD group reported significantly higher level of SDQ difficulties than the AO group with a large effect size ($d$ = 0.92 ), whereas the ECU group rated to be significantly more impaired than the AO ($d$ = 0.46). When the prosocial behavior was counted, the ECU group was found to be significantly lower than the other two offending groups as well as the control. Effect sizes of these differences were very large, ranging from 1.59 to 2.16. Thus, amongst the offending groups, the ECU group had much lesser prosocial behavior.

In the previous three-group comparison, the broad early-onset offending group was found to have lower IQ as compared to the control. In the current analysis, the IQ of the EADHD (mean IQ = 98.47) group but not the ECU group (mean IQ = 103.45) was found significantly lower than that of the control (mean IQ = 105.93), with effect size = 0.65. There was no significant difference between the ECU and the control on IQ. For the average time of repeating a class, the adolescent-onset group had the highest mean, which was significantly different from the control. There was no overall significant difference obtained on the previous history of receiving special training among groups.

In the previous three-group comparison, five significant differences were obtained between the broad early-onset group and the control out of six variables, indicating that the early-onset group suffered risks in the realm of family adversities. Fewer significant differences were evident in this four-group comparison. There was no significant difference between the ECU group and the control on all the six variables. This could be due to limited statistical power because of the group's small group size ($n$ = 11). However, an examination of the group's mean on these variables suggests that this is not likely the case. On four of the five variables (where significant differences were obtained between the broad EO group and the control), the mean scores of the ECU group are lower than the grand mean of the broad early-onset group (EO). Specifically, on parents' criminality, the mean is 0.27 for the ECU whereas the mean is 0.36 for the broad EO group; on years of single parenting before age 11, the mean is 0.78 for the ECU whereas the mean is 0.93 for the EO group; on number of family transition before age 11, the mean is 0.40 for the ECU whereas the mean is 0.52 for the broad EO group; on parents' child abuse, the mean is 0.10 for the ECU whereas the mean is 0.14 for the EO. Thus, the possibility is that the early-onset high CU group is not as worse as the broad early-onset group on these family adversities. On the contrary, the pattern is reverse for the EADHD group. The mean scores of the EADHD group were found to be higher than or

comparable to the grand mean of the broad early-onset group on five out of six variables in family adversities. Specifically, on parent's criminality, the mean is 0.43 for the EAHDH whereas the mean is 0.36 for the EO, on the low SES, the mean is 0.87 for the EADHD whereas the mean is 0.67 for the EO, on the number of family transition before age 11, the mean is 0.55 for the EADHD whereas the mean is 0.52 for the EO, on the parents' child abuse, the mean is 0.53 for the EADHD whereas the mean is 0.14 for the EO, on the parents' mental health problem, the mean is 0.42 whereas the mean is 0.18 for the EO. In fact, in the current comparison, the EADHD group was found to have significantly lower SES than the control and significantly more parents' child abuse than the control and also the adolescent-onset offending group, with all effect sizes > 0.8, being large. Thus, within the early-onset offending group, it is specifically the high ADHD group but not the high CU group that suffered higher family risks as compared to the control.

In terms of parenting risks, among the three offending groups, the ECU group showed significant worse risks as compared to the control and the AO group, prominently in terms of lacking positive side of parenting, and also having more poor parenting. Specifically, The ECU had significantly lesser mother's involvement ($d$ = 1.07), father's involvement ($d$ = 0.84) and positive parenting ($d$ = 1.03) than the

control. Within offending groups, the ECU had significantly lesser mother's involvement ($d$ = .71), father's involvement ($d$ = .97) and positive parenting ($d$ = 1.39) as compared to the AO. Similar to the ECU, the EADHD group had significantly lesser father's involvement than AO and control ($d$ = .88 for the EADHD vs. AO difference and d = .77 for the EADHD vs. control difference). Both the EADHD and ECU, and also the AO group were all significantly different from the control on poor parental monitoring, indicating that all these offending groups similarly had worse parent monitoring, with effect size being 1.31 for the EADHD versus control, 0.69 for the ECU versus control, and 0.79 for the AO versus control.

On the two variables pertaining to deviant peer influences, there were huge difference between the three offending groups and the control, that each group had significantly much more peers being delinquent, and that their best friend had much higher level of delinquency. The effect size of the six significant differences ranged from 1.85 to 5.52. There were no significant difference among the offending groups, except that, the EADHD group had peers with significantly higher delinquency as compared to the AO group ($d$ = .74).

Significant group differences were found on the card playing task. As

hypothesized, the ECU group played significantly more rounds in the card playing task (mean = 93.67), as compared to the control (mean = 78.33), and the other two offending groups (mean = 72.29 for the EADHD and mean = 74.37 for the AO). The ECU group obtained a minus score (-5) on the task as compared to the mean score of 4.18 for the EADHD, 1.95 for the AO and 2.08 for the control. The three effect sizes of the group differences between the ECU and each other group ranged from 0.6 to 0.83, being medium to large. There was no significant difference among the EADHD, the AO and the control on the card task. On the Stop Task, no significant overall group difference was found. As regarded anxiety, as hypothesized, the EADHD group had the highest mean and was significantly different from the control with a close to large effect size ($d$ = .79).

Gleaning from the above results of group comparisons, it can be seen that the two early-onset groups share certain similarities with the broad early-onset offending group but each of them has some distinct features. Common to the broad early-onset offending group, the two EADHD and the ECU groups reported significantly higher level of frequency and more variety of delinquent acts committed in adolescence, as compared to control. The two groups indicated much higher difficulties in adolescence, and reported more concomitant impairment, of which the

adolescent-onset group was lacked of. However, among offending groups, the EADHD group showed the highest level of variety of self-reported delinquency whereas the ECU group had the most favorable delinquent attitude. Notably, the ECU group also developed significantly less prosocial behavior in adolescence amongst all groups whereas the EADHD group was not different from the control. In terms of background risks, there is a lack of cognitive risk in terms of low IQ for the ECU group, but the IQ of the EADHD group was significantly lower than that of the control. There is an absence of risks on family adversities but presence of parenting risks for the ECU, whereas the EADHD showed a combination of risks from both family adversities and inadequate parenting. Only the EADHD group associated with a significantly higher level of anxiety as compared to the control. It is the ECU group that played significantly more cards than all the other groups, rendering evidence for reward dominance as its distinct group feature.

## Further test on the distinct association between callous-unemotional traits and reward dominance shown by the Card Task

As already shown, there is evidence from the group comparison that the ECU group is distinctly characterized by reward dominance reflected by the card task. The underlying theory assumes that it is callous unemotional trait which associates with reward dominance. To further test on the distinct relation between CU trait and reward dominance shown by the card task, correlation analyse was first conducted to examine the correlations between reward dominance and possibly correlated variables. Hierarchical multiple regressions were then performed with card round played as criterion. Variables shown to be significantly correlated with card playing would be entered into the regression to predict card round played to see whether CU traits would remain as significant predictor of reward dominance, when the effects of other predictors being accounted for. The analysis will be based on both full sample, and also separately on the offender sample, considering that many previous findings regarding callous unemotional traits and reward dominances were based on clinical samples (Barry, et al., 2000; Frick et al., 2003; Fisher & Blair, 1998; O'Brien & Frick, 1996). Table 24 lists the correlations between the card playing, CU traits and other potential confounding variables in the full sample.

For the full sample, among 11 variables, card playing was significantly correlated with CU trait, age, and DISC ADHD diagnosis. Specifically, correlation between the card round and CU trait was .16, $p < .05$, and correlation was - .16, $p < .05$ between CU trait and card score, indicating that higher CU trait, higher card round played and lower score obtained. Correlation between age and card round was -.18, $p < .05$, whereas correlation was .15, $p = .06$, marginally significant between age and card score, indicating older subjects played fewer rounds and obtained higher score. ADHD diagnosis was significantly correlated with card round, with $r_{pb} = .16$, $p = .05$, suggesting that those with ADHD diagnosis tended to view more cards (see table 24).

Table 24.
Correlation between card round and card score with CU traits and other variables in the full sample

| | Card round (*n*) | Card score (*n*) |
|---|---|---|
| Age | .-18* | .15 (*p*=.06) |
| | (162) | (162) |
| IQ | -.10 | .13 |
| | (161) | (161) |
| Class level | -.13 | .13 |
| | (162) | (162) |
| DISC CD symptom score | .007 | -.01 |
| | (162) | (162) |
| DISC ODD symptom | .01 | -.02 |
| score | (158) | (158) |
| DISC ADHD symptom | .07 | -.08 |
| score | (158) | (158) |
| DISC CD diagnosis[a] | .002 | -.008 |
| | (161) | (161) |
| DISC ODD diagnosis[a] | .03 | -.03 |
| | (158) | (158) |
| DISC ADHD diagnosis[a] | .16* | .-15 |
| | (159) | (159) |
| YSR anxiety | .-10 | .10 |
| | (161) | (161) |
| CU trait | .16* | -.16* (p=.54) |
| (ICU total) | (155) | (155) |

Note:
YSR = Youth Self Report, ICU total = total score on Inventory of Callous Unemotional Traits
DISC = Diagnostic Interview Schedule for Children
[a]: $r$ = point-biserial correlation ($r_{pb}$) computed for categorical and continuous variable
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

In the hierarchical regression, age was first entered, followed by the ADHD diagnosis in the second block and CU traits in the third block to predict card rounds. Only age showed to be a significant predictor, whereas the effect of ADHD diagnosis and CU traits was not significant (see table 25).

Table 25.
Hierarchical regression predicting card rounds played in the card task for full sample

| Step | Predictors | Beta | $R^2$ | $R^2$ change |
|---|---|---|---|---|
| 1 | Age | -.19* ($p$ = .02) | .04 | .04* ($p$ = .02) |
| 2 | ADHD diagnosis | .13 ($p$ = .11) | .05 | .02 ($p$ = .11) |
| 3 | CU trait | .12 ($p$ = .15) | .07 | .01 ($p$ = .15) |

Note:
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

A same set of analyses was repeated for the offender sample. Table 26 listed the correlations between card playing and the 11 variables. For the offender sample, card playing was significantly correlated with CU trait, with a greater strength. Specifically, ICU total was significantly correlated with the card round with $r$ = 0.25, $p$ = .02, and with card score $r$ = -.22, $p$ = .03. Thus higher CU traits were associated with higher card rounds played and fewer card score. Correlations between card task variables and other variables were not significant, except for age and anxiety. Correlation between age and card round was -.24, $p$

< .05 and correlation between age and card score was .22, $p < .05$, indicating that offender subjects with older age played fewer rounds and obtained higher score. Correlation between anxiety and card round was -.21, $p < .05$, and with card score was .21, $p < .05$, indicating that subjects with higher anxiety played fewer rounds and obtained higher score whereas subjects with less anxiety played more rounds and obtained fewer score.

Table 26.

Correlation between card round and card score with CU traits and other variables in offender sample

| | Card round (*n*) | Card score (*n*) |
|---|---|---|
| Age | -.24* | .22* |
| | (99) | (99) |
| IQ | -.05 | .06 |
| | (98) | (98) |
| Class level | -.17 | .16 |
| | (99) | (99) |
| DISC CD symptom | -.08 | .10 |
| score | (99) | (99) |
| DISC ODD symptom | .01 | .05 |
| Score | (98) | (98) |
| DISC ADHD Symptom | -.04 | .04 |
| score | (98) | (98) |
| DISC CD diagnosis[a] | -.02 | .05 |
| | (98) | (98) |
| DISC ODD diagnosis[a] | .02 | .007 |
| | (98) | (98) |
| DISC ADHD diagnosis[a] | .19 | -.17 |
| | (98) | (98) |
| YSR anxiety | -.21* | .21* |
| | (98) | (98) |
| CU trait | .25 | -.22* |
| (ICU total) | (92) | (92) |

Note:
YSR = Youth Self Report,
ICU total = total score on Inventory of Callous Unemotional Traits
DISC = Diagnostic Interview Schedule for Children
a : $r$ = point-biserial correlation ($r_{pb}$)
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

In the subsequent hierarchical regression analyses, age was entered in the first block, anxiety in the second block, and CU trait in the last block, to predict card rounds played (table 27).

Table 27.
Hierarchical regression predicting card rounds played in the card task for the offender sample

| Step | Predictors | Beta | $R^2$ | $R^2$ change |
|---|---|---|---|---|
| 1 | Age | -.25* ($p = .02$) | .06 | .06* ($p = .02$) |
| 2 | YSR anxiety | -.19 ($p = .07$) | .10 | .04 ($p = .07$) |
| 3 | CU trait | .25* ($p = .02$) | .16 | .06*($p = .02$) |

Note:
YSR = Youth Self Report, ICU total = total score on Inventory of Callous Unemotional Traits
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

The above results showed that CU trait remained to be a significant predictor after age and anxiety were entered into the model, with beta = .25, $p = .02$. The accompanied $R^2$ change was also significant when CU trait was entered into the final block. Thus CU trait maintained its specific association with card playing task even when the effect of age and anxiety was partialled out in the offender sample.

As the correlation pattern between card task and other variables appeared to be

different for offender sample and full sample, all the correlations were also computed for the control separately and were listed in table 28. It was found that for the control, card playing was significantly correlated with ADHD symptom score only, with $r = -.31$, $p < .02$ between card score and ADHD symptom score, and $r = .31$, $p < .02$ for card rounds and ADHD symptom score. There was no significant relationship between card task and other variables including CU traits.

The above results show that the distinct association between CU trait and reward dominance measured by the card task seemed to be specific to the offender sample, which had a significantly higher level of CU traits (mean CU traits = 29.54, $SD = 7.92$), as compared to the control group (mean CU traits = 25.04, $SD = 6.12$, $t(155) = -4.2$, $p < .001$).

Table 28.

Correlation between card round and card mark with CU traits and other variables in the control group

| | Card round (*n*) | Card score (*n*) |
|---|---|---|
| Age | .-78 | .02 |
| | (63) | (63) |
| IQ | -.20 ($p$ = .12) | .23 ($p$ = .07) |
| | (63) | (161) |
| Class level | -.12 | .05 |
| | (63) | (63) |
| DISC CD symptom score | .16 | -.13 |
| | (63) | (632) |
| DISC ODD symptom | .06 | -.10 |
| score | (60) | (60) |
| DISC ADHD symptom | .31* | -.31* |
| score | (60) | (60) |
| DISC CD diagnosis[a] | .004 | .01 |
| | (63) | (63) |
| DISC ODD diagnosis[a] | .14 | -.15 |
| | (60) | (60) |
| DISC ADHD diagnosis[a] | .10 | .-12 |
| | (61) | (61) |
| YSR anxiety | .12 | .09 |
| | (63) | (63) |
| CU trait | -.004 | -.02 |
| (ICU total) | (63) | (63) |

Note:

YSR = Youth Self Report, ICU total = total score on Inventory of Callous Unemotional Traits

DISC = Diagnostic Interview Schedule for Children

[a] : $r$ = point-biserial correlation ($r_{pb}$)

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

## Clarification on the relation between ADHD and inhibition deficit measured by the Stop Task

As response inhibition deficit shown on the stop task is characteristic of ADHD (Rapport, Chung, Shore, Denney, Isaacs, 2000), it was hypothesized that the early-onset group with high ADHD symptom would also show a longer stop signal reaction time in the task, relative to the non-ADHD offending groups and the control. In the previous group comparison, there was no significant difference in this regard. The early-onset ADHD group was formed based on the DISC ADHD symptom count in which subjects with ADHD symptom score above 75$^{th}$ percentile were assigned to the group. The age of onset requirement (symptom onset before age 7) had not been counted and thus even a high score will not necessarily warrant an ADHD diagnosis. It is in indeed the case for this study. Of 14 subjects with ADHD (based on combined sources of youth and parent DISC-IV), 13 of them was in the EO group, and only three of them were categorized in the EADHD group based on the ADHD symptom score. Of the six subjects with ADHD diagnosis (based on Y-DISC-IV), five of them were in the EO group and only two was classified in the EADHD group. Thus, a significant portion of the subjects with ADHD diagnosis was not included in

EADHD group despite that the group was defined by a high symptom score based on the distributional cut off. The subsequent analysis was done to examine whether using ADHD diagnosis for analysis would show different results with respect to the stop task. Firstly, partial correlations between the SSRT of the stop task and both dimensional measure and clinical diagnoses of ADHD, CD and ODD (all based on self report) were computed, controlling for the effect of age, educational level and IQ (see table 29).

Table 29

Correlations between SSRT (stop task) and DISC dimensional measure and diagnoses of ADHD, CD and ODD

| | ADHD* diagnosis | CD* diagnosis | ODD* diagnosis | ADHD symptom score | CD Symptom score | ODD Symptom score |
|---|---|---|---|---|---|---|
| SSRT Stop task | .22** $N = 140$ | -.10 $N = 140$ | -.07 $N = 140$ | .003 $N = 140$ | -.13 $N = 144$ | -.03 $N = 140$ |

Note:

*–$r$ between diagnosis and SSRT = point-biserial correlation ($r_{pb}$)

SSRT = stop signal reaction time

ADHD = Attention Deficit Hyperactivity Disorder; CD = Conduct disorder;

ODD = Oppositional Defiant Disorder

All correlations partialled out for age, education level and IQ

*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

As can be seen, the SSRT was significantly and positively correlated with ADHD diagnosis only but not CD and ODD, indicating that having diagnosis of past year DISC ADHD was associated with a longer SSRT. The SSRT was not related to any dimensional measure of clinical diagnosis, including that of ADHD. The unique association between ADHD diagnosis and inhibition deficit shown by the stop task was further supported by the result of group comparison between subjects with and without ADHD diagnosis on the SSRT (see table 30).

Table 30
Comparison between subject with and without DISC ADHD diagnosis (youth) on SSRT

| | Subjects with DISC ADHD<br>*n* = 6<br>*M / SD* | Subjects without DISC ADHD<br>*n* = 141<br>*M / SD* | *t / p* / effect size |
|---|---|---|---|
| SSRT<br>Stop task | 306.02 ms<br>(78.07 ms) | 255.85 ms<br>(43.16 ms) | -2.7, *p*=.008<br>Cohen's d = 1.13 |

Note:
ms = milli-seconds, SSRT = stop signal reaction time
**p* ≤ .05, ** *p* ≤ .01, *** *p* ≤ .001

The SSRT of the subjects with DISC ADHD was significantly longer than that of the subjects without the diagnosis, with a large effect size (Cohen's *d* = 1.13).

The above DISC ADHD diagnosis was ascertained based on the self report of DISC-IV (Y-DISC-IV) which parallels the ADHD symptom score used for defining group, noted that ADHD symptom score was also derived from Y-DISC-IV. If the combined sources of data (P-DISC-IV and Y-DISC-IV) were used, a total of 14 ADHD diagnoses were found. Comparison between groups based on such diagnosis found a similar pattern, with a trend-level statistical significance (*p* = .07) and a medium effect size (*d* = .51), that the ADHD group was associated with a slower SSRT, indicative of inhibition deficit (Table 31).

Table 31
Comparison between subject with and without DISC ADHD diagnosis (combined parent and youth) on SSRT

| | Subjects with DISC ADHD<br>*n* = 14<br>M / SD | Subjects without DISC ADHD<br>*n* = 134<br>M / SD | *t* / *p* / effect size |
|---|---|---|---|
| SSRT<br>Stop task | 278.61 ms<br>(53.3 ms) | 255.50 ms<br>(43.81 ms) | -1.8, *p*=.07<br>Cohen's *d* = .51 |

Note.
ms = milli-seconds, SSRT = stop signal reaction time
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

The number of subjects in the ADHD group is admittedly low ($n = 6$, and $n = 14$ respectively for the diagnosis based on single-source and combined-source), which cautions against any firm conclusion based on the results. On the other hand, the results render certain support to the possibility that the deficit inhibition as hypothesized distinguished feature of the early-onset ADHD offending group could not be shown in the previous group comparison might be a result of adopting only partial diagnostic criteria of ADHD in defining group.

## Common and specific predictors of different offending groups

We know from the previous group comparison that out of a list of background risks and external correlates chosen in this study, both the two broad offending groups, i.e., the early-onset group and the adolescent-onset group, are significantly different from the control group on similar variables, including the extent and variety of delinquency and criminal attitude, all the three variables of cognitive risks (IQ, repeating class and receiving follow-up service), variables of family adversities and parenting (years of single parenting before age 11 and family transition before age 11, poor parental monitoring), variables of deviant peer affiliation, and symptom of ADHD. On the contrary, risk variables that directly differentiate the two offending groups are relatively few (the four significant differences include: EO has more child abuse, higher criminal attitude and less prosocial behavior, AO has more family transitions before age 11). The findings seem to agree more with the assertion and findings that different offending groups are subject to the influence of a common set of risk factors in differing degree (Fergusson, Horwood, & Nagin, 2000; Chung, Hill, Hawkins, Gilchrist, & Nagin, 2002), and less agree with the theory of the presence of distinct etiological factors for different offending groups (Moffitt & Caspi, 2001; Moffitt, 2003). A third possibility though is there are both common risk factors and

distinct correlates for different offending groups.

Multinominal logistic regressions were performed to identify whether there are common and specific predictors associated with different offending groups. Apart from this purpose, regression analyses also give additional information to the previous group comparisons by identifying significant predictors among a set of correlated variables, noting that various risk variables in the same class such as those of family adversities or parenting variables were correlated in this study. There are several considerations in selecting predictors. Firstly, the selection of predictors will be guided by the results of the group comparison, that variables associated with significant group differences will be entered into the regression to predict group membership. Secondly, there is a need to balance the number of predictors relative to the numbers of subjects. Stevens (2009) shows that the ratio of 15 subjects per predictor could produce a reliable prediction model. Thus for example, for 10 predictors to form a regression model, 150 subjects will be required to make the model reliable. Thirdly, variables that have excessively high correlations with other predictors should not be entered into the regression model. Excessive correlations among predictors, known as multicollinearity, could enlarge the standard error of

regression coefficient and lead to inaccurate estimation of the relative importance of predictor variables. Thus, all the variables were checked against their multicollinearity, as indicated by Tolerance and Variance Inflation Factor (VIF) before regression, where Tolerance (T) is the regression of a particular independent variable on all the other independents and is the reciprocal of VIF (1/VIF). Variables with Tolerance smaller than .1 or VIF larger than nine indicate serious multicollineraity (Stevens, 2009). Other suggests that Tolerance smaller than .25 and VIF greater than four cause significant concern of multicollineraity (Garson, 2010). The present study will adopt a more stringent criterion in screening out multicollinearity of variables. In this study, among variables, Self-reported delinquency total score has T = .10 and VIF = 9.94, Self-reported delinquency variety score has T = .12 and VIF = 8.54, and delinquency of my best friend has T = .22 and VIF = 4.53. These variables were not selected as predictor.

In the first set of analyses, predictors were entered into three separate blocks of multinominal logistic regressions to predict the log-odds of membership of the early-onset and the adolescent-onset group using the control as reference group. The effects of each independent on predicting group membership are presented as Exp (B)

(odd ratios) and are displayed in the following tables. Odd ratio with value greater than one indicates an increase in the odds of belonging to a particular offending group relative to the reference group for every unit increase of the predictor. Conversely, an odd ratio less than one indicates a decrease in the odds of belonging to a particular offending group relative to the reference group for every unit increase of a predictor. An overall model fit is shown by the chi-square goodness-of-fit test whereas the importance of each predictor to the regression model is shown by the significant chi square by the likelihood ratio test, also listed in the table (table 32).

The first regression model includes four adolescent covariates, i.e. the SDQ total difficulties, SDQ impact score, SDQ prosocial behavior and criminal attitude. Both the EO and AO group were predicted by criminal attitude, with higher criminal attitude increases the odds of the EO membership by 60% and AO membership by 37%. Additionally, the SDQ impact score uniquely predicted the EO membership, with higher score increasing EO membership by 65%.

Ten variables conceptualized as background risk variables including those of cognitive risks, family adversities and parenting risk, and deviant peer influence were

entered into a second block of regression. It is noted that distribution of child abuse of parents was zero in both the AO and the control group. When this variable was entered into the regression, the logistic regression coefficient (B) was large and the Wald test was biased to large B, leading to large standard error and meaningless result on the odd ratio. Child abuse of parent was thus not selected for regression. Among the ten predictors in the model, three predictors were common to both the EO and AO group, which include IQ, poor parental monitoring and peer delinquency. Higher IQ predicted a decrease of the EO and AO membership respectively by 5% and 6%. More problems in parental monitoring increased the EO and AO membership respectively by 20% and 18%, whereas higher peer delinquency increases the odds of being in the EO and AO group respectively by 71% and 63%. Years of single parenting emerged as a significant predictor of the EO group, with odd ratio equal to 1.49. The odd ratios (1.46) associated with the AO group predicted by years of single parenting is similar to the odd ratio associated with the EO group, but it marginally exceeded statistical significance ($p = .07$).

In the third block of multinominal logistic regression, the dimensional and categorical measure of ADHD (symptom score and diagnosis), and the CU trait were

entered. Both the EO and AO were predicted by the ADHD symptom score but not the diagnosis, that higher ADHD symptom increased the EO and AO membership by 18% and 19% respectively. The callous-unemotional trait was a unique predictor of EO group, that higher CU trait increased the EO membership by 7%.

On the whole, from the three blocks of multinominal logistic regressions, five predictors including criminal attitude, IQ, poor parental monitoring, deviant peer influence, and ADHD symptoms are found to be common to both the EO and AO groups, whereas higher impairment and distress associated with adjustment problem (SDQ impact sore), more years of single parenting and higher CU traits are specific predictors to the EO group.

Table 32
Multinominal logistic regression predicting early-onset and adolescent-onset group

| Block 1<br>Predictors:<br>Adolescent covariates | Early-onset group<br>($n$ = 89)<br>Exp (B) / $p$ | Adolescent-onset<br>group ($n$ = 18)<br>Exp (B) / $p$ | Likelihood ratio test<br>($\chi^2$ / $df$ / $p$) |
|---|---|---|---|
| 1. SDQ total difficulties | .94 / .30 | .99 / .90 | 1.28 / 2 / .53 |
| 2. SDQ impact score | **1.65*** / .02 | 1.20 / .53 | 7.56* / 2 / .02 |
| 3. SDQ prosocial behavior | .84 / .16 | .92 / .60 | 2.06 / 2 / .36 |
| 4. criminal attitude | **1.60***** / <.001 | **1.37**** / .002 | 46.62*** / 2 / <.001 |
| Full model ($n$ = 164) | | | 75.04*** / 8 / <.001 |
| Block 2<br>Predictors:<br>Background risks of delinquency | Early-onset group<br>($n$ = 70)<br>Exp (B) / $p$ | Adolescent-onset<br>group ($n$ = 17)<br>Exp (B) / $p$ | Likelihood ratio test<br>($\chi^2$ / $df$ / $p$) |
| 1. IQ | **.95*** / .05 | **.94**** / .05 | 4.96 / 2 / .08 |
| 2. repeating a class | 1.84 / .26 | 2.96 / .09 [a] | 3.09 / 2 / .21 |
| 3. special follow-up<br>( 0 = no follow-up) | .40 / .35 | .31 / .32 | 1.07 / 2 / .59 |
| 4. parents' criminality | 120 / .71 | .86 / .79 | 0.87 / 2 / .64 |
| 5. SES | 1.40 / .40 | 1.04 / .93 | 1.19 / 2 / .55 |
| 5. years of single parenting<br>before age 11 | **1.49*** / .05 | 1.46 / .07 [b] | 4.93 / 2 / .09 |
| 7. number of family transition before age 11 | .99 / .99 | 4.79 / .09 [c] | 11.18** / 2 / .004 |
| 8. APQ mother's Involvement | 1.000 / .84 | 1.04 / .47 | .77 / 2 / .68 |
| 9. APQ poor parental Monitoring | **1.20**** / .006 | **1.18***/ .05 | 8.75** / 2 / .01 |
| 10. Peer delinquency total | **1.71**** / <.001 | **1.63***** / <.001 | 44.68*** / 2 / <.001 |
| Full model ($n$ = 151) | | | 153.52 / 16 / <.001 |

Table 32 (cont'd)

| Block 3<br>Predictors:<br>ADHD symptoms and CU trait | Early-onset group<br>($n$ = 89)<br>Exp (B) / $p$ | Adolescent-onset group ($n$ = 19)<br>Exp (B) / $p$ | Likelihood ratio test ($\chi^2$ / $df$ / $p$) |
|---|---|---|---|
| 1. CU trait | **1.07*** / .021 | 1.03 / .47 | 5.89* / 2 / .05 |
| 2. DISC ADHD diagnosis (past year) ( 0 = no diagnosis) | .34 / .33 | .78 / .87 | 1.63 / 2 / .44 |
| 3. DISC ADHD total symptom (past year) | **1.18***** / <.001 | **1.19**** / .006 | 16.43*** / 2 / <.001 |
| Full model 3 ($n$ = 166) | | | 35.33*** / 6 / <.001 |

Note:
Reference group: control
APQ = Alabama Parenting Questionnaire
SDQ = Strength and Difficulties Questionnaire
ADHD = attention deficit hyperactivity disorder
CU trait = callous-unemotional trait
DISC = Diagnostic Interview Schedule for Children
a = 95% confidence interval for the odd ratio (2.96) = 0.86 – 10.28, B = 1.09 and SE = 0.63
b = 95% confidence interval for the odd ratio (1.46) = 0.97 – 2.20, B = 0.38 and SE = 0.21
c = 95% confidence interval for the odd ratio (4.79) = 0.80 – 28.62, B = 1.57 and SE = 0.91
bolded predictors are significant predictors
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

In the second set of logistic regression analyses, the two early-onset groups, i.e., the early-onset ADHD group and the early-onset CU trait group were predicted with the control as the reference group by three separate blocks of multinominal logistic regressions (see table 33). Likewise, the selection of predictors is based on the results of the previous group comparison and the balance of predictor to sample size ratio. Model fit of all three blocks of regression was showed by the significant model chi square. In the first block of multinominal regression, five predictors including SDQ total difficulties, SDQ impact score, SDQ prosocial behavior, criminal attitude, and peer delinquency were entered to predict group membership with the control as reference category. Results of the previous group comparison showed that the ECU and the EADHD group were both similarly different from the control on four of these variables (criminal attitude, SDQ difficulties, SDQ impact score and peer delinquency) whereas only the ECU was significantly different from the control on prosocial behavior. Results of the regression show that amongst these five predictors, both the ECU and EADHD group were predicted by criminal attitude and peer delinquency. Higher criminal attitude increased the odds of being in ECU group and EADHD group respectively by more than 5 times (Exp B = 5.91 and 5.95). Higher peer delinquency increased the odds of being EADHD by 3.35 times (Exp B = 3.35) and ECU by 2.08 times (Exp B = 2.08). Prosocial behavior was a unique predictor of the ECU group,

that higher prosocial behavior reduced the ECU group membership by 88%.

In the second regression model, the background risk variables were entered as predictors. It is noted that the total number of subjects of these three groups are less than 100 and thus precludes entering all background risk variables associated with significant group differences. Given this constraint, the variables of family adversities (low SES and family transition before age 11) and parenting risks (mother's involvement, father's involvement, positive parenting and poor parental monitoring) were selected in the regression model to identify significant predictors among these correlated variables. Peer delinquency was entered into the first block of regression for testing. Of the six predictors entered, two were significant predictors of the EADHD group, where this group was predicted by higher family transition in childhood with a high odd ratio (11.87) and more poor parental monitoring (odd ratio = 1.3). No variable was significant predictors for the ECU group, despite that in previous group comparison the ECU was found to be significantly lower in positive side of parenting as compared to the AO and control. It is noted that odd ratios associated with the ECU group membership predicted by positive parenting (exp (B) = 0.72, $p = .06$) and poor parental monitoring (exp (B) = 1.15, $p = .06$) marginally exceeded the .05 significance level.

Three variables of individual attributes including IQ, reward dominance (shown

by card playing) and anxiety were entered into a separate block of regression. The EADHD group was predicted by IQ and YSR anxiety, that higher anxiety increased for the group membership by 91% and higher IQ decreased the group membership by 7%. No independent variable was independent predictor of the ECU group.

Taking the results of these three blocks of regression together, there are two predictors common to both the EADHD and ECU group, i.e., criminal attitude and peer delinquency, where higher criminal attitude and delinquency increased risk of being in both groups. Prosocial behavior uniquely predicted the ECU group inversely, whereas family transition in childhood, poor parental monitoring, IQ and anxiety uniquely predicted the EAHD membership.

For the sake of clarity, table 34 summarizes the significant predictors of the all the offending groups, noting that the reference category for all offending groups is equally the control group. As can be seen from the table, there are both common correlates (such as criminal attitude and peer delinquency for all groups) and specific correlates (such as prosocial behavior for the ECU group) associated with the four offending groups.

Table 33.
Multinominal logistic regression predicting EADHD and ECU group

| Block 1<br>Predictors:<br>Adolescent covariates | EADHD group<br>($n$ = 17)<br>Exp (B) / $p$ | ECU group<br>($n$ = 11)<br>Exp (B) / $p$ | Likelihood ratio test ($\chi^2$ / $df$ / $p$) |
|---|---|---|---|
| 1. SDQ total difficulties | .83/ .61 | 1.11 / .76 | 3.17 / 2 / .21 |
| 2. SDQ impact score | 1.87 / .54 | 3.22 / .25 | 2.54 / 2 / .28 |
| 3. SDQ prosocial behavior | 1.11/ .89 | **.12**** / .013 | 16.85** / 2 / <.001 |
| 4. criminal attitude | **5.91*** / .03 | **5.95***** / .03 | 18.93*** / 2 / <.001 |
| 5. peer delinquency | **3.35***** / .005 | **2.08***** / .04 | 46.80*** / 2 / <.001 |
| Full model<br>$n$ = 90 | | | 123.77*** / 10 / <.001 |
| **Block 2**<br>**Predictors:**<br>**Background risks of family and parenting risks** | **EADHD group**<br>**($n$ = 14)**<br>**Exp (B) / $p$** | **ECU group**<br>**($n$ = 11)**<br>**Exp (B) / $p$** | **Likelihood ratio test ($\chi^2$ / $df$ / $p$)** |
| 1. low SES | 1.50 / .32 | 2.15 / .09[a] | 2.92 / 2 / .23 |
| 2. number of family transition before age 11 | **11.87*** / .023 | 6.74 / .09[b] | 6.48* / 2 / .04 |
| 3. APQ mother's Involvement | .93 / .47 | 0.85 / .09 | 3.13 / 2 / .21 |
| 4. APQ father's involvement | .99 / .86 | 1.06 / .55 | .58 / 2 / .75 |
| 5. APQ positive parenting | .90 / .39 | .72 / .06 | 4.67 / 2 / .01 |
| 6. APQ poor parental Monitoring | **1.30***** / .001 | 1.15 / .06 | 17.18*** / 2 / <.001 |
| Full model<br>$n$ = 80 | | | 52.70***/ 12 / <.001 |

Table 33 (cont'd)

| Block 3 Predictors: Individual's characteristics | EADHD group ($n$ = 17) Exp (B) / $p$ | ECU group ($n$ = 9) Exp (B) / $p$ | Likelihood ratio test ($\chi^2$ / $df$ / $p$) |
|---|---|---|---|
| 1. IQ | **.93**** / .013 | 1.00 / .96 | 7.83* / 2 / .02 |
| 2. reward dominance (card round played) | .99 / .28 | 1.03 / .16 | 4.96 / 2 / .08 |
| 3. YSR anxiety | **1.91**** / .004 | 1.11 / .12 | 7.83* / 2 / .02 |
| Full model $n$ = 89 | | | 20.88**/ 6 / .002 |

Note:
Reference group: control
EADHD = early-onset high ADHD group
ECU = early-onset high CU trait group
CU trait = callous-unemotional trait
APQ = Alabama Parenting Questionnaire
SDQ = Strength and Difficulties Questionnaire
ADHD = attention deficit hyperactivity disorder
DISC = Diagnostic Interview Schedule for Children
a = 95% confidence interval for the odd ratio (2.15) = 0.88 – 5.23, B = 0.77 and SE = 0.45
b = 95% confidence interval for the odd ratio (6.74) = 0.74 – 61.55, B = 1.91 and SE = 1.13
bolded predictors are significant predictors
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 34
Significant predictors of different offending groups and the associated Exp (B)

| Predictors | EO group Exp (B) | AO group Exp (B) | EAHDH group Exp (B) | ECU group Exp (B) |
|---|---|---|---|---|
| Criminal attitude | 1.60*** | 1.37** | 5.91*** | 5.96*** |
| SDQ impact score | 1.65* | ns | ns | ns |
| SDQ prosocial behavior | ns | ns | ns | .12** |
| IQ | .95* | .94* | .93** | ns |
| Poor parental monitoring | 1.20** | 1.18* | 1.30*** | ns |
| Years of single parenting before age 11 | 1.49* | ns | *Not tested* | *Not tested* |
| Number of family transition before age 11 | ns | ns | 11.87* | Ns |
| Peer delinquency | 1.71** | 1.63*** | 3.35*** | 2.08*** |
| ADHD symptoms | 1.18*** | 1.19** | X | X |
| CU trait | 1.07* | ns | X | X |
| YSR anxiety | *Not tested* | *Not tested* | 1.19** | ns |

Note:
Reference group: control
EO = early-onset offending group, AO = adolescent-onset offending group
EADHD = early-onset ADHD group; ECU = early-onset CU trait group
*Not tested* – no significant group difference between offending group vs. control was found in the group comparisons, and thus these variables were not entered in the regression model; ns = non significant
X: ADHD symptoms and CU traits are defining characteristic of the EADHD and ECU group and thus were not entered to predict their group membership
*$p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

# Chapter IV

# Discussion

This study tests a taxonomy of antisocial and delinquent behaviors by examining whether different offending groups hypothesized could be distinguished by their associated group features hypothesized. Based on a growing and substantial literature on the differentiation between the early-onset and the adolescent-onset antisocial behavior, this study identified these two broad offending groups based on a priori criteria (age of first CD symptom on or before age 11) in 116 offender subjects. Together with 63 non-delinquent control subjects, a three-group comparison and multinominal logistic regression were adopted to test whether the two offending groups were characterized by different background risks and adolescent correlates. The selection of these external correlates and background risk variables were based on the theory of Moffitt and Patterson, who hypothesized different etiological factors of these offending groups. According to these etiological theories and previous findings, these two broad offending groups should show differential group features which could be tested in this study, even within the constraints of a cross-sectional design. Previous studies showed that inattention and hyperactivity, and callous-unemotional trait are two important differentiators that mark different subgroups

whose antisocial behavior could develop through different etiological mechanism with an early onset similarly. These two subgroups are mostly identified in separate sample in many previous studies. Within the same sample this study isolated these two independent subgroups hypothesized to coexist in the early-onset group based on the associated ADHD symptoms (n = 17) and callous-unemotional traits (n = 11). Multivariate statistical analyses including group comparison and multinominal regression then test the hypothesis that these two subgroups, despite both belonging to the early-onset group, are different from each other in terms of background risks and correlates.

## The differentiation between the two broad offending groups: the early-onset (EO) and the adolescent-onset (AO) group

*The proportion of the EO group and AO group.* According to Moffitt's two-trajectory model, the early-onset group is pathological and rare as compared to the adolescent-onset group which is near normative and much higher in number. This is true for the Dunedin's study, which recruited a representative sample, regardless whether the groups were derived by preset clinical criteria (displayed pervasive antisocial behavior at three assessment points out of four at ages 5, 7, 9, 11)

or the new modeling method that makes no a priori assumptions about the grouping. Specifically, 10% and 25% of the Dunedin male study members were classified in the LCP and AL group respectively using clinical criteria whereas 10.5% and 19.6% were classified into the LCP and AL group respectively by modeling method. In this study, based on the age of onset of CD symptom on or before 11, 82% of the offender subjects were grouped as EO members whereas 18% of them were in the AO group. It is not uncommon to find that the early-onset offending group, which is supposed to be a relatively rare group, will resemble or even outnumber the adolescent-group in size in studies with high-risk or forensic sample such as adjudicated sample in this study. In Oregon Youth Study which followed up a high risk sample of 206 boys, early-onset group included 43 subjects and late-onset group included 52 subjects, using age of first arrest before 14 as criteria (Patterson, 1996; Patterson & Yoerger, 1997). In a recent study also using forensic sample (78 pre-adjudicated males) and also the same onset age as group formation criteria (onset of delinquent act or symptom of DSM-IV conduct disorder on or before age 11), similarly, the subjects of the childhood-onset group members ( n = 47) outnumbered that of adolescent-onset group (n = 31) (Dandreau & Frick, 2009).

*Could the EO group be differentiated from the AO group in terms of background risk variables and external correlates?* Despite different hypotheses on the etiology of the early-onset versus the adolescent- onset offending trajectories, according to both Moffitt (Moffitt & Caspi, 2001; Moffitt, 2003) and Patterson (Patterson, 1996; Patterson & Yoerger, 1997), the early-onset offending group is the one group that suffers worst childhood risks and thus could be differentiated from the adolescent offending group in terms of background risk variables. Patterson also made it explicit that over the course of development the early-onset delinquents would become both deviant and most underdeveloped such as in prosocial behavior. Results of this study provide support to these hypotheses in several ways. Firstly, the results of the three-group comparison found that the EO but not the AO group had significantly more psychosocial difficulties (in terms of emotional symptoms, conduct problem and hyperactivity, and peer difficulties measured by the SDQ), as well as associated distress and impairment in adolescence than the control subjects. Also notably, the EO members had developed significantly less prosocial behavior (SDQ prosocial behavior) than both the control and the AO group and their criminal attitude was also signficantly higher than both the control and AO members. The EO group thus could be regarded as most deviant (in terms of criminal attitude) and that their

prosocial behavior was most underdeveloped, consistent with Patterson and Moffitt's descriptions. Secondly, in terms of background risks, the EO group was found to be the worst group, that it was significantly worse than the control on a total of 12 out of 16 background risk variables cutting across the realm of cognitive risk (lower IQ, more years of repeating a class, higher proportion of receiving special training), family adversities (more child abuse, more family transition and single parenting before age 11, lower SES, more parent's criminality), parenting risks (less mother's involvement and more poor parental monitoring), and deviant peer influence (higher peer delinquency and best friend being more delinquent). Relatively, the AO group was characterized by less background risks when compared to the non-delinquent controls (8 significant differences were found out of 16 background risks). Previous studies on the effect of cumulative risks on later developmental outcomes found evidence that the presence of more risks predicts more unfavorable developmental outcomes, independent of the presence or absence of a specific risk (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Rutter, 1979; Sameroff, 2000). That the EO group showed a higher number of background risks will imply unfavorable adult outcomes, relative to the AO group. Results from various longitudinal studies with representative sample or high-risk sample provide ample evidence on the poor

outcomes of the EO group, apart from their persistence of delinquent behaviors into adulthood. In the Dunedin study, the early-starters (LCP group) had the poorest outcomes in terms of mental health, physical health and economic problem when followed up to age 32 (Odgers & Moffitt, et al., 2008; Odgers et al, 2007; Piquero, Gibson, Daigle, Piquero, & Tibbetts, 2007). In the Cambridge Study in Delinquent Development which followed up 411 high-risk male subjects, the group of persistent offender (convicted at ages 10 – 20 and 21 – 50, similar to the LCP group) led the most unsuccessful lives at ages 32 and 48 (in terms of living accommodation, employment, use of drug and alcohol, general mental health, use of violence, intimate relationship, etc.) (Farrington, Ttofi, & Coid, 2009).

Findings derived from two lines of research investigation separately on conduct problem comorbid with ADHD (Green, Keenan, & Lahey, 1995; Loeber et al., 2000, Vermeiren, 2003; Waschbush, 2002) and more recently, on conduct problem associated with CU trait (Christian, Frick, Hill, Tyler, & Fraxer, 1997; Frick & White, 2008; Rowe, Maughan, Moran, Ford, Briskman, & Goodman, 2010; Silverthorn, Frick, & Reynolds, 2001) point to the importance of comorbid ADHD and CU traits in subtyping antisocial behavior. One of the findings from these studies is that

antisocial behavior comorbid with ADHD and antisocial behavior associated CU traits are both characterized by an early onset. This study thus hypothesized that both ADHD and CU traits are more prominent characteristics of the EO group, but less found of the AO group. Results render support to this hypothesis. Childhood hyperactivity, impulsivity and inattention (ADHD), and callous-unemotional trait (CU trait) were both found to be associated with the EO group in this study. Results of the three-group comparison showed significantly higher level of CU trait in the EO but not in the AO as compared to the control. · Also, proportion of having childhood hyperactivity, impulsivity and inattention (DISC ADHD diagnosis which requires onset of symptoms before age 7) was significantly higher than the control in EO subjects but not in the AO. This finding is important as it links up three lines of literature on antisocial behavior marked by comorbid ADHD, antisocial behavior marked by CU trait, and antisocial behavior marked by early-onset to form the base of a taxonomy of antisocial behavior in which the early-onset offending group comprises two distinct groups associated with different features and that their antisocial behaviors possibly developed through different developmental pathways. The subsequent analyses on these two early-onset subgroups further test this notion and will be discussed later.

Taking together, more adolescent adjustment difficulties, underdeveloped prosocial behavior (SDQ variables), higher deviancy (criminal attitude), more background risks, higher rate of childhood ADHD, and higher CU trait are features that characterize the EO group. However, support to the differentiation of the EO versus the AO group obtained by this study should be interpreted also with a view that their differentiation were shown by differential significant differences relative to the non-delinquent control. There were few significant differences between the EO group and the AO on their background risks, as that shown by Moffitt's team (Moffitt & Caspi, 2001; Odgers & Moffitt, 2008), and other studies (Aguilar, et al., 2000; Brennan, et al., 2003; Fergusson & Horwood, 2002; Fergusson & Nagin, 2000; McCabe, Hough, Wood, Yeh, 2001). In Dunedin study, the LCP group ($n$=53) was significant worst than the AL group ($n$ = 200) on 21 out of 26 background risks, including that of undesirable parenting and family processes, child-neuro-cognitive risks, child temperamental and behavior risks, with effect size being small for 15 risk factors, medium for 3 risk factors, large for 6 risk factors. The six large effect sizes all came from the significant differences on child temperament-behavior risks at childhood, most of which could not be measured in this study (including age 5 and 11 hyperactivity, fighting and peer rejected rated by both parent and teacher). Effect

sizes associated with parenting and family risk variables, and neuro-cognitive risks were mostly small, as found in the data reported by Moffitt et al. (2002). In this study, statistical power to detect group differences between the EO and AO group is relatively low, considering $n = 95$ for the EO group and $n = 21$ for the AO group. Specifically, there are five EO versus AO differences (EO > AO) with small effect size which could not be detected as significant, including SDQ impact score (Cohen's $d = .31$), APQ mother's involvement (Cohen's $d = 0.31$), APQ parents' poor monitoring (Cohen's $d = 0.38$), peer delinquency (Cohen's $d = 0.32$), CU traits (Cohen's $d = 0.28$).

Small statistical power partly explains the lack of statistically significant differences between the EO and AO group. On the other hand, the findings are similar to that found by various previous studies, that there was a lack of differentiation among different offending trajectory groups in terms of background correlates or risks (Chung, Hill, Hawkins, Gilchrist, & Nagin, 2002; White, Bates, & Buyske, S. 2001; Wiesner & Capaldi, 2003; Wiesner & Windle; 2004). For example, White et al. (2001) recruited 698 male community subjects aged from 12-18 and through modeling identified four offending trajectories including nondelinquents

(47%), adolescent-limited delinquents (33%) (similar to Moffitt's AL group), adolescent-to adulthood-life-course-persistent delinquents (7%) (similar to LCP group of Moffitt), and escalating delinquents (13%) (delinquent behaviors increased into adulthood). These offending groups were tested on their differences on delinquent behavior and risk factors including neuropsychological problems (birth risks, verbal abilities, and executive functioning), personality variables (impulsivity, harm avoidance and disinhibition), family adversities (family's SES, family structure – single vs. two-parent family, parental hostility), in which one set of analyses compared adolescence-limited offending versus persistent offending group. It was found that only one risk factor, i.e., disinhibition (measured by sensation-seeking scale of Zuckerman, 1979) differentiated between adolescence-limited offenders and persistent offenders (persistent offenders higher in disinhibition, odd ratio = 1.19 for persistent offending). Most of the significant findings on the differentiation between offending groups in this study came from the comparison between the two extreme groups, i.e., the non-delinquent versus the delinquent group (3 trajectory groups as a whole), that delinquents were associated with higher impulsivity, lower harm avoidance, higher disinhibition, higher parental hostility, more single-parent family.

As reviewed earlier in chapter I, Wiesner & Capaldi (2003) identified six

trajectory groups through modeling using the data set from Oregon Youth study (204 at-risks boy being followed up from 9-10 year to 23-24 years annually), which included: 1. chronic high-level (similar to the LCP group of Moffitt); 2. decreasing high level 3. chronic low-level, 4. decreasing low-level, 5. rare offenders (n = 23; less than one offense each year), and 6. non-offenders. These groups were then compared on childhood risk variables including parents' arrests, childhood antisocial behavior, childhood attention problem, academic achievement, poor parental discipline and low parental supervision, and adolescent's measures including depressive symptom, sensation seeking, substance use, risky sexual behavior and deviant peer association, using multinominal logistic regression. The rare offender and non-offender groups were combined to become very rare offender group. It was found that only the two extreme groups, that is, the very rare offender and the high level chronic offender could be distinguished by two childhood background risks (attention problem and low parental supervision). The other three offending groups (decreasing low level, decreasing high level and chronic low level) could not be distinguished from the high chronic level by any childhood risk. Apart from these longitudinal studies, one study that is similar to the present study in its cross-sectional design, sample used (86 institutionalized adolescents and 43 regular school students)

and statistical analysis methods (group comparison by ANOVA), found that the early-onset group and the late-onset group could not be distinguished by any variables pertaining to the external correlate of impulsivity (measured by performance-based tests including stroop colour and word test, time perception, accuracy game and risk-taking game and questionnaire, the Eysenck Impulsiveness questionnaire) (Carroll, et al, 2006).

The findings of the group comparison on the EO and the AO raises the question about whether different offending patterns manifested are subject to the influence of a common set of factors, if not caused by a same factor such as the same propensity of lack of self control as asserted by criminologist (Gottfredson & Hirschi, 1990), or whether different offending groups are associated with specific risks, or risk variables that exert different effects on specific group. There is no single-sided support to this argument. In this study, results of the multinominal logistic regression showed that increased bad parental monitoring, increased peer delinquency, higher criminal attitude, IQ, and behavioral manifestation of ADHD symptom (ADHD symptom score but not the diagnosis) commonly predicted both the EO and AO with on the whole similar odd ratios. On the other hand, the EO was found to be uniquely predicted

by increased impairment and distress associated with their psychosocial problem (SDQ impact score), more years of single parenting, and higher level of CU traits. It is entirely possible that certain common risk variables are potent factors that trigger and maintain antisocial behaviour, as what Patterson had stated on the important role of poor parenting and deviant peer in early-onset and late-onset offending behaviours (Patterson, 1996; Patterson & Yoerger, 1997), which shown to be associated with both the EO and the AO in this study . Disrupted parenting practices and direct training by deviant peers were both proximal mechanisms for the production of antisocial behaviour, analogues to the disease process for the progression of cancer symptom (Patterson et al., 1998). The importance of these variables to delinquency are also reflected by their strong correlation with offending status in the current study, considering that $r_{pb} = .47, p < .001$ between offending status and parental monitoring, and $r_{pb} = .62, p < .001$ and $r_{pb} = .73, p < .001$ between offending status and peer delinquency, and between offending status and delinquency of best friend respectively. However, the presence of common risk factors should not necessarily contradict the role of specific factors shown to be associated with specific offending group, such as in the case of CU traits as unique predictor of the EO group, because it possibly implies specific causal pathways of development of antisocial behaviour, which

remains to be tested.

*The nature of the AO group.* One of the major purposes of this study is to test the nature of the adolescent-onset group, based on different etiological theories of Moffitt and Patterson. Moffitt hypothesized that the adolescent-limited offending was initiated as an adaptation response of some youngsters who mimic the antisocial behavior of deviant peers to cope with the maturation gap occurred in the period of adolescence. These adolescents were characterized by normative background and normative pre-delinquent development in prosocial behavior and social skills. Patterson asserted that disrupted family and parenting processes still played a role for the late-onset offending as in the early-onset group but differed in degree, that the late-onset offending adolescents were intermediate between the early-onset group and the normative youngsters in terms of the family and parenting risks. The late-onset youngsters had marginal adjustment as adolescents such as prosocial behavior. This study found that the mean scores obtained by the AO group on variables signifying adjustment difficulties in adolescence (SDQ difficulties and distress score) and prosocial behavior were intermediate between the EO and the control, though the differences between the AO and the control group on these variables were not

statistically different (despite that effect sizes for their group difference were over 0.3 for SDQ total difficulties and prosocial behaviors). Also, there is clearly more evidence for Patterson's proposal in risk background of the AO group, that this group suffers certain degree of background risks and could be differentiated from the normative control. Result of the group comparison found that the AO group was significantly worse than the control group on a total of 8 variables including that of family adversities (more family transition and more years of single parenting), disrupted parenting (more parental monitoring), and deviant peer affiliation with most of the effect sizes being large (number of family transition: Cohen's $d = 1.57$; years of single parenting: Cohen's $d = 0.96$; poor parental monitoring: Cohen's $d = 0.79$; delinquency of peer: Cohen's $d = 1.83$ and delinquency of best friend: Cohen's $d = 2.35$). That the deviant peer influence associated with the adolescent-onset offending is both anticipated by Moffitt and Patterson. But in term of family and parenting risks, the AO group could not be said as normative as what Moffitt hypothesized and could be described as intermediate between the EO and the normative control. Similar to the EO group, the AO group had significantly more family transition and more years of single parenting as compared to the control. The AO group was also associated with poor parental monitoring but not to the degree as

that of the EO (Cohen's $d = 0.79$ for the AO vs. control difference and Cohen's $d = 1.19$ for the EO vs. control difference).

Apart from family adversities and parenting risks, the AO group was also found to show considerable cognitive risks, notably, lower IQ (mean = 95.02, *SD* = 13.23), which was not significantly different from that of the EO group (mean = 99.36, *SD* = 14.67) but significantly lower than that of the control group (mean = 105.93, *SD* = 11.89). This finding is not consistent with previous studies which reported a lower IQ for the early-onset offending but not the adolescent-onset offending group, supportive of Moffitt's hypothesis regarding specific association between neuropsychological deficits and the early-onset life-course-persistent delinquency. Moffitt (1993) proposed that LCP delinquency was associated with lower verbal functions, as one of the major manifestation of neuropsychological deficits. This association was found in the Dunedin study, that the LCP group had lower verbal IQ (Wechsler Intelligence Scale for Children -R measured at age 7, 9, 11) than that of the control group and the AL group (Moffitt & Caspi, 2001), whereas the adolescent-limited group had a normative IQ. Using the youngest cohort of the Pittsburgh Youth Study (335 boys recruited at age 7 and tested at 17 years), Raine, et

al. (2005) also found a lower verbal intelligence (estimated from the subtest of Information and Vocabulary of the Wechsler Intelligence Scale for Children-III) for the early-onset life-course-persistent group (n = 44), as compared to the adolescent-limited offending group ($n$ = 68). But it is noted that there was no significant difference between the two offending groups on total IQ (based on Information, vocabulary, Block design and Picture Completion) and both groups were significantly below than the control on total IQ. However, some other studies did not find difference between the two offending groups on verbal IQ or general IQ. White et al. (2001) found no difference between the adolescence-limited delinquents and the persisters on verbal abilities (measured by the vocabulary test of Shipley Institute of Living Scale, Zachary, 1986) in a sample of 698 males recruited at age 12-18 and followed up for 13 years. Aguilar, et al., (2000) found no difference between the early-onset/persistent group and adolescent-onset group on both the WISC-R verbal and performance scores (Vocabulary, Similarities and Block Design) administered in the third grade of 212 high-risk subjects followed from birth to their 16 years. Siverthorn, et al. (2001) compared male early-onset delinquent group ($n$ = 11) to the late-onset group ($n$ = 12) in a sample of 72 incarcerated adolescents (mean age of 15.37) and found no significant group difference between the two groups on IQ. In

the current study, both the EO and the AO group had significantly lower global IQ as compared to the control. The results are consistent with the findings reviewed above that the offender groups as a whole had lower IQ relative to the normal control. Correlation between IQ and offending status in this study is -.25, $p = .001$, also in line with the evidence for the negative correlation between IQ and delinquency reported in the literature (Lynam, Moffitt, & Stouthamer-Loeber, 1993). That lower IQ as a distinct risk to children with early-onset antisocial behaviors is less consistently found in the literature could be related to the heterogeneity of this group. As shown in this current study, within the early-onset group, the subgroup marked by ADHD had a low IQ but the subgroup marked by CU traits did not have IQ deficit.

Findings in the current literature are mixed regarding the difference between the early-onset and the adolescent-onset group on impulsivity, which is hypothesized also as a key dispositional risk characteristic of the early-onset group (Moffitt, 1993) but not the adolescent-onset group. Moffitt et al. (2001) found that the LCP group was higher than the AL group in observed under-controlled behavior at age 3 and teacher- and parent-rated impulsivity measured at age 5 and 11. Several other studies found no difference between the two offending groups in terms of impulsivity tapped by

different measures. Aguilar, et al. (2000) found no difference between the two offending groups on temperamental impulsivity measured early at 30 months of 212 high risk sample subjects (mother completed the EASI Temperament Survey of Buss and Plomin, 1975). White et al. (2001) found no difference between the early-onset and adolescent-onset groups on impulsivity measured at adolescence (age 12 to 18) by questionnaire (Jackson's Personality Research Form). In Carroll et al. study (2006), the early-onset and the late-onset groups derived in a forensic adolescent sample could not be distinguished by any variables of impulsivity measured by a list of both performance-based tests (such as stroop colour and word test and time perception) and questionnaire (Eysenck Impulsiveness questionnaire). Dandreaux et al. (2009) found no difference between the early- and adolescent-onset group with an adjudicated adolescent sample on impulsivity measured by two personality questionnaires (Multidimensional Personality Questionnaire, Patrick, et al., 2002 and Behavioral Assessment system for children – 2nd edition self-report of personality, Reynolds & Kamphaus, 2004). However, McCabe et al. (2001) did find difference between early-onset and the late-onset conduct disorder group on impulsivity indicated by DISC-IV ADHD diagnosis with a sample of 295 adolescent subjects (mean age 15.17 years). Raine et al. (2005) found no difference between the life-course persistent

offending and the adolescent-limited offending on cognitive measure of impulsivity (such as Continuous Performance Test and Wisconsin Card Sorting Test) but they did find that the early-onset group had a higher rate of DISC ADHD diagnosis. Similarly, this study also found a significantly higher rate of DISC-IV ADHD diagnosis in the EO but not the AO group (13.7% vs. 4.8%). But interestingly, the AO group was similarly characterized by higher level of impulsivity as the EO group, as indicated by a similar level of ADHD symptom score which counts the past-year symptoms with no restriction on onset age before 7. This could imply that in terms of behavioral manifestation at the stage of adolescence, both the EO and AO members could be impulsive, hyperactive and inattentive; and could be difficult to be distinguished by overt behaviours, without looking into their developmental history and background risks.

## Differential features of the two early-onset subgroups: Early-onset group with ADHD symptoms (EADHD) and early-onset group with callous-unemotional trait (ECU)

Based on a substantial line of studies on antisocial behavior comorbid with ADHD and a growing area of studies advancing callous-unemotional traits as important marker of antisocial behaviors, two groups were hypothesized to coexist in the broad early-onset offending group with different features.

*Similarities between the EADHD and ECU group.* Both the EADHD and the ECU group are similar to the broad early-onset group in terms of having significantly higher extent and variety of self-reported delinquency as compared to the control, but with much larger effect sizes. The effect sizes associated with the EO-control difference on the frequency and variety of self-reported delinquency are 2.84, and 2.5 respectively, whereas the effect sizes associated with the differences between the EADHD and ECU versus the control were huge, ranging from $d = 4.6$ to 5.21, showing a more pronounced difference between the two offending groups and the control on frequency and variety of delinquency. Also, the two groups had significantly more favorable criminal attitude as compared to the control, and again

with larger effect sizes, particularly for the ECU group. Effect size was 3.03 and 1.68 for the ECU-control and the EADHD-control difference respectively on criminal attitude, as compared to the effect size = 1.46 for the difference between the broad early-onset group and the control. Among offending groups, the criminal attitude of the ECU group was significantly higher than the AO group with large effect size ($d$ = 1.33), and the variety of delinquent act committed by the EADHD was significantly higher than the AO with large effect size ($d$= 0.87). Thus, in terms of delinquency, including frequency, variety and criminal attitude, the ADHD and the callous-unemotional traits demonstrate to isolate two individual severe groups even within the early-onset offenders. The findings are consistent with that found by many studies in two lines of literature on comorbid CD-ADHD and CU traits, that antisocial behavior comorbid with hyperactivity, and antisocial behaviors marked by CU traits are more severe (Loeber et al., 2000; Waschbusch, 2002; Sibley, et al., 2010; Vermeiren, 2003; Frick & Dickens, 2006; Frick & white, 2008). Additionally, this study shows that both markers isolate separate groups of adolescents in early-onset offenders with prominent psychosocial difficulties in adolescence (measured by the Strength and Difficulties Questionnaire). Specifically, the two groups reported a significantly higher psychosocial disturbances as indicated by the SDQ total

difficulties with more pronounced effect sizes (d = 1.21 for the EADH-control difference d = 1.02 for the ECU-control difference), as compared to the difference between the broad EO group and the control of a small effect size ($d$ = 0.42). Such findings bear similarities to a recent large-scale UK study which also found that CD group with high CU traits showed most severe difficulties (also measured by the Strength and Difficulties Questionnaire) as compared to control and CD group without CU traits (Rowe, et al., 2010). Thus, both markers are important and should not be ignored as far as the taxonomy of antisocial behavior is concerned. As discussed by Waschbusch et al., (2008), both ADHD and CU traits are important moderators of antisocial behaviors in prediction of outcomes, and here as shown in this study, specifically in predicting the severity and variety of antisocial behavior in terms of self-reported delinquency and criminal attitude, and also psychosocial difficulties in adolescence.

*Distinct features of the EADHD group*. Several distinct features hypothesized to associate with the EADHD group include association with cognitive impairment indicated by low IQ, association with family adversities and parenting risks, association with anxiety, and association with deficit in inhibition as showed by the

stop-task. Most of these hypothesized features were evident in this study. Results of the group comparison indicated that the IQ of EAHD group (mean IQ = 98.47, *SD* = 10.55) was significantly lower than that of the control (mean IQ = 105.93, *SD* = 11.89) and similar to the grand mean of the broad early-onset group (mean IQ = 99.36, *SD* = 14.67). As hypothesized, the EADHD group showed a combination of family adversities and parenting risks, including lower SES, more family transition before age 11, more child abuse, less father's involvement and poorer parental monitoring as compared to the non-delinquent control. Particularly, for all the family adversities variables, the significant differences between the EAHD and the control are of large effect sizes ($d$ = 0.81, 0.9 and 1.43 on low SES, family transition and child abuse), indicating the pronounced risks suffered by this group, as compared to the difference between the broad early-onset group vs. the control on these variables which have small to medium effect sizes. Amongst all offending groups, it is only the EADHD group that had significantly higher anxiety level as compared to the control. Results of regression analyses also showed that IQ, anxiety, family transition before age 11, and poor parental monitoring were significant predictors of this group. Taken together, the features associated with the EADHD are similar to that found for the comorbid condition of CD-ADHD reported in the literature of disruptive behavior

disorders already reviewed in this paper, and also similar to that of the childhood-onset antisocial behavior reported in literature of delinquency, only that this group showed to carry risks similar to the broad early-onset group with greater extent.

If CD comorbid with ADHD is an additive combination or hybrid of both CD and ADHD, bearing the unique deficits of each disorder, it is expected that antisocial behavior comorbid with ADHD will show deficit in response inhibition, a core deficit characteristic of ADHD. This hypothesis has not been tested by any previous study with forensic sample. However, deficient behavioral inhibition measured by the Stop Task was not found of the EADHD group as hypothesized. Subsequent analysis in this study gives certain evidence suggesting that the expected association between deficient inhibition and the ADHD diagnosis did exist. Specifically, group comparison showed that the SSRT of the group with ADHD diagnosis (youth version of DISC- IV) was significantly slower (mean SSRT = 306.02 ms, $SD$ = 78.07) than that of the non-ADHD group with large effect size (mean SSRT = 255.85, $SD$ = 43.16, $t$ (145) = -2.7, $p$ = .008, $d$ = 1.13). A similar pattern was found when the ADHD diagnosis based on the combined source (parent and youth) was used, with a trend-level statistical significance ($t$ (146) = -1.8, $p$ = .07, $d$ = .51). The lack of

significant finding for the EADHD group might be due to the fact that too few subjects with ADHD diagnosis were included. For the EADHD group, only 3 of 17 subjects had ADHD diagnosis (combined sources) and 2 of them had the diagnosis based on single source (DISC-youth), despite that these subjects were assigned to the group based on a high level of ADHD symptoms cutting above 75$^{th}$ percentile of the early-onset offenders.

Another possibility considered for lack of significant finding on the stop task associated with the EADHD is that this comorbid group does not bear the unique inhibition deficit of ADHD. The finding in this study indeed reflects the diversity in the literature on inhibition deficit of different disruptive behavior disorders and the nature of comorbid conditions of these disorders. An early review of Oosterlaan et al. (1998) found that subjects with comorbid ADHD and CD did not differ from the ADHD only subjects on deficient inhibition indicated by SSRT, rendering support to the hybrid hypothesis by showing that the comorbid ADHD and CD also has the characteristic of ADHD (longer SSRT). It is noted that the review provides no data on the ADHD+CD versus the control comparison. But in a further study of Schachar, Mota, Logan, Tannock, & Klim (2000), it was found that the pure ADHD group ($n$ =

72, 7-12-year old referred children) was significantly impaired on inhibitory control indicated by a longer SSRT on the Stop Task as compared to both the ADHD+CD group ($n$ = 47, 7-12-year-old) and the control ($n$= 33, 7-12-year old), where the ADHD+CD had no difference from the control, providing support to another hypothesis different from the hybrid model, that ADHD occurring in the condition of CD is a "phenocopy" of ADHD, in which CD children exhibit AHDH-like behaviors reflective of their CD symptoms but without the underlying cognitive deficits of ADHD. The finding of this study bears certain similarities to this picture.

There is as yet some evidence for a third hypothesis that the ADHD + CD is a distinct condition from the pure disorders by showing that it bears distinct features which are not found in pure ADHD (Schachar & Tannock, 1995; Lauman, Van noesel, Papanikolau, Van Oostenbruggen-Scheffer, Veugelers, Sergeant, et al., 2009). Lauman, et al. (2009) tested whether ADHD+ODD was a more severe form of ADHD in term of neuro-cognitive impairment, using also the stop task as one of the measures. It was found that only ADHD children ($n$=20, age 7-12) had significantly longer SSRT as compared to the control ($n$=50, age 7-12), where the ADHD+ODD group was not significantly different from the control. Thus the comorbid group was not

impaired in terms of response inhibition. But both the ADHD only group and the comorbid group significantly underestimated time in a timing task as compared to the control. The time estimation performance of the ADHD+ODD group was enhanced disproportionally with a reward condition, more than the control and the ADHD only group. In contrast, children with ADHD performed poorer in estimating time in the reward condition as compared to the control. As argued, these different cognitive profiles give support to the hypothesis for the distinct entity of the comorbid condition. But the study lacks a comparison between the comorbid condition and pure ODD, leaving it uncertain whether the profile shown for the comorbid condition indeed bears resemblance to that of pure ODD.

The current study did find that the early-onset offending ADHD group showed characteristics that typically associated with both ADHD (e.g., a relatively low IQ) and CD (e.g., family adversities and parenting risks). It is a severe subgroup even within the early-onset offending adolescents, in terms of the level and variety of delinquency, criminal attitude, psychosocial difficulties, and pronounced family adversities. Whether the EADHD also has the underlying neuro-cognitive deficit of the ADHD indicated by the stop task is uncertain considering the possible

confounds caused by the group formation, that most subjects in the EADHD group had no clinical diagnosis despite high level of ADHD symptoms. Further research is needed to understand the nature of the frequently co-occurring CD and ADHD as it exists in the early-onset antisocial / offending population, and so the knowledge will give guide as to what is required for the interventions. For example, there is suggestion for stimulant medication, an effective treatment known for ADHD (Abikoff et al., 2004; Gillberg, et al., 1997; MTA Cooperative Group, 1999; Pelham, Wheeler, & Chronis, 1998), as one of the treatments for children and adolescents who display antisocial behaviour and also ADHD, not only for the sake of reducing ADHD symptoms per se, but because the impulsivity associated with ADHD may lead to aggressive and poorly regulated behaviour (Frick, 2001; Frick, Barry, & Bodin, 2000) and there is certain support that medication improves conduct problems in the case of comorbid CD and ADHD (Connor, Glatt, Lopez, Jackson, & Melloni, 2002). In the study of Waschbusch, Carrey, Willoughby, King & Andrade (2007), 37 children of age 7 to 12 all diagnosed with ADHD and CD / ODD were divided into two groups based on the presence and absence of callous-unemotional trait. The study intended to test the moderating role of the CU trait on the effects of methylphenidate (stimulant) and behavioural modification on a list of social and academic behaviour including rule

violation in classroom, counsellor and teacher ratings of ADHD and ODD/CD symptoms, seatwork completion and accuracy in these two groups of children who attended an intensive summer treatment programme. A consistent finding in this study is the significant main effect of treatment, that the effect of behaviour modification coupled with medication was significantly better than the effect of behaviour modification alone on all the outcome variables, including reduction of many CD / ODD symptoms such as non-compliance, rule violation, verbal abuse to other, lying, stealing, aggression, destruction of property, etc. However, the use of stimulants for comorbid CD and ADHD has to be further determined (Ipser & Stein, 2007), particularly considering the possibilities that the comorbid condition is distinct entity and is physiologically distinct from either disorder (Banaschewski, Brandeis, Heinrich, Albrecht, Brummer, Rothenberger, 2003), or the comorbid condition is a phenocopy of ADHD without sharing the same the underlying neuro-physiological deficit (Herpertz, Wenning, Mueller, Qunaibi, Henning, Herpertz-Dahlmann, 2001).

*Distinct features of the ECU group.* Consistent with that reported in the previous studies (Frick & Dickens, 2006; Rowe, Maughan, Moran, Ford, Briskman, & Goodman, 2010), antisocial behaviors that associated with CU traits were found to be

severe. In this study, CU trait has marked a severe group within the early-onset offenders in terms of frequency and variety of self-reported delinquency, which was similarly severe in extent to that of the EADHD group. But strikingly, the ECU group had the highest mean in criminal attitude and the lowest mean in prosocial behavior amongst all groups. In the four-group comparison, the ECU group had much more favorable criminal attitude, not only relative to the control (effect size = 3.03), but also relative to the EADHD (effect size of group difference = 1.02) and the AO group (effect size of group difference = 1.33). The ECU had developed significantly less prosocial behavior, relative to the control (effect size = 2.16), and also the other two offending groups (effect size of ECU vs. AO difference = 1.91; effect size of ECU vs. EADHD difference = 1.59). Results of multinomial logistic regression also showed that prosocial behavior and criminal attitude were significant predictors of this group. High criminal attitude and low prosocial behavior obviously match the characteristic interpersonal / emotional style defining CU traits in being callous, uncaring to others and lack of empathy, adding support to the construct validity of this traits.

Based the theory of psychopathy and later findings with children and adolescents,

CU trait shows to associate with distinct affective and interpersonal correlates including emotional deficits such as decreased response to threatening and distressing stimuli, thrill-seeking and absence of anxiety, and deficient passive avoidance learning in favorable of reward dominance. Children and adolescents of high CU trait was found to be lacking cognitive deficits and their antisocial behaviors was found to be less associated with ineffective parenting. Some of these features were tested in this study and were largely evident. Firstly, different from the broad early-onset group and the EADHD group, the ECU group has no cognitive deficit. The mean IQ of ECU is 103.45 (*SD* = 15.62) which was not significantly different from that of the control (mean = 105.93, *SD* = 11.89) and contrasted with the mean IQ = 99.36 (*SD* = 14.67) of the broad EO group and the mean IQ = 98.47 (*SD* = 10.55) of the EADHD group. Secondly, as hypothesized, there is a lack of anxiety in this group as compared to the control, which contrasts with the EADHD which had significantly higher level of anxiety compared to control. And thirdly, it is only the ECU group that showed a reward dominance response showed in the card task. Despite increasing punishment, the ECU group viewed significantly more cards (mean = 93.67, *SD* 12.86) than all the other groups (EADHD: mean = 72.29, *SD* = 31.44, AO: mean = 74.37, *SD* = 32.23, control: mean = 78.33, *SD* = 27.05), and

consequently this group obtained a minus score (-5) as contrasted to the score of 4.18, 1.95, and 2.08 obtained by the EADHD, AO and control respectively. Subsequent hierarchical regression ruled out that the distinct relation between CU trait and reward dominance could be accounted for by other covariates including age and anxiety which were significantly correlated with reward dominance in this study. The absence of anxiety couple with the reward-oriented response style are consistent with behavioral features underlain by temperament of low behavioural inhibition, which is characterized physiologically by under-activity in the autonomic nervous system and behaviorally by low fearfulness in novel or threatening situations and as well as poor response to punishment cues (Frick, 2001, Frick & Morris, 2004). Understanding of the specific features of CU trait gives important implications for intervention strategies. For example, motivational strategies that capitalize on the reward-oriented response style and appeal to self-interest could be more effective than that of punishment-oriented / control strategy that builds on their effect on a negative emotional arousal in children and adolescent with prominent CU traits (Frick, 2001; Frick, 2006, Frick & Dantagnan, 2005).

There is hypothesis that because of their distinct affective-cognitive

characteristics such as fearlessness and low emotional reactivity, children with high CU traits are less responsive to typical parenting socialization, and their antisocial behaviors will grow independent of parenting and is less related to ineffective parenting. Some previous studies render certain support for this hypothesis, such as, Wotton et al. (1997) and Oxford et al. (2003) found that increased antisocial behavior was significantly related to ineffective parenting but only for those children with low CU traits. It is thus hypothesized that the ECU group will be distinguished from other offending groups without high CU traits by a lack of association with deficient parenting. However, in this study, the ECU group was found to associate with risk variables in parenting, prominently with all that pertaining to the positive side of parenting, and less prominently with poor parental monitoring. Specifically, results of the group comparison showed that the ECU, as compared to the control, reported a significantly lower level of positive parenting and parental involvement (both mother and father), with all effect sizes being large ($d$ = 0.84, 1.07 and 1.03 on father's involvement, mother's involvement, and positive parenting respectively). The ECU also had poorer parental monitoring than the control but with less pronounced effect size ($d$ = 0.69). This finding is quite distinct, considering that the overall non-significant to small correlation between offending status and variables pertaining

to positive side of parenting (the only significant $r = -.15$, $p = .05$ between mother's involvement and offending status), as contrasted to $r = .47$, $p < .001$ between offending status and parental monitoring in this study. And also it is only the ECU group who reported significantly lesser positive parenting with a pronounced effect size among all offending groups in this study.

Two important considerations are related to these findings. Firstly, the role of parenting in development of CU trait proves to be a complex issue that has not been tested by earlier studies, which focused on the association between parenting and antisocial behaviors in children with or without CU traits whereas the association between parenting and CU traits were not tested. There is emerging evidence for the importance of parental warmth and involved parent-child relationship in socializing children of low anxiety and fear (like children high on CU traits), as these children, being low in anxiety and fear in temperament, are insufficiently aroused by punitive parenting and have low motivation to internalize parental messages that intend to foster a prosocial value, resulting in increased risk for developing a callous interpersonal style. (Dadds & Salmon, 2003; Pardini, 2006; Pardini, Lochman, & Powell, 2007). There is also emerging support that these child factors, particularly

temperamental anxiety and fear, and parenting factors interact to affect the development and maintenance of CU trait. This interaction was tested in the study of Pardini, Lochman & Powell (2007) which followed a sample of 120 moderately to highly aggressive children (aged 9 to 12) over a 1-year period. Time 1 and Time 2 CU trait and antisocial behaviors were measured with 1-year interval to detect their changes over time. The effects of positive and negative side of parenting and anxiety on Time 2 CU traits were tested. It was found that children who reported their parents as warm and involved and who exposed to a low level of corporal punishment predicted decrease in CU traits and antisocial behaviour at time 2. And also low anxiety was uniquely associated with increase of CU trait over time for children who reported low parental warmth / involvement. In the current study, the ECU group reported a significantly lower level of parental involvement and positive parenting. Though not a direct analogy, the results seem to converge with the above reviewed theory and findings on the importance of parental warmth and positive parenting involved in the development of CU traits.

Secondly, there is also consideration on the role of parenting in mediating association between CU traits and other important variables contributing to antisocial behaviours. Kimonis, Frick & Barry (2004) tested whether the association with

deviant peers differed for youths (n=98, mean age = 12.36) with and without CU traits with a high risk community sample over a 4-year interval. Though not their major hypothesis, Kimonis, et al., (2004) found that adolescents high on CU traits and conduct problems showed significantly more dysfunctional parenting including poor parental monitoring and less parental involvement, as compared to adolescents with high conduct problem but low CU trait. It was also shown that ineffective parenting significantly mediated between the high conduct CU group and its subsequent association with deviant peers overtime, among all other mediators being tested including impulsivity and lack of involvement with prosocial peers. In this context, ineffective parenting is involved possibly because of its role to constrain / or fail to constrain the adolescents' association with deviant peers (particularly by parental monitoring), even if it is less directly related to development of antisocial behaviours of youths with CU traits. Whether negative parenting practices involve in the development and maintenance of CU traits is not certain and requires further research. Frick, Kimonis, Dandreaux and Farell (2003) found that negative parental practices reported by both parent and child were significant predictors of the child's level of CU traits in the 4-year interval, having controlled the initial level of the CU traits, in a sample of 98 non-referred children selected through stratified sampling procedure

from a pool of 1136 children. However, a recent MZ twin study found that there was no longitudinal effect for the age-7 negative parental discipline on level of age-12 CU traits rated by either parents or teacher, in a sample 2254 twin pairs representative of UK population (Viding, Fontaine, Oliver, & Plomin, 2009).

Relatively few studies look at the relation between CU traits and social context such as peers and family adversities. It is noted from Kimonis et al.'s study (2004) that adolescents with high CU traits did associate strongly with deviant peers, despite their callous interpersonal style. This study also similarly found that the ECU group, like the other offending groups, reported significantly higher level of peer delinquency, with large effect sizes ($d = 2.97$ for delinquency of peer group and 5.52 for delinquency of best friend). The current study found that the ECU group did not demonstrate any risk on variables pertaining to family adversities. The findings contrasted with that found by Frick, Kimonis, Dandreaux and Farell (2003), that SES was a significant predictor of the level of child's level of CU traits in a 4-year interval (apart from negative parental practices), but are in line with the literature on adult psychopathy that the quality of family environment had only limited impact on the course of antisocial behaviour, such as age of onset, of psychopaths (Viding, 2004).

On the whole, many previous studies have documented the affective and cognitive features of antisocial adolescents with high CU traits, some of which have also been shown in this study. Less prior studies reported on the relation between CU traits and social context. The findings on the association between the ECU group with parenting and deviant peers, and its lack of association with undesirable family situations (such as low SES, single parenting, family transitions) in this study add to this under-studied area regarding the possible social context in which antisocial behaviors of the high CU adolescents embedded.

## Theoretical and clinical implications

Results of the current study provide evidence to a taxonomy of adolescent antisocial behavior. Firstly, an early-onset antisocial group could be differentiated from the antisocial group with an adolescent-onset in terms of criminal attitude, adolescent adjustment and prosocial behavior, and a list of background risk variables that shown to be significantly correlated with offending status in this study, with the early-onset group showing more risks on these measures. The early-onset group was also higher than the adolescent-onset group in two important attributes important for subtyping antisocial behaviors, i.e., ADHD diagnosis and callous-unemotional traits. The demarcation of the two broad offending groups is based on age of onset (of CD symptoms), an important variable stressed by the developmental theory of antisocial behavior of Moffitt and Patterson. Secondly, two subgroups, that is, a high ADHD group and a high CU group are hypothesized to coexist in the broad early onset group. Based on the age of onset of CD symptoms, these two groups were both categorized in the early-onset group. ADHD and CU traits demonstrate to isolate two subgroups both severe in terms of having much higher frequency and variety of self-reported delinquency, criminal attitude and adolescent adjustment difficulties, but they were

characterized also by different features, such as a lower IQ of the ADHD group versus a lack of cognitive deficit of the ECU group, higher anxiety of the ADHD group versus a lack of anxiety of the ECU group. The taxonomy demonstrated here bears importance to the theory of antisocial behaviors and its application.

Firstly, the fact that several offending groups could be differentiated by different features as hypothesized adds support to the notion that antisocial behavior could not be seen as a unitary construct (Rutter et al., 1998c; Moffitt, 1993; 1996; Patterson & Yoerger, 1993; Patterson, 1996). Research that does not take into the heterogeneity of antisocial behavior and does not distinguish different groups will possibly obscure important findings about features, risks or etiologies of antisocial behaviors or possibly omit variables that important for a specific group but not the others. For example, there is a well established finding that delinquency is associated with lower IQ (Lynam, Moffitt, & Stouthamer-Loeber, 1993), and as shown in this study offenders as a whole was associated with a lower IQ ($r = -.25$, $p < .001$), but this is not the case for adolescents with high CU traits and antisocial behavior. Secondly, the differential risks and features shown to be associated with different subtypes of antisocial behavior such as those characterized the EADHD versus that of the ECU

render support to the existence of different pathways and causal mechanisms through which antisocial behaviors consequently develop and persist. Apart from the two-trajectory models of Moffitt or Patterson, there are other attempts to delineate these developmental pathways and the causal mechanisms. One example relevant to the taxonomy proposed in this study is the childhood-onset-CU pathway to antisocial behavior depicted by Frick (2006) and colleagues (Frick & Morris, 2004), in which temperamental vulnerabilities such as low behavioral inhibition had been linked up to impact on affective component of conscience and internalization of parental and societal norm, and then development of callous-unemotional trait and antisocial behavior. The other example relevant to the taxonomy here is the temperament-related pathways of ADHD posited by Nigg and colleagues (Nigg, 2006b; Nigg, Goldsmith, & Sachek, 2004), in which six etiological pathways to ADHD were described. Among these six etiological paths, two are relevant not only to development of ADHD but also CD, i.e., secondary ADHD with primary socialized CD and secondary ADHD with primary unsocialized CD. Interestingly, the secondary ADHD with primary unsocialized CD is a pathway possibly landing in psychopathy, which is preceded by temperamentally low anxiety (low negative withdrawal) and then later disruption of regulation, with a clinical profile

characterized by low arousal, heart rate and aggressive conduct problem. The posited features and temperamental precursors share similarities with that specified by Frick. The secondary ADHD with primary socialized CD is preceded by early high negative emotional reactivity and later disruption of affect regulation and executive functions, leading to prominent comorbid ODD and CD characterized by notable hostility and poor affect regulation. Obviously, integration from different lines of research is in need given the high overlap among disruptive behavior disorders.

Arising from the above, a third important implication from the taxonomy is, if there are different meaningful subtypes of antisocial behavior arising from different pathways and causal mechanisms, then treatment and intervention for children and adolescents with antisocial behavior may very possibly differ in their effectiveness across groups. There will be different mediators and moderators that affect treatment outcomes for these different subgroups, which are not well known in the literature of psychosocial treatment outcomes for children and adolescents with antisocial behaviors (Eyberg, Nelson, & Boggs, 2008). Consider for example, different target areas of intervention / prevention and different strategies of intervention / prevention might be in need for different subgroups. Helping parents

to acquire ways to foster children's empathic concern and perspective taking versus helping parents to foster children's effortful control and regulation will be differentially important for children who have high CU traits versus those with prominent temperamental hyperactivity, both in terms of prevention of severe antisocial behavior (Frick, 2006; Frick & Dickens, 2006). In Moffitt's terms, early intervention encompassing multiple modalities including family, child and school will respond to the needs of those highly vulnerable children who are in high-risk environment and stepping on the childhood-onset antisocial path (Moffitt, 1996). The adolescent-onset path of offending is not a benign group as posited by Patterson and also showed by this current study. This group of adolescents demonstrated to have considerable background risks and thus certain preventive measures should need to prevent poor developmental outcomes, apart from helping them to connect to prosocial peers and avoid snares after onset of their antisocial behaviors (such as unemployment, school dropout, planned pregnancy, drug addiction, Moffitt, 1996). The above implications are important not only for actual intervention but also for research on the effectiveness or efficacy of intervention, both commonly targeting on a broad category of clinical population such as "children with antisocial behavior" and broad intervention approach, such as CBT applied across children and adolescents

who might be on different pathways. Some of the key theorists have written explicitly on important principals for intervention generated from the developmental pathway of antisocial behavior, such as Frick and colleagues (Frick, 2001; Frick, 2006; Frick & Dickens, 2006; Frick & White, 2008; McMahon & Frick, 2005). But it is uncertain of how much of this knowledge generated from research has been considered and integrated into practice. An example that integrates theory and practice is Patterson's coercion model of antisocial behavior, which has formed the base of a widely used behavioral parent training though it targets mainly to replace coercive parent-child interchanges by appropriate parenting (Bank, Marlowe, Reid, Patterson, & Weinrott, 1991; Patterson & Forgatch, 1987). It is known to have a stronger effect for preschool and school-aged adolescents as shown by a meta-analysis of behavioral parent-training versus cognitive-behavioral therapy for antisocial youth (McCart, Priester, Davies, & Azen, 2006).

The current study adds support to the validity of using age of onset of CD symptoms to organize the heterogeneity of antisocial behavior, by showing that early-onset antisocial behaviors could be differentiated from adolescent-onset antisocial in terms of risks and external correlates. But the specific age to demarcate

early- versus late-onset could vary and as shown in many studies, defining age other than that specified by DSM-IV (age 10) also shows certain validity in differentiating early-onset from the late-onset antisocial behavior in terms of features, course of development and outcomes. These findings are important for subtyping antisocial behavior based on age of onset and could be considered to alter the age of onset specification in the DSM system (Olsson, 2009). While there is support for differentiation between the early-onset versus adolescent-onset type of antisocial behavior, this study also shows that such distinction could be too board, because as indicated, within the broad early-onset group, there are important variations with regard to clinical features and risks based on the presence or absence of high ADHD and CU traits. Coding these important differentiators into the classification system of disruptive behavior disorder should also be considered (Moffitt, et al., 2008).

Similar to many previous studies (such as Barry et al., 2000; Frick et al., 2005); this study defined high CU traits using a distributional cutoff (at above 75$^{th}$ percentile), in the absence of an established standard to guide to decide the cut off for a problematic level of CU traits. Even the same instrument is used, depending on nature of sample (clinic-referred, forensic, community high risk, normative); cut off

score could be different to define high CU traits. There is a lack of epidemiological cohort studies that report on the distribution of CU traits and the prevalence of abnormal CU traits in general population (Moffitt, et al., 2008). However, a recent UK study adopting a nationally representative sample of 7977 5-to-16-year old children provides relevant information here, that using a 7-item questionnaires measuring CU traits, the distribution is: 87% of the full sample scored on 0, 10% scored 1, 2% scored 2, 0.7% scored 3 and 0.8% scored 4 or more (Rowe, et al. 2010). A cut point of 2 or more was adopted to define high CU traits, resulting in 3.8% of the sample being classified as having high CU traits. Such cutoff is still a distributional cutoff, which can be argued for a lower or higher point. To help understanding and defining what is problematic level of CU, further research that tests whether CU traits are best treated as a taxon or a dimension should be important, in which a taxon implies qualitative difference between those high and low on the CU traits (Vasey, kotov, Frick, & Loney, 2005). Vasey et al. (2005) found initial evidence that CU traits are best conceptualized as a taxon. In this study, the distinct relationship between CU traits and reward dominance exists only in the offender group (which has a higher level of CU trait) but non-exists in the control (which has a lower level of CU trait) might reflect such possibility. The predictive validity for categorical versus

dimensional use of CU traits might also be different; all these are important information for both further research with respect to subtyping of antisocial behavior and for intervention.

## Limitations of this study

There are some limitations to this study that should be noted. First, only a single source of information from self report had been obtained, despite that the original plan was to solicit multisource data. The exception is for the diagnosis of ADHD. A special second-round effort was made to persuade the parents to participate in a limited fashion. After reducing the amount of time requested from them, 111 out of 181 parents eventually agreed to participate. This is considered not to be a bad turnout. On the other hand, there is support for increased validity of self-report on CU traits, delinquent behaviors, other psychological symptoms such as anxiety, and adjustment, as compared to other informants, and self-report is especially important for covert behavior or affective styles as they are not so easily observed by others (Kamphaus & Frick, 1996). It is also noted that alphas for all measures derived from self-report are satisfactory in this study.

Second, the sample size is small, particularly for the two early-onset subgroups ($n$ = 17 for EDHD and $n$ = 11 for ECU), limiting the statistical power to detect significant differences between them and increasing type-II error.

Third, in this study, the EADHD and the ECU subgroups were derived using an arbitrary distributional cutoff of above 75$^{th}$ percentile on ADHD symptom score and CU trait total score. Other studies had used a more stringent cut off, such as in the study of Rowe, et al., (2010). A sample of 7977 children was recruited in their study. Given such a large sample, they could afford to use the highest 3.8% of the sample on the CU traits to define as a high CU group and still obtained a large group size. This current study cannot afford such a stringent cutoff. The current cut off was chosen to strike a balance between deviancy and a minimally sufficient group size for statistical comparison.

Fourth, it should also be noted that for the sake of retaining a sufficient group size; the EADHD subgroup was derived based on high ADHD symptom score but not diagnosis. This study found that the overlap between those with high ADHD symptoms and those with ADHD diagnosis was low. Of the 13 subjects with ADHD diagnosis in the broad EO group, only 3 were included in the EADHD subgroup based on a high symptom score. The subjects in the EADHD subgroup who had high symptom score (above 75$^{th}$ percentile) but without the diagnosis were those who

either did not fulfill the age of onset criterion (symptoms onset before age 7) or did not fulfill the criterion of pervasiveness (i.e., their ADHD symptoms were limited to a single setting such as school or home). If the EADHD subgroup is defined based on diagnosis, after removing the subjects overlapping with those having high CU traits, the group size will be fewer than 10. This group size would not be viable for any statistical testing. It is not known whether the findings on EADHD subgroup would be altered if a sufficient number of subjects with a formal ADHD diagnosis could be included. Subsequent analysis on the stop task in subjects with and without a formal diagnosis of ADHD does suggest that the current negative finding with respect to stop task in EADHD subgroup many be reversed, given the significant positive relationship between a formal diagnosis of ADHD and the SSRT of the stop task. Further research might help to clarify this.

Lastly, the correlational and the cross-sectional nature of this study limit its ability to establish casual relationship. Despite that poor parental monitoring is demonstrated as a common and strong risk associated with different subtypes of antisocial behaviors, there is no basis to go further to prove its causal effect on adolescent antisocial behavior. It is in fact the theoretical reasoning of the author,

based upon past literature, to choose to interpret the direction of relationship between parenting and antisocial behavior as the former influencing the latter. It is beyond the correlational and cross-sectional nature of this study to test the potential causal complexities between parenting, and antisocial behavior and other mediating variables. For example, it is equally possible that ineffective parenting is a consequence of the antisocial or ADHD behaviors of the adolescents. Moffit (2006) argued that antisocial adolescents evaded the parents' supervision and control, despite the parents' effort to do so. This also produces a phenomenon of a correlation between adolescent antisocial behavior and lack of parental supervision and control. Nonetheless, the relationship between antisocial behaviors and parenting is likely to be bidirectional.

## Contributions to the literature

The current study contributes to the literature in several ways. First, since the proposal of the two prototypic type of antisocial behavior in 1993 (Moffitt, 1993), the theory has been tested by the Dunedin study and the differentiation between the early-onset versus the adolescent-onset offending had been reported from many other samples in various countries including the US, Sweden, UK, Australia, Dutch, apart from New Zealand (Moffitt, 2003). This study adds to this literature by testing whether these two prototypic offending could be discerned in Chinese sample, which has not been reported so far by any study.

Second, it has been noted that most research on the developmental taxonomy of Moffitt focused heavily on the LCP offending and the adolescent-offending group had been treated as a contrast group (Moffitt, 2006). Given that the adolescent-onset offending is relatively common and its consequences are not benign (Moffitt, et al., 2002; Odgers et al., 2008), it is important to have more understanding about the nature of this group so that appropriate intervention could be derived. This study adds knowledge to this under-studied area by testing whether the adolescent-offending

group shows features as predicted by Moffitt versus Patterson, who postulated different etiologies for this offending group with correspondingly different implications on intervention.

Third, this current study hypothesizes that ADHD and CU traits further subcategorize two distinct subgroups within the broader early-onset group. The construct validity of ADHD in Chinese sample had been shown by previous studies (e.g. Leung, et al., 1996). However, the applicability of the construct of CU traits, a downward extension of adult psychopathy construct to children and adolescents, is unknown in non-western culture. There is some initial evidence supporting the cross-cultural application of child and adolescent psychopathy: the 3-factor structure of the Antisocial Processing Device, assessing child and adolescent psychopathy, was confirmed by factor analysis in 3675 schoolchildren aged 11 to 16 in Hong Kong (Fung, Gao, & Raine, 2010). This current study would further test whether the CU group, defined by one key component of psychopathy, shows its hypothesized associations with external correlates, and thus forms a meaningful subgroup under the broad early-onset group.

Last but not the least, ADHD and CU traits have been found separately as important correlates of antisocial behavior (Rutter, et al., 1998c, Waschusch & Willoughby, 2008) but very few studies look at both in one single sample and contrast groups defined respectively by them with respect to their antisocial behaviors and other risk factors and correlates. This study adds knowledge to the role of ADHD and CU traits in subtyping antisocial behaviour by testing whether the two hypothesized early-onset subgroups marked by these two important correlates could be differentiated by different associated features.

**END of the thesis**

# References


Abikoff, H., Hechtman, L., Klein, R. G., Weiss, G., fleiss, K., Etcovitch, J., et al., (2004). Symptomatic improvement in children with ADHD treated with long-term methylphenidate and multimodal psychosocial treatment. *Journal of the American Academy of Child and Adolescent Psychiatry, 43(7)*, 802-811.

Abikoff, H. & Klein R. G. (1992). Attention-deficit hyperactivity and conduct disorder: comorbidity and implications for treatment. *Journal of Consulting Clinical Pyschology, 60*, 881-892.

Abrantes, A. M., Hoffmann, N. G., Anton, R. (2005). Prevalence of co-occurring disorders among juveniles committed to detention centres. *International Journal of Offender Therapy and Comparative Criminology, 49(2)*, 179-192.

Achenbach, T. M. (1991). *Manual for the Youth Self-Report and 1991 profile.* Burlington: University of Vermont.

Aguilar, B., Sroufe, L. A., Egland, B., & Carlson, E. (2000). Distinguishing the early-onset-persistent and adolescent-onset antisocial behavior types: from birth to 16 years. *Development and Psychopathology, 12*, 109-132.

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders-IV-TR.* Washington, DC: Author.

Andrews, D. A., & Bonta, J. (2003). *The Psychology of Criminal Conduct.* (3rd ed.). Cincinnati, OH: Anderson.

Andrews, D. A., Bonta, J., & Wormith, J. S. (2006). The recent past and near future of risk and/or need assessment. *Crime and Delinquency, 52*, 7-27.

Andrews, D. A., & Dowden, C. (2006). Risk principle of case classification in correctional treatment. *International Journal of Offender Therapy and Comparative Criminology, 50*, 88-100.

Angold, A., Costello, E. J., Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40 (1)*, 57-87.

Angold, A., Erkanli, A., Costello, e. J., and Rutter, m. (1996). Precision, reliability and accuracy in the dating of symptom onsets in child and adolescent psychopathology. *Journal of child Psychology and Psychiatry Allied Discipline, 37*, 657 – 664.

Appleyard, K., Eagland, B., van Dulmen, M. H. M., & Sroufe, A. (2005). When more is not better: the role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry, 46(3)*, 235 – 245.

Avila, C., Cuenca, I., Felix, V., Parcet, M. A., & Miranda, A., (2004). Measuring Impulsivity in school-aged boys and examining its relationship with ADHD and ODD ratings. *Journal of Abnormal Child Psychology, 32*, 295-304.

Banaschewski, T., Brandeis, D., Heinrich, H., Albrecht, B., Brummer, E., Rothenberger, A. (2003). Association of ADHD and conduct disorder – brain electrical evidence for the existence of a distinct subtype. *Journal of Child Psychology and Psychiatry, 44*, 356-376.

Bank, L., Marlowe, J. H., Reid, J. B., Patterson, G R., & Weinrott, M. R. (1991). A comparative evaluation of parent training interventions for families of chronic delinquents. *Journal of Abnormal Child Psychology, 19*, 15-33.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive function: constructing a unified theory of ADHD. *Psychological Bulletin, 121*, 65-94.

Barkley, R. A., Fischer, M., Edelbrock, C. S., Simallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria, I: an 8-year prospective follow-up study. *Journal of the American Academy Child and Adolescent Psychiatry, 29*, 546-557.

Barkley, R. A., Fischer, M., Edelbrock, C. S., Simallish, L. (1991). The adolescent outcome of hyperactive children diagnosed by research criteria, III: mother child interactions, family conflicts and maternal psychopathology. *Journal of Child Psychology and Psychiatry, 32*, 233-255.

Barkley, R. A., McMurray, M. B., Edelbrock, C. S., & Robbins, K. (1989). The response of aggressive and nonaggressive ADHD children to two doses of methylphenidate. *Journal of the American Academy Child and Adolescent Psychiatry, 28*, 873-881.

Barry, C. T., Frick, P. J., DeShazo, T. M., Grooms, T., McCoy, M. G., Ellis, M. l., & Loney, B. R. (2000). The importance of callous-unemotional traits for extending the concept of psychopathy to children. *Journal of Abnormal Psychology, 109*, 335-340.

Beauchaine, T. P., & Neuhaus, E. (2008). Impulsivity and vulnerability to psychopathology. In Beauchaine T. P. & Hinshaw, S. P. (Eds). *Child and Adolescent Psychoapthology* (pp. 129-156), Wiley.

Biederman, J., Faraone, S. V., & Lapey, K. (1992). Comorbidity of diagnosis in attention-deficit hyperactivity disorder. In G Weiss (Ed.), *Child and adolescent psychiatry clinics of North America: Attention deficit disorder* (pp. 335-360). Philadelphia: W. B. Saunders.

Blader, J. C. (2004). Symptom, family and service predictors of children's psychiatric rehospitalization within one year of discharge. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*, 440-451.

Blair, R. J. R. (1999). Responsiveness to distress cues in the child with psychopathic tendencies. *Personality and Individual Differences, 27(1)*, 135-145.

Blair, R. J. R. (2005). Applying a cognitive neuroscience perspective to the disorder

of psychopathy. *Development and Psychopathology, 17*, 865-891.

Blair, R. J., R., Peschardt, K. S., Budhani, S., Mitchell, D. G. V., & Pine, D., S. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry, 47*, 262 – 275.

Borger, N., & Van der Meere, J. (2000). Motor control and state regulation in children with ADHD: A cardiac response study. *Biological Psychology, 51*, 247-267.

Boucher, L., Palmeri, T. J., Logan, G. D., & Schall, J. D. (2007). Inhibitory control in mind and brain: An interactive race model of countermanding saccades. *Psychological Review, 114*, 376-397.

Boylan, K., Vaillancourt, T., Boylan, M., & Szatmari (2007). Comorbidity of internalizing disorders in children with oppositional defiant disorder. *European Journal of Adolescent Psychiatry, 16*, 484-494.

Brennan, P., A. and Hall, J., Bor, W., Najman, J. M., & Williams, G. (2003). Integrating biological and social processes in relation to early-onset persistent aggression in boys and girls. *Developmental Psychology, 39*, 309-323.

Buss, A. H., & Plomin, R. A., (1975). *A Temperamental Theory of Personality Development*. New York: Wiley.

Cantwell D. P. & Baker, L. (1992). Attention deficit disorder with and without hyperactivity: a review and comparison of matched groups. *Journal of the American Academy Child and Adolescent Psychiatry, 31*, 125-144.

Capaldi, D. M., & Patterson, G. R. (1996). Can violent offenders be distinguished from frequent offenders? Prediction from childhood to adolescence. *Journal of Research in Crime and Delinquency, 33*, 206-231.

Caputo, A. A., Frick, P. J. & Brodsky, S. L. (1999). Family violence and juvenile sex offending: potential mediating roles of psychopathic traits and negative attitudes toward women. *Criminal Justice and Behavior, 26*, 338-356.

Carroll, A., Hemingway, F., Bower, J., Ashman, A., Houghton, S., & Durkin, K. (2006). Impulsivity in juvenile delinquency: differences among early-onset, late-onset, and non-offenders. *Journal of Youth and Adolescence, 35 (4)*, 519-529.

Caron, C., & Rutter, m. (1991). Comorbidity in child psychopathology: concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry, 232*, 1063-1080.

Christian, R. E., Frick, P. J., Hill, N. L., Tyler, L., Frazer, D. R. (1997). Psychopathy and conduct problems in children: II. Subtyping children with conduct problems based on their interpersonal and affective style. *Journal of the American Academy of Child and Adolescent Psychiatry, 36(2)*, 233-241.

Chung, I. J., Hill, K. G., Hawkins, J. D., Gilchrist, L., D., & Nagin, D. S. (2002).

Childhood predictors of offense trajectories. *Journal of Research in Crime and Delinquency, 39*, 60 – 90.

Cleckley, H., (1941). *The mask of Sanity: an attempt to reinterpret the so-called psychopathic personality*. Oxford, England: Mosby.

Cohen, B. H. (2001). *Explaining Psychological Statistics* (2nd ed.). John Wiley & Sons, Inc.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences (2nd ed.)*. Hillsdale, NJ: Erlbaum.

Coleman, J. C., & Hendry, L. B. (1999). The Nature of Adolescence, 3rd edition, Routledge.

Conduct Problems Prevention Research Group (2004). The effects of the FAST Track Program on serious problem outcomes at the end of elementary school. *Journal of Clinical Child Adolescent Psychology, 33*, 650-651.

Connor, D. F., Glatt, S. J., Lopez, I. D., Jackson, D., & Melloni, R. H. (2002). Psychopharmacology of aggression I: A meta-analysis of stimulant effects on overt/covert aggression-related behaviours in ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*, 253-261.

Connor, D. F., Steeber, J., & McBurnett, K. (2010). A review of Attention-Deficit/Hyperactivity Disorder complicated by symptoms of Oppositional Defiant Disorder. *Journal of Developmental and Behavioral Pediatrics, 31(5)*, 427-440.

Cooke, d. J., Kosson, D. S., & Miche, C. (2001). Psychopathy and ethnicity: Structural, item, and test generalizabiltiy of the Psychopathy Checklist-Revised (PCL-R) in Caucasian and African American participants. *Psychological Assessment, 13(4)*, 531-542.

Cooke, D. J. & Michine, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment, 13*, 171-188.

Copeland, W. E., Miller-Johnson, S., Keeler, G., Angold, A., & Costello, E. J. (2007). Childhood psychiatric disorders and young adult crime: A prospective, population-based study. *American Journal of Psychiatry, 164*, 1668-1675.

Dadds, M. R., Maujean, A., & Fraser, J. A. (2003). Parenting and conduct problems in children: Australian data and psychometric properties of the Alabama Parenting Questionnaire. *Australian Psychologist, 38*, 238-241.

Dandreaux, D. M. & Frick, P. J. (2009). Developmental pathways to conduct problems: a further test of the childhood and adolescent-onset distinction. *Journal of Abnormal Child Psychology, 37*, 375-385.

Davis, M. H. (1983). Measuring individual differences in empathy: evidence for a multidimensional approach. *Journal of Personality and Social Psychology, 44*, 113-126.

Dishion, T. J., McCord, J., & Poulin, F. (1999). Iatrogenic effects in interventions that aggregate high-risk youth. *American Psychologist, 54*, 1-10.

Dishion, T. J., & Patterson, G R. (2006). The development and ecology of antisocial behavior. In D. Cicchetti & D. Cohen (Eds.), *Developmental Psychopathology. Vol. 3: Risk, Disorder, and Adaptation* (2nd ed., pp. 503-541). New York: Wiley & Sons.

Drerup, L. c., Croysdale, A., & Hoffmann, H. G. (2008). Patterns of behavioral health conditions among adolescents in a juvenile justice system. *Professional Psychology: Research and Practice, 39*, 122-128.

Elliott, D. S. (1994). Serious violent offenders: Onset, developmental course, and termination – The American Society of Criminology 1993 presidential address. *Criminology, 32*, 1-21.

Elliott, D. S., Huizinga, D., Ageton, S. S. (1985). *Explaining Delinquency and Drug Use*. Sage Publications.

Elliott, D. S. & Huizinga, D., & Morse, B. (1987). Self-reported violent offending: a descriptive analysis of juvenile violent offenders and their offending careers. *Journal of Interpersonal Violence, 1*, 472-514.

Elliott, D. S. & Huizinga, D. (1989). Improving self-reported measures of delinquency. In Malcolm W. Klein (Ed.), *Cross-Notional Research in Self-Reported Crime and Delinquency* (pp. 155-186). Norwell, MA: Kluwer Academic.

Essau, C. A., Sasagawa, S., Frick, P. J. (2006). Callous-unemotional traits in a community sample of adolescents. *Assessment, 13(4)*, 454-469.

Eyberg, S. M., Nelson, M. M., & Boggs, S. R. (2008). Evidence-based psychosocial treatements for children and adolescents with disruptive behaviour. *Journal of Clinical Child and Adolescent Psychology, 37(1)*, 215-237.

Farrington, D. P. (1991). Childhood aggression and adult violence: early precursors and later life outcomes. In D. J. Pepler & K. H. Rubin (Edss), *the Development and Treatment of Childhood Aggression* (pp. 5-29). Hillsdale, NJ: Lawerence Erlbaum.

Farrington, D. P. (1995). The Twelfth Jack Tizard Memorial Lecture: the development of offending and antisocial behaviour from childhood: key findings from the Cambridge Study in Delinquent Development. *Journal of Child Psychology and Psychiatry, 36*, 929-964.

Farrington, D. P. (2009). Conduct disorder, aggression and delinquency. In Lerner, R. M. & Steinberg, L. (Eds). *Handbook of Adolescent Psychology*, 3rd edition (pp.683-722). Wiley.

Farrington, D. P., Coid, J. W., Harnett, L., Jolliffe, D., Soteriou, N., Turner, R., & West, D. J. (2006). Criminal careers and life success: new findings from the

Cambridge Study in Delinquent Development. Retrieved August 10, 2009 from http://www.homeoffice.gov.uk/rds/pdfs06/r281.pdf

Farrington, D. P., Lambert, S., West, D. J., (1998). Criminal careers in two generations of family members in the Cambridge Study of Delinquent development. *Studies on Crime and Crime Prevention, 7*, 1-22.

Farrington, D. P., Loeber, R., & van Kammen, W. B. (1990). Long-term criminal outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood. In Robins, L. N. & Rutter, M. (Eds.). *Straight and Devious Pathways from Childhood to Adulthood*, pp. 62-81. Cambridge University Press.

Farrington, D. P., Ohlin, L., & Wilson, J. Q. (1986). *Understanding and Controlling Crime*. New York: springer Verlag.

Farrington, D. P., Ttofi, M. M. & Coid, J. W. (2009). Development of adolescent-limited, late-onset and persistent offenders from age 8 to age 48. *Aggressive Behavior, 35*, 150-163.

Fazel, S., Doll, H., & Langstrom, N. (2008). Mental Disorders among adolescents in juvenile detention and correctional facilities: a systematic review and metaregression analysis of 25 surveys. *Journal of the American Academy Child and Adolescent Psychiatry, 47(9)*, 1010-1019.

FBI (2007). Table 39, retrieved May 21, 2009 from http://www.fbi.gov/ucr/cius2007/data/table_39.html http://www.fbi.gov/ucr/cius2007/documents/arreststopicpage.pdf

Fergusson, D. M. & Horwood, L. J. (1995). Predictive validity of categorically and dimensionally scored measures of disruptive childhood behaviours. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*, 477-485.

Fergusson, D. M., Horwood, L. J., & Nagin, D. S. (2000). Offending trajectories in a New Zealand birth cohort. *Criminology, 38*, 525-552.

Fergusson, D., M., & Horwood, L., J. (2002). Male and female offending trajectories. *Development and Psychopathology, 14*, 159-177.

Fergusson, D. M. & Horwood, L. J., & Ridder, E. M. (2005). Show me the child at seven: the consequences of conduct problems in childhood for psychosocial functioning in adulthood. *Journal of Child Psychology and Psychiatry, 46*, 837-849.

Fergusson, D. M. Lynskey, M. T., & Horwood, L. J., (1996). Factors associated with continuity and changes in disruptive behaviour patterns between childhood and adolescence. *Journal of Abnormal Child Psychology, 24*, 533-553.

Fisher, L., & Blair, R. J. R. (1998). Cognitive impairment and its relationship to psychopathic tendencies in children with emotional and behavioural difficulties.

*Journal of Abnormal Child Psychology, 26*, 511-519.

Frick, P. J. (2001). Effective interventions for children and adolescents with conduct disorder. *Canadian Journal of Psychiatry, 46*, 597-608.

Frick, P. J., (2006). Developmental pathways to conduct disorder. *Child and Adolescent Psychiatric Clinics of North America, 15(2)*, 311-331.

Frick, P. J., Barry, C. T., & Bodin, S. D. (2000). Applying the concept of psychopathy to children: implications for assessment of antisocial children and adolescents. In C. B. Gacono (Ed.), *The Clinical and Forensic Assessment of Psychopathy: a Practitioner's Guide*, LEA.

Frick, P. J., Bodin, S. D., & Barry, C. T. (2000). Psychopathic traits and conduct problems in community and clinic-referred samples of children: further development of the Psychopathy Screening Device. *Psychological Assessment, 12(4)*, 382 – 393.

Frick, P., J, Cornell A. H., Bodin S. D., Dane H. E., Barry, C. T., & Loney, B. R. (2003). Callous-unemotional traits and developmental pathways to severe conduct problems. *Developmental Psychology, 39*, 246-260

Frick, P. J., & Dantagnan, A. L. (2005). Predicting the stability of conduct problems in children with and without callous-unemotional traits. *Journal of child and Family Studies, 14(4)*, 469-485.

Frick, P. J., & Dickens, C. (2006). Current perspectives on conduct disorder. *Current Psychiatry Reports, 8*, 59-72.

Frick, P. J., & Ellis, M. L. (1999). Callous-unemotional traits and subtypes of conduct disorder. *Clinical Child and Family Psychology Review, 2*, 149-168

Frick, P. J., & Hare, R., D. (2001). *The antisocial Process Screening Device (APSD)*. Toronto: Multi-Health Systems

Frick, P. J., Kimonis, E. R., Dandreaux, D. M., & Farell, J. M. (2003). The 4 year stability of psychopathic traits in non-referred youth. *Behavioral Sciences and the Law, 21*, 713-736.

Frick, P. J., Lilienfeld, S. O., Ellis, M. L., Loney, B. R., & Silverthorn, P. (1999). The association between anxiety and psychopathy dimensions in children. *Journal of Abnormal Child Psychology, 27*, 381 – 390.

Frick, P. J. & Loney, B. R. (2000). The use of laboratory and performance-based measures in the assessment of children and adolescents with conducts disorders. *Journal of Clinical Child Psychology, 29*, 540-554.

Frick, P. J., & Morris, A. S. (2004). Temperament and developmental pathways to conduct problems. *Journal of Clinical Child and Adolescent Psychology, 33(1)*, 54-68.

Frick, P. J., O'Brien, B. S., Wootton, J., M., & McBurnett, K. (1994). Psychopathy and conduct problems in children. *Journal of Abnormal Psychology, 103*,

700-707.

Frick, P. J., Stickle, T. R., Dandreaux, D. M., Farrell, J. M., & Kimonis, E. R. (2005). Callous-unemotional traits in predicting the severity and stability of conduct problems and delinquency. *Journal of Abnormal Child Psychology, 33(4)*, 471-487.

Frick, P. J., & White, S. F. (2008). Research review: the importance of callous-unemotional traits for developmental models of aggressive and antisocial behaviour. *Journal of Child Psychology and Psychiatry, 49(4)*, 359-375.

Fung, A. L. C., Gao, Y, & Raine, A. (2010). The utility of the child and adolescent psychopathy construct in Hong Kong, China. *Journal of Clinical Child and Adolescent Psychology, 39(1)*, 134-140.

Garson, G. D. (2010). Statnotes: topics in multicollinearity and logistic regression. Retrieved on 5/20/2010 from http://faculty.chass.ncsu.edu/garson/PA765/logistic.htm.

Gaultney, J. F., Kipp, K., Weinstein, J., & McNeil, J. (1999). Inhibition and mental effort in attention deficit hyperactivity disorder. *Journal of Developmental and Physical Disabilities*, 11, 105-114.

Gillberg, C., Melander, H., von knorring, a., Janols, L., Thernlund, G., Hagglog, B., Eidevall-Wallin, L., Gustafsson, P., Kopp, S. (1997). Long-term stimulant treatment of children with attention-deficit hyperactivity disorder symptoms: A randomized, double-blind, Placebo-controlled trial. *Archives of General Psychiatry, 54(9)*, 857-864.

Goodman, R. (1997). The strengths and difficulties questionnaire: a research note. *Journal of Child Psychology and Psychiatry, 38*, 581-586.

Goodman, R. (2001). Psychometric Properties of the Strengths and Difficulties Questionnaire. *Journal of the American Academy Child and Adolescent Psychiatry, 40(11)*, 1337-1345.

Goodman, R. & Scott, S. (1999). Comparing the Strengths and Difficulties Questionnaire and the Child Behavior Checklist: is mall beautiful? *Journal of Abnormal Child Psychology, 27*, 17-24.

Gottfredson, M. R., & Hirschi, T. (1990). *A General Theory of Crime*. Stanford University Press, Stanford, CA.

Granic, I., & Patterson, G R. (2006). Toward a comprehensive model of antisocial development: a dynamic systems approach. *Psychological Review, 113(1)*, 101-131.

Hare, R. D. (1970). *Psychopathy: Theory and research*. New York: Wiley.

Hare, R. D. (1985). A comparison of procedures for the assessment of psychopathy. *Journal of Consulting and Clinical Psychology, 53*, 716.

Hare, R. D. (1991). *The Hare Psychopathy Checklist-Revised*. Toronto:

Multi-Health systems.

Hare, R. D. (2003). *The Hare Psychopathy Checklist-Revised (2nd ed.)*. Toronto: Multi-Health systems.

Harpur, T. J., Hakstian, A., R., & Hare, R. D. (1988). Factor structure of the Psychopathy Checklist. *Journal of Consulting and Clinical Psychology, 56*, 741- 747.

Harpur, T. J., Hare, R. D, & Hakstian, A., R. (1989). Two-factor conceptualisation of psychopathy: construct validity and assessment implications. *Psychological Assessment, 1*, 6-17.

Hawes, D. J., & Dadds, M. R. (2005). The treatment of conduct problems in children with callous-unemotional traits. *Journal of Consulting and Clinical Psychology, 73*, 737-741.

Hawes, D. J., & Dadds, M. R. (2006). Assessing parenting practices through parent-report and direct observation during parenting training. *Journal of Child and Family Studies, 15*, 555-568.

Hawes, D. J., Dadds, M. R., (2007). Stability and Malleability of Callous-unemotional traits during treatment for childhood conduct problems. *Journal of Clinical Child and Adolescent Psychology, 36(1)*, 347-355.

Hemphill, J. F. (2007). The Hare psychopathy checklist and recidivism: methodological issues and critically evaluating empirical evidence. In H. Herve and J. C. Yuille (Eds.), *The Psychopath: Theory, Research, and Practice* (pp.141-170). Mahwah, NJ: Lawrence Erlbaum Associates

Hemphill, J. F., Hare, R. d., & Wong, S. (1998). Psychoapthy and recidivism: a review. *Legal and Criminological Psychology, 4*, 139-170.

Herpertz, S. C., Wenning, B., Mueller, B., Qunaibi, M., Henning, S., Herpertz-Dahlmann, B. (2001). Psychophysiological responses in ADHD boys with and without conduct disorder: implications for adult antisocial behaviour. *American Academy of Child and Adolescent Psychiatry, 40(10)*, 1222-1230.

Hiatt, K. D., & Dishion, T. J. (2008). Antiosical Personality Development. In Beauchaine T. P. & Hinshaw, S. P. (Eds). *Child and Adolescent Psychoapthology* (pp. 370 -404), Wiley.

Hinshaw, S. P. (1987). On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin, 101*, 443-463.

Hinshaw, S. P. (1994). *Attention deficits and hyperactivity in children*. London: Sage Publications.

Ho, T. P., Leung, P. W. L, Lee, C. C., Tang C. P., Hung, S. F., Kwong S. L., Lucas, C. P., Lieh-Mak, F., Shaffer, D. (2005). Test-retest reliability of the Chinese version of the Diagnostic Interview Schedule for Children – Version 4 (DISC-IV).

*Journal of Child Psychology and Psychiatry, 46*, 1135-1138.

Hong Kong Police Force (2007). Hong Kong Police Review, appendix 8: persons arrested by age group and sex. Retrieved May 16, 2009 from http://www.police.gov.hk/review/2007

Hong Kong Police Force (2009). Brief report on Hong Kong's law and order situation in 2008. Paper for special meeting of the panel on Security of the Legislation Council on 21st January 2009. Retrieved May 16, 2009 from www.legco.gov.hk/yr08-09/english/.../se0121cb2-678-1-e.pdf

Howell, J. C., & Hawkins, J. D. (1998). Prevention of youth violence. *Crime and Justice: A Review of Research, 24*, 263-316.

Ipser, J., & Stein, D. J., (2007). Systematic review of pharmacotherapy of disruptive behaviour disorders in children and adolescents. Psychopharmacology, 191, 127-140.

Ivanova, M. Y., Achenbach, T. M., Dumenci, L., Rescorla, L. A., Almqvist, F., Weintraub, S., et al. (2007). Testing the 8-syndrome structure of the child behavior checklist in 30 societies. *Journal of Clinical Child and Adolescent Psychology, 36*, 405-417.

Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: implications for research, practice, and DSM-IV. *Journal of the American Academy Child and Adolescent Psychiatry, 36(8)*, 1065-1079.

Jensen, P. S., Hinshaw, S. P., Kraemar, H. C., Lenora, N., Newcorn, J. H., Abikoff, H. B., (2001). ADHD comorbidity findings from the MTA Study: comparing comorbid subgroups. *Journal of the American Academy Child and Adolescent Psychiatry, 40(2)*, 147-158.

Johnson, J. H., McCaskill IV, J. W., & Werba, B. E. (2001). Aggressive, antisocial, and delinquent behavior in childhood and adolescence. In Walker, C.E. & Roberts M. C. (Eds.), *Handbook of Clinical Child Psychology* (pp. 393-412), Wiley.

Junger-Tas, J. (1994). *Delinquent Behavior Among Young People in the Western World*. Amsterdam: Kugler Publications.

Kamphaus, R. W. & Frick, P. J., (1996). *Clinical Assessment of Child and Adolescent Personality and Behavior*. Boston: Allyn & Bacon.

Keenan, K., Loeber, R., Zhang, Q., Stouthamber-Loeber, M., & Kammen, W. B. (1995). The influence of deviant peers on the development of boy's disruptive and delinquent behaviour: a temporal analysis. *Development and Psychopathology, 7*, 715-726.

Kelly, B. T., Loeber, R., Keenan, K., & Delamatre, M.(1997). Developmental pathways in boys' disruptive and delinquent behaviour. Juvenile Justice Bulletin, Office of Juvenile Justice and Delinquency Prevention. Retrieved

May 15, 2009 from www.ncjrs.gov/pdffiles/165692.pdf.

Kerr, M. & Stattin, H. (2000). What parents know, how they know it, and several forms of adolescent adjustment: further support for a reinterpretation of monitoring. *Developmental Psychology, 36*, 360-380.

Kimonis, E. R., Frick, P. J., & Barry, C. T. (2004). Callous-unemotional traits and delinquent affiliation. *Journal of Consulting and Clinical Psychology, 72(6)*, 956-966.

Kimonis, E. R., Frick, P. J., Faxekas, H., & Loney, B. R. (2006). Psychopathy, aggression, and the emotional processing of emotional stimuli in non-referred girls and boys. *Behavioral Sciences and the Law, 24*, 21-37.

Kimonis, E. R., Frick, P. J., Skeem, J. L., Marsee, M. A., Cruise, K., Munoz, L. C. et al. (2008). Assessing callous-unemotional traits in adolescent offenders: validation of the inventory of callous-unemotional traits. *International Journal of Law and psychiatry, 31*, 241-252.

King, S., Waschbusch, D. A., Frankland, B. W., Andrade, B. F., Thurston, C. M., McMutt, L., et al. (2005). *Journal of Psychopathology and Behavioral Assessment, 27(2)*, 77-87.

Kirk, D. S. (2006). Examining the divergence across self-report and official data sources on inferences about the adolescent life-course of crime. *Journal of Quantitative Criminology, 22*, 107-129.

Klein, R. G., Abikoff, H., Klass, E., Ganeles, D., Seese, L. M., & Pollack, S. (1997). Clinical efficacy of methylphenidate in conduct disorder with and without attention deficit hyperactivity disorder. *Archives of General Psychiatry*, 54, 1073 – 1080.

Kotler, J. S., & McMahon, R. J. (2005). Child psychopathy: theories, measurement, and relations with the development and persistence of conduct problems. *Clinical Child and Family Psychology Review, 8(4)*, 291-325

Kratzer, L. & Hodgins, S. (1999). A typology of offenders: a test of moffitt's theory among makes and females from childhood to age 30. *Criminal Behaviour and Mental Health, 9(1)*, 57-75.

Kruh, I. P., Frick, P. J., & Clements, C. B. (2005). Historical and personality correlates to the violence patterns of juveniles tried as adults. *Criminal Justice and Behavior, 92*, 69-96.

Kuntsi, J., Oosterlaan, J., & Stevenson, J. (2001). Psychological mechanisms in hyperactivity: I response inhibition deficit, working memory impairment, delay aversion or something else? *Journal of Child Psychology and Psychiatry and the Allied Disciplines*, 42(2), 199-210.

Kuhne, M., Schachar, R., & Tannock, R. (1997). Impact of comorbid oppositional or conduct problems on attention-deficit hyperactivity disorder. *Journal of the*

*American Academy of Child and Adolescent Psychiatry, 36*, 1715-1725.

Lahey, B. B. (2008). Oppositional Defiant Disorder, Conduct Disorder, and Juvenile Delinquency. In Beauchaine T. P. & Hinshaw, S. P. (Eds). *Child and Adolescent Psychoapthology* (pp. 335-369), Wiley.

Lahey, B. B., Goodman S. H., Waldman, I. D., Bird, H, Canino, G., Jensen, P., Regier, D., Leaf, P. J., Gordon, R., & Applegate, B. (1999). Relation of age of onset to the type and severity of child and adolescent conduct problems. *Journal of Abnormal Child Psychology, 27(4)*, 247-260.

Lahey, B. B., Loeber, R., Quay, H. C., Applegate, B.,Shaffer, D., Waldman, I., Hart, E., McBurnett, K., Frick, P. J., Jensen, P. S., Dulcan, M. K., Canino, G., & Bird, H. R. (1998). Validity of DSM-IV subtypes of conduct disorder based on age of onset. *Journal of the American Academy of Child and Adolescent & Psychiatry, 37(4)*, 435-442.

Lahey, B. B., McBurnett, K., Loeber, R. (2000). Are attention-deficit / hyperactivity disorder and oppositional defiant disorder developmental precursors to conduct disorder? In Lewis, M & Sameroff, A (Eds.). *Handbook of Developmental Psychopathology*, pp. 431-446. New York: Plenum.

Lauman, M., Van Noesel, S., J., P., Papanikolau, A., Van Oostenbruggen-Scheffer, J., Veugelers, D., Sergeant, J. A., Oosterlaan, J. (2009). Inhibition, reinforcement sensitivity and temporal information processing in ADHD and ADHD+ODD: evidence of a separate entity? *Journal of Abnormal Child Psychology, 37*, 1123-1135.

Leech, N. L., Barrett, K. C., & Morgan, G. A. (2005). *SPSS for Intermediate Statistics*, 2nd ed. LEA.

Leung, P. W. L., Ho, T. P., Luk, S. L., Taylor, E., et al. (1996). Separation and comorbidity of hyperactivity and conduct disturbance in Chinese schoolboys. Journal of Child Psychology and Psychiatry, 37(7), 841-853

Leung, P. W. L., Kwong, S. L., Tang, C. P., Ho, T. P., Hung, S. F., Lee, C. C., Hong, S. L., Chiu, C. M., & Liu, W. S. (2006). Test-retest reliability and criterion validity of the Chinese version of CBCL, TRF, and YSR. *Journal of Child Psychology and Psychiatry, 47*, 970-973.

Lijffijt, M., Kenemans, J. L., Verbaten, M. N., & Engeland, H. (2005). A meta-analytic review of stopping performance in Attention-Deficit/Hyperactivity Disorder: deficient inhibitory motor control? *Journal of Abnormal Psychology, 114(2)*, 216-222.

Lipsy, M. W., & Wilson, D., B. (1988). Effective intervention for serious juvenile offenders. A synthesis of research. In R. Loeber & D. P. Farrington (Eds.), *Serious and Violent Offenders: Risk factors and Successful Interventions* (pp. 313-345). Thousand Oaks, CA: Sage.

Loeber, R., Burke, J., D., Lahey, B. B., Winters, A., & Zera, M. (2000). Oppositional defiant and conduct disorder: a review of the past 10 years, Part I. *Journal of the American Academy Child and Adolescent Psychiatry, 39(12)*, 1468-1484.

Loeber, R., Burke, J., & Pardini, D. A.(2009). Perspectives on oppositional defiant disorder, conduct disorder, and psychopathic features. *Journal of Child Psychology and Psychiatry, 50*, 133-142.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., Moffitt, T. E., Caspi, A. (1998). The development of male offending: key findings from the first decade of the Pittsburgh youth study. *Studies in Crime and Crime Prevention, 7*, 141-171.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., Moffitt, T. E., Caspi, A., & Lynam, D. (2001). Male mental health problems, psychopathy, and personality traits: key findings from the first 14 years of the Pittsburgh Youth Study. *Clinical Child and Family Psychology Review, 4*, 273 – 297.

Loeber, R., Farrington, D. P., Stouthamer-Loebr, M., & van Kammen, W. B. (1998). *Antisocial Behavior and Mental Health Problems: Explanatory factors in Childhood and Adolescence*. Mahwah, NJ: Lawrence Erlbaum.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & White, H. R. (2008). *Violence and Serious Theft: Development and Prediction from Childhood to Adulthood*. New York: Routledge.

Loeber, R., Green, S. M., Keenan, K., Lahey, B. B. (1995). Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. *Journal of the American Academy Child and Adolescent Psychiatry, 34*, 499-509

Loeber, R., Pardini, D., Homish, D., L., Wei, E. H., & Crawford, A. M., Farrington, D. P., et al. (2005). The prediction of violence and homicide in young men. *Journal of Consulting and Clinical Psychology, 73*, 1074 – 1088.

Loeber, R. & Stouthamer-Loeber, M. (1998). Development of Juvenile aggression and violence: some common misconceptions and controversies. *American Psychologist, 53*, 242-259.

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H., Carr (Eds.), *Inhibitory Processes in Attention, Memory, and Language* (pp. 189-239). San diego: Academic Press

Loney, B. R., Frick, P. J., Clements, C. B., Ellis, M. L., & Kerlin, K. (2003). Callous-unemotional traits, impulsivity, and emotional processing in adolescents with antisocial behavior problems. *Journal of Clinical Child and Adolescent Psychology, 32 (1)*, 66-80.

Loney, B. R., Frick, P. J., Ellis, M. L., & McCoy, M. G. (1998). Intelligence,

callous-unemotional traits, and antisocial behavior. *Journal of Psychopathology and Behavioral Assessment, 20(3)*, 231-247.

Lynam, D., Moffitt, T., & Stouthamer-Loeber, M. (1993). Explaining the relation between IQ and delinquency: class, race, test motivation, school failure, or self-control? Journal of Abnormal Psychology, 102, 187-196.

Lynam, D. R., Caspi, A., Moffitt, T. E., Loeber, R., & Stouthamer-Loeber, M. (2007). Longitudinal evidence that psychopathy scores in early adolescence predict adult psychopathy. *Journal of Abnormal Psychology, 116*, 155-165.

Marcus, D. K., John, S. L., & Edens, J. F. (2004). A taxometric analysis of psychopathic personality. *Journal of abnormal Psychology, 113*, 626-635.

Maughan, B., Rowe, R., Messser, J., Goodman, R., & Meltzer, H. (2004). Conduct disorder and oppositional defiant disorder in a national sample: developmental epidemiology. *Journal of Child Psychology and Psychiatry, 45*, 609-621.

McArdle, J. J., Nesselroade, J. R. Schinka, J. A., & Velicer, W. F. (2003). Growth curve analysis in contemporary psychological research. *Handbook of Psychology: Research Methods in Psychology, Vol. 2* ( pp. 447-480). New York: Wiley.

McCabe, M. K., Hough, R., Wood, P. a., Yeh, M. (2001). Childhood and adolescent onset conduct disorder: a test of the developmental taxonomy. *Journal of Abnormal Child Psychology, 29*, 305 – 316.

McCart, M. R., Priester, P. E., Davies, W. H., & Azen, R. (2006). Differential effectiveness of behavioral-training and cognitive-behavioral therapy for antisocial youth: a meta-analysis. *Journal of Abnormal Child Psychology, 34(4)*, 527-543.

McMahon, R. J., & Frick, P. J., (2005). Evidence-based assessment of conduct problems in children and adolescents. *Journal of Clinical Child and Adolescent Psychology, 34(3)*, 477-505.

McGuire, J. (2002). Integrating findings from research reviews. In J. McGuire (Ed.), *Offender rehabilitation and treatment: effective programmes and polices to reduce re-offending* (pp.3-38). Chichester, England: Wiley.

Moffitt, T. E., (1989). Accommodating self-report methods to a low-delinquency culture: experience from New Zealand. In M. W., Klein (Ed.), *Cross-national research in self-reported Crime and Delinquency* (pp.43-66). Norwell, Ma: Kluwer Academic.

Moffitt, T. E. (1990). Juvenile delinquency and attention deficit disorder: boys' developmental trajectories from age 13 to age 15. *Child Development, 61*, 893-910.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behaviour: a developmental taxonomy. *Psychological Review, 100(4)*, 674-701

Moffitt, T. E. (1997). Adolescence-limited and life-course-persistent offending: A complementary pair of developmental theories. In T. Thornberry (Eds.), *Advances in Criminological Theory: Developmental Theories of Crime and Delinquency* (pp. 11-54). London: Transaction Press.

Moffitt, T. E. (2003). Life-course-persistent and adolescent-limited antisocial behavior: a 10-year research review and a research agenda. In B.B., Lahey, T. E. Moffitt., & A., Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp.49-75). The Guildford Press

Moffitt, T. E. (2006). Life-course-persistent versus adolescent-limited antisocial behavior. In Cicchetti & Cohen (Eds.), *Developmental Psychopathology: Risk, Disorder, and Adaptation* (pp. 570-598). Wileys.

Moffitt, T. E. (2009). A review of research on the taxonomy of life-course persistent versus adolescent-limited antisocial behaviour. In Cullen, F. T., Wright, J. P., & Blevins, K. R. (Eds.), *Taking Stock: the Status of Criminological Theory* (pp.277-312). Transaction Publishers.

Moffitt, T. E., Arseneault, L., Jaffee, S. R., Kim-Cohen, J., Koenen, K. C., Odgers, C. L., Slutske, W. S., Viding, E. (2008). Research Review: DSM-V conduct disorder: research needs for an evidence base. *Journal of Child Psychology and Psychiatry, 49(1)*, 3-33.

Moffitt, T. E. & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology, 13*, 355-375.

Moffitt, T. E., Caspi, A., Dickson, N., Silva, P. A., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct in males: Natural history from age 3 to 18. *Development and Psychopathology, 8*, 399-424.

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

MTA Cooperative Group (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 56*, 1073-1086.

Muthen, B. O. (2004). Latent variable analysis: Growth mixture modelling and related techniques for longitudinal data. In D. Kaplan (Ed.), *Handbook of Quantitative Methodology for the Social Sciences* (pp.345 – 368). Newbury Part, CA: Sage.

Muthen, B., O., & Shedden, K., (1999). Finite mixture modelling with mixture outcomes using the EM algorithm. *Biometrics, 55*, 463-469.

Nagin, D. S., (1999). Analyzing developmental trajectories: a semiparametric, group-based approach. *Psychological Methods, 4*, 139 – 177.

Nagin, D S & Tremblay, R E (2001) Analyzing developmental trajectories of distinct but related behaviours a group-based method *Psychological Medicine, 6*, 18-34

Narcotic Division, Security Bureau, HK (2009) Retrieved May 16, 2008 from http //www nd gov hk/en/statistics_list htm

Nathalie, F, Rijsdijk, A, Fruhling, V, McCrory, E J P, & Viding, E (2010) Etiology of different developmental trajectories of callous-unemotional traits *Journal of the American Academy of Child and Adolescent Psychiatry, 49(7)*, 656-664

Neale, M C, & Kendler, K S (1995) Models of comorbidity for multifactorial disorders *American Journal of Human Genetics, 57*, 935-953

New Century Forum (2006) Report on adolescent antisocial behaviour Retrieved January 16, 2008 from http //www ncforum org hk/news_image/20060528 pdf

Newman, J P, & Kosson, D S (1986) Passive avoidance learning in psychopathic and non-psychopathic offenders *Journal of Abnormal Psychology, 95*, 252-256

Newman, J P, Patterson, C M, & Kosson, D S (1987) Response perseveration in psychopaths *Journal of Abnormal Psychology, 96*, 145-148

Newman, J P, & Wallance, J F, (1993) Diverse pathways to deficit self-regulation implications for disinhibitory psychopathology in children *Clinical Psychology Review, 13*, 688-720

Newman, J P, Widom, C S, & Nathan, S (1985) Passive avoidance in syndromes of disinhibition psychoapthy and extraversion *Journal of Personality and Social Psychology, 43*, 1316-1327

Nigg, J T (2000) On inhibition/disinhibition in developmental psychopathology views from cognitive and personality psychology and a working inhibition taxonomy *Psychological Bulletin*, 126(2), 220-246

Nigg, J T (2001) Is ADHD a disinhibitory disorder? *Psychological Bulletin*, 127(5), 571-598

Nigg, J T (2006) Executive functioning or cognitive control In Nigg, J T (Ed ), *What Causes ADHD* (pp 106-141) Guildford Press

Nigg, J T (2006b) Multiple pathways In Nigg, J T (Ed ), *What Causes ADHD* (pp 175-192) Guildford Press

Nigg, J T, Goldsmith, H H, & Sachek, J, (2004) Temperament and attention deficit hyperactivity disorder the development of a multiple pathway model *Journal of Clinical Child and Adolescent Psychology, 22(1)*, 42-53

Nigg, J T & Nikolas M, (2008) Attention-Deficit / Hyperactivity Disorder In Beauchaine T P & Hinshaw, S P (Eds) *Child and Adolescent Psychoapthology* (pp 370-404), Wiley

Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2007). Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the National Comorbidity Survey Replication. *Journal of Child Psychology and Psychiatry, 48*, 703-713.

O' Brien, B. S. & Frick, P. J. (1994). Reward dominance among children with disruptive behaviour disorders. *Journal of Psychopathology and Behavioral Assessment, 16(2)*, 131 – 145.

O' Brien, B. S. & Frick, P. J. (1996). Reward dominance: association with anxiety, conduct problems, and psychopathy in children. *Journal of Abnormal Child Psychology, 24*, 223-239.

Odgers, C. L., Caspi, A., Poulton, R., Harrington, H. L., Thomson, M., Broadbent, J., Dickson, N., Sears, M., Hancox, B., & Moffitt, T. E. (2007). Conduct problem subtypes predict differential adult health burden. *Archives of General Psychiatry, 64*, 476-484.

Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R. Thomson, W. M., & Caspi, A., (2008). Female and male antisocial trajectories: from childhood origins to adult outcomes. *Development and Psychopathology, 20*, 673-716.

OJJDP, Department of Justice, U. S. (2009). Juvenile Justice Bulletin (April, 2009). Retrieved May 16, 2009 from www.ojp.usdoj.gov/ojjdp.

Olsson, M. (2009). DSM diagnosis of conduct disorder (CD) – a review. *Nordic Journal of Psychiatry, 63(2)*, 102-112.

Oosterlaan, J., Logan, G. D., Sergeant, J. A. (1998). Response inhibition on AD/HD, CD, comorbid AD/HD+CD, Anxious, and control children: a meta-analysis of studies with stop task. *Journal of Child Psychology and Psychiatry, 39(3)*, 411-425.

Oxford, Cavell, & Haghes (2003). Callous/Unemotional traits moderate the relation between ineffective parenting and child externalizing problems: a partial replication and extension. Journal of Clinical Child and Adolescent Psychology, 32(4), 577-585.

Palanczyk, G., Caspi, A., Houts, R., Lollins, S. H., Rohde, L. A., & Moffitt, T. E. (2010). Implications of extending the ADHD age-of-onset criterions to age 12: results from a prospectively studied birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry, 49(3)*, 210-216.

Pardini, D. A., (2006). The callousness pathway to severe violent delinquency. *Aggressive Behavior, 32*, 590-598.

Pardini, D. A., Lochman, J. E., & Powell, N. (2007). The development of callous-unemotional traits and antisocial behaviour in children: are there shared and / or unique predictors? *Journal of Clinical Child and Adolescent*

*Psychology, 36(3)*, 319-333.

Paradini, D. A., Lochman, J. E., & Frick, P. J. (2003). Callous/unemotional traits and social cognitive processes in adjudicated youths. *Journal of American Academy of Child and Adolescent Psychiatry, 42*, 364 – 371.

Patrick, C. J., Curtin, J. J., & Tellegen, A. (2002). Development and validation of a brief form of the multidimensional personality questionnaire. *Psychological Assessment, 14*, 150-163.

Patterson, G. R. (1996). Some characteristics of developmental theory for early onset delinquency. In M. Lenzenweger & J. J. Haugaard (Eds.), *Frontiers of Developmental Psychopathology* (pp.81-124). New York: Oxford University Press.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behaviour. *American Psychologist, 44*, 329 – 335.

Patterson, G. R., DeGarmo, D. S., & Knutson, N. (2000). Hyperactive and antisocial behaviors: Comorbid or two points in the same process? *Development and Psychopathology, 12*, 91-106.

Patterson, G. R., & Forgatch, M. (1987). *Parents and adolescents living together: Part 1*. Eugene, OR: Castalia.

Patterson, G. R., & Forgatch, M. S., Yoerger, K. L., & Stoolmiller, M. (1998). Variables that initiate and maintain an early-onset trajectory for juvenile offending. *Development and Psychopathology, 10*, 531-548.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *A social learning approach: Vol. 4. Antisocial boys*. Eugene, OR: Castalia.

Patterson, G. R. & Yoerger, K. (1993). Developmental models for delinquent behavior. In S. Hodgins (Ed.), *Mental Disorder and Crime* (pp.140-172). Newbury Part, CA: Sage.

Patterson, G. R., & Yoerger, K. (1997). A developmental model for late-onset delinquency. In R. Dienstbier & D. w. Osgood (Eds.), *The Nebraska Symposium on Motivation, vol. 44: Motivation and Delinquency*, PP. 119-177.

Pekarik, E. G, Prinz, R. J., Liebert, D. E., Weintraub, S., & Neale, J. M. (1976). The Pupil Evaluation Inventory: A sociometric technique of assessing children's social behaviour. *Journal of Abnormal Child Psychology, 4*, 83-97.

Pelham, W. E., Wheeler, T., & Chronis, A. M. (1998). Empirically supported psychosocial treatment for attention deficit hyperactivity disorder. *Journal of Clinical Child Psychology, 27*, 190-205.

Pfiffner, L., J., McBurnett, K., Rathouz, P. J., & Judice, S. (2005). Family correlates of oppositional and conduct disorders in children with attention deficit / hyperactivity disorders. *Journal of Abnormal Child Psychology, 33(5)*, 551-563.

Piquero, A. R. (2000). Frequency, specialization, and violence in offending careers. *Journal of research in crime and delinquency, 37*, 392-418.

Piquero, A. R. (2008). Taking stock of developmental trajectories of criminal activity over the life course. In A. M. Liberman (Eds), *The Long View of Crime: A Synthesis of Longitudinal Research* (pp.23-78). New York: Springer.

Piquero, A. R., Farrington, D. P., & Blumstein, A. (2007). *Key Issues in Criminal Career Research: New Analyses of the Cambridge Study in Delinquent Development*: Cambridge: Cambridge University Press

Piquero, A. R., Gibson, C. L., Daigle, L. E., Piquero, N., & Tibbetts, S. G. (2007). Are life-course-persistent offenders at risk for adverse health outcomes? *Research in Crime and Delinquency, 44*, 185-207.

Pliszka, S. R. (2000). Patterns of psychiatric comorbidity with attention-deficit / hyperactivity disorder. *Child and Adolescent Psychiatric Clinics of North American, 9(3)*, 525-540.

Porter, S., & Woodworth, M. (2006). Psychopathy and aggression. In C. J. Patrick (Eds.), Handbook for Psychopathy (pp.481-494). New York: Guildford Press.

Puzzanchera, C., Adams, B., and Kang, W. (2008). *Easy Access to FBI Arrest Statistics 1994-2006*. Retrieved January 16 2009 from http://ojjdp.ncjrs.gov/ojstatbb/ezaucr/

Quay, H. C. (1987). Patterns of delinquent behaviour. In H. C., Quay (Ed.), *Handbook of Juvenile Delinquency* (pp.118-138). New York: Wiley.

Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder: a theoretical perspective. *Development and Psychopathology, 5*, 165-180.

Ramtekkar, U. P., Reiersen, A. M., Todorov, A. A. & Todd, R. D. (2010). Sex and age difference in Attention-Deficit/Hyperactivity Disorder Symptoms and Diagnoses: implications for DSM-V and ICD-11. *Journal of the American Academy of Child and Adolescent Psychiatry, 49(3)*, 217-228.

Rapport, M. D., Chung, K. M., Shore, G., Denney, C. B., Isaacs, P. (2000). Upgrading the science and technology of assessment and diagnosis: laboratory and clinic-based assessment of children with ADHD. *Journal of Clinical Child Psychology, 29(4)*, 555-568.

Reynolds, C. R., & Kamphaus, K. W. (2004). *Behavioral Assessment System for Children – 2*. Circle Pines, MN: AGS.

Reynolds, C. R., & Richmond, B. O. (1985). *Revised Children's Manifest Anxiety Scale Manual*. Los Angeles: Western Psychological Services.

Rhee, S. H., Willcutt, E. g., Hartman, C. A., Pennington, B. F., & DeFried, J. C. (2008). Test of alternative hypotheses explaining the comorbidity between Attention-Deficit/Hyperactivity Disorder and Conduct Disorder. *Journal of*

*Abnormal Child Psychology, 36*, 29-40.

Rowe, R., Maughan, B., Costello, E. J., & Angold, A. (2005). Defining oppositional defiant disorder. *Journal of child Psychology and Psychiatry, 46*, 1309-1316.

Rowe, R., Maughan, B., Moran, P., Ford, t., Briskman, J., & Goodman, R. (2010). The role of callous and unemotional traits in the diagnosis of conduct disorder. *Journal of Child Psychology and Psychiatry, 51(6)*, 688-695.

Rowe, R., Maughan, B., Pickles, A., Costello, E. J., & Angold, A. (2002). The relationship between DSM-IV oppositional defiant disorder and conduct disorder: findings from the Great Smoky Mountains Study. *Journal of Child Psychology and Psychiatry, 43*, 365-373.

Russell, S. & Rosemary, T. (2005). Test of four hypotheses for the comorbidity of attention-deficit hyperactivity disorder and conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 34(5)*, 639 – 648.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M. W. Kent & J. E. Rolf (Eds.), *Primary prevention of psychopathology III: Social competence in children* (pp. 49 – 74). Hanover, NH: University Press of New England.

Rutter, M. (1997). Comorbidity: concepts, claims and choices. *Criminal Behaviour and Mental Health, 7*, 265-285.

Rutter, M. (2003). Crucial Paths from Risk Indicator to Causal Mechanism. In Lahey, B. B., Moffitt, T. E., & Caspi, A. (Eds.), *Causes of Conduct Disorder and Juvenile Delinquency* (pp.3-24). The Guilford Press.

Rutter, M., Giller, H., & Hagell, A. (1998a). Concepts and Measures of Crime. In Rutter, M., Giller, H., & Hagell, A (Eds.), *Antisocial Behavior by Young People* (pp.25-61). Cambridge University Press.

Rutter, M., Giller, H., & Hagell, A. (1998b). Introduction. In Rutter, M., Giller, H., & Hagell, A (Eds.), *Antisocial Behavior by Young People* (pp.1-24). Cambridge University Press.

Rutter, M., Giller, H., & Hagell, A. (1998c). Varieties of antisocial behavior. In Rutter, M., Giller, H., & Hagell, A (Eds.), *Antisocial Behavior by Young People* (pp.95-126). Cambridge University Press.

Rutter, M., Maughan, B., Meyer, J., Pickles, A., Silberg, Simonoff, E., & Taylor, E. (1997). Heterogeneity of antisocial behavior: causes, continuities, and consequences. In R. Dienstbier & D. w. Osgood (Eds.), *The Nebraska Symposium on Motivation, vol. 44: Motivation and Delinquency* (PP. 45-118).

Salekin, R. T., Rogers, R., & Sewell, K. w., (1996). A review and meta-analysis of the Psychopathy Checklist and Psychopathy Checklist Revised: predictive validity of dangerousness. *Clinical Psychology: Science and Practice, 3*, 203-215.

Sameroff, A. J. (2000). Dialectical processes in developmental psychopathology. In A. Sameroff, M., Lewis, & S. Miller (Eds.), *Handbook of Developmental Psychology (2nd edition)* ( pp. 23-40). New York: Kluwer Academic / Plenum Publishers.

Sanford, M., Boyle, M. H., Szatmari, P., Offord, D. R., Jamieson, E., & Spinner, M. (1999). Age-of-onset classification of conduct disorder: reliability and validity in a prospective cohort study. *Journal of the American Academy of Child Psychiatry, 38(8)*, 992-999.

Satterfield, J. H. & Schell, A. (1997). A prospective study of hyperactive boys with conduct problems and normal boys: adolescent and adult criminality. *Journal of the American Academy Child and Adolescent Psychiatry, 36*, 1726 – 1735.

Sattler, J. M. & Ryan J. R. (2001). Wechsler Adult Intelligence Scale-III (WAIS-III): description. In Sattler, J. M (Ed.), *Assessment of Children: Cognitive Applications (4th edition)* (pp. 375-414). Jerome M. Sattler, Publisher, Inc.

Schachar, R. J., Tannock, R.(1995). Test of four hypotheses for the comorbidity of attention-deficit hyperactivity disorder and conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*, 639-648.

Schachar, R. J., Tannock, R., Marriott, M., & Logan, G (1995). Deficient inhibitory control in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 23*, 411-438.

Schweitzer, J. B. & Sulzer-Azaroff, B. (1995). Self-control in boys with attention-deficit hyperactivity disorder: Effects of added stimulation and time. *Journal of Child Psychology and Psychiatry, 36*, 671-686.

Semrud-Clikeman, M., Steingard, R. J., Filipek, P., Biederman, J., Bekken, K., & Renshaw, P. (2000). Using MRI to examine brain-behavior relationships in makes with attention deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 477-484.

Shaffer, D, Fisher, P., Dulcan M. K., Davies, M., Piacentini, J., & Schwab-Stone, M. E., et al. (1996). The NIMH Diagnostic Interview Schedule for Children, Version 2.3 (DISC-2.3): description, acceptability, prevalence rates, and performance in the MECA study. *Journal of American Academy Child and Adolescent Psychiatry, 35 (7)*, 865-877.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwabstone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): description, differences from pervious versions, and reliability of some common diagnoses. *Journal of American Academy Child and Adolescent Psychiatry, 39(1)*, 28-38.

Shaw, D. S., Lacourse, E., & Nagin, D. S. (2005). Developmental trajectories of conduct problems and hyperactivity from ages 2 to 10. *Journal of child*

*Psychology and Psychiatry, 46*, 931-942

Shelton, K. K., Frick, P. J., & Wootton, J. (1996). The assessment of parenting practices in families of elementary school-aged children. *Journal of Clinical Child psychology, 25*, 317-327.

Sibley, M. H., Pelham, W. E., Molina, B. S. G, Gnagy, E. M., Waschbusch, D. A., Biswas, A. Et al. (2010). The delinquent outcomes of boys with ADHD with and without comorbidity. *Journal of Abnormal Child Psychology, 38(6)*, published online on 10 August 2010.

Silberg, J. L., Rutter, M., Tracy, K., Maes, H. H., & Eaves, L. (2007). Etiological heterogeneity in the development of antisocial behavior: the Virginia Twin Study of Adolescent Behavioral development and the young adult follow-up. *Psychological Medicine, 37*, 1193-1202.

Siverthorn, P., Frick, P. J., & Reynolds, R. (2001). Timing of onset and correlates of severe conduct problems in adjudicated girls and boys. *Journal of Psychopathology and Behavioral Assessment, 23*, 171-181.

Stattin, H. & Kerr, M. (2000). Parental monitoring: a reinterpretation. *Child Development, 71*, 1072-1085.

Stevens, J. P. (2009). *Applied Multivariate Statistics for the Social Sciences* – 5th edition. Routledge.

Stewart, M. A., Cummings, C., Singer, S., & deBlois, C. S. (1981). The overlap between hyperactive and unsocialized aggressive children. *Journal of child Psychology and Psychiatry, 22(1)*, 35-45.

Szatmari, P. (1992). The epidemiology of attention-deficit hyperactivity disorders. In G. Weiss (Ed.), *Child and adolescent psychiatry clinics of North America: Attention deficit disorder* (pp.361-372). Philadelphia: W. B. Saunders.

Szatmari, P., Boyle, M. H. & Offord, D. R. (1989). ADDH and conduct disorder: degree of diagnostic overlap and differences among correlates. *Journal of the American Academy Child and Adolescent Psychiatry, 28*, 865-872.

Taylor, E. (1999). Developmental neuropsychopathology of attention deficit and impulsiveness. *Development and Psychopathology, 11*, 6-7-628.

Taylor, E., Chadwick, W., Heptinstall, E., & Danckaerts, M. (1996). Hyperactivity and conduct problems as risk factors for adolescent development. *Journal of the American Academy Child and Adolescent Psychiatry, 35(9)*, 1213-1226.

Taylor, E., Iacono, W. g., & McGue, M. (2000). Evidence for a genetic eiotiology for early-onset delinquency. *Journal of Abnormal Psychology, 109*, 634-643.

Taylor, E., Schachar, R., Thorley, G., & Wieselberg, M. (1986a). Conduct disorder and hyperactivity: I, separation of hyperactivity and antisocial conduct in British child psychiatric patients. *British Journal of Psychiatry, 149*, 760-767.

Taylor, E., Schachar, R., Thorley, G., & Wieselberg, M. (1986b). Conduct disorder

and hyperactivity II: a cluster analytic approach to the identification of a behavioural syndrome. *British Journal of Psychiatry, 149*, 768-777.

Vagg, J., Bacon-Shone, J., Gray, P., & Lam, D. (1995). *The Final Report on the Social Causes of Juvenile Crime*. Hong Kong, China: Fight Crime Committee.

Van Lier, P. A., C., Van Der Ende, J., Koot, H., M., Verhulst, F. C. (2007). Which better predicts conduct problems? The relationship of trajectories of conduct problems with ODD and ADHD symptoms from childhood into adolescence. *Journal of Child Psychology and Psychiatry, 48(6)*, 601-608.

Van Lier, P. A. C., Wanner, B., & Vitaro, F. (2007). Onset of antisocial behavior, affiliation with deviant friends, and childhood maladjustment: a test of the childhood and adolescent-onset models. *Development and psychopathology, 19*, 167-185.

Vasey, M. W., Kotov, R., Frick, P. J., & Loney, B. (2005). The latent structure of psychopathy in youth: a taxometric investigation. *Journal of Abnormal Child Psychology, 33*, 411-429.

Verbruggen, F., & Logan, G. D. (2008). Response inhibition in the stop-signal paradigm. *Trends in Cognitive Sciences, 12 (11)*, 418-424.

Verbruggen, F., & Logan, G. D. (2009). Model of response inhibition in stop-signal and stop-change paradigms. *Neuroscience and Biobehavioral Review, 33*, 647-661.

Verburggen, F., Logan, G. D., & Stevens M. A. (2008). STOP IT: Windows executable software for the stop-signal paradigm. *Behavior Research Methods, 40(2)*, 479 – 483.

Vermeiren, R. (2003). Psychopathology and delinquency in adolescents: a descriptive and developmental perspective. *Clinical Psychology Review, 23*, 277-318.

Viding, E. (2004). Understanding the development of psychopathy. *Journal of Child Psychology and Psychiatry, 45(8)*, 1329-1337.

Viding, E., Blair, R. J. R., Moffitt, T. e., & Plomin, R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry, 46*, 592-597.

Viding, E. Fontaine, N. M. G, Oliver, B. R., & Plomin, R. (2009). Negative parental discipline, conduct problems and callous-unemotional traits: monozygotic twin differences study. *The British Journal of Psychiatry, 195*, 414-419.

Viding, E., Frick, P. J., & Plomin, R. (2007). Aetiology of relationship between callous-unemtional traits and conduct problems in childhood. *British Journal of Psychiatry, 190 (49)*, 33-38.

Viding, E., Jones, A. P., Frick, P. J., Moffitt, T. E., & Plomin, R (2008). Heritability of antisocial behavior at 9: do callous-unemotional traits matter? *Developmental*

*Science,* 11, 17-22.

Vieno, A. & Nation, M. (2009). Parenting and antisocial behavior: a model of relationship between self-disclosure, parental closeness, parental control, and adolescent antisocial behavior. *Developmental Psychology, 45(6)*, 1509-1519.

Vitaro, F., Bredngen, M., & Tremblay, R. E. (2000). Influence of deviant friends on delinquency : searching for moderator variables. Journal of Abnormal Child Psychology, 28, 313-325.

Wan, M. P. (2006). Application of Chinese Wechsler Adult Intelligence Scale-III: an evaluation of its psychometric properties. Graduate thesis of the University of Hong Kong.

Waschbusch, D. A. (2002). A meta-analytic examination of comorbid hyperactivity-impulsive-attention problems and conduct problems. *Psychological Bulletin, 128*, 118-149.

Waschbusch, D. A., Carrey, N. J., Willoughby, M. T., King, S., & Andrade, B. F. (2007). Effects of methylphenidate and behavior modification on the social and academic behavior of children with disruptive behavior disorders: the moderating role of callous-unemotional traits. *Journal of Clinical Child and Adolescent Psychology, 36(4)*, 629-644.

Waschbusch, D. A., & Willoughby (2008). Attention-deficit/Hyperactivity Disorder and callous-unemotional traits as moderators of conduct problems when examining impairment and aggression in elementary school children. *Aggressive Behavior, 34*, 139-153.

Wechsler, D. (1974). Wechsler Intelligence Scale for Children-Revised. San Antonio: the Psychological Corporation.

Wechsler, D., (1981). *Hong Kong Wechsler Intelligence Scale for Children Manual.* The Psychological Corporation, N. Y., USA.

Wechsler, D., (2002). *Wechsler Adult Intelligence Scale – third edition, Chinese version Manual.* The Psychological Corporation, N. Y., USA.

White, H. R., Bates, M. E. & Buyske, S. (2001). Adolescent-limited versus persistent delinquency: extending Moffitt's hypothesis into adulthood. *Journal of Abnormal Psychology, 110*, 600-609.

White, J. L., Moffitt, T. E., Caspi, A., Bartusch, D. J., Needles, D. J., & Stouthamer-Loeber, M. (1994). Measuring impulsivity and examining its relationship to delinquency. *Journal of Abnormal Psychology, 103(2)*, 192-205.

Wiesner, M., Capaldi, D. M. (2003). Relations of childhood and adolescent factors to offending trajectories of young man. *Journal of Research in Crime and Delinquency, 40 (3)*, 231-262.

Wiesner, M., and Windle, M., (2004). Assessing covariates of adolescent delinquency trajectories: a latent growth mixture modeling approach. *Journal*

*of Youth and Adolescence, 33(5)*, 431-442.

Wootton, J. M., Frick, P. J., Shelton, K. K., and Siverthorn, P. (1997). Ineffective parenting and childhood conduct problems: the moderating role of callous-unemotional traits. *Journal of Consulting and Clinical Psychology, 65(2)*, 301-308.

Zachary, R. A. (1986). *Shipley Institute for Living Scale: Manual.* Los Angeles, CA: Western Psychological Services.

Zaparniuk J. & Taylor (1997). Impulsivity in children and adolescents. In Webster, C. D. & Jackson, M. A. (Ed.), *Impulsivity: theory, assessment and treatment* (pp. 158-179). Guilford Press.

Zuckerman, M., Kolin, E. A., Price, L., & Zoob, I. (1964). Development of a sensation seeking scale. *Journal of Consulting and Clinical Psychology, 28*, 477-482.

Zuckerman, M. (1979). Sensation Seeking: Beyond the optimal level of arousal. Hillsdale, NJ: Erlbaum.

**END**

# Appendix I: data collection sequence and standardized instructions

***List of data collection sequence and time estimated***

| Time required | Sequence of data collection |
|---|---|
| Session 1 | |
| 60' | Structured interview 1: DISC-IV (for ADHD, CD and ODD) |
| 15' | Interview 2: for 1. demographic information, 2 background risks |
| 10' | Interview 3: for 1. peer delinquency 2. self delinquency |
| 15' | Administration of HK-WISC<br>Short form (similarities, vocabulary, block design, object assembly) if age = or below 15, administration of short form of WAIS-III-chinese version if age = or > 16 |
| Time for session 1 | 100 minutes |
| 10' | Questionnaire 1: Inventory of Callous unemotional trait (ICU): 24-items |
| 20' | Questionnaire 2: Alabama Parenting Questionnaire – 42 items |
| 10' | Questionnaire 3: strengths and difficulties questionnaire – self report, 25 items |
| 5' | Questionnaire 4: anxious /depressed scale / YSR – 14 items |
| 20' | Computer task 1: stop task |
| 15' | Computer task 2: card playing task |
| Time for session 2 | 80 minutes |
| Total time | 180 minutes |

**Appendix I**

***Standardized instructions 1***

instruction for data collection with adolescent subjects

青少年行為及成長研究計劃

_____同學，多謝你參與這個研究 ，尼個研究係關於青少年的行為既，研究的資料搜集會在呢度(屯門院 or school) 進行；我會同你面談、填寫問卷、同埋做幾個以電腦進行的行為測試，時間要大約三個小時，會分別係二節進行。如果你對我問你的問題或填寫的問卷有不明白的地方，記住話比我知，有任何其他需要，亦話比我知啦

留意，你所講所填既資料全部保密，只作研究用途，沒有你同意，你的資料不會向其他人發放

有沒有問題？

**Appendix I**

***Standardized instructions 2 : instructions for the two computerized tasks***

## 1 Card-playing task

以下係一個啤牌o既遊戲。如果你禁呢個制 (demonstration)，張牌就會打開。如果你揭開o既啤牌係公仔o既話，你會贏一分。如果你揭開o既啤牌係數字，你會輸一分。如果你最後贏到"24"分，你就會得到呢大獎 – 呢排塊朱古力 (show the chocolate) 。如果你最後贏到"20"分，你就會得到中獎 - 呢排塊朱古力 (show the chocolate)。如果你最後贏到 "16"分，你就會得到呢份細獎- 呢塊朱古力 (show the chocolate)。如果你得到o既分數係少過"16"分，你會得不到任何o既獎品。你可以除時停止揭牌，終止遊戲，只要你禁呢個制就可以啦。

你要記住呢個遊戲係無得跳牌架, 只可以揀開牌, 或者放棄遊戲, 一禁左放棄，遊戲就終止， 咁電腦就用你噤"放棄"制時o既分數去計分架啦.

呢副唔係一副普通o既啤牌，唔止有52張。係玩o既過程你唔需要數有幾多張啤牌啦。

## 2 Stop Task

「稍後，在電腦螢光幕中間會連續出現一個「□」或一個「○」，你需要快而準作出反應：如果是「□」，請用左手食指按鍵盤的「Z」鍵；如果是「○」，請用右手食指按「/」鍵，請留意，反應要快而準確。每次在「□」或「○」出現前，螢光幕中間會先出現一個「＋」，提示你集中留意跟著出現的符號。」

「有時候，在「□」或「○」出現後，你會聽到「嘟」一聲。當你一聽到「嘟」一聲，請不要按任何鍵。有時，這「嘟」聲在符號出現後不久就出現，你會發現較容易去停止按鍵；但有時這聲響會出現得較遲，你會發現較難，甚至沒有可能去停止按鍵。無論如何，請你不要等，不要等沒聽到聲響時才去按鍵。當你一看見「□」或「○」出現，便要立即以你最快的速度去按鍵：記著，如果是「□」，按左邊的「Z」鍵；如果是「○」，按右邊的「/」鍵。有冇問題？」

「如果冇問題，我們先練習一下。請將左手食指放在「Z」鍵，右手食指放在「/」鍵。準備好未？準備好就請按「Z」鍵或「/」鍵開始！」

**Appendix I**

**Practice Block (60 trials)**

Give quick short feedback when necessary to optimize performance:
- (Subject's response time slow down) 「記住要快！」
- (Following a commission error) 「要按對。」
- (Subject tends to wait for stop-signal) 「不要等！」

When subject asks how long to go, don't give a specific response
– e.g. say, *"你做得快D，就快D完" The faster you work, the faster you'll finish"*

「你做得好好！現在測試正式開始，請將手指放在鍵上。記著，集中精神，要快而準！準備好未？好！請按「Z」鍵或「/」鍵開始！」

**Experimental Block 1 (60 trials) –after 30 trials:** 「好, 集中 D 玩埋佢啦」

「做得好好！我地繼續做下一組。請將手指放在鍵上。要盡力做到快而準！準備好未？好！集中精神！開始！」

**Experimental Block 2 (60 trials) –after 30 trials:** 「好, 比心機玩埋佢呀」

「做得非常好！現在做最後一組。記著集中精神！好！開始！」

**Experimental Block 3 (60 trials) –after 30 trials:** 「係啦, 集中 D 玩埋佢啦」

「測試完畢。」

<END>