Antisocial Behaviour in Boys and Girls: A Review and Two Longitudinal Studies of the Developmental Origins

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Antisocial Behaviour in Boys and Girls:

A Review and Two Longitudinal Studies of the Developmental Origins

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STATEMENT OF ORIGINALITY

This work has not been submitted previously for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made.

Juliane Pariz Teixeira

14 July 2020

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ABSTRACT

Research seeking to understand the developmental origins of antisocial behaviour (ASB) has depended on complex theories and analyses, as well as multidisciplinary approaches. Such complexity comes from considering how different aspects of individuals' lives may interact to explain the change in their ASB over the lifespan. Overall, developmental theories support that ASB originates in a mix of adverse socio-environmental circumstances and individual characteristics (e.g., temperament and mental health conditions) that vary in number and seriousness between people but also within the same individual over the life course. However, one of the main challenges of studying ASB development is the substantial heterogeneity in its manifestation, which includes distinct aggression forms (e.g., physical, verbal, relational) and specific rule-breaking behaviours (e.g., vandalism, gang membership, stealing). In this respect, much is yet to be understood about specific risks, which could be successfully identified in childhood, for distinct types of ASB into adolescence. Therefore, focusing on ASB and its developmental origins, this thesis is composed of one systematic review and two prospective longitudinal studies. The systematic review summarised the results from 31 prospective longitudinal studies reporting on childhood predictors of distinct ASB trajectories (Study 1). Drawing from a 7-wave national Australian sample, the two prospective longitudinal studies tested age four childhood predictors of boys' and girls' physical aggression (Study 2) and rule-breaking (Study 3) into late adolescence. The predictors tested in Studies 2 and 3 were effortful control deficits and specific features of both negative emotionality (anger, fear, and sadness dysregulation) and callous-unemotional traits (callous-lack of empathic concern for others). In Studies 2 and 3, in addition to direct effects of the tested predictors, possible catalyst effects were also tested, whereby the presence of callous-lack of empathic concern for others at age four was expected to intensify associations of effortful control deficits and anger, fear and sadness dysregulation

with physical aggression and rule-breaking behaviours. Furthermore, the examination of sex differences was a particular focus in both Studies 2 and 3.

The main results of the systematic review (Study 1) suggested that, overall, higher levels of chronic ASB, relative to no/low-stable ASB, can be predicted by a wide range of risk factors measured in childhood. Also, there was no evidence of sex differences in the socio-environmental risks for ASB. Still, a few sex-specificities were found in individual-level risks (i.e., the risks that were identified as significantly related to ASB in one sex, but not the other), as their higher level in childhood was associated with a higher level and more chronic ASB for girls more consistently than for boys.

The main results from Studies 2 and 3 showed that all tested individual-level risks measured at age four were associated with either a higher intercept or with change (i.e., linear or U-shaped pattern of change over time) in aggression (Study 2) and with more frequent rule-breaking (Study 3) into adolescence. In Study 2, the significance of associations did somewhat differ depending on whether physical aggression was reported by mothers or self-reported. Also, callous-lack of empathic concern for others enhanced the positive association of effortful control deficits and anger dysregulation with physical aggression into late adolescence (Study 2). This catalyst effect was not observed for rule-breaking behaviour (Study 3). Notably, in both Studies 2 and 3, there was no evidence of significant sex differences in associations of any of the age four risk factors tested and either physical aggression or rule-breaking behaviours over time. Still, a few specific individual-level risks tested did seem to posit an increased risk for ASB in boys only or girls only.

Two general conclusions can be drawn from these studies. First, although many risk factors were identified across all three studies, the lack of consensus in the literature about the distinct descriptions of ASB development, and the many ways of identifying and measuring risk factors, pose challenges to the consolidation of the findings. Second, risks for

ASB development can be identified in childhood, and risks for physical aggression and rule-breaking into adolescence can be identified as early as age four. However, the degree to which the considerable sex differences in ASB levels could be explained/predicted by socio-environmental and individual-level risks measured in childhood is still not clear. Findings in this thesis can be applied to inform risk assessment and intervention services for young children most at-risk for chronic or elevated ASB into adolescence and adulthood. These include, for instance, upskilling young children's effortful control, anger regulation, and empathic concern for others to mitigate both physical aggression and rule-breaking during adolescence, when the prevalence of ASB increases substantially for both boys and girls. Also, sex-specific prevention initiatives could be useful, mainly concerning the trait of callous-lack of empathic concern for others in girls and effortful control deficits in boys. Still, anger dysregulation is a core risk factor, hence, a relevant specifier of chronic ASB for both sexes. As such, it should be targeted in prevention programs and child- or family-based interventions with both boys and girls. Study limitations and directions for future research are also discussed within each study chapter and also in the final chapter of this thesis.

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STATEMENT OF ETHICAL PROTOCOL

I confirm that this research was exempted of ethical approval by the Griffith Human Research Ethics Committee. The empirical studies in this thesis (Studies 2 and 3) were conducted using seven waves of data from the Longitudinal Study of Australian Children (LSAC). My access to LSAC data was granted by the Australian National Centre for Longitudinal Data under the Griffith University's organizational license. The completion of this thesis was supported by both the Griffith University International Postgraduate Research Scholarship and the Griffith University Postgraduate Research Scholarship.

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14 July 2020

LIST OF OUTPUTS PRODUCED DURING THE PHD CANDIDATURE

PUBLISHED OR PREPARED FOR SUBMISSION

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- **Pariz, J.**, Zimmer-Gembeck, M. J., & Modecki, K. (Manuscript prepared for submission). Childhood predictors of antisocial behaviour trajectories: A review of 10 years of longitudinal research and comparisons of boys to girls.
- **Pariz, J.**, Zimmer-Gembeck, M. J., & Modecki, K. (under review). The roles of Early Effortful Control, Negative Emotionality, and Callous-unemotional Traits in Boys' and Girls' Physical Aggression into Adolescence**.**
- **Pariz, J.**, Zimmer-Gembeck, M. J., & Modecki, K. (under review). The roles of early childhood effortful control deficits, negative emotionality, and callous-unemotional traits in adolescent boys' and girls' rule-breaking behaviour.

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- Presenter**, Pariz, J.** (2018, July). *Trajectories of antisocial behaviour in the Australian population from early childhood to adolescence*. Poster session presented at the meeting of the International Society for the study of Behavioral Development, Gold Coast, Australia.
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- Presenter**, Pariz, J.** (2020, conference cancelled due to COVID-19). *Interactions Between self-regulators and Callousness, Predicting growth in boy's and girl's Physical*

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- Rovinski, S.L.R, Schneider, A., **Pariz, J.,** Santos, A. Z., & Bandeira, D.R. (2018). Rorschach aggression responses in male perpetrators of conjugal violence. *Avaliação Psicológica, 17*(2), 199-204. http://dx.doi.org/10.15689/ap.2018.1702.13998.05
- **Pariz, J.,** Haddade, E., & Machado, W. L. (2016). Convergent and criterion-related validity for brief scale in the Five-Factor Model. *Avaliação Psicológica, 15*(3), 346-351. https://doi.org/10.15689/ap.2016.1503.07

Poster Accepted at International Conference Reporting Outcomes of Analyses that are Not Included in this Thesis

- Presenter**, Pariz, J.** (2020, March - conference postponed to 2021 due to COVID-2019). *Body Image and Anxiety: Body Dysmorphic Symptoms as Compensatory Responses to Appearance Rejection Sensitivity*. Poster accepted for presentation and indicated for a student's award at the meeting of the Society for Research in Adolescence, San Diego, California/USA.

ACKNOWLEDGEMENT OF PUBLICATIONS INCLUDED IN THE THESIS

Included in this thesis are papers in Chapters 4, 5, and 6, which are co-authored with other researchers, my two PhD supervisors. My contribution to each co-authored paper alongside their bibliographic details are outlined at the front of the relevant chapter. Whenever it was the case, appropriate acknowledgements of those who contributed to the research but did not qualify as authors are included in each paper.

None of the three papers, in Chapters 4, 5, and 6, respectively has been accepted for publication yet. The publication status of Study 1 is outlined at the front of Chapter 4.

Studies 2 and 3 have been submitted to peer-review journal four times, but acceptance for publication had been unsuccessful upon this thesis' submission. These two studies have been updated in preparation for re-submission to other journals. Studies 2 and 3 are now under review for publication.

(Signed)_____ (Date) 14/07/2020

Juliane Pariz Teixeira

(Countersigned)_____ (Date) 14/07/2020

Supervisor: Prof Melanie Zimmer-Gembeck

(Countersigned)_____ (Date) 14/07/2020

Supervisor: Dr Kathryn Modecki

CHAPTER 1

Antisocial Behaviour: Definition, Prevalence Rates, and Impact

Antisocial behaviour (ASB) is a heterogeneous set of externalising behaviours encompassing several mental health disorders related to social, emotional, and behavioural problems (Dodge, Coie, & Lynam, 2008; Muratori et al., 2016). Investigations of the distinct forms of ASB in the literature have found two sub-dimensions of ASB: Aggression and rule-breaking behaviour (Burt, 2012; Frick et al., 1993; Niv, Tuvblad, Raine, & Baker, 2013). Aggressive behaviour is physical (e.g., hitting, pushing, slapping, biting, kicking), indirect (e.g., spreading negative rumours about others to harm others' relationships), or verbal (e.g., using offensive words and phrases to threaten others) actions that are enacted with the intentions of causing harm and/or injury to other people and/or objects (Aloia & Solomon, 2016; Crick & Grotpeter, 1995; Niv et al., 2013). Rule-breaking behaviour is a broad pattern of breaking rules that includes property violations, such as theft, vandalism and fire-setting, and status violations, such as truancy, swearing, and running away, encompassing non-aggressive and delinquent behaviours (Niv et al., 2013).

It is important to point out that many terms have been used in the literature to refer to ASB and its two sub-dimensions. As shown in Figure 1.1, in addition to ASB, the terms delinquency (e.g. Evans, Simons, & Simons, 2016), externalizing behaviour (e.g. Hale et al., 2016; Lansford et al., 2011), maladjustment (e.g. Ehrenreich, Beron, & Underwood, 2016), conduct problems (Baskin-Sommers, Waller, Fish, & Hyde, 2015; Fontaine, Mccrory, Boivin, & Moffitt, 2011), and behaviour problems (e.g. Van Meurs, Reef, Verhulst, & Van Der Ende, 2009) have also been used to refer to ASB. For aggressive behaviour, the terms overt behaviour or aggression (Di Giunta et al., 2010), aggressive delinquency (e.g. Sittner & Hautala, 2016), and aggression (e.g. Logan-Greene & Semanchin Jones, 2015) are the most frequent synonyms used in the literature. For rule-breaking behaviour, the terms delinquency

(e.g. Church II et al., 2012), offending behaviour (Dynes, Domoff, Hassan, Tompsett, & Amrhein, 2015; Van Domburgh, Loeber, Bezemer, Stallings, & Stouthamer-Loeber, 2009), conduct problems (e.g. Okado & Bierman, 2015), behaviour problems (Muñoz, Pakalniskiene, & Frick, 2011), non-aggressive conduct problems (Maughan, Pickles, Rowe, Costello, & Angold, 2000), and covert behaviour (Di Giunta et al., 2010; Snyder, Schrepferman, Bullard, McEachern, & Patterson, 2012) have been used as synonyms often.

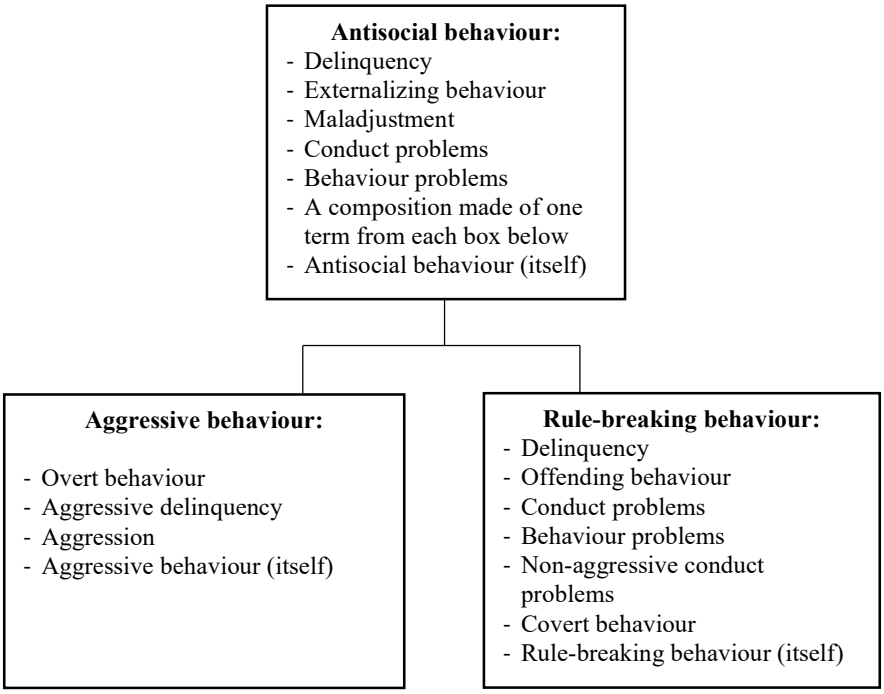

Figure 1.1. Terms used in the scientific literature to refer to antisocial behaviour and its two sub-dimensions, aggressive and rule-breaking behaviours.

Overall, regarding the prevalence of ASB, there is now an emerging consensus that, within the general population of youth, more than half of children and adolescents will *not* engage in levels of ASB that are considered to be problematic at any time of life (Loeber, Capaldi, & Costello, 2013; Odgers et al., 2008). However, it is also known that around 7% to 12% of children (most often defined as age 12 or younger) are expected to engage in

problematic and high levels of ASB (Odgers et al., 2008; Smart et al., 2005). Of these children who exhibit high levels of ASB, approximately 50% will meet the criteria for antisocial personality disorder in adulthood (Fairchild, Van Goozen, Calder, & Goodyer, 2013).

When adolescents are considered (often defined as between age 12 to 19; World Health Organisation, 2014), the prevalence rates of ASB are higher, with some studies reporting that a majority of youth (e.g., prevalence rates vary from 25% to 80% depending on the sample studied) will engage in some form of ASB, many for the first time (Hemphill et al., 2007; Moffitt, 2006; Odgers et al., 2008). The considerable variation, and generally high prevalence rates of ASB among adolescents, has led researchers to hypothesise that ASB could be normative in adolescence (Moffitt, 1993). However, referring to engagement in ASB in adolescence as normative has been a matter of debate. On the one hand, this view of the normalcy or typicality of ASB during adolescence has been challenged by evidence of its potentially serious consequences (Fairchild et al., 2013). For instance, of the whole group of adolescents who exhibit ASB, around 30% will have some contact with the justice system as a result (i.e., being arrested, detained or sentenced; Broidy et al., 2015). Further, about 15% of adolescents who are high in ASB will meet the criteria for antisocial personality disorder in adulthood (Fairchild et al., 2013). On the other hand, fortunately, studies have also suggested that about 50% of adolescents who exhibit ASB will gradually reduce their ASB as they move into adulthood (Cox et al., 2016). In any case, these pieces of evidence suggest that ASB onset in childhood may foreshadow more chronic conduct problems than ASB that onsets in adolescence, but ASB onset in adolescents can also have negative consequences.

In addition to high rates of ASB that start either in childhood or in adolescence, researchers have also reported on prevalence rates and consequences of the continuation of ASB in a subgroup of serious antisocial youth (i.e., young who engage in high levels of

chronic ASB). Overall, the highest rates of ASB occur in around 10% of children and 25% of adolescents (Van Lier, Vitaro, Barker, Koot, & Tremblay, 2009b) for whom the chronicity of their ASB has serious and negative consequences for youth themselves and the social environment in which they live. One example of such consequences is that adolescents who exhibit high levels and chronic ASB are responsible for about half of the criminality rates in any given society (Hemphill, Heerde, Herrenkohl, & Farrington, 2015; Howell, Krisberg, & Jones, 1995; Jolliffe, Farrington, Piquero, MacLeod, & Van de Weijer, 2017; Moffitt, 2006). What is most alarming here is that chronic ASB usually unfolds into a range of negative costs and outcomes for the youth themselves, the society, and the economy globally (Foster, Jones, & the Conduct Problems Prevention Research Group, 2005; Niv et al., 2013; Tremblay, 2010). The main negative outcomes of ASB for youth themselves include low academic performance, peer problems, and continuation in aggression, violence, and drug-use (Cox et al., 2016), and criminality, delinquency, and vandalism (Salzinger, Feldman, Stockhammer, & Hood, 2002) over time. The main negative outcomes for the society include topics which also weight from public health, policymaking, and economic perspectives. For example, providing physical and mental health services for both offender and non-offender antisocial youth and their victims, providing law assistance and justice services, and behavioural intervention programs either preventing or treating ASB (Hemphill et al., 2015; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt, 2006). For the economy, the high costs of dealing with the outcomes of high ASB stand out. For instance, the costs of antisocial youth's involvement with criminality are estimated at around $36 billion per year in Australia (Smith, Jorna, Sweeney, & Fuller, 2014), $60 billion per year in the USA (Kuklinski, Briney, Hawkins, & Catalano, 2012), and £8000 a year per offender youth (the total amount per year in the UK could not be found in the literature) in the UK (National Audit Office, 2011).

Further consideration of prevalence rates and consequences of ASB also must be addressed separately for boys and girls. Overall, consensus exists that both males and females engage in ASB (Jordan, 2011; Odgers et al., 2008; Urben et al., 2017), but, after the age of two, boys are considerably more numerous than girls among children and adolescents who engage in physical aggression and rule-breaking behaviours (Alink et al., 2006, Baillargeon et al., 2007). In addition, the chronicity of high levels of ASB is also more characteristic of males compared to females (Odgers et al., 2008). This evidence is consistent across the literature and justifies the larger number of studies examining ASB in samples of boys to date, whereas less (but a growing volume of) research exists addressing high and chronic levels of ASB in samples of girls (Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009; Odgers et al., 2008; Pepler et al., 2010).

Despite sex differences in the level of ASB, with high and chronic levels of ASB more prevalent among boys than girls, evidence suggests that the severity of girls' ASB does not usually differ from that of boys' ASB (Pepler et al., 2010; Tiet, Wasserman, Loeber, McReynolds, & Miller, 200; Waschbusch, 2002; Wasserman, McReynolds, Ko, Katz, & Carpenter, 2005). Such a similarity in the severity of ASB between boys and girls has led to calls for more research, especially longitudinal studies, focused on girls-only or separate samples of boys and girls. A handful of these studies have been emerging. For instance, a study of at-risk girls from the USA (mean age = 11.5, Sample size = 100) and the UK (mean age = 11.7, Sample size = 145) found 20% of US girls and 30% of UK girls presented with clinical levels of behaviour problems (Harold et al., 2014). In addition, the gap between rates of boys and girls in the population of offender youth seems to have been narrowing over the years, and some authors have claimed that the prevalence of offending among girls has increased 50% in the last couple of decades (Puzzanchera & Adams, 2011). Some have argued that such an increase in the proportion of female offenders could be due to fewer

males engaging in ASB today relative to past years (Lauritsen, Heimer, & Lynch, 2009; Zahn, 2007). Alternatively, researchers have also argued that this could be a result of more people reporting female-offender incidents to the police and/or police officers being more prone to arrest females compared to in the past (Lauritsen, Heimer, & Lynch, 2009; Zahn, 2007). In any case, while the importance of focusing research efforts on understanding ASB in boys seems unquestionable, the increasing rate of ASB among girls is also concerning and warrants research focus to report on more accurate prevalence rates and possible specificities in origins and consequences of ASB for the female population.

In short, the prevalence rates of ASB and the broad spectrum of its negative consequences, which affect not only youth who engage in ASB themselves, but also their families, their local communities, and the whole of society, support the need for additional research on the development of ASB. In particular, further research is needed to identify early socio-environmental experiences and individual-level child characteristics that can account for ASB in childhood, ASB in adolescence, and a trajectory of high or increasing ASB across life periods.

CHAPTER 2

Developmental Research and Theories on Antisocial Behaviour

Developmental science is the study of human development across the lifespan. Within developmental science, the lifespan developmental psychology is a framework that draws attention to the description, explanation, and optimization (i.e., improving, promoting or supporting) of human development from its conception to death (Baltes, Reese, & Virginia, 1980; Lerner, Agans, De Souza, & Gasca, 2013). Relevant contemporary theories within the lifespan developmental psychology framework describe and explain child and adolescent behavioural development through relational developmental systems models (Damon & Lerner, 2008; Lerner et al., 2013). In particular, such models uncover how factors at multiple contextual levels (e.g., neurological, social, family, school, historical) both influence and are influenced by each other to affect changes in behavioural development over time (Damon & Lerner, 2008; Lerner et al., 2013).

Relational developmental systems models are unique because developmental theories addressing any variable within one or another contextual level in isolation would hardly account for the diversity in child and adolescent behavioural development (Cairns & Cairns, 2006). Still, accommodating such diversity in youth's behavioural development is a challenge for any developmental theoretical model because, for each youth, the relationships between their singular life history and personal characteristics, such as temperament, brain structure, and personality, create unique patterns of change in their behavioural development; and these interrelations are also expected to show age-related changes (Damon & Lerner, 2008). Hence, aligned with these views, influential theoretical models of ASB development are those that successfully account for the many risky and protective influences to explain the diversity of patterns of ASB development (e.g., Cairns & Cairns, 2006; Damon & Lerner, 2008; Dodge et al., 2008; Forgatch, Patterson, Degarmo, & Beldavs, 2009; Moffitt, 1993; Sampson & Laub,

2005). Generally, the most influential theoretical models of ASB development have had a dual focus: 1 – describing how changes in ASB occur at an intra-individual (i.e., ASB change within a person) and interindividual level (i.e., between-person diversity in intra-individual change), and 2 – articulating and integrating knowledge from various disciplines (e.g., neurology, physiology, sociology, anthropology, education, criminology) to explain which and how individual-contextual factors may affect developmental changes in ASB. In the following sections, some of the most relevant developmental theoretical models of ASB are briefly cited, but this chapter is primarily designed to introduce the three models that formed the framework for the longitudinal examinations of the origins of ASB in this thesis.

Developmental Theoretical Models of Antisocial Behaviour

Overall, many theories and models of the development of ASB have been proposed. These include, for instance, neuroscience-based models (Ernst & Fudge, 2009); criminological theories (Brownfield & Sorenson, 1993); the age-crime curve (Farrington, 1986); the Oregon delinquency model (Forgatch et al., 2009); the social learning theory of ASB (Akers, 1998; Akers & Jensen, 2006); the life-course theories (Elder, 1998; Sampson & Laub, 2005); the social control/social bonding theory (Gottfredson & Hirschi, 2003; Hirschi & Gottfredson, 2001); the social cognitive theory (Bandura, 1986); the integrated cognitive antisocial potential theory (ICAP), which integrates strain, control, learning, labelling and rational choice theories (Farrington, 2014); the social development model of ASB (Catalano & Hawkins, 1996); the social information processing model (Crick & Dodge, 1996); the interactional theory of offending (Thornberry, 1987); the coercion theory (Patterson, DeBaryshe, & Ramsey, 1989); age-graded theory of informal social control (Sampson & Laub, 1993); the integrated multilayered control theory (Le Blanc, 2017); the temperament-based theory of ASB (DeLisi & Vaughn, 2014); the developmental psychopathology model of ASB (Frick & Viding, 2009); and the developmental taxonomic theory (Moffitt, 1993).

Propositions integral to the last three developmental theories of ASB mentioned above (DeLisi & Vaughn, 2014; Frick & Viding, 2009; Moffitt, 1993) provided an overarching theoretical framework that underpinned the examination of the developmental origins of boys' and girls' ASB in this thesis. In particular, the developmental taxonomic theory proposed by Moffitt (1993) provided a solid background for a detailed description of both the within and between-person patterns of change in ASB and for a preliminary explanatory model of both the origins and chronicity of ASB over time. Complimentarily, propositions from the temperament-based theory of ASB (DeLisi & Vaughn, 2014) and from the developmental psychopathology model of ASB focused on callous-unemotional traits (CU traits; Frick & Viding, 2009) were brought together to extend upon the developmental taxonomic theory. This integration of the three theories helped to address two gaps in the developmental taxonomic theory of the development of ASB and sex-specificities in risks for boys' and girls' ASB. In particular, the temperament-based theory of ASB and the developmental psychopathology model of ASB guided investigation of the role of specific individual characteristics that could be identified in childhood as significant predictors of boys' and girls' ASB in the present thesis. Notably, each of these three developmental theoretical models of ASB also integrates knowledge from various disciplines, including neuropsychology, developmental psychology, developmental psychopathology, and developmental criminology.

The Developmental Taxonomic Theory

The developmental taxonomy of ASB proposed by Moffitt (1993, 2006) is often acknowledged as the most influential theoretical model of ASB development. In both her dual taxonomic theory (1993) and the revised taxonomy (2006), Moffitt describes that the within-person diversity in patterns of change in ASB from childhood to adolescence could be managed in scientific research by grouping individuals who follow similar trajectories into

distinct groups. In this description, the members of each distinct group (i.e., each ASB trajectory) would share an ASB developmental pathway that differs substantively from that of other groups (i.e., other ASB trajectories). Notably, because this methodological approach has been used to identify mutually exclusive ASB trajectories (i.e., distinct ASB groups), the developmental taxonomy also allows for the study of between-person differences in the predictors or correlates of ASB developmental patterns. In this respect, Moffitt's developmental taxonomic theory is notable for the description of three trajectory groups of ASB: The life-course-persistent (LCP), defined as few, persistent and pathological antisocial youth with high levels of ASB that onsets during childhood and continues over time; the adolescent-limited (AL), defined as low or absent ASB during childhood that increases considerably or emerges during early-to-middle adolescence and declines in early adulthood, and the childhood-limited (CL), defined as ASB restricted to childhood years.

Importantly, in her taxonomic theory, Moffitt (1993, 2006) also theorised about the socio-environmental and neuropsychological origins of the LCP, the AL, and the CL ASB trajectory groups. More specifically, because Moffit considered LCP, AL, and CL as distinct categories of ASB (i.e., distinct trajectory groups), she also proposed that each of such categories would have distinct and specific origins. For instance, the LCP trajectory would be primarily determined by childhood neuropsychological characteristics such as cognitive deficits (Moffitt & Caspi, 2001; Odgers et al., 2008) and hyperactivity and impulsivity characteristics (Fontaine et al., 2011; Moffitt & Caspi, 2001; Odgers et al., 2008). Additionally, social-environmental factors, such as low SES, poverty, harsh and inconsistent parenting discipline, and family conflict (Fontaine et al., 2011; Moffitt & Caspi, 2001; Odgers et al., 2008), and poor social relationships (Moffitt, 2006) would be risks for the LCP trajectory. In contrast, the AL trajectory would originate primarily – but not only - from a normative 'maturity gap' in youth development that resulted from the asymmetry between

adolescents' advanced biological maturation and their limited access to adult privileges and responsibilities (Moffitt, 1993; 2006). That is, AL would not necessarily be indicative of future problem behaviour, but it would be found among a large proportion of adolescents because this population used ASB to pursue anticipation of adult privileges. Because AL ASB is a behaviour aligned with pursuing early adult privilege, it would also be the case that AL antisocial youth would decrease their ASB levels as they approach adulthood and can take on legitimate adult roles and autonomy. Regarding CL, Moffitt (2006) posited that, despite its apparent recovering characteristic compared to both LCP and AL trajectories, the CL trajectory does not always signify complete desistance from early ASB problems. Instead, Moffit also considered that CL might have its origins in the same neuropsychological determinants as the LCP trajectory (Moffitt, 2006; Raine et al., 2005). Further similarities between CL and LCP that support such an assumption are also reported by Moffitt. For instance, evidence shows that, across adolescence, CL boys are at an increased risk to intermittently engage in crimes or present internalizing disorders such as social phobias or depression (Moffitt, Caspi, Rutter, & Silva, 2001; Moffitt, 2006).

Overall, Moffitt's descriptions of LCP, AL and CL patterns, and the qualitative differences in their origins (i.e., specific predictors for each trajectory) suggest that ASB present since childhood would be a stronger prognostic indicator of poor adjustment into adolescence and beyond - in relation to ASB starting in adolescence. Therefore, there may not be a 'recovery' pattern for ASB when it begins in early childhood, even when ASB itself seems limited to the childhood years. Importantly, regarding sex-specificities in the origins of the LCP, AL, and CL trajectories, Moffitt also has posited that her developmental taxonomic theory would apply to description and explanation of the development of ASB of both boys and girls alike (Moffitt & Caspi, 2001).

The origins of ASB. According to the developmental taxonomic theory, high levels of and chronic ASB that start in childhood originate from children's early appearing neuropsychological deficits (i.e., also referred to as individual-level risks) and adverse socio-environmental circumstances (i.e., also referred to as external and familial level risks) (Fairchild et al., 2013; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt et al., 2001; Raine et al., 2005). To date, research findings have supported these hypotheses. For instance, evidence has shown that external and family risks for ASB include parents' lower educational level, no occupation and low income, greater family adversity (i.e., single parenthood; father's ASB in adolescence, neighbourhood adversity/disadvantage, and mother's mental health issues), and parental practices of low warmth and higher hostility, neighbourhood disadvantage, peer exclusion and delinquency (Alink et al., 2006; Baillargeon et al., 2007; Campbell, Spieker, Vandergrift, Belsky, & Burchinal, 2010; Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006; Givens & Reid, 2019; Gutman, Joshi, Parsonage, & Schoon, 2018; Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008, 2013; Kim & Um, 2018; Martino, Ellickson, Klein, McCaffrey, & Edelen, 2008; Pitzer, Esser, Schmidt, & Laucht, 2010; Teymoori et al., 2018). However, considerably more studies exist that have examined adverse socio-environmental circumstances as risks for ASB, compared to individual-level risks. This imbalance in research attention is such that researchers have argued that individual-level characteristics in the origin of ASB still are vastly under-investigated and more studies examining them are urgently needed (DeLisi, 2011; DeLisi & Vaughn, 2014; Denson, DeWall, & Finkel, 2012; Givens & Reid, 2019; Hay, 2017; Hay et al., 2014; Nigg, 2017). Also, the particular relevance of new studies focusing on individual-level risks to further explain the origins of ASB and advance both theory and empirical research is supported by Moffitt (Moffitt et al., 2011; Moffitt, 2006). Attending to such calls for more research, a two-step approach to the examination of the origins of boys' and girls' ASB was followed in this

thesis. Firstly, a focus was placed on describing both socio-environmental and individual-level childhood predictors of ASB trajectories by conducting a systematic review of the literature (Study 1). Second, in two prospective longitudinal studies drawing on a large Australian sample (Growing Up in Australia: The Longitudinal Study of Australian Children), the focus was narrowed to place a lens on individual-level risks that could be identified in early childhood to predict developmental patterns (i.e., intraindividual growth curves) of aggression from early childhood through adolescence (Study 2) and frequency of rule-breaking behaviour measured three times during adolescence (Study 3).

Two gaps emerging from research drawing upon the developmental taxonomic theory. Despite the unquestionable relevance of the developmental taxonomic theory to the study of ASB development, some of its assumptions have not been fully supported by recent research results, which could indicate two gaps in this theory. One gap concerns Moffitt's description of developmental patterns of ASB. The other gap concerns the possible sex-specificities in explanatory predictors of ASB that had originally been posited as inexistent in the developmental taxonomy. To both build upon and better inform current theory and research aiming at the optimization of youth's development, these two gaps are discussed next. Such discussion will underpin the rationale and overview of the three studies in the present PhD thesis, which will be summarised later, in Chapter 3.

The first gap concerns Moffitt's descriptions of the LCP, the AL, and the CL as mutually exclusive categories of ASB development (i.e., trajectories) and that AL is normative. Regarding ASB categories, when researchers have applied latent variable factor analysis in samples of LCP and AL ASB, a one-dimensional structure across distinct ASB trajectories has been found consistently (Raine et al., 2005; Walters, 2011; Walters & Ruscio, 2013). This evidence has been described as presenting a challenge to the proposals within the developmental taxonomic theory because, if ASB trajectories are not distinct categories, but

variations within the same continuum, the onset of ASB at a particular age (in childhood or in adolescence) may not be the best criteria to account for both within and between-person diversity in ASB development. Regarding AL being normative, studies of ASB trajectories that have more extended follow-up have found that ASB starting in adolescence may also persist into adulthood, much like a later-starting LCP trajectory (Fairchild et al., 2013, Roisman et al., 2010). Moreover, there is evidence that individuals classified as LCP or AL may not always differ in outcomes of mental and physical impairments and/or their history of conviction resulting from their ASB (Burt, Donnellan, Iacono, & McGue, 2011; Fairchild et al., 2013; Odgers et al., 2007, Roisman et al., 2010). The challenge posited to the developmental taxonomic theory by these pieces of evidence regarding AL ASB is that, if ASB starting in adolescence is not always limited to the adolescent years and have similar negative consequences compared to LCP, then AL may not be normative to youth's development. Therefore, its origins would require a new explanation by the theory.

All such challenges together suggest that, if ASB is better considered as a continuum (not categories) of the same trait, regardless of its age-of-onset, and no ASB trajectory can be reliably described as developmentally normative in adolescence, then differences in the explanatory model of the origins of LCP, AL, and CL may not be qualitative (i.e., specific factors predicting specific ASB trajectory groups), as Moffit (1993, 2006) had firstly posited. Instead, any ASB trajectory may originate from similar socio-environmental and neuropsychological risk factors, but differences in the number (i.e., quantitative differences) of such risks in childhood or adolescence, would suit better to explain high levels of ASB starting in one or another developmental stage. Knowledge of such challenges and similar hypotheses have been increasing in the literature as they have been highlighted in recent reviews (Fairchild et al., 2013; Farrington, Gaffney, & Ttofi, 2017; Jolliffe, Farrington, Piquero, Loeber, & Hill, 2017).

The second gap concerns Moffitt's explanations that the same socio-environmental and neuropsychological characteristics would explain ASB origins in boys and girls alike (Moffitt & Caspi, 2001). Overall, evidence suggests that LCP, AL, and CL ASB trajectories, as Moffitt has proposed them in the developmental taxonomic theory, may have a better fit for boys with ASB, in relation to girls with ASB for two reasons. First, because boys are considerably more likely than girls to first engage in ASB in childhood (Fairchild et al., 2013). Second, compared to girls, boys engage in a greater variety of ASB at any developmental stage (Jordan, 2011), which may result in greater diversity; therefore resulting in the LCP and AL ASB trajectory group distinction as a better fit for boys than girls. In this respect, evidence also suggests that girls with more serious ASB would show one specific LCP trajectory, namely 'adolescent-delayed-onset pathway' (Fairchild et al., 2013; Fontaine et al., 2009; Silverthorn & Frick, 1999). Taken together, these findings represent a challenge to the taxonomic theory because Moffitt's definitions of LCP and AL seem not to apply as readily to ASB development in girls as they do to boys. Such a challenge also makes room for hypothesising possible sex-specificities in the origins of boys' and girls' ASB development. Overall, prospective longitudinal research assessing sex-specific socio-environmental and individual-level risk factors to the development of ASB are scarce in the literature (Fairchild et al., 2013). Therefore, much is yet to be understood about possible sex-specificities in the origins of the well-known diversity of boys' and girls' patterns of change in ASB over time. Potentially, this understanding could open new avenues for assessment and intervention services on girls' ASB.

In this thesis, while keeping these two gaps in mind, three studies were conducted. The first was a systematic review of longitudinal studies that had prospectively examined predictors of ASB trajectory groups (Study 1). The remaining two studies drew upon a 7-wave national Australian sample to test early childhood individual-level factors as predictors

of ASB development (Studies 2 and 3). These two empirical studies differed in the type of ASB studied (aggression in the first study, and rule-breaking in the second study), in the time frame covered (aggression from childhood into adolescence in the first study, rule-breaking across adolescence in the second study), in the length of follow-up data available, and in the methodological approach used (latent variable growth curve modelling in the first study, and multiple/hierarchical regressions in the second study).

Origins of ASB: Developmental Theories of Early Individual-level Characteristics

Relevant developmental theories of the role of early occurring (i.e., childhood) individual-level characteristics in the development of ASB usually merge evidence from various disciplines (e.g., developmental psychology, psychopathology, criminology, as well as evolutionary and social psychology) to propose an explanatory model of interindividual differences in intraindividual change in ASB over childhood, adolescence, or adulthood. Most challenging to such theories, however, is to identify specifically what and how individual characteristics that are present in early childhood may present an increased risk for boys and girls to engage and persist in ASB over time. For example, Moffitt had proposed in her developmental taxonomy that the same deficits in neuropsychological characteristics/deficits would increase risk of higher levels of and chronic ASB for both boys and girls (Moffitt & Caspi, 2001; Moffitt et al., 2001). Still, less clarity exists in Moffitt's description of what precisely such deficits and/or characteristics would be, and how they could contribute to explaining the onset and continuation of high levels of ASB over time (Fairchild et al., 2013; Raine et al., 2005; Walters, 2011; Walters & Ruscio, 2013). Fortunately, two other theories complement the developmental taxonomy theory by pointing to deficits in specific self-regulatory processes, defined here as deliberate, dynamic, and adaptive modulation of behaviour, emotions, and cognition (Nigg, 2017), that could be relevant risks for ASB. These are both the temperament-based theory of ASB (DeLisi &

Vaughn, 2014) and the developmental psychopathology theory of ASB (Frick & Viding, 2009).

The temperament-based theory of ASB posits that effortful control deficits and negative emotionality characteristics are the most relevant self-regulatory processes that can be assessed in childhood to predict high levels of ASB over time (DeLisi & Vaughn, 2014; Eme, 2018). Effortful control is defined in this theory as one's dispositional (as opposed to automatic) use of high levels of self-regulation to control emotions and behaviours to achieve optimum levels of social competence. Negative emotionality is a pattern of experiencing the environment negatively, through emotion dysregulation of anger, fear, and sadness. According to this theory, neurological, social, and developmental aspects prompt a normative shift in self-regulation that occurs at ages three to four, when effortful control skills emerge and children acquire a level of executive function skills (i.e., attentional, cognitive, and inhibitory control) that allow them to subordinate their dominant impulses (i.e., regulate attention, emotions, and behaviour) in favour of socially appropriate ones (DeLisi & Vaughn, 2014). Such a view suggests that, when they occur by around the age of five, effortful control deficits and negative emotionality characteristics are likely to increase the risk of ASB in later childhood, adolescence and adulthood. Although the temperament-based theory of ASB is fairly new, its ideas also have been expanded to discuss how sex differences in the role of early deficits in effortful control and negative emotionality may contribute to massive sex differences in ASB development (Eme, 2018). These include, for instance, consideration of biological mechanisms shaping temperament and favouring naturally higher levels of fear, which may act as a shield against ASB in girls (Eme, 2018). In contrast, higher physical activity levels and deficits in effortful control would be linked with increased ASB in boys (Eme, 2018). All these ideas underpinned the hypotheses in Studies 2 and 3 of this thesis (see Chapters 5 and 6). More specifically, effortful control deficits and features of negative

emotionality (i.e., anger, fear, and sadness dysregulation) at age four were investigated in Studies 2 and 3 with the aim of understanding whether these were risks for boys' and girls' ASB into adolescence.

Complimentarily, to both the developmental taxonomic theory and the temperament-based theory of ASB, Frick and his colleagues (Frick, Ray, Thornton, & Kahn, 2014a; Frick & Viding, 2009) also propose an influential theoretical model that has explained the early onset, high level and chronicity of ASB in a particular group of severe antisocial youth through the lens of developmental psychopathology. According to such model, the presence of callous-unemotional traits since childhood affects the normative development of the child's brain resulting in an increased risk of more severe and chronic levels of ASB over time (Frick, Ray, Thornton, & Kahn, 2014a; Frick & Viding, 2009). In this literature, callous-unemotional traits have been defined as a lack of guilt, carelessness, shallow emotions, and absence of empathy. Research shows that the presence of callous-unemotional traits flags a subgroup of children who grow up at greater risk for conduct or oppositional defiant disorders in childhood and adolescence and antisocial personality disorder in adulthood (see Frick, Ray, Thornton, & Kahn, 2014a, and Frick & White, 2008, for reviews). In addition, the presence of callous-unemotional traits has been included as criteria for diagnosing conduct disorder within the DSM-5 by specifying the child's lack of prosocial emotions (American Psychiatric Association, 2013). This followed consistent evidence that callous-unemotional traits predict ASB even after accounting for conduct disorder symptoms themselves (Frick, Ray, Thornton, & Kahn, 2014b). Such evidence is complementary to both the developmental taxonomic theory and the temperament-based theory of ASB because they further explain the diversity in the development of ASB as a function of the presence of callous-unemotional traits (Frick, 2012). Despite the relevance of callous-unemotional traits as an individual-level risk for ASB development, only a few prospective studies were found examining its

longitudinal associations in childhood with ASB development into adolescence separately for boys and girls (Hawes, Byrd, Waller, Lynam, & Pardini, 2017; Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011; Waller et al., 2016). Most importantly, no study could be located examining sex-specificities in the role of callous-unemotional traits alongside effortful control deficits and features of negative emotionality in early childhood on boys' and girls' ASB into late adolescence. Therefore, these were the aims of Studies 2 and 3 of this thesis (see Chapters 5 and 6). In particular, Studies 2 and 3 were designed to advance knowledge on the roles of effortful control deficits and features of both negative emotionality and callous-unemotional traits in the roots of higher and chronic levels of ASB in the first two decades of life.

CHAPTER 3

General Aims and Overview of Studies in the Present Thesis

Drawing together theory and research presented and discussed in Chapters 1 and 2, the overarching purpose of the three studies in chapters 4, 5, and 6 of this doctoral thesis was to review what is known about childhood correlates of ASB trajectory profiles (Study 1) and to investigate early childhood individual-level correlates of boys' and girls' ASB development into adolescence (Studies 2 and 3). More specifically, Study 1 (Chapter 4) was a systematic review of prospective longitudinal studies that reported on childhood predictors of distinct ASB trajectories (referred to as ASB profile groups). Overall, the aims of Study 1 were to (a) summarise the findings of studies that had examined socio-environmental and individual-level childhood predictors of trajectories of ASB, as they have been reported for samples of boys and girls together and separately, and (b) draw attention to the similarities and differences in findings across studies, with a focus on variability in age and length of prospective follow-up across studies, differences in the sets of ASB trajectory profiles identified across studies, and sex differences in study findings.

Next, Studies 2 and 3 are presented in Chapter 5 and 6. These studies further addressed gaps in theory and research described in Chapter 2 and also expanded on Study 1 (Chapter 4) findings by attending to a particular need for additional, prospective longitudinal studies of early childhood individual-level variables as predictors of the development of specific types of ASB into late adolescence in samples of boys and girls separately. Studies 2 (Chapter 5) and 3 (Chapter 6) are prospective longitudinal examinations of early childhood predictors of boys' and girls' aggressive (Study 2) and rule-breaking (Study 3) behaviours into late adolescence. Data analysed in Studies 2 and 3 was drawn from a large, 7-wave national sample of Australian youth. The two sub-dimensions of ASB examined were mother and child self-reported physical aggression from childhood into late adolescence (Study 2)

and self-reported rule-breaking behaviour from early to late adolescence (Study 3). Regarding the investigation of early childhood predictors in Studies 2 and 3, this focused on the direct and interactive effects of caregiver-reported effortful control deficits and features of negative emotionality (emotion dysregulation of anger, fear, and sadness) with a specific trait of callous-unemotional (callous-lack of empathic concern for others), all measured at age four. Thus, Studies 2 and 3 were novel in examining a set of hypotheses, which were derived from jointly considering the developmental taxonomic theory, the temperament-based theory of ASB, and the developmental psychopathology model of callous-unemotional traits as a risk for ASB. In addition, sex specificities in the predictive effects of all the tested early childhood predictors on the development of aggression and rule-breaking behaviours were also examined in these two studies.

Finally, following Chapters 4, 5, and 6, Chapter 7 presents a general discussion of the main in this thesis and their implication for theory, assessment, and intervention on ASB development. More specifically, Chapter 7 begins with a brief restatement of this thesis' general aims followed by a summary of the main findings in chapters 4, 5, and 6 and how they addressed such aims. Next, two main conclusions that were drawn from all findings are discussed in more detail. Finally, the implications of these two conclusions are discussed in the context of the limitations of the three studies included in this thesis. Although specific directions for future research are presented within each study following their particular scope, additional future research needs are also proposed in Chapter 7.

CHAPTER 4

Study 1

Childhood Predictors of Antisocial Behaviour Trajectories: A Review of 10 Years of Longitudinal Research and Comparisons of Boys to Girls

Statement of Contribution to Co-authored Paper

This chapter includes a full version of a co-authored manuscript of a systematic review. It consists of a version of the review that had been prepared for submission during the PhD candidature. However, because the search of studies occurred in April/2018, this version of the manuscript will be updated to include years 2019 and 2020 in the search of studies before its submission to a peer-reviewed journal. The bibliographic details of this paper, including all authors, are:

Pariz, J., Zimmer-Gembeck, M. J., & Modecki, K. (Manuscript prepared for submission). Childhood predictors of antisocial behaviour trajectories: A review of 10 years of longitudinal research and comparisons of boys to girls.

My contribution involved: Review of literature, lead and completion of the search of studies and data extraction, lead and completion of data summaries and tabulation, lead in writing of the paper, corresponding author of the paper.

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Supervisor: Prof Melanie Zimmer-Gembeck

Supervisor: Dr Kathryn Modecki

Abstract

The main goal of this study was to systematically review ten years (2008-2018) of prospective longitudinal studies that investigated childhood predictors of the onset and continuation patterns of antisocial behaviour (ASB) over the lifespan (i.e., trajectories). The ASB trajectories identified across includes studies were no/low-stable, NL; child-limited, CL; adolescent-limited, AL; life-course-persistent, LCP; adolescent-onset, AO; high-stable, HS; antisocial-desisters, AD; medium-level, ML. Overall, 31 prospective studies were located that had used group-based analysis to identify ASB trajectories plus examined their possible childhood predictors. Predictors were summarised according to type (static demographics, socio-environmental, or individual characteristics) and sample sex (boys and girls together or separated). Also, quantitative and qualitative differences in predictors across both distinct ASB trajectories and sample sex were described. Main results suggest that trajectories of higher levels and more chronic ASB (e.g., LCP, HS) are predicted by a greater number of risks, in relation to all other ASB trajectories, regardless of age at ASB onset or time frame studied. There was some evidence that specific early individual-level, but not socio-environmental level, risks were associated with LCP and HS trajectories. Regarding sex-specificities, both socio-environmental and individual-level risks differentiated ASB trajectories for both sexes, but individual-level factors were slightly more numerous and had stronger associations with more chronic or higher levels of ASB in girls. Findings for protective factors by sex were also described. Risks for ASB may be prospectively identified in childhood for both sexes, but such identification of risks may be equally important for assessment and intervention with girls. The study of individual-level risks for girls' ASB is scarce and requires further investigation.

Keywords: Antisocial behaviour, trajectory, longitudinal, childhood predictors, risk factors.

Childhood Predictors of Antisocial Behaviour Trajectories: A Review of 10 Years of Longitudinal Research and Comparisons of Boys to Girls

Developmental trajectories of antisocial behaviour (ASB) are generally defined by the age at onset, the pattern of change (e.g., decline, increase), and the severity (e.g., high/low levels) of aggressive, rule-breaking (also referred to as delinquency), or offending behaviours over all or a part of the life-course (Burt, 2012; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt et al., 2001; Niv et al., 2013). The negative consequences of a high and stable ASB trajectory over time include increased criminality rates and high personal and financial costs to antisocial youth and their community within the law, prevention, and treatment/rehabilitation spheres. Such negative consequences justify the importance of ASB prevention and intervention programs (Assink et al., 2015; Cox et al., 2016; Jolliffe & Farrington, 2007; Piquero, Jennings, Farrington, Diamond, & Gonzalez, 2016). However, the efficacy of these treatments and programs rely on correctly identifying at-risk youth as early in life as possible. Such identification is informed by evidence from studies of the factors that bring greater risk or protection. In this respect, research has been crucial that (1) describes and compares different trajectory profiles of ASB and (2) explains membership in ASB trajectory profile groups associated with earlier risk and protective factors across multiple levels, especially within socio-environmental and individual characteristics. Thus, research describing and explaining ASB development helps researchers, practitioners, and policymakers to act towards optimizing youth development in two ways: (a) by identifying children and adolescents most at risk for high and chronic levels of ASB and (b) by targeting those risk and protective factors that will have the greatest impact on reducing and even preventing ASB in at-risk populations of youth.

Fortunately, many prospective longitudinal studies exist to inform optimization of youth development by describing the trajectories of the different types of ASB, while also

providing evidence of possible predictors of patterns of ASB change over time. However, these studies differ greatly in the age ranges covered (e.g., sample age at baseline and at last follow up of data collection) and in the types of ASB examined (e.g., aggression, rule-breaking, offending). Such differences result in a broad and diverse body of evidence. Therefore, there is a need for continued systematic reviews to organize longitudinal studies of ASB and deliver useful information to researchers, practitioners, and anyone dealing with ASB in youth's population (Farrington et al., 2017). Answering this call, this novel systematic review of prospective longitudinal studies extends on current research in two specific ways. First, by describing sex-specific childhood predictors of membership in distinct ASB trajectories (also referred to here as trajectory profiles) reported in prospective longitudinal studies. Second, by comparing and reporting on similarities and differences in childhood predictors across distinct ASB trajectories and child sex.

Describing ASB Trajectory Profiles and Identifying them in Longitudinal Studies

There has been a long history of describing and identifying developmental patterns of ASB in both developmental psychopathology and criminology research. In these fields, Moffitt's developmental taxonomy (1993; 2006) has been one of the most widely applied theories to describe distinct trajectories of ASB. Three ASB trajectories were described in both Moffitt's original dual-taxonomy (Moffitt, 1993) and revised taxonomic theory (Moffitt, 2006), namely childhood-limited (CL; ASB restricted to childhood years), life-course-persistent (LCP; ASB that onsets in either childhood or early adolescence and remains high over time, ideally with evidence that it continues into adulthood), and adolescent-limited (AL; ASB restricted to the adolescent years[1]). There have also been expansions to identify additional ASB trajectories in the literature, such as an ASB profile starting in adulthood,

[1] As stated in a previous review (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017), there is consensus in the literature that adolescence extends into the age of 20. However, Moffitt has also suggested that a stretch to age 25 would also be adequate given young people are living at their family house for longer and getting married later.

namely adult-onset (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). However, this profile has been described in Moffitt's taxonomy as also having a childhood-onset and the same socio-environmental predictors of the LCP profile (Beckley et al., 2016).

Many longitudinal studies have supported Moffitt's theoretical descriptions of the distinct ASB trajectories. Such studies have usually used group-based trajectory modelling (GBTM) techniques (Brame, Paternoster, & Piquero, 2012; Nagin, 2014) to estimate the intraindividual change in ASB over time alongside identifying groups of individuals within the same sample who are similar in their patterns of change in ASB but differ from others' patterns. The use of GBTM allows for the possibility of both single and multiple-group model structures, with individuals classified in groups according to the maximum probability (e.g., > .70) that they belong to one or another developmental trajectory (Nagin, 2014, 2015). Longitudinal research that used some form of GBTM to report on prevalence rates of youth in distinct ASB trajectories is summarised in previous reviews (Jennings & Reingle, 2012; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt, 2006). Overall, these studies revealed that 3% to 17% (depending on the study sample) of youth in any population had been classified as LCP (Jennings & Reingle, 2012; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017), reflecting their more frequent and severe ASB during the time of follow-up. Notably, although LCP is the minority of the youth population, it is estimated that youth in the LCP group may be responsible for 50% of crimes in any society (Moffitt, 2006).

Regarding AL, literature presents no consensus about its prevalence rates. Moffitt had initially posited that this would be a normative ASB trajectory, therefore justifying that AL ASB would be expected in youth; hence observed in more than 50% of such population (Moffitt, 2006). However, recent studies revealed prevalence rates of AL ranging from 2% to 80% depending on the study (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). Nevertheless, consensus also exists about a large group of youth who follow a consistent

no/low ASB trajectory profile, and account for 30% to 98% of such population in any sample (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt, 2006).

Importantly, a review of studies using GBTM points to similar prevalence rates between LCP and a group of youth who will account for a disproportionately higher number of ASB in any cohort - chronic ASB (Jolliffe et al., 2017). That is, although both LCP and chronic ASB groups may overlap (i.e., the same youth might be in both groups), they are not the same. The chronic ASB group is composed by youth who have higher levels of ASB relative to others within any cohort, but many of they may not have persisted in such higher levels of ASB over time had a longer period of follow up been completed, which is a requirement for being in the LCP group. Hence, although youth in the group of chronic ASB may not be in the LCP, youth in the LCP group are likely to be in the chronic ASB group when using GBTM approaches (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Piquero, Sullivan, & Farrington, 2010).

Notably, research has also reported that the prevalence of boys and girls across all ASB trajectories differs for each ASB profile grouping. For example, studies consistently show a preponderance of males relative to females in the LCP trajectory profile (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt et al., 2001). This evidence has increased the relevance of the sample-sex in studies using GBTM by establishing the male sex as the primary risk characteristic for the development of ASB. Therefore, justifying the priority given in the literature to study ASB in the male population. However, other study findings have suggested that, despite lower prevalence rates of girls in LCP ASB, a female-specific LCP trajectory profile may exist (Fontaine et al., 2009; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). Thus, a growing body of literature now considers ASB trajectories in boys and girls separated and highlights the importance of considering sex-specific risk and protective factors that may be common or unique risks to males and females high levels of

chronic ASB (Fontaine et al., 2009; Jennings & Reingle, 2012).

Identifying Risk and Protective Factors Associated with ASB Trajectory Profiles

According to Moffitt's (2006) taxonomic theory, characteristics that could be useful for identifying individuals classified into different ASB trajectory profiles (e.g., CL, AL or LCP) are found across multiple socio-environmental (e.g., family, school, neighbourhood factors) and neuropsychological (e.g., individual-level conditions such as IQ and self-control) factors. In addition, Moffitt also posited that the same such characteristics would be equally relevant to both boys and girls (Moffitt et al., 2001). What is most important here is a great deal of longitudinal studies that have followed to test Moffitt's hypotheses. Fortunately, six reviews could be located that summarised what is now known about the predictive correlates of distinct ASB trajectories in the literature. Three of these reviews addressed ASB trajectories as proposed by Moffitt in her taxonomy (Assink et al., 2015; Fairchild et al., 2013; Jolliffe, Farrington, Piquero, Loeber, et al., 2017). One review addressed factors associated with the onset and continuation of criminal careers (Farrington, 2015). Another review addressed factors associated with patterns of offending and violent behaviour in high and low at-risk samples (Ttofi, Farrington, Piquero, & DeLisi, 2016). Finally, the sixth review was a systematic review of quantitative reviews on predictors of ASB trajectory profiles (Farrington et al., 2017).

Overall, in the first review of 61 studies (Fairchild et al., 2013), the researchers confirmed Moffitt's ideas about the relevance of both socio-environmental and individual-level risks for ASB trajectory profiles. What was particularly relevant was the finding that the AL profile showed onset in adolescents, but ASB was rarely limited to adolescence. Further, individuals in the AL profile did not differ in the negative and chronic prognosis when compared to those in the LCP profile. Consequently, individuals who were placed in the AL profile group could have some of the same neuropsychological and environmental risks as

those in the LCP profile groups. The authors concluded that a revision of Moffitt's taxonomy was in order by suggesting the idea that the AL and LCP profile groups differed quantitatively (the number of risks) rather than qualitatively (specific risks to specific ASB trajectories). In addition, conclusions also suggested that adverse socio-environmental risks in childhood could moderate the associations between individual-level risks and ASB trajectories. The authors argued that future longitudinal studies focusing on risk factors for ASB should include more girls. Specifically, they should examine girls separated from boys to identify why childhood-onset of ASB appears to be rare in girls, whereas a considerable reduction in the sex-ratio in ASB is observed in adolescence.

In the second review of ASB trajectory studies (Assink et al., 2015), researchers conducted a meta-analysis of the effects of different risk domains (i.e., broader categories grouping risk factors by similarity) for LCP relative to AL from 55 studies. The results showed that risk factors within both physical health and neighbourhood domains did not differ between LCP and AL. However, more numerous risks were found for the LCP relative to the AL across the domains of family, neurocognitive, emotion/behavioural, school/employment, and attitudes. Still, effects were generally small, except for a larger effect of childhood peer relationship. Overall, findings in this review corroborated previous findings (Fairchild et al., 2013) that the differences in risk domains between LCP and AL may be quantitative rather than qualitative, but that peers may be a particular source of risk. In particular, the authors concluded that the assessment of risks to identify youth in the LCP ASB trajectory should differ according to sex, age, and sociocultural background, as well as focus primarily on previous ASB and both emotional/behavioural (e.g., being depressed, having emotional problems or ADHD diagnosis) and school/employment (e.g., poor academic achievement, lack of interest in school, unstable job record) problems.

A third review (Jolliffe, Farrington, Piquero, Loeber, et al., 2017) examined and

compared early risk factors for LCP, AL, and late-onset (LO) trajectories offending in seven prospective longitudinal studies of community samples with a sample size of at least 300 youth. Overall, the results showed little evidence supporting the notion that specific risk factors predicted each trajectory (i.e., qualitative differences in predictors) profile of offending, even after adjusting the definitions of the trajectories to account for longer criminal career duration. Instead, the findings supported that a greater number of risks (i.e., quantitative differences in predictors) that also had the strongest effects (poor housing, having a convicted parent, child abuse, being old for the school-grade, low academic achievement, lack of guilt, hyperactivity) differentiated between LCP and AL and between AL and LO. In particular, LCP had a greater number of risks that were also of a greater magnitude than AL, and the same was observed for AL relative to LO. The authors concluded that much more research on risk factors of specific offending types are needed for a reliable examination of the extent to which different trajectories of criminal careers can be predicted.

A fourth review (Farrington, 2015) was not based on Moffit's taxonomic theory but reported on risk factors for the onset and continuation of criminal careers in 30 prospective longitudinal studies of community samples with a sample size of at least 300 youth. Major findings in this review suggest that variables within the socio-environmental level are the most critical risks for criminal career continuation or an increase in the likelihood of convictions. Such variables included poor child-rearing, poor parental supervision, younger mothers, child abuse, parental conflict, disrupted families, low socioeconomic status, delinquent peers, and disadvantaged neighbourhoods. Within the individual-level, low self-control (or high impulsivity), low intelligence, and low school attainment are the most critical risks. However, the author concluded that more prospective longitudinal studies starting from childhood and examining the within-individual change in delinquent behaviour over time are

still needed. Such studies may provide more convincing conclusions on causal factors of onset and continuation of criminal careers.

Protective factors for high and chronic ASB were addressed in two reviews. In the first review (Ttofi et al., 2016), researchers focused on individuals' low ASB in 11 prospective longitudinal studies that had analysed both risk-based protective factors (i.e., direct ameliorative effects in high-risk samples) and buffering protective factors (i.e., interactive protective effects in both high and low-risk samples) against offending and violence. Findings showed that IQ, high academic achievement, positive teacher-child relationship, and self-control are protective factors for ASB in high-risk samples. The authors concluded that results were consistent with the literature on resilience and that future reviews should narrow their focus to uncover age-specific, sex-specific, and context-specific predictors of resilience to high and chronic ASB trajectory profiles to inform best practice in intervention and research. In the second review (Farrington, 2015), a differentiation was made between promotive factors (i.e., variables that predict a low probability of offending in any sample) and protective factors (i.e., variables that predict the low likelihood of offending among at-risk youth). Overall, low ADHD, good parental supervision, low physical punishment, and boys' high involvement in family activities were promotive factors. Academic achievement (e.g., good reading, reasoning and problem-solving skills) at age 10, good attachment to parents, and a good neighbourhood were protective factors. The author concluded that more research on promotive and protective factors in at-risk samples is needed to find true protective factors.

Finally, one review summarised findings from 42 systematic reviews reporting on explanatory risk factors for violence, offending, and delinquency (Farrington et al., 2017). The reviews in this study were 24 on sex offending/dating/intimate partner violence, 11 on criminal behaviour, and seven on delinquency/violence. The vast majority of studies focused

either only on socio-environmental risks or merged them with individual-level risks. Overall, the strongest risk factors for criminal behaviour were low parental education, poor parental supervision, poor child-rearing skills, large family size, and cognitive, but not affective empathy. The strongest risk factors for delinquent behaviour were low parental affection and involvement. The researchers called for more quantitative reviews of longitudinal studies focusing on explanatory risk factors and their effect sizes both before and after controlling for other risk factors.

The Present Review

Findings from these six past reviews suggest that a range of socio-environmental and individual-level risk and protective factors are relevant to understand the development of ASB. Further, the reviews summarised above also highlighted three specific gaps in current research and reviews:

(a) A summary of what is known about predictors of ASB trajectory profiles beyond LCP and AL is not currently available.

(b) Attention to study features, such as age range of participants, study duration, and reports of sex-specific predictors of ASB trajectory profiles are scarce; therefore, it is unknown whether targeting sex-specific risk and protective factors for ASB will have a greater impact on reducing and/or even preventing high and chronic ASB in boys separate than girls.

(c) There is little information on the risk and protective factors in childhood and their association with the range of known ASB trajectory profiles. This was only addressed in one of the six reviews, and no review described protective factors assessed in childhood specifically. However, evidence suggests that childhood risk and protective factors for ASB may differ from those in adolescence.

In addition to answering the call for more systematic reviews of predictors of ASB,

the general aim of the present review was to address such gaps in current literature by describing and comparing childhood predictors of youth's membership in several theoretically derived ASB trajectory profiles. First, to describe childhood predictors of ASB trajectory profiles, variables studied as possible correlates of seven distinct ASB trajectory profile (in comparison to a no/low-stable ASB profile) are reported. These findings are summarised for samples of boys and girls together and separated, whenever information was available. Among the seven distinct ASB trajectory profiles examined here, three are the CL, AL, and LCP, as described in Moffit's taxonomic theory (Moffitt et al., 1996; Moffitt, 2006) and additional research (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). Two are additional ASB trajectories that are also often described in the literature: Adult-onset and high-chronic ASB (Farrington, Ttofi, & Coid, 2009; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; McGee & Farrington, 2010). One is a profile of no/low ASB that is expected to be found in any community cohort (Moffitt, 2006) when using GBTM. The seventh ASB trajectory is a group of high ASB that starts in adolescence, namely adolescent-onset. This profile group exists in studies that did not follow their participants through to the end of adolescence; hence this profile possibly encompasses members of both LCP and AL trajectory groups.

Second, to look further into possible explanations of developmental origins of ASB in current literature, a series of comparisons of similarities and differences in significant childhood predictors across both distinct ASB trajectories (beyond AL and LCP) and sex-specific samples (i.e., boys and girls mixed and separated) are reported. Importantly, only prospective longitudinal studies that started assessment of ASB in childhood (age 12 or earlier) and used GBTM techniques were included in this review. This was done to focus on childhood predictors of ASB trajectory profiles within or from childhood and into adolescence or beyond.

Method

The following criteria were applied to identify eligible prospective longitudinal studies for this systematic review:

1. The study must be an empirical article that considered any sample kind, size, and sex, was written in languages in which the PhD candidate has reading proficiency (English, Portuguese, or Spanish), and was published in a peer-reviewed scientific journal from January 2008 to April 2018.
2. The study must have started to collect data during the participant's childhood (from 0 to 12 years). When age is not reported, data collection must have started in grade 6 or earlier.
3. The study must have at least three waves of follow-up information using repeated measures from the same source (i.e., self/mother/teacher-report, or criminal records) to assess aggressive behaviour, rule-breaking behaviour (delinquency) or offending behaviour. These were assessed separately from trajectories of recidivism[2], deviant peer affiliation, dating and sexual aggression, cyberbullying, gambling, drug use, ADHD, internalizing symptoms/behaviours, or any other mental health condition[3].
4. The study must have used a statistical approach consistent with GBTM to report on groups of individuals with similar trajectories of ASB.
5. The study must have focused on a limited age range at each wave of assessment to better identify age-specific patterns of change in ASB. Thus, studies were excluded if they reported that participants had an age range greater than two years within a single wave of data collection.
6. The study must have investigated childhood predictors of independent trajectories of ASB (as opposed to joint) by using multinomial logistic regression (s) to reliably identify

[2] Longitudinal studies that focused on trajectories of recidivism will be excluded according to what was done in previous reviews (Farrington et al., 2017).
[3] Although several mental health conditions are highly associated with manifestation of antisocial behaviour, they are not part of the definition of antisocial behaviour investigated in this study and each of them is addressed by a specific and separate body of literature.

childhood predictors that significantly differentiated between a problematic and a less or not at all problematic ASB trajectory profile. For this reason, the reference group in the multinomial logistic regression must have been either a no/low-stable ASB trajectory or a fully decreasing trajectory from any level to no/low-stable ASB levels. Also, the investigated predictors are not chromosomes or genotypes.

Search Strategy for Longitudinal Studies

The search was conducted from 29/03/2018 to 14/04/2018 in Medline, PsycINFO, Embase, and Web of Science online databases. The following search terms and Boolean operators were used: aggression OR "aggressi* behavior" OR "aggressi* behaviour" OR "antisocial behavior" OR "antisocial behaviour" OR delinquen* OR "rule break*" OR rule-break* OR offend* AND longitud* OR prospective OR trajector* OR pathway*.

Figure 1 shows the steps followed from the database search through to the identification of the eligible studies using the *Prisma statement* (Moher, Liberati, Tetzlaff, & Altman, 2009). A two-step approach underpinned the review of the 366 abstracts screened after duplicated studies were removed (Figure 1). In the first step, 269 studies were excluded after consideration of the inclusion/exclusion criteria numbers one to five (above): Fifteen studies were not empirical research and/or had not been published in a scientific journal from January 2008 to April 2018; in 67 studies, the first wave of data collection started when youth were older than 12 years old and/or were in grade seven or later at school; 81 studies had less than three waves of follow-up information and/or did not use their longitudinal data to examine ASB trajectories; 81 studies did not use GBTM methods to examine the trajectories reported; in four studies, sample age at wave one was a wide range of 11 to 17 years, 12 to 19 years, 4 to 17 years, or 9 to 14 years; 12 studies did not report on trajectories, but investigated childhood predictors of ASB in adolescence solely; four studies focused only on trajectories of behaviours that were beyond the scope of this review (e.g., internalizing behaviours,

ADHD, or drug use).

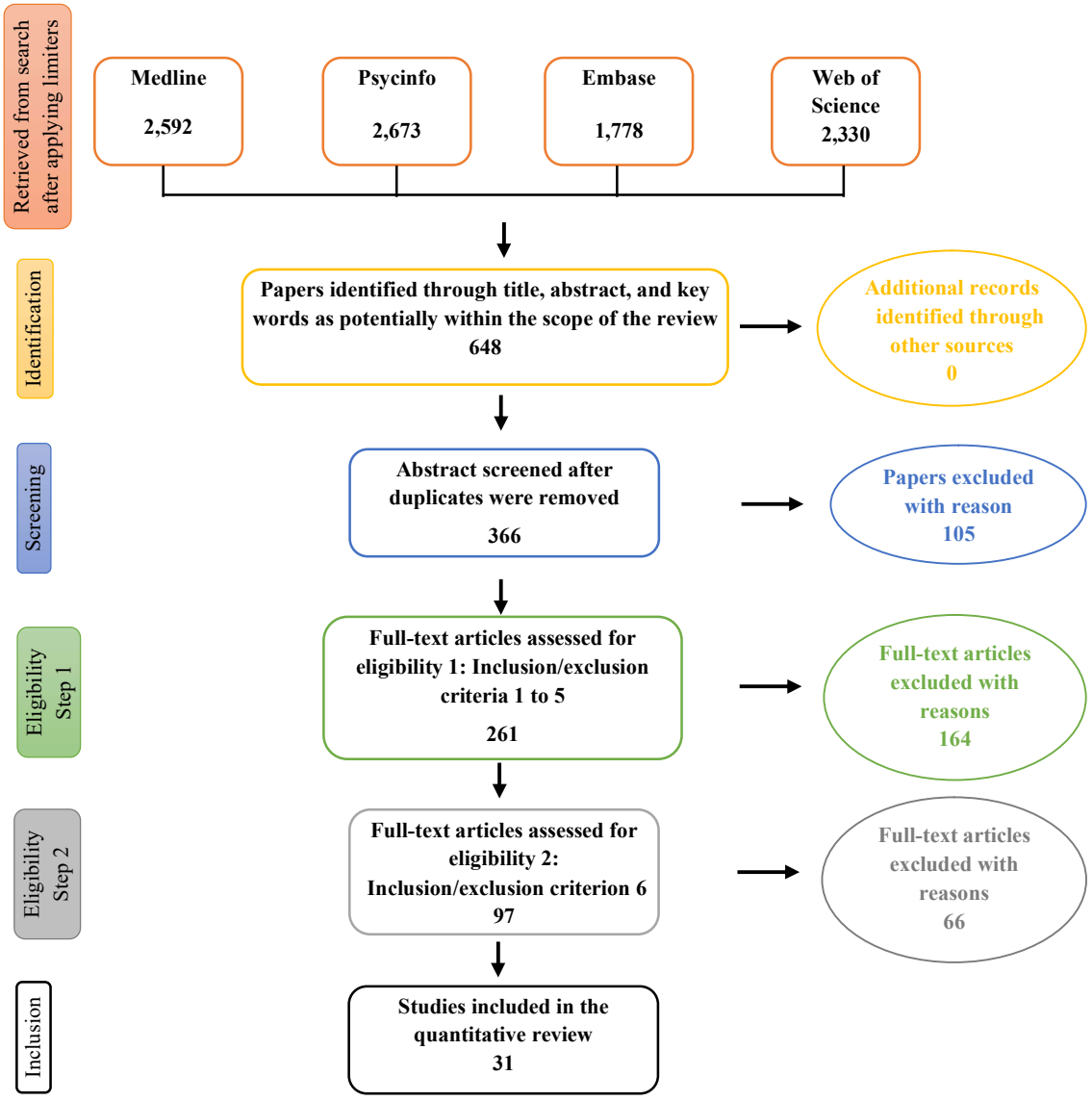

Figure 4.1. PRISMA flow diagram of the search strategy and process of identification of eligible studies.

In the second step, the application of the sixth inclusion/exclusion criterion resulted in 31 studies that met all inclusion criteria for this review (Figure 1). The 66 studies excluded in this second step had either not investigated childhood predictors of ASB trajectories or not used multinomial logistic regression to report on predictors of ASB trajectory membership. Two studies that had used multinomial logistic regression to investigate childhood predictors of ASB trajectories were excluded because the reference group in the regression analyses was out of the scope of this review (Baglivio, Wolff, Piquero, & Epps, 2015; Livingston, Stewart, Allard, & Ogilvie, 2008).

Data Extraction Strategy

As described in other reviews (Jolliffe, Farrington, Piquero, Loeber, & Hill, 2017; Jolliffe, Farrington, Piquero, MacLeod, & Van de Weijer, 2017), labels used to name trajectories of ASB usually vary across studies as researchers focus more on the age of onset, duration, and/or declining patterns of the trajectories they found. Thus, in the present review, a procedure from a previous meta-analytic review (Assink et al., 2015) was followed to assign each trajectory labels within each of the 31 studies into distinct, theoretically-derived definitions of ASB profile groups. Such profile groups were seven profiles of ASB that were carefully chosen to represent well-established definitions of ASB trajectories, as they have been described in Moffit's developmental taxonomic theory (Moffitt et al., 1996; Moffitt, 2006) and in previous studies (Farrington et al., 2009; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; McGee & Farrington, 2010). This process of choosing the seven definitions of ASB profiles was conducted after a thorough review of all eligible studies and before the data extraction process to generate the following mutually exclusive ASB profile groups:

- ASB profile group 1: *No/low-stable* (NL) – Stable low or no ASB.
- ASB profile group 2: *Child-Limited* (CL) – Childhood ASB levels above low that declined to no/low-stable at or before the age of 12.

- ASB profile group 3: *Adolescent-Limited* (AL) – High ASB starting sometime between the typical age of puberty and age 18, and a return to no/low-stable ASB by the age of 20[4].
- ASB profile group 4: *Life-course-persistent* (LCP) – High ASB starting at age 12 or earlier that was maintained or increased between ages 12 – 17 and continued beyond the age of 20.
- ASB profile group 5: *Adolescent-onset* (AO) – High ASB that started some time from age 12 and was maintained or increased; however, the study follow-up did not extend beyond age 19.
- ASB profile group 6: *High-stable* (HS) – The highest levels of ASB that remained high over most of data collection[5]; however, the study follow-up did not extend beyond age 19.
- ASB profile group 7: *Adult-onset* – High ASB starting at age 18 or after.

Importantly, to identify purer AL and LCP trajectory profiles more confidently, only studies that reported the continuation of ASB across all adolescent years (puberty to 18, for AL) and from childhood to adulthood (12 or earlier to 20+, for LCP) were included in ASB profile groups 3 and 4, respectively. Thus, these seven ASB profile groups are expected to account for seriousness (low/high), duration (limited/chronic), and age-of-onset of ASB; hence providing a more homogeneous definition of the trajectories of ASB assigned to each of them. Further, the setting up of these more homogeneous trajectory groups for comparison in this review also allowed for a more confident framework in which the childhood predictors of each ASB profile group could be classified. Notably, during data extraction, a trajectory label within an eligible study was coded as *other* when it did not fit in any of the seven ASB profile groups mentioned above. Trajectory labels within the *other* category were dealt with after data

[4] Such extended definition of adolescence (beyond age 20) is consistent with a previous study (Jolliffe, Farrington, Piquero, Loeber, et al., 2017) and also with a personal communication from Professor Moffitt to the authors in that same study.

[5] Trajectories within the high-stable profile may report a slightly increasing or decreasing in antisocial behaviour over time, but still remain within the high range - see high increasing trajectory (Maldonado-Molina et al., 2009) and high desisting trajectory (Ehrenreich et al., 2014) for examples.

extraction was completed and during the coding process. Also, information on ASB profile groups and their predictors were extracted separately for samples of boys and girls, and both sexes pooled together, whenever available. This was done to obtain data on sex-specific childhood predictors of ASB profile groups. In this process, the effect sizes extracted for each predictor of an ASB profile group were those reported in the most comprehensive model within each eligible study (e.g., models with all investigated predictors entered simultaneously in the regression analyses within each study).

The PhD candidate extracted data on all variables within each eligible study, extracting the same data from each study three times to check for errors. The primary supervisor also checked all data extracted on the ASB profile groups and their corresponding predictive variables. Variables names and effects' significance and size were then thoroughly re-inspected by both the PhD candidate and the primary supervisor after tables in the results section were put together.

Coding of Profile Groups of Antisocial Behaviour in Eligible Studies

After data extraction was completed, trajectory labels from eligible studies that had been placed in the *other* category during data extraction (i.e., they did not fit in any of the seven ASB profile groups) were examined separately and then grouped by similarity according to trajectory trends (decreasing/increasing), severity (low/medium/high), age at onset, and labels. This resulted in two new additional ASB profile groups:

- ASB profile group 8: *Antisocial-desistors* (AD) – ASB above the low trajectory in the study that showed a full decreasing path to a no/low-stable level[6].
- ASB profile group 9: *Medium-level* (ML) – ASB that was medium in level and generally stable over time in comparison to all other trajectories within the original study.

[6] When a full decreasing trajectory was observed within childhood years (0-12 years) this was classified into Group 2: *Childhood-limited* and not into Group 8.

Results

Overview of Eligible Studies

Table 4.1 presents sample sizes and age-ranges from the first to the last wave of data collection, the numbers of ASB trajectories found in each study, the final classification of each trajectory label into ASB profile groups, the proportion of participants in the corresponding trajectories, and the number of predictors alongside participants' ages when they were assessed within each study. The ASB trajectory used as the reference group in the multinomial logistic regression models conducted in each study is marked with an * in Table 4.1. No eligible study reported a trajectory within the *Adult-onset* ASB profile group (i.e., group 7 above). Studies in Table 4.1 are listed in alphabetical order by the first author's surname.

Across the 31 included longitudinal studies, the sample size ranged from 192 (López-Romero, Romero, & Andershed, 2015) to 120,133 (Ferrante, 2013), and studies varied in the age period covered (see Table 4.1). The youngest age period covered was from the age 2 to 5 (Degnan, Calkins, Keane, & Hill-Soderlund, 2008), with most other studies following youth from just prior to adolescence (e.g., age 8 to 11) until the mid- to late-teens. The oldest age at the last wave was 32 years (Ward et al., 2010).

Also shown in Table 4.1, 28 of the 31 studies (90%) designated the no/low-stable ASB trajectory as the reference group for the multinomial logistic regression, whereas a decreasing/desisting trajectory was the reference group in one study (Ehrenreich, Beron, Brinkley, & Underwood, 2014). One study reported only on predictors of membership in the highest ASB trajectory in relation to the rest of the sample in lower trajectories (i.e., not the high ASB trajectory) as the reference group (Petitclerc, Boivin, Dionne, Zoccolillo, & Tremblay, 2009).

Table 4.1

Part 1 Characteristics of the 31 Included Studies: Samples, Trajectories, Antisocial Behaviour (ASB) Profile Groups, and Predictors in Each Study

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
1. (Becht, Prinzie, Deković, Van Den Akker, & Shiner, 2016)	290	9-15	2 (rule breaking)	G1: NL - Low rule-breaking*- G6: HS - High rule-breaking	92 8	26	9
		9-15	3 (aggression)	G1: NL - Low decreasing aggression* G6: HS - High increasing aggression G8: AD - High decreasing aggression	85 6 9		
2. (Boutin, Verlaan, Denault, & Déry, 2017)	347	8-11	3	G2: CL - Low-decreasing aggression* G6: HS - Mean-increasing aggression G8: AD - Mean- decreasing aggression	36 19 45	12	8
3. (Bowers et al., 2011)	626	11-17	4	G1: NL - No-low offending G5: AO - Low-moderate offending G6: HS - Adolescent-peaked offending* G9: ML- Moderate-unstable offending	55 26 4 15	13	11
4. (Day et al., 2012)	386	10-29	7	G1: NL - Low persister offending G3: AL - Low-desister offending* G4: LCP - Moderate Late persister offending G4: LCP - High late offending G4: LCP - High early offending G4: LCP - Moderate adolescent-peaked offending G9: ML - Moderate early persister offending	32 29 4 4 4 12 15	2	C
5. (Degnan et al., 2008)	318	2-5	4	G1: NL - Low disruptive behaviour* G2: CL - Normative disruptive behaviour G6: HS - High disruptive behaviour* G9: ML - Moderate disruptive behaviour	14 44 8 34	6	2
6. (Ehrenreich, et al., 2014)	296	9-18	3 (social	G1: NL - Low decreasing aggression* G6: HS - High desisting aggression	37 17	7	9

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
			aggression)	G8: AD - Middle desisting aggression	46		
			3 (physical aggression)	G1: NL - No aggression*	36	7	9
				G1: NL – Stable-low aggression	46		
				G8: AD - High declining aggression	18		
7. (Evans et al., 2016)	354	10-19	4	G1: NL - Negligible delinquency*	39	8	10
				G5: AO - Late start delinquency	23		
				G6: HS - Early starter/ chronic delinquency	14		
				G8: AD - Early starter/ declining delinquency	24		
8. (Ferrante, 2013)	120,133	10-30	3 (boys)	G1: NL - Low rate offending*	84	11	8
				G4: LCP - High-rate offending	3		
				G9: ML - Mid-rate offending	13		
			2 (girls)	G1: NL - Low rate offending	91		
				G4: LCP - Mid-rate offending	9		
9. (Higgins, Ricketts, Marcum, & Mahoney, 2010)	407	12-16	4	G1: NL - Non-offenders*	19	8	12
				G5: AO - Linear increasing offending	50		
				G6: HS - High offending	6		
				G9: ML - Low/ progressing offending	25		
10. (Hoeve et al., 2008)	503	10-19	5	G1: NL - Nondelinquents*	27	5	9
				G3: AL - Serious desisters delinquents	14		
				G6: HS - Serious persisters delinquents	24		
				G8: AD - Moderate desisters delinquents	7		
				G9: ML - Minor persisters delinquents	28		
11. (Jennings, Maldonado-Molina, & Komro, 2010)	3,038	12-14	7 (boys)	G1: NL - Non-delinquent*	10	8	12
				G5: AO - Moderate-rate increasing delinquency	5		
				G5: AO - Low-rate increasing delinquency	4		
				G6: HS - High rate increasing delinquency	20		
				G6: HS - Highest-rate increasing delinquency	6		
				G9: ML - Low rate stable delinquency	24		
				G9: ML - Moderate-rate stable delinquency	31		
		12-14	7	G1: NL - Non-delinquent*	8	8	12

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
			(girls)	G5: AO - Moderate-rate increasing delinquency	21		
				G5: AO - Low-rate increasing delinquency	6		
				G6: HS - High rate increasing delinquency	31		
				G6: HS - High rate stable delinquency	14		
				G8: AD - Low-rate decreasing delinquency	9		
				G8: AD - Moderate-rate decreasing delinquency	11		
12. (Jennings, Maldonado-Molina, Piquero, et al., 2010)	1,414	10-14	4 (Puerto Rican boys from New York)	G1: NL - Non- offending*	72	5	10
				G2: CL - Declining offending	10		
				G6: HS - High-increasing offending	2		
				G5: AO - Low-increasing offending	16		
		10-14	4 (Puerto Rican girls from New York)	G1: NL - Non-offending*	69	5	10
				G1: NL - Low offending	25		
				G2: CL - High Declining offending	2		
				G6: HS - High increasing offending	4		
	1,525	10-14	3 (Boys from Puerto Rico)	G1: NL - Non-offending*	74	5	10
				G1: NL - Low offending	24		
				G2: CL - Declining offending	2		
		10-14	3 (Girls from Puerto Rico)	G1: NL - Non-offending*	81	5	10
				G1: NL – Low-stable offending	18		
				G2: CL - High-declining offending	1		
13. (Kolivoski, Shook, Goodkind, & Kim, 2014)	794	12-22	5	G1: NL - No or low offending*	70	9	C
				G3: AL - Early age offending	6		
				G3: AL - Short-term high offending	8		
				G4: LCP - Chronic offending	8		
				G4: LCP - Late adolescence-early adult offending	8		
14. (López-Romero et al., 2015)	192	6-14	3	G1: NL - Stable low conduct problems*	63	4	C
				G6: HS - Stable high conduct problems	19		
				G8: AD - Decreasing conduct problems	18		
15. (Maldonado-Molina, Jennings, & Komro, 2010)	3,038	12-14	4	G1: NL - Non-aggressive*	16	11	11
				G6: HS - Chronic aggressive	55		

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
				G5: AO - Escalators aggressive	20		
				G8: AD - Desistors aggressive	9		
16. (Maldonado-Molina, Piquero, Jennings, Bird, & Canino, 2009)	1,525	10-14	4 (Puerto Rican youth from Puerto Rico)	G1: NL - Non-offending*	59	15	10
				G1: NL - Very low delinquency	35		
				G2: CL - Rapid decline delinquency	2		
				G9: ML - Low-stable delinquency	4		
	1,414	10-14	5 (Puerto Rican youth from New York)	G1: NL - Non-offending*	49	15	10
				G1: NL - Very low-decreasing delinquency	37		
				G2: CL - Desisting delinquency	4		
				G5: AO - Increasing delinquency	8		
				G6: HS - High-increasing delinquency	2		
17. (Maldonado-Molina, Reingle, Tobler, Jennings, & Komro, 2010)	731	12-18	5	G1: NL - Non-Aggressive*	14	10	11
				G1: NL - Low-stable aggression	23		
				G3: AL - Early-rapid desistors aggression	26		
				G6: HS - High / moderate desistors aggression	21		
				G9: ML - Escalators aggression	16		
18. (Malti, Averdijk, Ribeaud, Rotenberg, & Eisner, 2013)	1,028	8-11	5	G1: NL - Low-stable aggression*	30	6	8
				G2: CL - Decreasing aggression	10		
				G6: HS - High stable aggression*	4		
				G6: HS - Increasing aggression	9		
				G9: ML - Medium stable aggression	47		
19. (Malti, McDonald, Rubin, Rose-Krasnor, & Booth-LaForce, 2015)	230	10-12	3	G1: NL - Low stable aggression*	81	9	10
				G5: AO - Increasing aggression	4		
				G8: AD - Decreasing aggression	15		
20. (Odgers et al., 2008)	1,037	7-26	4 (boys)	G1: NL - Low conduct problems*	46	13	C
				G4: LCP - Life-course persistent conduct problems	10		
				G4: LCP - Adolescent-onset conduct problems	20		
				G8: AD - Child-limited conduct problems	24		
		7-15	4 (girls)	G1: NL - Low conduct problems*	55	13	C
				G5: AO - Adolescent Onset conduct problems	17		

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
				G6: HS - Early-onset persisters conduct problems	8		
				G8: ML - Mean- decreasing conduct problems	20		
21. (Park, Lee, Sun, Vazsonyi, & Bolland, 2010)	556	11-16	3	G1: NL - Low steady antisocial behaviour*	77	7	11
				G5: AO - Incremental antisocial behaviour	15		
				G8: AD - High starter antisocial behaviour	8		
22. (Petitclerc et al., 2009)	2,120	2-6	4	G1: NL - Very low disregard for rules	9	15	2
				G1: NL - Low disregard for rules	57		
				G6: HS - Chronic disregard for rules [b]	30		
				G9: ML - Moderate disregard for rules	4		
23. (Petts, 2009)	2,472	10-25	3	G1: NL - Low level delinquency*	60	19	10
				G3: AL - Early Adolescent Limited delinquency	33		
				G4: LCP - Late adolescent limited delinquency	7		
24. (Reingle, Maldonado-Molina, Jennings, & Komro, 2012)	3,038	12-14	4 (African Americans)	G1: NL - Low aggression*	8	11	12
				G5: AO - Escalators aggression	20		
				G6: HS - Consistent aggression	64		
				G9: ML - Moderate aggression	8		
		12-14	4 (Hispanics)	G1: NL - Low aggression*	17	11	12
				G5: AO - Escalators aggression	22		
				G6: HS - Consistent aggression	43		
				G8: AD - Desistors aggression	18		
25. (Roisman et al., 2010)	990	3-15	5	G1: NL - Low externalising*	42	6	EC
				G1: NL - Moderate externalising	34		
				G5: AO - Adolescent onset externalising	12		
				G6: HS - Early-onset persister externalising	5		
				G8: AD - Childhood limited externalising	7		
26. (Shaw, Hyde, & Brennan, 2012)	268	10-17	4	G1: NL - Low stable delinquency*	62	5	2
				G5: AO - Late increasing delinquency	23		
				G6: HS - High increasing delinquency	10		
				G8: AD - High decreasing delinquency	5		
27. (Silver, Measelle,	241	5-10	3	G1: NL - Low externalising*	85	7	5

Study ID and reference	Sample size	Sample age[a]	No of trajectories	ASB profile group (number: name) – trajectory label in the original study	%	No of predictors	Age at predictors
Armstrong, & Essex, 2010)				G6: HS - Chronic high externalising	6		
				G9: ML - Low Increasing externalising	9		
28. (Sittner & Hautala, 2016)	646	10-19	5	G1: NL - No offenders*	22	8	10
				G2: CL - Moderate desistors offending	20		
				G3: AL - Adolescent-limited offending	22		
				G6: HS - Chronic offending	19		
				G8: AD - High desistors offending	17		
29. (Tabone et al., 2011)	827	4-10	5	G1: NL - Lowest externalising*	33	24	4
				G6: HS - High-chronic externalising	4		
				G6: HS - Increasing predicted externalising	8		
				G8: AD - Decreasing-predicted externalising	12		
				G9: ML - Low-medium externalising	43		
30. (Ward et al., 2010)	378	8-32	4	G3: AL - Low-rate offending*	65	2	C
				G4: LCP - High-rate adolescent peaked offending	5		
				G4: LCP - High-rate adult peaked offending	8		
				G9: ML - Mid-rate offending	22		
31. (Yessine & Bonta, 2009)	235	12-31	2 (Aboriginal)	G4: LCP - Chronic high offending	19	9	C
				G8: AD - Stable-low offending*	81		
	204	12-31	2 (no-Aboriginal)	G4: LCP - Chronic high offending	12	9	C
				G8: AD - Stable-low offending*	88		

Note. ASB Profile Groups are mutually exclusive theoretically-derived profiles of antisocial behaviour that represent groups of specific ASB trajectories. ID numbers link to values in brackets within Tables 4.3 to 4.18. G1: NL = No/low-stable. G2: CL = Child-limited. G3: AL = Adolescent-limited. G4: LCP = Life-course-persistent. G5: AO = Adolescent-onset. G6: HS = High-stable. G8: AD = Antisocial-desistors. G9: ML = Medium-level. EC / C = The predictive variables were assessed before the study's first wave and averaged or different predictors were assessed at different times before age 3 (EC) or age 12 (C).

[a] Sample age indicates the age range of participants from the first to the last wave of assessment within each study. No study had less than three waves of assessment.

[b]The study only reported on childhood predictors of the chronic trajectory using sample children in all other three trajectories within the study as the reference group.

*Indicates the no/low-stable ASB trajectory that was used as the reference group in the multinomial logistic regression. In studies 3, 5, and 18, the high stable ASB trajectory was also used as the reference group to examine predictors of no/low-stable ASB trajectory membership.

Also, one study reported on predictors of no/low-stable ASB trajectory in relation to the highest trajectory - used as the reference group within the study (Bowers et al., 2011). In addition, two of the 31 studies reported an additional regression analysis using the reference group with the highest level of ASB to test predictors of no/low-stable ASB (Degnan et al., 2008; Malti et al., 2013).

Table 4.1 also shows that prevalence rates of participants in ASB trajectories ranged from 8% to 92% within the no/low-stable ASB group; 1% to 44% within the child-limited ASB group; 6% to 65% within the adolescent-limited; 3% to 20% within the life-course-persistent ASB group; 4% to 50% within the adolescent-onset; 2% to 65% within the high-stable ASB group; 5% to 88% within the antisocial-desister ASB group; and 4% to 47% within the medium-level ASB group.

Table 4.2 presents additional methodological details about the 31 included studies. Overall, the studies came from seven countries with most from the USA (n = 19; 61%) and two had cross-national samples. As can be seen, the studies ranged in the types of ASB examined. The ASB type more frequently considered was rule-breaking behaviours (9 studies; 29%) and aggressive behaviours (8 studies; 26%; being 2 of physical aggression only, 1 of a composite of physical aggression and oppositional defiant disorder (ODD) symptoms, 2 of a composite of physical and relational aggression, 2 of a composite of physical and verbal aggression, and 1 of overt aggression). In addition, five studies (16%) accessed official records of offending (being 1 of records of police charges and arrest and 4 of official records of court conviction). A further three studies (9%) focused on a composite of rule-breaking behaviours merged with official records of convictions (n = 1), with physical aggression (n = 1), or with physical and verbal aggression (n = 1).

Table 4.2

Part 2 Characteristics of the 31 Included Studies: Publication Year, Country of Origin, ASB Definition, Recruitment Source, Sex and Ethnicity

Authors	Year	Country	ASB Investigated	Recruitment source	% of males	Sample ethnicity[a]
Becht, Prinzie, Deković, Van Den Akker, & Shiner	2016	Belgium	Rule-Breaking behaviour, Physical and verbal aggression	General Population	48.6	NR
Boutin, Verlaan, Denault, & Déry	2017	Canada	Malicious and exclusionary behaviours	At-risk Girls	0	NR
Bowers et al.	2011	USA	Rule-breaking behaviour	General population	49	35% Latinos; 35% European American; 7% African American; 5% Asian American; 18% NR
Day et al.	2012	Canada	Records of conviction	Offenders	100	NR
Degnan, Calkins, Keane, & Hill-Soderlund	2008	USA	Physical aggression, ODD	Children with at least one year out of home welfare placement	48	67% European American; 27% African American; 4% Biracial; 2% Hispanic
Ehrenreich, Beron, Brinkley, & Underwood	2014	USA	Physical and relational aggression	General population	46.6	21% African American; 5% Asian; 52% Caucasian; 21% Hispanic
Evans, Simons, & Simons	2016	USA	Rule-breaking behaviour	General Population	100	100% African American
Ferrante	2013	Australia	Police charge and arrest	Offenders	72	White 86%; Aboriginal 14%
Higgins, Ricketts, Marcum, & Mahoney	2010	USA	Rule-breaking behaviour	General population	41.7	NR

Authors	Year	Country	ASB Investigated	Recruitment source	% of males	Sample ethnicity[a]
Hoeve et al.	2008	USA	Rule-breaking behaviour + records of convictions	Low-income boys	100	57% African American; 43% NS
Jennings, Maldonado-Molina, & Komro	2010	USA	Rule-breaking behaviour	General population	50.6	37% Black; 30% white/other; 33% Hispanic/Latino
Jennings, Maldonado-Molina, Piquero, et al.	2010	USA	Rule-breaking behaviour	General population	51.5	100% Latino
Kolivoski, Shook, Goodkind, & Kim	2014	USA	Records of conviction	Children with at least one year out of home welfare placement	51	48% African American; 46% Caucasian; 6% Biracial
López-Romero, Romero, & Andershed	2015	Spain	CD and ODD symptoms	General population	72.4	A not ethnically diverse sample
Maldonado-Molina, Jennings, & Komro	2010	USA	Physical and verbal aggression	Low-income youth	50.6	38% Black; 29% white/other; 33% Hispanic/Latino
Maldonado-Molina, Piquero, Jennings, Bird, & Canino	2009	USA/Puerto Rico	Rule-breaking behaviour	General population	51.5	100% Latino
Maldonado-Molina, Reingle, Tobler, Jennings, & Komro	2010	USA	Physical aggression	General Population	49.5	100% Hispanic/Latino
Malti, Averdijk, Ribeaud, Rotenberg, & Eisner	2013	Switzerland	Overt aggression	General Population	NR	NR

Authors	Year	Country	ASB Investigated	Recruitment source	% of males	Sample ethnicity[a]
Malti, McDonald, Rubin, Rose-Krasnor, & Booth-LaForce	2015	USA	Physical and relational aggression	General population	50	55% European American; 13% African American; 16% Asian American 9% Latin American; 8% bi-multiracial
Odgers et al.	2008	New Zealand	Theft, Vandalism, and Physical aggression	General population	52	NR
Park, Lee, Sun, Vazsonyi, & Bolland	2010	USA	Rule-breaking behaviour	Poor inner-city African Americans	48.2	100% African American
Petitclerc, Boivin, Dionne, Zoccolillo, & Tremblay	2009	Canada	Disregard for rules	General population	50.3	NR
Petts	2009	USA	Vandalism, theft,	General population	NR	Black and Hispanic oversampled and minority white
Reingle, Maldonado-Molina, Jennings, & Komro	2012	USA	Physical aggression	General population	48	57% African Americans; 43% Hispanics
Roisman et al.	2010	USA	Rule-breaking behaviour	General population	49.7	NR
Shaw, Hyde, & Brennan	2012	USA	Rule-breaking behaviour	Low-income boys	100	53% European American; 36% African American; 5% biracial
Silver, Measelle, Armstrong, & Essex	2010	USA	CD and ODD symptoms	General population	48.5	90% European American not of Hispanic origin; 3% African American not of Hispanic origin; 3% Native American; 2% Latino; 1% Asian; American; 1% NR
Sittner & Hautala	2016	Canada/USA	Physical and verbal aggression	Families living in indigenous reserves	NR	100% Indigenous

Authors	Year	Country	ASB Investigated	Recruitment source	% of males	Sample ethnicity[a]
Tabone et al.	2011	USA	Rule-Breaking behaviour and Physical aggression	Children reported for child protective services for maltreatment	NR	31% White; 44% African American; 25% NR
Ward et al.	2010	Canada	Records of conviction	Offenders	100	NR
Yessine & Bonta	2009	Canada	Records of conviction	Offenders	100	54% Aboriginal; 46% NR

Note. ASB = Antisocial Behaviour. ODD = Oppositional Defiant Disorder. NR = Not reported in the paper. CD = Conduct Disorder

[a] The reporting of the sample ethnicity is worded as it was written in the each of the original papers and not all papers presented percentages.

Finally, two studies (6%) considered a composite of conduct disorder and ODD symptoms, another two studies (6%) considered theft and vandalism (one of these also measured physical aggression and combined it with theft and vandalism), one study (3%) measured disregard for rules, and one study (3%) focused on malicious and exclusionary behaviours.

The recruitment source of each study is also summarised in Table 4.2. Thirteen studies were based on selected samples of youth (4 studies of offenders, 5 studies of low income or at-risk youth, 3 studies of children in welfare placements of protective services, and 1 study of youth in Indigenous reserves), whereas the remaining 18 studies were of community samples. Regarding sample sex, 20 studies were mixed, six included boys only, four analysed data from boys and girls separately, and one focused on girls only. The socio-cultural background of each study is also shown in Table 4.2.

Predictors of Antisocial Behaviour Trajectories in 31 Studies

As in a previous review (Jolliffe, Farrington, Piquero, Loeber, et al., 2017), differentiation was made here between explanatory and non-explanatory predictive variables of ASB trajectories to only report the first type. Non-explanatory childhood predictors include criminal history, aggression, youth's favourable attitudes towards ASB, and drug use. They were not reported because they reflect the same underlying construct, therefore being a tautological explanation of the continuity of ASB (Jolliffe, Farrington, Piquero, Loeber, et al., 2017). In contrast, only explanatory predictors tested in prospective longitudinal studies are reported here because they provide possible causal information on each ASB profile group, which is relevant for theory, interventive, and preventive initiatives targeting the continuation of ASB (Farrington & Welsh, 2007; Jolliffe, Farrington, Piquero, Loeber, et al., 2017). Notably, socio-demographic variables of sex, SES, ethnicity, and place of birth also have causal relevance for the onset and continuity of ASB but examining them was beyond the scope of this review. Thus, for informative purposes, all socio-demographic variables

reported in the studies were grouped into a separate category of *static background factors*, as suggested by Assink and colleagues (2015), and are summarised in Appendix A.

The pie chart shown in Figure 2 reports the proportion of included studies that had a focus on socio-environmental and individual levels, while also specifying whether the tested effects were either direct-only or both direct and interaction. Overall, 16 (52%) of the eligible studies examined predictors at the socio-environmental level; 11 (35%) examined socio-environmental and individual-level predictors; two (6%) examined socio-environmental and individual-level predictor variables alongside interactions effects between them; one (3%) examined direct effects and interaction among socio-environmental variables, and one study (3%) examined predictors at the individual level.

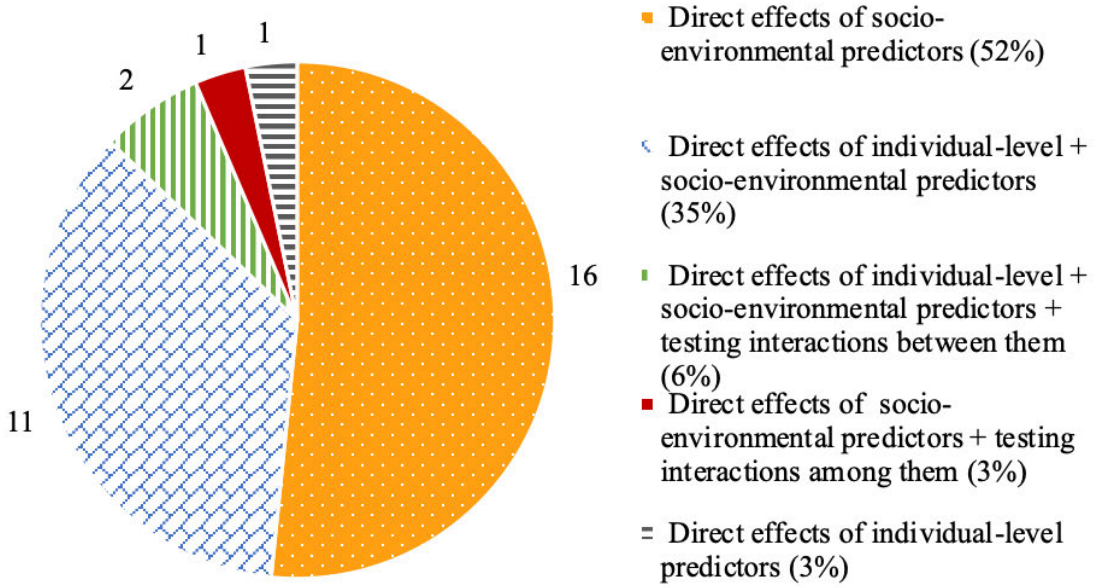

Figure 4.2. Prevalence of types of explanatory predictors in the 31 included studies. The predictors were categorised according to (1) socio-environmental vs. individual-level (2) whether either direct effects-only or direct effects + interaction effects were tested as predictors of membership in trajectories of antisocial behaviour.

Tables 4.3 to 4.18 show both significant ($p < 0.05$) and non-significant odds ratios (ORs) for each predictive variable within 31 eligible studies. Regression coefficients (*B*s)

from multinomial logistic regression results were reported in three studies rather than ORs (Hoeve et al., 2008; Malti et al., 2013; Shaw, Hyde & Brennan, 2012). In these cases, Bs were converted to ORs by calculating the exponent of the B so that all findings are on the same metric. The variables presented in each table were classified into domains within socio-environmental and individual levels. A list of these domains is shown in Figure 4.3. The domains within the socio-environmental level included Family (e.g., variables relating to familial conflict, change, relationship, and conflict); School (e.g. variables relating to education and school behaviour and environment); Peer (e.g., variables relating to the relationship with peers); Neighbourhood/Community (e.g., variables related to the quality, resources, and circumstances of child's living environment); Sexual behaviour (e.g., variables relating to sexual behaviour and dating). The domains within the individual-level included Physical/Physiological (e.g., variables relating to child's physical health and physiological functioning/responses); Emotional/personality (e.g., factors relating to internalizing and self/emotion-regulation problems, psychological diagnosis, temperament, and personality traits); (Neuro)cognition/academic achievement (e.g., variables relating to neuropsychological factors, cognitive functioning, and child's academic achievement). Such classification of childhood predictors into various domains was guided by procedures in previous reviews (Assink et al., 2015; Jolliffe, Farrington, Piquero, Loeber, et al., 2017).

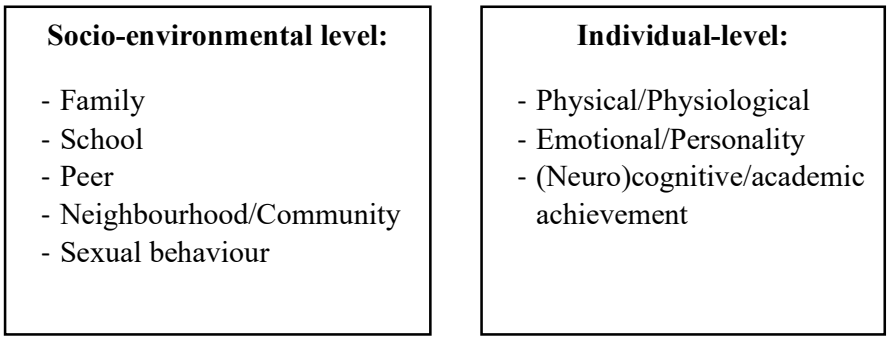

Figure 4.3. The framework of levels of predictors of antisocial behaviour trajectory groups and their corresponding domains which guided the reporting of results in this systematic review.

Childhood Predictors of Sex-Specific Antisocial Behaviour Profile Groups

Next, a detailed description of childhood predictors of ASB profiles is reported separately for each of the nine profile groups. For each ASB profile group, environmental and individual-level of explanatory childhood predictors are reported separately, as their association with membership in nine distinct ASB profile groups had been tested in relation to no/low-stable ASB in original studies. Furthermore, to also report on sex-specificities of all such variables, childhood predictors are presented here for samples of boys and girls separated and/or pooled together, whenever they were available. Samples composed of boys and girls together are referred to here as mixed samples.

Antisocial behaviour profile group 1: No/low-stable (NL). Three studies examined socio-environmental and/or individual-level predictors of membership in the NL ASB profile group relative to a high ASB in mixed samples. Results from these three studies are shown in Tables 4.3 and 4.4. Eleven socio-environmental factors were examined in two studies, and two of these factors were significant (see Table 4.3). Having more access to teachers and school resources and more participation in recreation and external academic programs at age 11 increased the chance of membership in the NL profile group, relative to high ASB. These effects were found in an ethnically diverse mixed sample of 11- to 17-years-old youth in the general population.

Possible individual predictors of NL ASB were tested in two studies (see Table 4.4). In one study, youth that had been rated by their peers as high in trustfulness and trustworthiness at age eight had a higher odds of membership in the NL ASB group relative to high ASB. These effects were found in a mixed sample of 8- to 11-years-old youth in the general population. In the second study, physiological emotion regulation and reactivity to frustration were not associated with being a member of the NL ASP profile in a mixed sample of 2- to 5-years-old youth in out-of-home welfare placement.

Table 4.3

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of No/low-Stable ASB Relative to High-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Maternal control and punitiveness toward the child [5]	1.18[a]	-
	Child often eats dinner with family [3]	1.05[b]	-
	Mother years of education [3]	0.97[b]	-
School			
Mixed sample	School accessibility: lower size and lower student-teacher ratio [3]	-	1.84[b]
	Youth participation in recreation and external academic programs [3]	-	1.47[b]
	Teacher education and experience levels [3]	0.60[b]	-
	Communication school-parents [3]	0.58[b]	-
Neighbourhood/Community			
Mixed sample	Presence of community groups/ organizations in the neighbourhood [3]	1.52[b]	-
	Child-adult ratio in the neighbourhood [3]	1.36[b]	-
	% of employed males and college-educated residents in the neighbourhood [3]	1.29[b]	-
	Neighbourhood physical and institutional resources [3]	0.86[b]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined.
[a](Degnan et al., 2008). [b](Bowers et al., 2011).

Table 4.4

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of No/low-Stable ASB Relative to High-Stable ASB

		Odds ratio	
Sample / Domain / Predictive variable		Non-significant	Significant (p <.05)
Mixed sample	**Physical/Physiological**		
	Physiological markers of emotion regulation[a] [5]	1.76[b]	-
	Emotional/Personality		
	High trustworthiness [18]	-	162.40[c]
	High trustfulness [18]	-	3.06[c]
	High reactivity to frustration [5]	0.38[b]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined.
[a]Decrease in Respiratory sinus arrhythmia. [b](Degnan et al., 2008). [c](Malti et al., 2013).

Antisocial behaviour profile group 2: Child-limited (CL). Six of the 31 eligible studies reported socio-environmental (see Table 4.5) and/or individual-level (see Table 4.6) predictors of CL ASB. One study followed children from ages two to five (Degnan et al., 2008), whereas in the other five studies, the age range of participants at the first assessment was eight to 10 years and at the last wave it was 11, 14, or 19 years.

As shown in Table 4.5, a total of 15 possible socio-environmental predictor variables were examined in studies considering mixed samples, and one study tested three predictors in boys and girls separately. Of the tested predictors, six (40%) in mixed samples and two (67%) in samples of boys and girls were found to significantly predict membership in CL, relative to no/low-stable ASB. All such significant predictive variables were found in two studies examining rule-breaking behaviour from ages 10 to 14 in three community samples of Latino youth: A mixed sample, a sample of boys, and a sample of girls. Neighbourhood/community predictors were the most often tested and also the most numerous among the significant predictors in the mixed sample (n = 5 / 3 significant: Exposure to violence, acculturation of Latino youth into USA language and culture- risks, and social support - protective), boys (n = 2 / 1 significant: Exposure to violence), and girls (n = 2 / 2 significant: Exposure to violence, acculturation of Latino youth into USA language and culture). Significant predictive variables were also reported in the family (parent coercive discipline), school (negative school environment), and peer (delinquent peer association) domains in the mixed sample and the family domain in the sample of boys (parent coercive discipline).

Table 4.5

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Child-limited ASB Relative to No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Parent coercive discipline [16]	1.44[a]	4.41[a]
	Perceived parental rejection [28]	1.17[b]	-
	Good parent-child interaction [16]	0.83[a] / 0.18[a]	-
	Maternal control and punitiveness toward the child [5]	0.71[c]	-
Boys	Parent coercive discipline [12]	-	2.01[d] / 1.91[d]
Girls	Parent coercive discipline [12]	2.30 [d] / 1.27 [d]	-
School			
Mixed sample	Negative school environment [16]	1.09[a]	1.11[a]
	Failed a class at school [28]	1.33[b]	-
	Positive school adjustment [28]	0.83[b]	-
Peer			
Mixed sample	Delinquent peer association [16,28]	1.41[b] / 0.59[a]	2.27[a]
	Peer relationships [16]	1.10[a] / 1.03[a]	-
Neighbourhood/Community			
Mixed sample	Exposure to violence [16]	-	1.46[a] / 1.21[a]
	Acculturation of Latino youth into USA language and culture [16]	1.35[a]	2.26[a]
	Social support [16]	0.86[a]	0.46[a] [LR]
	Stressful life events [16]	1.14[a] / 1.05[a]	-
	Welfare [16]	0.95[a] / 0.73[a]	-
Boys	Exposure to violence [12]	-	1.34[d] / 1.11[d]
	Acculturation of Latino youth into USA language and culture [12]	1.43[d] / 1.15[d]	-
Girls	Exposure to violence [12]	-	1.74 [d] / 1.19 [d]
	Acculturation of Latino youth into USA language and culture [12]	1.84 [d]	5.08 [d]
Sexual behaviour			
Mixed sample	Had/has a steady boy/girlfriend [28]	1.30[b]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group.
[a](Maldonado-Molina et al., 2009). [b](Sittner & Hautala, 2016). [c] (Degnan et al., 2008). [d] (Jennings, Maldonado-Molina, Piquero, et al., 2010).

Table 4.6 shows seven individual-level predictors examined in three mixed samples and one individual-level predictor tested in boys and girls separately. Variables that significantly predicted CL relative to no/low-stable ASB were found in three studies and were all in the emotional/personality domain. Three (43%) of such variables were tested in mixed samples and one in samples of boys and girls significantly. In two studies that considered mixed samples of youth in the general population, one of the three significant variables was found to predict rule-breaking behaviour from 10 to 14 years old (sensation-seeking), the other two reduced the likelihood of overt aggression from eight to 11 years old (high trustfulness, high trustworthiness). The third study examined rule-breaking behaviour from 10 to 14 years reported the same significant predictor for separated samples of Latino boys and girls who live in the USA (sensation-seeking).

Table 4.6

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of Child-limited ASB Relative to No/Low-Stable ASB

		Odds ratio	
Sample / Domain / Predictive variable		Non-significant	Significant (p <.05)
Mixed sample	**Physical/Physiology**		
	Physiological marker of emotion regulation[a] [5]	0.78[b]	-
	Emotional/Personality		
	Sensation seeking [16]	-	1.28[c] / 1.34[c]
	High trustfulness [18]	-	0.33[d] [LR]
	High trustworthiness [18]	-	0.01[d] [LR]
	High reactivity to frustration [5]	2.99[b]	-
	Locus of control [16]	1.25[c] / 0.74[c]	-
	Self-esteem [16]	0.92[c] / 0.83[c]	-
Boys	**Emotional/Personality**		
	Sensation-seeking [12]	1.01[e]	1.12[e]
Girls	**Emotional/Personality**		
	Sensation-seeking [12]	-	1.78[e] / 1.45[e]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group.
[a]Decrease in Respiratory sinus arrhythmia. [b](Degnan et al., 2008). [c](Maldonado-Molina et al., 2009). [d](Malti et al., 2013). [e](Jennings, Maldonado-Molina, Piquero, et al., 2010).

Antisocial behaviour profile group 3: Adolescent-limited (AL). AL ASB trajectories in studies with follow-up into adulthood (18+) were found in seven studies (see Table 4.7). In all seven studies, the first assessment occurred between ages eight to 12 years, and the last wave occurred between the ages of 19 to 32. Visual inspection of AL ASB trajectories suggests that the age in which ASB levels started to increase above the low level (i.e., age of onset) ranged from 10 to 15 years-old among them.

As shown in Table 4.7, 30 possible socio-environmental predictors were examined in mixed samples, and two predictors were investigated in boys. Among the examined variables, eight (27%) in mixed samples and one (50%) in boys significantly differentiated AL from the no/low-stable ASB. Variables in the family domain were the most studied and more numerous among the significant predictors in two studies. One study examined vandalism and theft from ages 10 to 25 in a mixed sample of Hispanic and black youth in the general population (n = 16 / 3 significant: child lives with a single caregiver, parent-child arguments

Table 4.7

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Adolescent-Limited ASB Relative to No/Low-Stable ASB

		Odds Ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Lives with a single caregiver [23]	-	1.84[a]
	Parent-child arguments over rules [23]	-	1.40[a]
	More educated parents [23]	-	0.95[a] [LR]
	Lives with stepfamily [23]	1.70[a]	-
	Perceived parental rejection [28]	1.26[b]	-
	Hours without adult supervision [17]	1.18[c]	-
	Mother/child have different religious affiliations [23]	1.14[a]	-
	Parental engagement in activities with child (i.e. movies, dinner, schoolwork, play) [23]	1.03[a]	-
	Mother age at birth [23]	1.00[a]	-
	Parent praise and show affection toward child [23]	1.00[a]	-
	Parental involvement [17]	0.98[c]	-
	Live with both parents in the house [17]	0.97[c]	-
	Parent rules about child TV watching, dating, going out, doing homework [23]	0.93[a]	-
	Parent-child collaboration in decision-making [23]	0.91[a]	-
	Attends religious services with parents and parents	0.86[a]	-

Domain / Sample / Predictive variable		Odds Ratio: Non-significant	Odds Ratio: Significant (p <.05)
	think this is important [23]		
	Latino background and speaks Spanish at home [17]	0.59[c]	-
Boys	Neglectful parenting style [10]	-	7.10[d]
	Authoritarian parent style [10]	1.32[d]	-
School			
Mixed sample	Failed a class at school [28]	1.43[b]	-
	Positive school adjustment [28]	0.84[b]	-
Peer			
Mixed sample	Number of friends who drink alcohol [17]	-	2.47[c]
	Delinquent peer association [23,28]	0.84[b]	1.83[a]
Neighbourhood/Community			
Mixed sample	Adult alcohol use in the neighbourhood [17]	-	1.49[c]
	Ever utilised the drug and alcohol treatment system [13]	1.82[e]	3.06[e]
	Number of child welfare placements [13]	0.97[e]	1.07[e]
	Ever utilised the public mental health system [13]	3.03[e] / 0.62[e]	-
	Ever in group and/or regular residential facilities [13]	1.68[e] / 0.97[e]	-
	More years in out-of-home placement [13]	1.10[e] / 0.91[e]	-
	Higher frequency of religious participation [23]	1.02[a]	-
	Age at first placement [13]	0.98[e] / 0.98[e]	-
	Welfare case closed before age 13[13]	0.89[e] / 0.57[e]	-
Sexual behaviour			
Mixed	Had/has a steady boy/girlfriend [28]	2.31[b]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. [a](Petts, 2009). [b](Sittner & Hautala, 2016). [c](Maldonado-Molina, Reingle, et al., 2010). [d](Hoeve et al., 2008). [e](Kolivoski et al., 2014).

over rules - risks, having more educated parents - protective). The other study examined rule-breaking behaviour and records of conviction from ages 10 to 19 in a sample of low-income boys (n = 2 / 1 significant: neglectful parenting style). Significant predictive variables were also reported in peer and neighbourhood/community domains in three studies of mixed samples. The first two studies examined physical aggression from ages 12 to 18 (greater number of friends who drink alcohol, adult's alcohol use in the neighbourhood) and vandalism and theft from ages 10 to 25 (delinquent peer association) in Hispanic and black youth in the general population. The third study examined records of conviction from ages 12 to 22 in youth in out-of-home welfare placement (ever involved with the drug and alcohol treatment system, number of child welfare placements). No variables in the family, school, and sexual behaviour domains were significant predictors of AL profile membership.

For individual-level predictors (data not shown in a Table), only the presence of depressive symptoms at the age of 11 was considered in one of the 31 included studies. Such a study examined physical aggression from age 12 to 18 years in a mixed sample of Hispanic youth (Maldonado-Molina, Reingle, Tobler, Jennings, & Komro, 2010) and found strong associations between depressive symptomatology and membership in AL ASB profile group (OR = 2.28).

Antisocial behaviour profile group 4: Life-course-persistent (LCP). Predictors of membership in LCP ASB profile group were studied in seven of the 31 eligible studies (see Tables 4.8 and 4.9). In all seven studies, the first assessment occurred between ages seven to 12 years, and the last wave occurred between the ages of 22 to 32. Visual inspection of LCP ASB trajectories suggests that, in some cases, LCP trajectories had high levels of ASB since the first age of assessment. However, in other cases, the age in which ASB levels started to increase above the low level (i.e., age of onset) ranged from 10 to 15 years old.

Table 4.8 summarises results from testing 20 socio-environmental predictors in studies considering mixed samples, as well as 19 predictors studied in boys, and six studied in girls. Out of such variables, eight (40%) in mixed samples, 15 (79%) in boys, and three (50%) in girls significantly differentiated between LCP and no/low-stable ASB. Overall, predictors in both family and neighbourhood/community domains were the most studied and numerous among the significant variables. For instance, across these two domains, the following variables had both the most consistent (no study reported non-significant estimates for them) and the strongest effects (OR => 2.00[7]): Mixed sample - Child lives with single caregiver (OR = 3.14), ever utilized the drug and alcohol treatment system (OR = 2.33 / 2.17). Boys – More quantity of family-related maltreatment indicators (OR = 14.50 / 4.00),

[7] Because odds ratios are measures of effect that are centered around 1, a rule of thumb for an important association has been described as an odds ratio of 2 for a “risk” factor (see Cohen (1996); Jolliffe, Farrington, Piquero, Loeber, et al. (2017)).

higher risk in the PRA-V1 (OR = 7.96 / 4.07, see Yessine and Bonta (2009) for details on this measure), involvement with alternative care (OR = 3.82 / 3.14).

Table 4.8

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Life-Course-Persistent ASB Relative to No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Lives with single caregiver [23]	-	3.14[a]
	Lives with stepfamily [23]	3.31[a]	-
	Mother and child have different religious affiliations [23]	1.37[a]	-
	Parent-child arguments over rules [23]	1.29[a]	-
	Attends to religious services with parents and parents think this is important [23]	1.06[a]	-
	Having more educated parents [23]	1.05[a]	
	Parent praise and show affection toward child [23]	1.01[a]	-
	Parental engagement in activities with child (i.e. movies, dinner, schoolwork, play) [23]	0.99[a]	-
	Parent set rules about TV watching, dating, going out, doing homework [23]	0.99[a]	-
	Parent-child collaboration in decision making [23]	0.97[a]	-
	Mother's age at birth [23]	0.95[a]	-
Boys	Quantity of maltreatment indicators (rejecting mother-child interaction, harsh discipline, 2+ changes in primar caregiver, physical or sexual abuse) [20]	-	14.50[b] / 4.00[b]
	Family physical and verbal conflict [20]	-	NR[b] / NR[b]
	Volatile Family [31]	1.74[c]	2.53[c]
	Parent self-report of a criminal conviction [20]	1.10[b]	3.40[b]
	Mother low IQ [20]	1.10[b]	2.50[b]
	Inconsistent discipline (mother vs. father) [20]	NR[b]	NR[b]
	Mother's affective distress and somatic response [20]	NR[b]	NR[b]
	Broken home or family transitions (parental separation/divorce, change in caregiver, frequent moves) [30]	0.96[d] / 0.45[d]	-
School			
Boys	=> 2 years below education norm at school [31]	2.64[c] / 0.75[c]	-
Peer			
Mixed sample	Delinquent peer association [23]	-	1.78[a]
Boys	Delinquent peer association [31,20]	2.08[c]	4.63[c] / NR[b]
Neighbourhood/Community			
Mixed sample	Ever utilised the drug and alcohol treatment system [13]	-	2.33[e] / 2.17[e]
	Higher frequency of religious participation [23]	-	1.20[a][LR]
	Ever in group home and/or regular residential facilities [13]	1.64[e]	2.78[e]
	Ever utilised public mental health system [13]	1.72[e]	0.44[e]

Domain / Sample / Predictive variable		Odds ratio	
		Non-significant	Significant (p <.05)
	Greater number of child welfare placements [13]	0.98[e]	1.12[e]
	More years in stable out-of-home placement [13]	0.93[e]	0.85[e] [LR]
	Age at first placement [13]	1.00[e] / 0.99[e]	-
	Welfare case closed before age 13 [13]	0.43[e] / 0.35[e]	-
Boys	High risk in the PRA-V1 [31]	-	7.96[c] / 4.07[c]
	Involvement with alternative care (institutional, foster, child welfare care) [30]	-	3.82[d] / 3.14[d]
	Onset diversion [8]	-	1.50[f]
	Onset-resource (deficient educational and occupational characteristics in the community) [8]	-	1.16[f]
	Onset-city (Location of first offence: Perth) [8]	-	0.90[f] [LR]
	Onset-north (Location of first offence: western Australia) [8]	-	0.52[f] [LR]
	Unstable living arrangements [31]	1.63[c]	3.48[c]
	Onset-edocc (neighbourhood educational and occupational disadvantage) [8]	NR[f]	-
	Onset-disadvantage (neighbourhood economic disadvantage) [8]	NR[f]	-
Girls	Onset-resource (deficient educational and occupational characteristics in the community) [8]	-	1.33[f]
	Onset-city (Location of first offence: Perth) [8]	-	1.19[f] [LR]
	Onset-north (Location of first offence: western Australia) [8]	-	0.66[f] [LR]
	Onset-disadvantage (neighbourhood economic disadvantage) [8]	0.75[f]	-
	Onset-edocc (neighbourhood educational and occupational disadvantage) [8]	NR[f]	-
	Onset diversion [8]	NR[f]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. Onset refers to the variable measured at onset and the terms used in the paper are maintained here. Onset diversion = Whether the (first offence was dealt with via diversionary processes, which aims to reduce the effects of labelling and stigmatization associated with juvenile offending. PRA-V1 = Primary Risk Assessment - version 1 is a revised version of the Wisconsin Risk and Needs classification system, see Yessine and Bonta (2009) for details on this measure.

[a](Petts, 2009). [b](Odgers et al., 2008). [c](Yessine & Bonta, 2009). [d](Ward et al., 2010). [e](Kolivoski et al., 2014). [f](Ferrante, 2013).

A variable in the peer domain (delinquent peer association) was a significant predictor of LCP in three studies: One study examining vandalism and theft from ages 10 to 15 in a mixed sample of Hispanic and black youth in the general population, one study examining records of convictions from ages 12 to 31 in a sample of offender boys, and one study examining theft, vandalism, and physical aggression together from seven to 26 years in a community sample of boys.

Table 4.9 presents results for the six individual-level predictive variables examined in two studies considering samples of boys. All such variables significantly differentiated membership in LCP from no/low-stable ASB. Five of such predictors were found across the physical/physiology (low resting heart rate), emotion/personality (ADHD diagnosis, uncontrolled temperament), and (neuro)cognition/academic achievement (low IQ, reading) domains in a study examining theft, vandalism, and physical aggression together from seven to 26 years in a community sample of boys. One significant predictor (low academic achievement) was reported in a study examining records of conviction from ages 10 to 29 in a sample of male offenders.

Table 4.9

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of Life-Course-Persistent ASB Relative to No/Low-Stable ASB

		Odds ratio	
Sample / Domain / Predictive variable		Non-significant	Significant (p <.05)
Boys	**Physical/Physiology**		
	Low resting Heartrate [20]	NR[a]	NR[a]
	Emotional/Personality		
	ADHD diagnosis [20]	2.00[a]	18.7[a]
	Undercontrolled temperament [20]	-	3.50[a] / 3.20[a]
	(Neuro)Cognition/Academic achievement		
	Low IQ [20]	-	5.80[a] / 2.30[a]
	Low reading achievement [20]	-	NR[a] / NR[a]
	Low academic achievement [4]	0.84[b] / 3.08[b] / 0.30[b]	0.36[b] [LR]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.

[a](Odgers et al., 2008). [b](Day et al., 2012).

Antisocial behaviour profile group 5: Adolescent-onset (AO). Thirteen of the 31 studies identified trajectories of high ASB in adolescence, but follow-up of participants in these studies was not available either up to age 19 or beyond (see Tables 4.10 and 4.11). In all 13 studies, the first assessment occurred before age 12 years, and the last wave occurred between the ages of 12 to 19. Visual inspection of AO ASB suggests that the age of ASB onset ranged from age 11 to 14.

Table 4.10 summarises 36 socio-environmental level predictive variables examined in studies considering mixed samples, with 18 studied in samples of boys, and 11 studied in samples of girls. From these, seven (19%) in mixed samples, five (26%) in boys, and five (45%) in girls significantly differentiated AO from no/low-stable ASB. Family domain predictors were the most studied and most numerous among the significant variables when considering mixed samples (*n* = 12 / 2 significant: Low maternal sensitivity to child's needs - risk, living with both mother and father in the house - protective), boys (*n* = 9 / 2 significant: Family physical and verbal conflict - risk, both biological parents at home - protective), and girls (*n* = 8 / 3 significant: More quantity of family-related maltreatment indicators, low mother IQ, mother's affective distress and somatic response).

Table 4.10

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Adolescent-Onset ASB Relative to a No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Living with both mother and father in the house [15]	-	0.65[a] [LR]
	Low maternal sensitivity to child's needs [25]	-	0.59[b]
	Parent-child interaction [16]	1.41[c]	-
	Coerciveness of parent's disciplining techniques [16]	1.19[c]	-
	Daily amount of time without adults around [15,24]	1.19[d] / 1.14[a] / 1.04	-
	Poor parental monitoring of child's behaviour [21]	1.11[e]	-
	Parental involvement with child's life [24]	1.03[d] / 0.99[d]	-
	Child-mother positive attachment [9]	1.00[f]	-
	Child-father positive attachment [9]	0.97[f]	-
	Having access to alcohol at home [24]	0.81[d]	-
	Lives with single caregiver [25]	0.72[b]	-

Domain / Sample / Predictive variable		Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
	Latino background and speaks Spanish at home [24]	0.71[d]	-
Boys	Family physical and verbal conflict [20]	-	NR[g]
	Both biological parents at home [11]	0.60[h]	0.44[h] [LR]
	lives with a single parent [7]	1.54[i]	-
	Lack of adult supervision [11]	1.27[h] / 1.14[h]	-
	Coerciveness of parent's disciplining technics [12]	1.15[j]	-
	Authoritative parenting [7]	1.01[i]	-
	High interparental violence and aggression [26]	0.89[i]	-
	Supportive parenting and quality home environment [26]	0.98[i]	-
	Maternal depressive symptoms [26]	0.94[i]	-
Girls	Quantity of maltreatment indicators (rejecting mother-child interaction, harsh discipline, 2+ changes in primary caregiver, physical or sexual abuse) [20]	-	3.90[g]
	Mother low IQ [20]	-	2.40[g]
	Mother affective distress and somatic response [20]	-	NR[g]
	Parent self-report of a criminal conviction [20]	1.60[g]	-
	Lack of adult supervision [11]	1.12[h] / 1.06[h]	-
	Both biological parents at home [11]	0.61[h] / 1.08[h]	-
	Family physical and verbal conflict [20]	NR[g]	-
	Inconsistent discipline (mother vs. father) [20]	NR[g]	-
School			
Mixed sample	School suspension [21]	-	2.37[e]
	Negative school environment [9,16]	1.04[c]	1.12[f]
	School expulsion [21]	1.99[e]	-
Boys	Receives free or low-price lunches at school [11]	1.14[h]	0.47[h] [LR]
	Participation in advanced classes at school [7]	1.02[i]	-
Girls	Receives free or low-price lunches at school [11]	1.09[h]	2.00[h]
Peer			
Mixed sample	Better understanding of friendship trust [19]	-	0.16[k] [LR]
	Delinquent peer association [9,16]	1.30[c]	1.34[f]
	Friend-reported good-quality friendship [19]	2.44[k]	-
	Self-reported good-quality friendship [19]	1.89[k]	-
	Having peers who use alcohol [24]	1.76[d] / 1.22[d]	-
	Friend's aggressive behaviours [19]	1.50[k]	-
	Understanding of friendship conflict resolution [19]	1.40[k]	-
	Greater number of friends who drink alcohol [15]	1.38[a]	-
	Gang membership [24]	1.23[d] / 0.79[d]	-
	Peer relationships [16]	1.07[c]	-
	Better understanding of reasons for friends [19]	0.73[k]	-
	Better understanding of friendship termination [19]	0.71[k]	-
	Better understanding of friendship closeness [19]	0.60[k]	-
Boys	Number of delinquent friends [7]	-	1.16[i]
	Number of friends who drink alcohol [11]	1.31[h] / 0.98[h]	-
Girls	Delinquent peer association [20]	-	NR[g]
	Number of friends who drink alcohol [11]	1.60 [h] / 0.86[h]	-
Neighbourhood/Community			
Mixed	Youth exposure to violence [16]	-	1.16[c]

Domain / Sample / Predictive variable		Odds ratio	
		Non-significant	Significant (p <.05)
sample	Number of off-premise alcohol outlet in the neighbourhood [24]	2.03[d]	-
	Adult alcohol use in the neighbourhood [24]	1.36[d] / 1.03[d]	-
	Acculturation of Latino youth into USA language and culture [16]	1.34[c]	-
	Youth welfare [16]	1.16[c]	-
	Stressful life-events [16]	1.01[c]	-
	Social support [16]	0.65[c]	-
Boys	Youth exposure to violence [12]	-	1.07[j]
	Moving houses in the past year [7]	1.17[i]	-
	Frequency of racial discrimination [7]	1.02[i]	-
	Acculturation of Latino youth into USA language and culture [12]	0.97[j]	-
	Death of friends/family [7]	0.66[i]	-
	Neighbourhood impoverishment (low income, unemployment, low education) [26]	1.00[l]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported. Onset diversion = Whether the first offence was dealt with via diversionary processes, which aims to reduce the effects of labelling and stigmatization associated with juvenile offending.
[a](Maldonado-Molina, Jennings, et al., 2010). [b](Roisman et al., 2010). [c](Maldonado-Molina et al., 2009). [d](Petts, 2009). [e](Park et al., 2010). [f](Higgins et al., 2010). [g](Odgers et al., 2008). [h](Jennings, Maldonado-Molina, & Komro, 2010). [i](Evans et al., 2016). [j](Jennings, Maldonado-Molina, Piquero, et al., 2010). [k](Malti et al., 2015). [l](Shaw et al., 2012).

Also, three studies reported significant effects in the school domain. Two of them considered mixed samples (school suspension, negative school environment) and one considered two samples of boys and girls (receiving free or low-price lunches at school, for both sexes). Significant effects in the peer domain were reported in four studies; two considering mixed samples (delinquent peer association and more mature understanding of friendship trust), one study of boys (a greater number of friends who engage in delinquent behaviours), and one study of girls (delinquent peer association). In the neighbourhood/community domain, two studies reported one significant effect in a mixed sample (youth exposure to violence) and one in a sample of boys (youth exposure to violence).

Table 4.11 shows 12 individual-level predictive variables examined in mixed samples, with two predictors studied in boys, and five in girls. Of these, three (25%) in mixed samples and one (50%) in boys significantly differentiated AO from no/low-stable ASB.

Table 4.11

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of Adolescent-Onset ASB Relative to a No/Low-Stable ASB

Sample / Domain / Predictive variable		Odds ratio	
		Non-significant	Significant (p <.05)
Mixed sample	**Physical/Physiology**		
	Worse physical health [25]	-	0.63[a]
	Emotional/Personality		
	Sensation seeking [16]	-	1.22[b]
	Self-esteem [16]	-	0.85[b] [LR]
	Locus of control [16]	1.87[b]	-
	Depressive symptoms [24]	1.38[c] / 1.07[c]	-
	Difficult temperament [25]	1.30[a]	-
	Risk-taking [9]	1.08[d]	-
	High level of Impulsivity [9]	1.05[d]	-
	Hopelessness [21]	0.97[e]	-
	Self-worth perception [21]	0.93[e]	-
	(Neuro)Cognition/Academic achievement		
	Low academic achievement [24]	0.86[c] / 1.60[c]	-
	Low cognitive functioning [25]	1.03[a]	-
Boys	**Emotional/Personality**		
	Sensation-seeking [12]	-	1.11[f]
	(Neuro)Cognition/Academic achievement		
	Low academic achievement [4]	1.11[g]	-
Girls	**Physical/Physiology**		
	Low resting Heartrate [20]	NR[h]	-
	Emotional/Personality		
	ADHD diagnosis [20]	3.00[h]	-
	Undercontrolled temperament [20]	0.90[h]	-
	(Neuro)Cognition/Academic achievement		
	Low IQ [20]	1.80[h]	-
	Low reading achievement [20]	NR[h]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported. [a](Roisman et al., 2010). [b](Maldonado-Molina et al., 2009). [c](Reingle, Maldonado-Molina, et al., 2012). [d](Higgins et al., 2010). [e](Park et al., 2010). [f](Jennings, Maldonado-Molina, Piquero, et al., 2010). [g](Day et al., 2012). [h](Odgers et al., 2008).

One significant predictor in the physical/physiology domain was reported in one study that examined rule-breaking behaviour from ages 0 to 15 in a mixed sample of youth in the general population (worse physical heath). Significant predictors in the emotional/personality domain were found in two studies that examined rule-breaking behaviour from 10 to 14 in community samples of Hispanic youth. One study considered a mixed sample (sensation seeking, self-esteem) and the other study considered a sample of boys (sensations seeking).

Antisocial behaviour profile group 6: High-stable (HS). Twenty-three of the 31 eligible studies had trajectories identified within the HS ASB group, whereby ASB was higher than all other trajectories and remained high across all or most of the time points (see Tables 4.12 and 4.13). However, because the follow-up period never extended past age 20 for any given study, HS should be considered a mix of CL, AL and LCP. In all 23 studies, the first assessment occurred before age 12, and the last wave occurred between the ages of five to 19. Therefore, HS ASB profile primarily represents the highest level of ASB (i.e., the severity of ASB) at any given time within each study.

Table 4.12 provides a summary of 53 possible socio-environmental predictor variables examined in studies considering mixed samples, 20 in samples of boys, and 22 in samples of girls. Of these, 23 (43%) in mixed samples, seven (35%) in boys, and 10 (45%) in girls significantly differentiated HS from no/low-stable ASB. Childhood predictors in the family domain were the most studied and most likely reported as significant predictors when considering mixed samples (n = 32 / 11 significant), boys (n = 10 / 4 significant), and girls (n = 15 / 8 significant). For instance, the following variables had both the most consistent (no study reported non-significant estimates for them) and the strongest effects (OR => 2.00) among all significant variables within the family domain: Mixed samples – Maternal permissive parenting (OR = 4.54). Boys – Neglectful parenting style (OR = 2.03). Girls – More quantity of family-related maltreatment indicators (OR = 7.80), parent's self-report of criminal conviction (OR = 3.90), mother's low IQ (OR = 3.10).

Significant predictors of membership in HS were also observed in the school domain in two studies, one examining conduct disorder and ODD from five to 10 years in a mixed sample of children in the general population (more frequency of teacher-child interpersonal conflict) and one examining a composite of physical and verbal aggression from 10 to 19 years in a mixed sample of Indigenous youth (positive school adjustment). Within the peer

domain, the greatest number of significant variables was reported by studies that considered mixed samples (n = 5; a greater number of friends who drink alcohol, gang membership, having peers who drink alcohol, delinquent peer association, peer relationships – risks, peer acceptance - protective). Still, in the peer domain, two significant predictors were also found in two other studies. One study examined rule-breaking behaviour from 10 to 19 years in a community sample of African American boys (greater number of friends who engage in delinquent behaviour) and the other study examining theft, vandalism, and physical aggression together from 7 to 15 years in a community sample of girls (delinquent peer association). Within the neighbourhood/community domain again the greatest number of significant variables was reported in studies considering mixed samples (n = 4; child ever utilized mental health services, adult's alcohol use in the neighbourhood, exposure to violence - risks, neighbourhood safety and social support – protective). In addition, significant predictors in this domain were also found in one study examining rule-breaking behaviour from 10 to 14 years in two separate community samples of Hispanic boys and girls (exposure to violence for both sexes), and in another study examining rule-breaking behaviour from 10 to 19 years in a community sample of African American boys (higher frequency of experiencing racial discrimination). Finally, one significant predictor in the sexual behaviour domain was found in a study examining a composite of physical and verbal aggression from 10 to 19 years in a mixed sample of Indigenous youth (the child had/has a steady boy/girlfriend).

Table 4.12

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of High-Stable ASB Relative to a No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed	Maternal permissive parenting style [6]	-	4.54[a]
sample	Child perceived parental rejection [28]	-	1.36[b]
	Latino background and speaks Spanish at home [24,17]	-	0.56[c] [LR] / 0.35[d] [LR]
	Low maternal sensitivity to child's needs [25]	-	0.38[e]
	Married parents [6]	-	0.14[a] [LR]
	Overreactive parenting [1]	2.45[f]/ 2.20[f] /1.53[f] / 1.01[f]	18.45[f] / 3.79[f]
	Hours without adult supervision [24,15,17]	1.39[c]/ 1.20[g] /1.14[g]	1.21[d]
	Caregiver alcoholism [29]	1.11[h]	1.42[h]
	Paternal depressive symptoms [22, 29]	1.02[h]	1.90[i] / 1.10[h]
	Parental involvement with child [24,17]	0.99[c]	0.95[c] [LR] / 0.94[d] [LR]
	Lives with both parents [15,17]	0.89[d]	0.67[g] [LR]
	Maternal authoritarian parenting style[6]	2.33[a]	-
	Mother first pregnant before 20 [22]	1.73[i]	-
	Maternal depressive symptoms [22]	1.63[i]	-
	Maternal antisocial behaviour [22]	1.60[i]	-
	Mother smoked during pregnancy [22]	1.53[i]	-
	Hostile-reactive paternal behaviour [22]	1.47[i]	-
	Caregiver low education level [22, 29]	1.39[i] / 0.95[h] /1.05[h]	-
	Paternal antisocial behaviour [22]	1.38[i]	-
	Hostile-reactive maternal behaviour [22]	1.31[i]	-
	Maternal history of depression [22]	1.27[i]	-
	Having access to alcohol at home [24]	1.26[c]	-
	Coercive parent discipline [16]	1.16[j]	-
	Low maternal responsiveness [27]	1.14[k]	-
	Negative mother-child interaction [22]	1.10[i]	-
	Mother conflict and problem-solving skills [6]	1.06[a]	-
	Child-father positive attachment [9]	0.97[l]	-
	was separated from the caregiver [29]	0.95[h] / 1.02[h]	-
	Child-mother positive attachment [9]	0.94[l]	-
	Paternal history of depression [22]	0.88[i]	-
	lives with single caregiver [25]	0.58[e]	-
	Positive parent-child interaction [16]	0.26[j]	-
Boys	Neglectful parenting style [10]	-	2.03[m]
	Authoritarian parenting style [10]	-	1.62[m]
	Mother depressive symptoms [26]	-	1.07[n]
	Both biological parents at home [11]	0.66[o]	0.51[o] [LR]
	Lack of adult supervision [11]	1.17[o] / 1.11[o]	-
	Supportive parenting and home environment [26]	0.98[n]	-
	Authoritative parenting style [7]	0.96[p]	-
	Interparental violence and aggression[26]	0.93[n]	-
	lives with a single parent [7]	0.69[p]	-
	Parent coercive discipline [12]	0.50[q]	-
Girls	Quantity of maltreatment indicators (rejecting mother-child interaction, harsh discipline, 2+ changes in	-	7.80[r]

Domain / Sample / Predictive variable		Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
	primary caregiver, physical or sexual abuse) [20]		
	Parent self-report of a criminal conviction[20]	-	3.90[r]
	Mother low IQ [20]	-	3.10[r]
	Parent coercitive discipline [12]	-	1.86[q]
	Family physical and verbal conflict [20]	-	NR[r]
	Inconsistent discipline (mother vs. father) [20]	-	NR[r]
	Mother affective distress and somatic symptoms [20]	-	NR[r]
	Lack of adult supervision [11]	1.15[o]	1.29[o]
	Parental neglect [2]	2.72[s]	-
	Parental hostility [2]	1.72[s]	-
	Blended or single-parent family [2]	1.66[s]	-
	Number of family moves [2]	1.06[s]	-
	Parental rejection [2]	0.87[s]	-
	Both biological parents at home [11]	0.77[o] / 0.70[o]	-
	Parental warmth [2]	0.57[s]	-
School			
Mixed	Teacher-child interpersonal conflict [27]	-	12.95[k]
sample	Positive school adjustment [28]	-	0.65[b] [LR]
	Failed a class at school [28]	2.15[b]	-
	Negative school environment [9,16]	1.16[j] / 0.94[l]	-
	Teacher-child closeness [27]	0.49[k]	-
Boys	Participation in advanced classes at school [7]	1.19[p]	-
	Receives free or low-price school lunch [11]	1.03[o] / 0.88[o]	-
Girls	Receives free or low-price school lunch [11]	1.48[o] / 1.36[o]	-
Peer			
Mixed	Number of friends who drink alcohol [15,17]	-	3.43[d] / 1.73[g]
sample	Gang membership [24]	-	2.94[c] / 2.00[c]
	Peer acceptance [27]	-	0.09[k] [LR]
	Peers use alcohol [24]	1.86[c]	2.28[c]
	Delinquent peer association [9,16,28]	0.52[j]	2.94[b] / 1.51[l]
	Peer relationships [16]	0.77[j]	-
Boys	Number of delinquent friends[7]	-	1.20[p]
	Number of friends who drink alcohol[11]	1.60[o] / 1.54[o]	-
Girls	Delinquent peer association [20]	-	NR[r]
	Number of friends who drink alcohol [11]	2.50[o] / 2.20[o]	-
	Peer rejection [2]	1.31[s]	-
	Peer acceptance [2]	0.82[s]	-
Neighbourhood/Community			
Mixed	Ever utilized mental health services [29]	-	4.78[h] / 2.72[h]
sample	Adult alcohol use in the neighbourhood [24,17]	-	1.87[d] / 1.75[c] / 1.55[c]
	Youth exposure to violence [16]	-	1.21[j]
	Neighbourhood safety and social support [29]	0.97[h]	0.95[h] [LR]
	Child welfare receipt [29,16]	2.77[h]/ 1.04[j]/ 0.99[h]	-
	Acculturation of Latino youth into USA language and culture [16]	1.41[j]	-
	Number of off-premise alcohol outlet in the neighbourhood [24]	1.49[c]	-
	Stressful life-events [16]	1.29[j]	-
	Child social support [16]	0.95[j]	-
Boys	Youth exposure to violence [12]	-	1.16[q]

Domain / Sample / Predictive variable		Odds ratio	
		Non-significant	Significant (p <.05)
	Frequency of racial discrimination [7]	-	1.07[p]
	Moving houses in the past year [7]	1.13[p]	-
	Neighbourhood impoverishment (low income, unemployment, low education) [26]	0.91[n]	-
	Death of friends/family [7]	0.84[p]	-
	Acculturation of Latino youth into USA language and culture [12]	0.83[q]	-
Girls	Youth exposure to violence [12]	-	1.20[q]
	Acculturation of Latino youth into USA language and culture [12]	1.41[q]	-
Sexual behaviour			
Mixed sample	Had/has a steady boy/girlfriend [28]	-	2.65[b]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.
[a](Ehrenreich et al., 2014). [b](Sittner & Hautala, 2016). [c](Reingle, Maldonado-Molina, et al., 2012). [d](Maldonado-Molina, Reingle, et al., 2010). [e](Roisman et al., 2010). [f](Becht et al., 2016). [g](Maldonado-Molina, Jennings, et al., 2010). [h](Tabone et al., 2011). [i](Petitclerc et al., 2009). [j](Maldonado-Molina et al., 2009). [k](Silver et al., 2010). [l](Higgins et al., 2010). [m](Hoeve et al., 2008). [n](Shaw et al., 2012). [o](Jennings, Maldonado-Molina, & Komro, 2010). [p](Evans et al., 2016). [q](Jennings, Maldonado-Molina, Piquero, et al., 2010). [r](Odgers et al., 2008). [s](Boutin et al., 2017).

As shown in Table 4.13, 29 individual-level predictive variables were examined in mixed samples, one in a sample of boys, and 10 in samples of girls. Overall, 18 (62%) of such variables in mixed samples, one (100%) in boys, and six (60%) in girls, significantly differentiated HS from no/low-stable ASB. Childhood predictors in the emotional/personality domain were the most studied and most likely to be significant predictors when considering mixed samples (n = 25 / 15 significant), boys (n = 1 / 1 significant), and girls (n = 7 / 4 significant). For instance, among all significant variables in the emotion/personality domain, the following had the most consistent (no study reported non-significant estimates for them) predictive effects: Mixed samples - Sensation seeking, behavioural psychopathy, risk-taking, high trustfulness, high trustworthiness, high optimism, and high compliance. Boys - sensation seeking. Girls - ADHD diagnosis, uncontrolled temperament, high surgency/extraversion, and sensation seeking.

Table 4.13

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of High-Stable Relative to No/Low-Stable ASB

Sample / Domain / Predictive variable	Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
Mixed sample **Physical/Physiology**		
Developmental disability [29]	-	3.28[a] / 2.39[a]
Worse physical health [25]	-	0.43[b]
Emotional/Personality		
Sensation seeking [16]	-	1.81[c]
Behavioural psychopathy [14]	-	1.50[d]
Risk-taking [9]	-	1.40[e]
High Trustfulness [18]	-	0.40[f] [LR]
High Trustworthiness [18]	-	0.03[f] [LR]
Depressive symptoms [17,24]	1.26[g]	1.63[g] / 2.68[h]
High impulsivity [14,9]	1.03[e]	1.28[d]
Affective and interpersonal psychopathy [14]	1.13[d]	-
Self-esteem [16]	0.92[c]	-
High Empathy [14]	0.89[d]	-
Difficult temperament [22,25]	0.88[i] / 1.44[b]	-
Locus of control [16]	0.77[c]	-
Facets of Extroversion: [1]		
High Optimism	-	0.14[j] [LR] / 0.06[j] [LR]
High Expressiveness	1.04[j]	17.08[j]
High Energy	1.66[j]	4.70[j]
High Shyness	2.26[j] / 0.42[j]	-
Facets of Benevolence:[1]		
High Compliance	-	0.16[j] [LR] / 0.05[j] [LR]
High Irritability	2.94[j]	8.92[j]
High Altruism	2.30[j] / 1.17[j]	-
High Egocentrism	2.28[j] / 1.21[j]	-
High Dominance	1.28[j] / 1.03[j]	-
Facets of Conscientiousness: [1]		
High Perseverance	0.78[j]	0.10[j] [LR]
High Orderliness	0.57[j]	0.19[j] [LR]
High Concentration	0.25[j]	0.21[j] [LR]
Achievement motivation	1.41[j] / 0.81[j]	-
(Neuro)Cognition/Academic achievement		
Low academic achievement [24]	1.04[g]	1.59[g]
Low cognitive functioning [25]	0.70[b]	-
Boys **Emotional/Personality**		
Sensation-seeking [12]	-	1.62[k]
Girls **Physical/Physiology**		
Low resting Heartrate [20]	NR[l]	-
Emotional/Personality		
ADHD diagnosis [20]	-	51.80[l]
Undercontrolled temperament [20]	-	2.40[l]
High Surgency/extraversion (high activity level, impulsivity, high intense pleasure-	-	1.67[m]

Sample / Domain / Predictive variable	Odds ratio	
	Non-significant	Significant (p <.05)
seeking, low shyness) [2]		
Sensation-seeking [12]	-	1.37[k]
High negative affectivity (sadness, discomfort, fear, frustration, unshoothability) [2]	1.26[m]	-
Low Effortful control [2]	0.87[m]	-
Low empathy [2]	0.83[m]	-
(Neuro)Cognition/Academic achievement		
Low IQ [20]	-	5.10[l]
Low reading achievement [20]	-	NR[l]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.
[a](Tabone et al., 2011). [b](Roisman et al., 2010). [c](Maldonado-Molina et al., 2009). [d](López-Romero et al., 2015). [e](Higgins et al., 2010). [f](Malti et al., 2013). [g](Reingle, Maldonado-Molina, et al., 2012). [h](Maldonado-Molina, Reingle, et al., 2010). [i](Petitclerc et al., 2009). [j](Becht et al., 2016). [k](Jennings, Maldonado-Molina, Piquero, et al., 2010). [l](Odgers et al., 2008). [m](Boutin et al., 2017).

Significant predictors were also observed in the physical/physiology domain in two studies that considered mixed samples. One examined rule-breaking behaviour from 0 to 15 years in the general population (worse physical health), and the other one examined rule-breaking and physical aggression together from 4 to 10 years among children reported for child protective services for maltreatment (developmental disability). Significant predictors within the (neuro)cognition/academic achievement domain were reported in two studies. One study examined physical aggression from 12 to 14 years in a mixed sample of African American and Hispanic youth in the general population (low academic achievement), and the other study examined theft, vandalism, and physical aggression together from 7 to 15 years in a community sample of girls (low IQ and low reading achievement).

Antisocial behaviour profile group 8: Antisocial-desistors (AD). In 17 of the 31 included studies, AD trajectories were identified after ensuring that the ASB profile did not fit within the CL ASB profile group (see Tables 4.14 and 4.15). Table 4.14 summarises results from these 17 studies, whereby 31 possible socio-environmental predictive variables were examined in studies considering mixed samples, 20 in samples of boys, and 19 in samples of girls. Of such variables, 17 (55%) in mixed samples, nine (45%) in boys, and five

(26%) in girls significantly differentiated AD from no/low-stable ASB. More specifically, variables in the family and peer domains were the most studied and more numerous among these significant variables. For instance, the following variables had both the most consistent (no study reported non-significant estimates for them) and the strongest effects (OR => 2.00) across family and peer domains: Mixed samples - A better understanding of reasons for having friends (OR = 4.39), a better understanding of friendship closeness (OR = 2.84), having friends who use alcohol (OR = 2.57). Boys - Neglectful parenting style (OR = 11.47), more quantity of family-related maltreatment indicators (OR = 6.70), authoritarian parent style (OR = 5.47). Girls - Parental hostility (OR = 4.05), more quantity of family-related maltreatment indicators (OR = 2.80).

Table 4.14 also shows that significant variables were reported across the school, neighbourhood/community, and sexual behaviour domains in three studies that considered mixed samples. The first study found one significant predictor in the school domain (school suspension) in examining rule-breaking behaviour from 11 to 16 years in poor inner-city African Americans. The second study found two significant predictors in both the school and the sexual behaviour domains (positive school adjustment and child had/has a steady boy/girlfriend, respectfully) in examining physical and verbal aggression together from 10 to 19 years in Indigenous youth. The third study found one significant predictor in the neighbourhood/community domain (living with both mother and father in the house) in examining a composite of physical and verbal aggression from 12 to 14 years among youth from low-income families.

Table 4.14

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Antisocial-Desisters ASB Relative to a No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p <.05)
Family			
Mixed sample	Poor parental monitoring of child behaviour [21]	-	1.30[a]
	Access to alcohol at home [24]	-	0.65[b] [LR]
	Living with both mother and father in the house [15]	-	0.61[c] [LR]
	Latino background and speaks Spanish at home [24]	-	0.54[b] [LR]
	Low maternal sensitivity to child's needs [25]	-	0.45[d]
	Lives with a single caregiver [25]	-	0.38[d]
	Married parents [6]	-	0.32[e] [LR] / 0.14[e] [LR]
	Overreactive parenting [1]	2.13[f]	7.90[f] / 6.76[f]
	Daily amount of time without adults around [24,15]	1.03[c] / 0.94[c]	1.69[b]
	Maternal authoritarian parenting [6]	1.83[e] / 2.22[e]	-
	Mother good conflict and problem-solving skills [6]	1.71[e] / 2.02[e]	-
	Maternal permissive parenting [6]	1.39[e] / 3.09[e]	-
	Perceived parental rejection [28]	1.06[g]	-
	Parental involvement with child's life [24]	0.98[b]	-
Boys	Neglectful parenting style [10]	-	11.47[i]
	Quantity of maltreatment indicators (rejecting mother-child interaction, harsh discipline, 2+ changes in the primary caregiver) [20]	-	6.70[h]
	Authoritarian parent style [10]	-	5.47[i]
	Supportive parenting and quality home environment [26]	-	0.87[j] [LR]
	Family physical and verbal conflict [20]	-	NR[h]
	Inconsistent discipline (mother vs. father) [20]	-	NR[h]
	Mother affective distress and somatic response [20]	-	NR[h]
	Mother low IQ [20]	1.80[h]	-
	High Interparental violence and aggression [26]	1.55[j]	-
	Lives with a single parent [7]	1.05[k]	-
	Maternal depressive symptoms [26]	1.04[j]	-
	Authoritative parenting style [7]	0.98[k]	-
	Parent self-report of a criminal conviction [2]	0.80[l]	-
Girls	Parental hostility [2]	-	4.05[l]
	Quantity of maltreatment indicators (rejecting mother-child interaction, harsh discipline, 2+ changes in the primary caregiver) [20]	-	2.80[h]
	Mother low IQ [20]	-	1.80[h]
	Inconsistent discipline (mother vs. father) [20]	-	NR[h]
	Mother affective distress and somatic response[20]	-	NR[h]
	Parental Neglect [2]	2.53[l]	-
	Parent's self-report of a criminal conviction [20]	1.90[h]	-
	Lack of adult supervision [11]	1.12[m] / 0.98[m]	-
	Non-intact family (blended or single-parent family)[2]	1.08[l]	-
	Number of family moves [2]	1.03[l]	-
	Both biological parents at home [11]	0.81[m] / 0.68[m]	-
	Low Parental warmth [2]	0.46[l]	-
	Parental rejection [2]	0.36[l]	-

Domain / Sample / Predictive variable		Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
	Family physical and verbal conflict [20]	NR[h]	-
School			
Mixed sample	School suspension [21]	-	3.56[a]
	Positive school adjustment [28]	-	0.67[g] [LR]
	Failed a class at school [28]	1.54[g]	-
	School expulsion [21]	1.08[a]	-
Boys	Participation in advanced classes at school [7]	1.36[k]	-
Girls	Receives free or low-price school lunch [11]	1.46[m] / 1.18[m]	-
Peer			
Mixed sample	Better understanding of reasons for friends [19]	-	4.39[n]
	Better understanding of friendship closeness [19]	-	2.84[n]
	Having friends who use alcohol [24]	-	2.57[b]
	Greater number of friends who drink alcohol [15]	-	1.90[c]
	Delinquent peer associations [28,24]	1.20[b]	2.05[g]
	Better understanding of friendship trust [19]	1.64[n]	-
	Self-reported good-quality friendship [19]	1.40[n]	-
	Friend-reported good-quality friendship [19]	1.32[n]	-
	Better understanding of friendship termination [19]	1.23[n]	-
	Friend aggressive behaviour [19]	1.11[n]	-
	Understanding of friendship conflict resolution [19]	1.03[n]	-
Boys	Greater number of delinquent peers [7]	-	1.10[k]
	Delinquent peer association [20]	-	NR[h]
Girls	Number of friends who drink alcohol [11]	1.53[m] / 2.42[m]	-
	Peer rejection [2]	1.09[l]	-
	Peer acceptance [2]	1.06[l]	-
	Delinquent peer association [20]	NR[h]	-
Neighbourhood/Community			
Mixed sample	Adult alcohol use in the neighbourhood [24]	0.81[b]	-
Boys	Neighbourhood impoverishment (low income, unemployment, low education) [26]	1.35[j]	-
	Death of friends/family [7]	1.22[k]	-
	Frequency of Experiencing racial discrimination [7]	1.04[k]	-
	Moving houses in the past year [7]	0.72[k]	-
Sexual behaviour			
Mixed sample	Had/has a steady boy/girlfriend [28]	-	2.76[g]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.

[a](Park et al., 2010). [b](Reingle, Maldonado-Molina, et al., 2012). [c](Maldonado-Molina, Jennings, et al., 2010). [d](Roisman et al., 2010). [e](Ehrenreich et al., 2014). [f](Becht et al., 2016). [g](Sittner & Hautala, 2016). [h](Odgers et al., 2008). [i](Hoeve et al., 2008). [j](Shaw et al., 2012). [k](Evans et al., 2016). [l](Boutin et al., 2017). [m](Jennings, Maldonado-Molina, & Komro, 2010). [n](Malti et al., 2015).

As shown in Table 4.15, 24 individual-level predictive variables were examined in mixed samples, with five studied in samples of boys, and nine in samples of girls. Out of such variables, seven (29%) in mixed samples, four (80%) in boys, and three (33%) in girls significantly differentiated AD from no/low-stable ASB. In particular, predictors in the emotional/personality domain were the most studied and more numerous among the significant effects when considering mixed samples (*n* = 21 / 4 significant: Behavioural psychopathy, hopelessness, high energy - risks, high compliance - predictors), boys (*n* = 2 / 2 significant: Uncontrolled temperament, ADHD diagnosis), and girls (*n* = 6 / 1 significant: ADHD diagnosis). Significant variables were also reported in physical/physiology and (neuro)cognition/academic achievement domains in two studies considering mixed samples; one study examined rule-breaking from birth to 15 years in a community sample (worse physical health and low cognitive functioning), and the other study focused on physical aggression from 12 to 14 years in a community sample of African American and Hispanic youth (low academic achievement). The same two significant variables were reported in the (neuro)cognition / academic achievement domain in a study of two community samples that examined a composite of theft, vandalism, and physical aggression from 7 to 26 years in boys and from 7 to 15 in girls (child's low IQ and low reading achievement).

Table 4.15

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of Antisocial-Desisters ASB Relative to No/Low-Stable ASB

Sample / Domain / Predictive variable	Odds ratio	
	Non-significant	Significant (p <.05)
Mixed sample **Physical/Physiology**		
Worse physical health [25]	-	0.39[a]
Emotional/Personality		
Behavioural psychopathy [14]	-	1.72[b]
Hopelessness [21]	-	1.54[c]
Facets of Extroversion: [1]		
High Energy	-	8.69[d]
High Expressiveness	1.85[d]	-
High Shyness	1.46[d]	-
High Optimism	0.27[d]	-
Facets of Benevolence:[1]		
High Compliance	-	0.12[d] [LR]
High Irritability	5.09[d]	-
High Dominance	2.19[d]	-
High Egocentrism	1.24[d]	-
High Altruism	1.13[d]	-
Facets of Conscientiousness:[1]		
High Perseverance	1.68[d]	-
Achievement motivation	0.96[d]	-
High Concentration	0.76[d]	-
High Orderliness	0.30[d]	-
High Impulsivity [14]	1.24[b]	-
Depressive symptoms [24]	1.10[e]	-
Affective and interpersonal psychopathy [14]	1.06[b]	-
Difficult temperament [25]	0.90[a]	-
Self-worth perception [21]	0.86[c]	-
High Empathy [14]	0.80[b]	-
(Neuro)Cognition/Academic achievement		
Low academic achievement [24]	-	1.82[e]
Low cognitive functioning [25]	-	0.42[a]
Boys **Physical/Physiology**		
Low resting Heartrate [20]	NR[f]	-
Emotional/Personality		
Uncontrolled temperament [20]	-	4.70[f]
ADHD diagnosis [20]	-	4.30[f]
(Neuro)Cognition/Academic achievement		
Low IQ [20]	-	4.00[f]
Low reading achievement [20]	-	NR[f]
Girls **Physical/Physiology**		
Low resting Heartrate [20]	NR[f]	-
Emotional/Personality		
ADHD diagnosis [20]	-	14.40[f]

Sample / Domain / Predictive variable	Odds ratio	
	Non-significant	Significant (p <.05)
Uncontrolled temperament [20]	2.00[f]	-
High Surgency/extraversion (high activity level, impulsivity, high intense pleasure-seeking, low shyness) [2]	1.31[g]	-
High negative affectivity (sadness, discomfort, fear, frustration, unshoothability) [2]	1.18[g]	-
Low Empathy [2]	0.95[g]	-
Low Effortful control [2]	0.65[g]	-
(Neuro)Cognition/Academic achievement		
Low IQ [20]	-	2.90[f]
Low Reading achievement [20]	-	NR[f]

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.
[a](Roisman et al., 2010). [b](López-Romero et al., 2015). [c](Park et al., 2010). [d](Becht et al., 2016). [e](Reingle, Maldonado-Molina, et al., 2012). [f](Odgers et al., 2008). [g](Boutin et al., 2017).

Antisocial behaviour profile group 9: Medium-level (ML). Fifteen of the 31 studies found an ML ASB profile group, which did not conform to any of the other ASB profile groups, as primarily defined in this review (see Tables 4.16 and 4.17). As shown in Table 4.16, 26 possible socio-environmental predictors of ML ASB were examined in studies of mixed samples, and 14 were studied in samples of boys - none in samples of girls. In total, nine of such variables (35%) in mixed samples and five (36%) in boys significantly differentiated ML from no/low-stable ASB. Such significant variables were reported in the family (n = 2; Parent coercive discipline - risk, the child is from a Latino background and speaks Spanish at home - protective), school (n = 2; teacher-child interpersonal conflict, negative school environment), peer (n = 2; a greater number of friends who drink alcohol, delinquent peer association), and neighbourhood/community (n = 3; exposure to violence, adult's alcohol use in the neighbourhood – risks, social support - protective) domains in studies examining ASB in mixed samples of youth in the general population.

Table 4.16

Results from Studies using Multinomial Regression to Test Socio-Environmental Level Predictors of Medium-Level ASB Relative to No/Low-Stable ASB

		Odds ratio	
Domain / Sample / Predictive variable		Non-significant	Significant (p<.05)
Family			
Mixed sample	Parent coercive discipline [16]	-	2.18[a]
	Latino background and speaks Spanish at home [17]	-	0.39[b] [LR]
	Hours without adult supervision [17,24]	1.16[b] / 0.94[d]	-
	Live with both parents in the house [17]	1.05[b]	-
	Parent-child interaction [16]	1.03[a]	-
	Child-mother positive attachment [9]	1.01[c]	-
	Parental involvement in child's life [17,24]	0.99[d] / 0.96[b]	-
	Child-father positive attachment [9]	0.95[c]	-
	Maternal control and punitiveness toward the child[5]	0.76[e]	-
	Low maternal responsiveness [27]	0.73[f]	-
Boys	Broken home or family transitions (parental separation/divorce, change in caregiver, frequent moves) [30]	-	1.82[g]
	Lack of adult supervision [11]	1.23[h] / 1.19[h]	-
	Both biological parents at home [11]	0.60[h] / 0.60[h]	-
	Authoritarian parent style [10]	B = 0.59[i]	-
	Neglectful parenting style [10]	B = 0.55[i]	-
School			
Mixed sample	Teacher-child interpersonal conflict [27]	-	2.17[f]
	Negative school environment [9,16]	0.99[c]	1.11[a]
	Teacher-child closeness [27]	0.92[f]	-
Boys	Receives free or low-price school lunch [11]	0.90[h] / 1.11[h]	-
Peer			
Mixed sample	Number of friends who drink alcohol [17]	-	2.23[b]
	Delinquent peer association [9,16]	1.90[a]	1.44[c]
	Having friends who use alcohol [24]	1.44[d]	-
	Gang membership [24]	1.42[d]	-
	Peer relationships [16]	1.17[a]	-
	Peer acceptance [27]	1.04[f]	-
Boys	Greater number of friends who drink alcohol [11]	1.34[h] / 1.19[h]	-
Neighbourhood/Community			
Mixed sample	Youth exposure to violence [16]	-	1.18[a]
	Social support [16]	-	0.47[a] [LR]
	Adult alcohol use in the neighbourhood [17,24]	1.35[d]	1.41[b]
	Acculturation of Latino youth into USA language and culture [16]	1.22[a]	-
	Stressful life-events [16]	1.06[a]	-
	Number of off-premise alcohol outlet in the neighbourhood [24]	0.82[d]	-
	Youth's welfare [16]	0.82[a]	-
Boys	Onset diversion [8]	-	1.83[j]
	Onset-edocc (neighbourhood educational and occupational disadvantage) [8]	-	1.32[j]

Domain / Sample / Predictive variable	Odds ratio	
	Non-significant	Significant (p<.05)
Onset-city (Location of first offence: Perth) [8]	-	0.93[j] [LR]
Onset-north (Location of first offence: western Australia [8]	-	0.91[j] [LR]
Involvement with alternative care (institutional, foster, child welfare care) [30]	1.17[g]	-
Onset-resource (deficient educational and occupational characteristics in the community) [8]	NR[j]	-
Onset-disadvantage (neighbourhood economic disadvantage) [8]	NR[j]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.
[a](Maldonado-Molina et al., 2009). [b](Maldonado-Molina, Reingle, et al., 2010). [c](Higgins et al., 2010). [d](Reingle, Maldonado-Molina, et al., 2012). [e](Degnan et al., 2008). [f](Silver et al., 2010). [g](Ward et al., 2010). [h](Jennings, Maldonado-Molina, & Komro, 2010). [i](Hoeve et al., 2008). [j](Ferrante, 2013).

Significant predictors were also reported in both the family and neighbourhood/community domains for offender boys in two studies. One of them examined records of conviction from 8 to 32 years (broken home or multiple family transitions), and the other study examined police charge and arrest from 10 to 30 years (Onset diversion – risk, see Ferrante, 2003 for definitions, neighbourhood educational and occupational disadvantage; locations of first offence: Perth and Western Australia - protectives).

Table 4.17 summarises findings for 11 possible individual-level predictors; all tested in studies considering mixed samples. Overall, five significant associations were found within the emotional/personality domain in four studies: Sensation-seeking, risk-taking, depressive symptoms – risks, and high trustfulness and trustworthiness - protectives. No study considered individual-level predictors in samples of boys or girls.

Table 4.17

Results from Studies using Multinomial Regression to Test Individual-Level Predictors of Membership in Medium-level ASB Relative to No/Low-Stable ASB

Sample / Domain / Predictive variable		Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
Mixed sample	**Physical/Physiology**		
	Physiological marker of emotion regulation [a] [5]	0.79[b]	-
	Emotional/Personality		
	Sensation seeking [16]	-	1.46[c]
	Risk-taking [9]	-	1.22[d]
	High Trustfulness [18]	-	0.61[e] [LR]
	High Trustworthiness [18]	-	0.09[e] [LR]
	Depressive symptoms [17,24]	1.23[g]	1.91[f]
	High reactivity to frustration [9]	2.16[d]	-
	High Impulsivity [9]	1.07[d]	-
	Locus of control [16]	1.05[c]	-
	Self-esteem [16]	0.97[c]	-
	(Neuro)Cognition/Academic achievement		
	Low academic achievement [4,24]	1.04[g] / 0.58[h]	-

Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group.
[a]Decrease in Respiratory sinus arrhythmia. [b](Degnan et al., 2008). [c](Maldonado-Molina et al., 2009). [d](Higgins et al., 2010). [e](Malti et al., 2013). [f](Maldonado-Molina, Reingle, et al., 2010). [g](Reingle, Maldonado-Molina, et al., 2012). [h](Day et al., 2012).

Interaction effects predicting membership in various antisocial behaviour profile groups. Table 4.18 summarises the findings for interaction effects. All such effects were tested and reported in three of the 31 studies reviewed; all focused on mixed samples. Overall, most interaction effects were not significant. The exceptions were five significant effects reported in three studies for trajectories within ASB profile groups 1 (*no/low-stable*: NL ASB), 2 (*child-limited:* CL ASB), 4 (*life-course-persistent:* LCP ASB), and 6 (*high-stable:* HS ASB). In the first study, the authors examined a composite of physical aggression and ODD in children that were two to five years in out-of-home placement. Two interactions were significant. In the first interaction (maternal control × reactivity to frustration), high maternal control combined with a higher level of reactivity to frustration increased the likelihood of membership in NL relative to HS ASB.

Table 4.18

Results from Studies using Multinomial Regression to Test Interactions between Variables as Predictors of Eight different ASB Profile Groups Relative to No/Low-Stable ASB

		Odds ratio	
Profile group / Sample / Predictive variable		Non-significant	Significant (p <.05)
ASB Profile Group 1: No /low stable			
Mixed sample	Maternal control × Reactivity to frustration [5]	-	0.25[b] [LR]
	Maternal control × Physiological emotion regulation[a] [5]	0.75[b]	-
	Reactivity to frustration × Physiological emotion regulation[a] [5]	0.33[b]	-
ASB Profile Group 2: Child-limited			
Mixed sample	Reactivity to frustration x Physiological emotion regulation[a] [5]	-	11.87[b]
	Maternal control × reactivity to frustration [5]	2.33[b]	-
	Maternal control × Physiological emotion regulation[a] [5]	0.97[b]	-
ASB Profile Group 3: Adolescent-limited			
Mixed sample	Attends to religious services with parents and parents think this is important × parent praises and show affection towards child [23]	1.00[d]	-
	Higher frequency of religious participation × living with a single caregiver [23]	0.91[d]	-
	Higher frequency of religious participation × living with stepfamily [23]	0.89[d]	-
ASB Profile Group 4: Life-course persistent			
Mixed sample	Attends to religious services with parents and parents think this is important × parent praises and show affection towards child [23]	-	0.95[d] [LR]
	Higher frequency of religious participation × living with a single caregiver [23]	-	0.80[d] [LR]
	Higher frequency of religious participation × living with stepfamily [23]	0.88[d]	-
ASB Profile Group 6: High stable			
Mixed sample	OVR × Compliance [1]	2.23[c]	21.50[c]
	OVR × Perseverance [1]	8.00[c] / 1.22[c]	-
	OVR × Optimism [1]	4.74[c] / 1.45[c]	-
	OVR × Expressiveness [1]	3.51[c] / 0.12[c]	-
	OVR × Orderliness [1]	3.32[c] / 2.81[c]	-
	OVR × Shyness [1]	2.35[c] / 0.14[c]	-
	OVR × Dominance [1]	2.16[c] / 1.29[c]	-
	OVR × Energy [1]	1.41[c] / 0.45[c]	-
	OVR × Egocentrism [1]	1.31[c] / 0.70[c]	-
	OVR × Irritability [1]	1.30[c] / 0.53[c]	-
	OVR × Concentration [1]	1.16[c] / 0.48[c]	-
	OVR × Altruism [1]	1.08[c] / 0.78[c]	-
	OVR × Achievement motivation [1]	0.78[c] / 0.47[c]	-
ASB Profile Group 8: Antisocial - desistors			

Profile group / Sample / Predictive variable	Odds ratio	
	Non-significant	Significant (p <.05)
Mixed sample OVR × Compliance [1]	9.38[c]	-
OVR × Expressiveness [1]	4.07[c]	-
OVR × Achievement motivation [1]	3.42[c]	-
OVR × Irritability [1]	2.48[c]	-
OVR × Orderliness [1]	2.41[c]	-
OVR × Dominance [1]	2.18[c]	-
OVR × Egocentrism [1]	2.04[c]	-
OVR × Altruism [1]	1.08[c]	-
OVR × Concentration [1]	0.59[c]	-
OVR × Shyness [1]	0.56[c]	-
OVR × Energy [1]	0.48[c]	
OVR × Optimism [1]	0.26[c]	-
OVR × Perseverance [1]	0.20[c]	-
ASB Profile Group 9: Medium-level-stable		
Mixed sample Reactivity to frustration × Physiological emotion regulation[a] [5]	2.78[b]	-
Maternal control × Reactivity to frustration [5]	1.50[b]	-
Maternal control × Physiological emotion regulation[a] [5]	0.90[b]	-

Note. Note. ASB = antisocial behaviour. Numbers in brackets link to Table 4.1. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group.
NR = Estimate not reported. OVR = Overreactive (irritability, anger and frustration) parenting.
[a]Decrease in Respiratory sinus arrhythmia. [b](Degnan et al., 2008). [c](Becht et al., 2016). [d](Petts, 2009).

In the second interaction (reactivity to frustration × physiological emotion regulation), high reactivity to frustration combined with high physiological emotion regulation increased the risk of CL relative to NL ASB. In the second study, the authors examined a composite of vandalism and theft from ages 10 to 25 in a community sample of black and Hispanic youth and found that two measures of religion were relevant when combined with family factors. In the first interaction, a combination of a high frequency of attending religious services and receiving praise and affection from parents was interactive protection against LCP. In the second interaction, a combination of a high frequency of attending religious services and living with a single caregiver was protective against LCP.

Finally, in the third study, the authors examined a composite of rule-breaking behaviour, and physical and verbal aggression from ages nine to 15 in a community sample and found that youth with a combination of parents who overreact to situations with high

irritability, anger, and frustration and a high level of child compliance had an elevated risk of membership in HS, relative to NL ASB.

Comparing Significant Childhood Predictors Across Distinct ASB Profile Groups

Along the previous sections, a detailed description was provided of sex-specific childhood predictors tested for their association with an increase in the likelihood of membership in nine distinct ASB profile groups in relation to no/low-stable ASB. Next, to provide a closer look into childhood predictors of distinct ASB profile groups that is beyond LCP and AL, all significant variables reported for the profile groups with the highest level and most chronic ASB (i.e., LCP and HS) in relation to other ASB profile groups (i.e., AL, CL, ML) will be examined within two sets of comparisons: 1- LCP, AL, and CL and 2- HS and ML. Such examinations consisted of reporting on similarities and differences across the predictors of the trajectories within each set of ASB profile groups. More specifically, the two sets of comparisons focused on the strength and magnitude of overlapping childhood predictors (i.e., qualitative differences) and the number of significant predictive variables (i.e., quantitative differences) for each ASB profile.

Importantly, the decision about the compatibility of the specific ASB profile groups compared within each of these two sets was made with basis on (a) their theoretical definition, to ensure they would be mutually exclusive groups, and (b) the number of studies in this review that reported ASB trajectories within each ASB profile group, to minimize, as much as possible, that eventual differences in the number of predictors found between ASB groups were due to a greater number of studies testing predictors of one or another ASB group. Also, given that all included studies in this review had examined ASB trajectories from childhood, the following comparisons aim to be an overview of childhood predictors of overall developmental change in ASB from (or, in few cases, within) childhood years. For information purposes, a summary table of all significant predictors reported for each ASB

profile group in this review, which has guided the following comparison analyses is provided in Appendix B.

Comparison of predictors of LCP, AL and CL ASB profile groups. Overall, regarding the strength of effects, the three strongest predictors of the LCP group were two individual-level factors of ADHD diagnoses (OR = 18.70) and low IQ (OR = 5.80), and one socio-environmental factor – the quantity of family-related maltreatment indicators (OR = 14.50). The three strongest predictors of the AL group were all socioenvironmental, including neglectful parenting style (OR = 7.10), ever having utilized the drug and alcohol treatment system (OR = 3.06), a greater number of friends who drink alcohol (OR = 2.47). The three strongest predictors of the CL group were also within the socio-environmental level. These included acculturation of Latino youth into USA language and culture (OR = 5.80), parent coercive discipline (OR = 4.41), and delinquent peer association (OR = 2.27). Thus, the strongest predictors reported here tended to be factors within the individual-level in the LCP group and within the socio-environmental level (i.e., in the family, peer, and neighbourhood/community domains) in both the AL and the CL ASB groups. Also, the strongest effects were found in analyses predicting LCP or CL compared to analyses of AL.

Notably, and in line with previous reviews (Fairchild et al., 2013; Farrington, 2015; Jolliffe, Farrington, Piquero, Loeber, et al., 2017), the differences in childhood predictors across LCP, AL, and CL profile groups were generally quantitative. However, contrary to such studies and consistent with the developmental taxonomic theory (Moffitt, 1993; 2006), some evidence of qualitative differences in childhood predictors of ASB were also found between LCP and AL. The most consistent evidence of such quantitative differences was found among significant predictors within the socio-environmental level for all LCP, AL, and CL ASB profile groups. However, the domain of influence did differ across the three ASB groups, which may also indicate qualitative differences in the predictive domain - but not in

the variable level. Significant predictors tended to be in the neighbourhood/community domain for LCP (*n* = 13; e.g., a higher score in the PRA-V1 (Primary Risk Assessment - version 1, see Yessine & Bonta, 2009, for details on this measure), involvement with alternative care such as foster or child welfare care, and unstable living arrangements – risks) and CL (*n* = 3; exposure to violence, acculturation of Latino youth into USA language and culture – risks, and more social support – protective) ASB profile groups. In contrast, significant predictive variables for the AL group tended to be in the family domain (n = 4; living with a single caregiver, more parent-child arguments over rules, neglectful parenting style – risks, and higher education level of parents – protective). Interestingly, only one variable in the school domain (negative school environment) significantly predicted membership in the CL group, but none was significantly associated with LCP or AL ASB profiles. Also, variables in the sexual behaviour domain were not significantly associated with these three ASB profile groups.

Notably, the one predictor within the socio-environmental level that overlapped across CL, AL, and LCP ASB groups was delinquent peer association (OR = 4.63, LCP; OR = 1.83, AL; OR = 2.27, CL). Other socio-environmental level predictors overlapped across two ASB profile groups (LCP / AL, respectively). These were living with a single caregiver (OR = 3.14 / OR = 1.84), a greater number of child welfare placement (OR = 1.12 / OR = 1.07), ever having utilized the drug and alcohol treatment system (OR = 2.33 / OR = 3.06). These results support that the strongest effects are generally found in the LCP, but this may change for different predictors.

Child individual-level predictors tended to be in the (neuro)cognition/academic achievement domain for the LCP (*n* = 3; low IQ, reading achievement, and academic achievement – risks) and in the emotional/personality domain for both AL (*n* = 1; depressive symptoms – risk) and CL (*n* = 3; sensation-seeking – risk, high trustfulness and

trustworthiness – protective) groups. Interestingly, variables in the physical/physiological (low resting rate) and (neuro)cognition/academic achievement (see above) domains were only related to LCP. Also, regarding comparing significant effects, no predictors were found within the individual-level that overlapped across the three ASB profile groups. Therefore, in line with the developmental taxonomic theory (Moffitt, 1993, 2006), these could be evidence that possible qualitative differences that are in addition to quantitative differences exist among individual-level predictors of LCP compared to AL and CL. However, given much less prospective studies were found that have tested individual-level compared to socio-environmental predictors of ASB groups, it may also be premature to assert that qualitative differences in predictors of ASB profile groups do exist among individual-level, but not among socio-environmental variables.

In sum, although most differences in childhood predictors across LCP, AL, and CL ASB profile groups tended to be quantitative, evidence for qualitative differences in the effects are found when specific domains of influence are considered. Childhood predictors that most strongly differentiated between the LCP and the AL group tended to be in both neighbourhood/community and (neuro)cognition/academic achievement domains. Also, the childhood predictors that most strongly differentiated between the AL ASB group and both LCP and CL ASB groups tended to be in both family and emotional/personality domains. Furthermore, the strongest effect sizes were reported for the LCP group, but this was not unanimous across all predictors. Generally, significant predictors of these three ASB profile groups tended to be within the socio-environmental level compared to the individual-level. However, this could also be a result of the greater attention placed on socio-environmental variables across the included studies.

Comparison of childhood predictors between HS and ML ASB profile groups. Individuals in trajectories within the HS ASB profile group had the highest levels of ASB

within their corresponding sample that remained high (i.e., did not decline) across all or almost all waves of data collection. Hence, ASB trajectories in this profile may be a mix of LCP and AL, but a decision about one or the other simply could not be made due to study duration limitation. In contrast, trajectories within the ML profile showed ASB levels that were just above low but that never reached the high range, being stable within the medium level in comparison to other trajectories identified within the same sample. Thus, consideration of study duration or age of onset was not the focus of comparing HS and ML groups. Instead, these two ASB profile groups are compared here to shed light on possible differences in childhood predictors reported as significant for two levels of stable ASB (the highest and the moderate level), whenever they were reported in prospective studies that started their data collection in childhood, regardless of the age range of the study.

Regarding the strength of significant effects, the strongest predictors tended to be variables within the individual-level in the HS group. For instance, ADHD diagnoses (OR = 51.80), overreactive parenting (OR = 18.45), and high levels of the personality trait of expressiveness (OR = 17.08). In contrast, the strongest significant predictors of the ML ASB group were variables within the socio-environmental level. These included a greater number of friends who drink alcohol (OR = 2.23), parent coercive discipline (OR = 2.18), and teacher-child interpersonal conflict (OR = 2.17). In addition, the effects sizes were generally much stronger for the HS group compared to the ML group. Regarding the number of significant predictors of HS and ML, a considerably greater quantity of variables within both the socio-environmental and the individual level were significant predictors of HS (n = 59, n = 23, respectively) when compared to the number of significant predictors of ML (n =18, n = 5, respectively). Although such findings corroborate previous evidence that the strongest effect sizes are consistently reported to highest levels of chronic ASB in relation to low ASB levels (Assink et al., 2015; Jolliffe, Farrington, Piquero, Loeber, et al., 2017). Interpretation

of the quantitative differences in childhood predictors found between HL and ML requires taking into consideration that a greater number studies in this review have reported ASB trajectories within the HS (*n* = 23) compared to ML (*n* = 15). Although less probable, the quantitative differences found here could also just indicate that possibly a fewer number of predictors have been tested for ML compared to HS ASB.

A closer look at the number of significant variables within the socio-environmental level across the HS and ML profile groups shows that most childhood predictors of HS tended to be in the family domain (*n* = 21; e.g., overreactive parenting, a greater quantity of family-related maltreatment indicators, and maternal permissive parenting style). In comparison, childhood predictors for the ML group tended to be in the neighbourhood/community domain (*n* = 6; e.g., neighbourhood educational and occupational disadvantage, adult's alcohol use in the neighbourhood, and exposure to violence). The following socio-environmental level predictors were tested for both HS and ML in prospective studies (OR reported respectively). Parent coercive discipline (OR = 1.86 / OR = 2.18), being from a Latino background and speaking Spanish at home (OR = 0.56 / OR = 0.39), teacher-child interpersonal conflict (OR = 12.95 / OR = 2.17), more number of friends who drink alcohol (OR = 3.43 / OR = 2.23), delinquent peer association (OR = 2.94 / OR = 1.44), exposure to violence (OR = 1.21 / OR = 1.18), adult's alcohol use in the neighbourhood (OR = 1.87 / OR = 1.41). As can be seen by examining these ORs, the effects are generally stronger when focused on predicting HS rather than ML, but predictors are found across most domains within the socio-environmental level for both profile groups.

Within the individual level, the greatest numbers of childhood predictors tended to be in the emotional/personality domain for both the HS (*n* = 18; e.g., ADHD diagnose, high expressiveness, and high irritability) and the ML (*n* = 5; e.g., depressive symptoms, sensation-seeking, and risk-taking) profile groups. Interestingly, in both

physical/physiological (worse physical health and developmental disability) and (neuro)cognition/academic achievement (low IQ and both academic and reading achievement) domains, predictors were only significant when the analyses focused on HS. Individual-level predictors that were tested for both HS and ML included sensation-seeking (OR = 1.81 / OR = 1.46), depressive symptoms (OR = 2.68 / OR = 1.91), risk-taking (OR = 1.40 / OR = 1.22), high trustfulness (OR = 0.40 / OR = 0.61), and high trustworthiness (OR = 0.03 / OR = 0.09).

To summarise, the results for comparing childhood predictors tested for HS and ML suggested that, just as was found for LCP predictors relative to AL and CL predictors, there are a greater number of risks within both socio-environmental and individual levels for HS, in relation to the number of significant predictors found for ML. In particular, childhood predictors that most strongly differentiated between the HS and the ML groups tended to be in family, physical/physiological, and (neuro)cognition/academic achievement domains. However, variables within the emotional/personality domain had considerably stronger effects in predicting the HS group when compared to the ML group.

Comparing Significant Childhood Predictors of ASB across Sex-Specific Samples

The previous set of comparative analyses presented a closer look into possible differences and similarities between significant predictive variables of the highest and more chronic ASB (i.e., LCP and HS) in relation to other ASB profile groups (i.e., CL, AL, and ML); hence expanding knowledge beyond the comparisons between LCP and AL ASB groups, usually reported in previous reviews. In the next set of comparison analyses, a different approach to the same data was taken to look further into possible sex-specificities in childhood predictors of ASB profiles by separately comparing variables that were significant risk and those that were protective factors for membership in an ASB trajectory above the no/low-stable levels of ASB across samples of boys and girls.

Comparison of sex-specific risk factors. Table 4.19 summarises the 59 significant risk factors that were consistent (i.e., no study reported a non-significant effect for them within the same profile group) predictors of at least one ASB profile group according to sample sex (mixed samples, boys-only, girls-only). These were reported in 25 of the 31 studies included in this review. Comparisons of sex-specific risks for ASB profiles are reported within the next paragraphs in the following order: First, an overview of the number and magnitude of the effects across the three sex-specific samples is presented. Next, childhood risks are compared across samples of boys and girls in terms of (a) number and kind of risks and (b) the magnitude of overlapping risks, while also attending to the specific ASB profile group predicted by them. In addition, such comparisons are presented separately for socio-environmental and individual-level risks. A summary of sex-specificities in risk factors for membership in ASB profile groups is also provided at the end of this section.

Overview of childhood risks across three sex-specific samples. Overall, a considerably greater number of risks was reported by studies considering mixed samples in relation to samples of boys, and the same was observed for samples of boys in relation to girls (Table 4.19). The three risk factors measured in childhood that most strongly predicted membership in ASB profile groups among mixed samples were teacher-child interpersonal conflict (OR = 12.95, HS), child's high energy (OR = 8.69, AD), and ever utilized the public mental-health care system (OR = 4.78, HS). The three risk factors that were the strongest predictors of boys' ASB were more quantity of family-related maltreatment indicators (OR = 14.50, LCP), neglectful parenting style (OR = 11.47, AD), and a higher score in the PRA-V1 (OR = 7.96, LCP; Primary Risk Assessment - version 1, see Yessine and Bonta (2009) for details on this measure). The three risk factors that were the strongest predictors among girls were ADHD

Table 4.19

Summary of Significant Results (p < .05) from 31 Studies using Multinomial Regression to Test Socio-Environmental and Individual-level risks for ASB Profiles in Relation to No/Low-Stable ASB by Sample Sex

	Odds ratio (ASB profile group)		
Domain / Sample / Predictive variable	Mixed samples	Samples of Boys	Samples of Girls
Socio-environmental level			
Family			
Maternal permissive parenting style	4.54[d] (HS)	-	-
Child lives with single caregiver	3.14[a] (LCP); 1.84[a] (AL); 0.38[b] (AD)	-	-
Parent Coercive discipline	2.18[f] (ML)	2.01[g], 1.91[g] (CL)	1.86[g] (HS)
Parent-child arguments over rules	1.40[a] (AL)	-	-
Child's perceived parental rejection	1.36[c] (HS)	-	-
Poor parental monitoring of child's behaviour	1.30[e] (AD)	-	-
Low maternal sensitivity to child's needs	0.59[b] (AO); 0.45[b] (AD); 0.38[b] (HS)	-	-
More quantity of maltreatment indicators	-	14.50[i], 4.00[i] (LCP); 6.70[i] (AD)	3.90[i] (AO); 7.80[i] (HS); 2.80[i] (AD)
Neglectful parenting style	-	11.47[h] (AD); 7.10[h] (AL); 2.03[h] (HS)	-
Authoritarian parenting style	-	5.47[h] (AD); 1.62[h] (HS)	-
Broken home or family transitions	-	1.82[k] (ML)	-
Mother's depressive symptoms	-	1.07[j] (HS)	-
Family's physical and verbal conflict	-	NR[i] (LCP, AO, AD)	NR[i] (HS)
Inconsistent discipline (child vs. mother)	-	NR[i] (AD)	NR[i] (LCP, AD)
Mother's affective distress and somatic response	-	NR[i] (AD)	NR[i] (AO, HS, AD)
Parental hostility	-	-	4.05[l] (AD)
Parents' self-report of criminal conviction	-	-	3.90[i] (HS)
Mother's low IQ	-	-	3.10[i] (HS); 2.40[i] (AO); 1.80[i] (AD)
School			
School suspension	3.56[e] (AD); 2.37[e] (AO)	-	-
Teacher-child interpersonal conflict	12.95[m] (HS); 2.17[m] (ML)	-	-
Peer			
More number of friends who drink alcohol	3.43[n], 1.73[o] (HS); 2.47[n] (AL); 2.23[n] (ML); 1.90[o] (AD)	-	-

Domain / Sample / Predictive variable	Odds ratio (ASB profile group)		
	Mixed samples	Samples of Boys	Samples of Girls
Having friends who drink alcohol	2.57[p] (AD)	-	-
Delinquent peer association	1.78[a] (LCP)	NR[i] (AD)	NR[i] (AO, HS)
Better understanding of reasons for having friends	4.39[q] (AD)	-	-
Gang membership	2.94[p], 2.00[p] (HS)	-	-
Better understanding of friendship closeness	2.84[q] (AD)	-	-
More number of delinquent friends	-	1.20[r] (HS); 1.16[r] (AO); 1.10[r] (AD)	-
Neighbourhood/Community			
Ever utilized public mental health care system	4.78[t], 2.72[t] (HS)	-	-
Ever utilized the drug and alcohol treatment system	2.33[s], 2.17[s] (LCP)	-	-
Adult's alcohol use in the neighbourhood	1.87[n], 1.75[p], 1.55[p] (HS); 1.49[n] (AL)	-	-
Youth exposure to violence	1.46[f], 1.21[f] (CL); 1.21[f] (HS); 1.18[f] (ML); 1.16[f] (AO)	1.34[g], 1.11[g] (CL); 1.16[f] (HS); 1.07[g] (AO)	1.74[g], 1.19[g] (CL); 1.20[g] (HS)
Onset resource (deficient educational and occupational characteristics in the community)	-	1.16[u] (LCP)	1.33[u] (LCP)
Onset edocc (neighbourhood educational and occupational disadvantage)	-	1.32 [u] (ML)	-
Onset diversion	-	1.83[u] (ML); 1.50[u] (LCP)	-
High risk in PRA-V1	-	7.96[v], 4.07[v] (LCP)	-
Involvement with alternative care	-	3.82[k], 3.14[k] (LCP)	-
Frequency of experiencing racial discrimination	-	1.07[r] (HS	-
Sexual Behaviour			
Child has/had a steady boy/girlfriend	2.76[c] (HS); 2.65[c] (HS)	-	-
Individual-level			
Physical/Physiological			
Worse physical health	0.63[b] (AO); 0.43[b] (HS); 0.39[b] (AD)	-	-
Developmental disability	3.28[t] / 2.39[t] (HS)	-	-
Emotional/personality			
High energy	8.69[w] (AD)	-	-
Depressive symptoms	2.28[n] (AL)	-	-

Domain / Sample / Predictive variable	Odds ratio (ASB profile group)		
	Mixed samples	Samples of Boys	Samples of Girls
Sensation-seeking	1.81[f] (HS); 1.46[f] (ML); 1.34[f] (CL); 1.28[f], 1.22[f] (AO)	1.62[g] (HS); 1.11[g] (AO)	1.78[g], 1.45[g] (CL); 1.37[g] (HS)
Behaviour psychopathy	1.72[x] (AD); 1.50[x] (HS)	-	-
Hopelessness	1.54[e] (AD)	-	-
Risk-taking	1.40[z] (HS); 1.22[z] (ML)	-	-
Uncontrolled temperament	-	4.70[i] (AD); 3.50[i], 3.20[i] (LCP)	2.40[i] (HS)
ADHD diagnoses	-	4.30[i] (AD)	51.80[i] (HS); 14.14[i] (AD)
High surgency/extraversion	-	-	1.67[l] (HS)
(Neuro)Cognition/Academic Achievement			
Low academic achievement	1.82[p] (AD)	-	-
Low cognitive functioning	0.42[b] (AD)	-	-
Low IQ	-	5.80[i], 2.30[i] (LCP); 4.00[i] (AD)	5.10[i] (HS); 2.90[i] (AD)
Low reading achievement	-	NR[i] (LCP, AD)	NR[i] (HS, AD)

Note. ASB = antisocial behaviour. In this table, only variables that were consistently reported as risks are reported. Consistent means that no study reported non-significant effects for them within the same ASB profile group. Mixed sample = boys and girls combined. NR = Estimate not reported. CL = Child-limited. AL = Adolescent-limited. LCP = Life-course-persistent. AO = Adolescent-onset. HS = High-stable. AD = Antisocial-desistors. ML = Medium-level. Onset diversion = Whether the first offence was dealt with via diversionary processes, which aims to reduce the effects of labelling and stigmatization associated with juvenile offending. PRA-V1 = Primary Risk Assessment - version 1 is a revised version of the Wisconsin Risk and Needs classification system, see Yessine and Bonta (2009) for details on this measure.
[a](Petitclerc et al., 2009). [b](Roisman et al., 2010). [c](Sittner & Hautala, 2016). [d](Ehrenreich et al., 2014). [e](Park et al., 2010). [f](Maldonado-Molina et al., 2009). [g](Jennings, Maldonado-Molina, Piquero, et al., 2010). [h](Hoeve et al., 2008). [i](Odgers et al., 2008). [j](Shaw et al., 2012). [k](Ward et al., 2010). [l](Boutin et al., 2017). [m](Silver et al., 2010). [n](Maldonado-Molina, Reingle, et al., 2010). [o](Maldonado-Molina, Jennings, et al., 2010). [p](Reingle, Maldonado-Molina, et al., 2012). [q](Malti et al., 2015). [r](Evans et al., 2016). [s](Kolivoski et al., 2014). [t](Tabone et al., 2011). [u](Ferrante, 2013). [v](Yessine & Bonta, 2009). [x](López-Romero et al., 2015). [z](Higgins et al., 2010). [w](Becht et al., 2016).

diagnoses (OR = 51.80, HS), more quantity of family-related maltreatment indicators (OR = 7.80, LCP), low IQ (OR = 5.10, HS). These results show that the effects tended to be stronger for both the LCP and HS profile groups across the three sex-specific samples. In addition, in samples of boys, the strongest effects were found in a mix of variables within the socio-environmental level (i.e., in both the family and neighbourhood/community domains), whereas in samples of girls, the strongest effects tended to be found in variables within the individual-level.

Socio-environmental level risks. As shown in Table 4.19, socio-environmental level risks in studies considering mixed samples tended to be a balanced mix of variables in both family (n = 7; e.g., maternal permissive parenting style, living with a single caregiver, and parent coercive discipline) and peer (n = 7; e.g., a better understanding for reasons for having friends, having more friends who drink alcohol, and gang membership) domains. In contrast, socio-environmental risks tended to be in the family domain in both samples of boys (n = 9; e.g., more quantity of family-related maltreatment indicators, and both neglectful and authoritarian parenting styles) and girls (n = 8; e.g., more quantity of family-related maltreatment indicators, parental hostility, and parents' self-report of criminal conviction). In addition to the relevance of variables in the family domain to predict boys' and girls' ASB, a similar number of risks was also found for both sexes in this domain. Also, a greater quantity of family-related maltreatment indicators was a common predictor between both sexes. Notably, significant risks in both school (school suspension and teacher-child interpersonal conflict) and sexual behaviour (child has/had a steady boy/girlfriend) domains were only reported in studies considering mixed samples.

As also shown in Table 4.19, the following variables are the socio-environmental risks that overlapped across sex-specific samples in predicting membership in distinct ASB profile groups (boys/girls, respectively): Parent coercive discipline (OR = 2.01, CL / OR =

1.86, HS), more quantity of family-related maltreatment indicators (OR = 14.50, LCP; 6.70, AD / OR = 7.80, HS; 3.90, AO; 2.80, AD), family's physical and verbal conflict (OR = not reported, LCP, AO, AD / OR = not reported, HS), inconsistent discipline across mother and father (OR = not reported, AD / OR = not reported, HS, AD), mother's affective distress and somatic response (OR = not reported, AD / OR = not reported, HS, AO, AD), delinquent peer association (OR = not reported, AD / OR = not reported, AO, HS), youth exposure to violence (OR = 1.34, CL; 1.07, AO; 1.16, HS / OR = 1.74, CL; 1.20, HS), deficient educational and occupational characteristics in the community (variable name: Onset edocc, OR = 1.16, LCP / OR = 1.33, LCP). Although odds ratio were not reported for some variables, generally, the strongest effects were not exclusive of a sex-specific sample but were often found predicting boys' and girls' membership in both the CL and LCP groups. Interestingly, such findings also show that most risks predicted membership in both the LCP and HS groups for boys and girls. However, some variables seem to have predicted more severe ASB groups in girls compared to boys (i.e., parent coercive discipline inconsistent discipline across mother and father, mother's affective distress and somatic response, delinquent peer association).

Individual-level risks. As shown in Table 4.19, individual-level risks tended to be in the emotional/personality domain in studies considering mixed samples (n = 6; high energy, depressive symptoms, sensation-seeking, behaviour psychopathy, hopelessness, and risk-taking), boys (n = 3; ADHD diagnoses, uncontrolled temperament, and sensation-seeking), and girls (n = 4; ADHD diagnoses, uncontrolled temperament, sensation-seeking, and high surgency/extraversion). Interestingly, significant risks in the physical/physiological domain were only reported in studies considering mixed samples (worse physical health and developmental disability). These findings support the relevance of aspects within the emotional/personality domain in childhood for both boys' and girls' ASB over time. In

addition, it also shows that a similar number of risks was found for both sexes in this domain. This is interesting because, among the 31 studies included in this review, only five analysed data for girls only, but ten analysed data for boys only.

The following individual-level risks overlapped in predicting membership in distinct ASB profile groups across sex-specific samples (boys/girls, respectively): Sensation-seeking (OR = 1.11, AO; 1.62, HS / OR = 1.78, CL; 1.37, HS), uncontrolled temperament (OR = 3.50, LCP; 4.70, AD / OR = 2.40, AD), ADHD diagnoses (OR = 4.30, AD / OR = 51.80, HS; 14.14, AD), low IQ (OR = 5.80, LCP; 4.00, AD / OR = 5.10, HS; 2.90, AD), low reading achievement (OR = not reported, LCP, AD / OR = not reported, HS, AD). These results suggest that, in general, the strongest effects for any risk factor are neither exclusive of a sex-specific sample nor do they exclusively predict membership in more chronic and severe ASB trajectories - as what was observed in variables within the socio-environmental level. Instead, the strongest effects tended to be found for the presence of a psychological diagnosis (ADHD) and risk factors in the (neuro)cognition/academic achievement domain.

Summary of sex-specificities in risk factors. The results of comparing significant childhood risk factors of ASB profile groups in relation to a no/low-stable ASB trajectory across mixed samples and samples of boys and girls uncovered similarities and differences in the number and magnitude of such risks for both sexes. When considering domains that posited the greater number of risks, variables within both family and emotional/personality domains were the most relevant for predicting membership in ASB trajectories for both boys and girls. In complement, a similar number of predictive variables was found for both sexes in both such domains. However, a greater quantity of family-related maltreatment indicators seemed to be most relevant as it was a common predictor for both sexes within the family domain. When considering the magnitude of sex-specific risks for membership in ASB profile groups across socio-environmental and individual levels, three aspects are worth

highlighting. First, when the effects of the risks found in samples of boys and girls were analysed separately, most variables among those that had the strongest effects tended to be within the individual level for girls. For boys, the strongest effects were found in a mix of domains within the socio-environmental level. Second, the examination of the effects of risks that had been tested in samples of boys and also in girls (i.e., overlapping variables) suggested that the strongest effects were not exclusive of a sex-specific sample. Third, the effects of variables within the socio-environmental level tended to be stronger for both the LCP and HS profile groups for mixed samples and samples of boys and girls. In contrast, the strongest effects of variables within the individual level were found not for ASB profile groups, but for a specific kind/domain of risks (i.e., psychological diagnose (ADHD) and in the (neuro)cognition/academic achievement domain).

In short, few sex-specificities in risk factors for membership in ASB profile groups probably exist. However, most of it is still unclear in the literature because risk factors for boys' and girls' membership in ASB profile groups are mostly dissolved in mixed samples. Also, although these findings only represent overall tendencies, the number of studies examining samples of girls compared to boys in this review was considerably less. Therefore, the balanced number of risks found in the family and the emotional/personality domains for both sexes is concerning because it may indicate that these two domains are either more relevant for predicting girls' ASB than assumed here. Still, another possibility could be that they have just been the most studied in the few prospective longitudinal studies that considered girls. In either of these cases, very little is known about risks for ASB in samples of girls; hence, it seems safe to say that the extent to which whether sex-specific predictors of ASB trajectories really exist is mostly unknown in the literature and more longitudinal studies are needed in this regard. In addition, these studies should preferably be prospective longitudinal studies considering boys and girls separated, and they may also broaden the

examined risks to include factors within the school, sexual behaviour, (neuro)cognition/academic achievement, and physical/physiological domains, which have rarely been investigated in prospective longitudinal research.

Comparison of sex-specific protective factors. In the present review, predictive variables that were significantly associated with a reduced likelihood of membership in any ASB profile group in relation to no/low-stable ASB are referred to as protective factors. Table 4.20 shows 29 predictive variables that were reported as protective factors in 17 out of the 31 included studies and are presented here according to sample-sex in each study (mixed samples, boys-only, girls-only).

Overall, significant protective factors were predominantly reported within the socio-environmental level compared to individual-level. Also, protective factors were almost exclusively reported in studies that analysed data from mixed samples compared to those that analysed data from boys or girls only. For example, the following were four protective factors reported within the socio-environmental level as most strongly associated with specific ASB profile groups in mixed samples. Both higher frequency of religious participation (OR = 1.20, LCP) and parent's higher educational level (OR = 0.95, AL) were reported in a sample of black and Hispanic youth in the general population in the USA. Positive self-perception of neighbourhood safety and social support (OR = 0.95, HS) were reported in an ethnically diverse sample of maltreated children reported for protective services in the USA. Parental involvement with the child's life (OR = 0.94, HS and 0.94, HS) was reported in two community samples, one of Latino-only and another of black and Latino youth in the USA. Thus, considering the at-risk characteristic of all such samples, the predictors mentioned above could be considered as authentic protective factors for LCP, HS,

Table 4.20

Summary of Significant Results (p < .05) from 31 Studies using Multinomial Regression to Test Socio-Environmental and Individual Level Variables Associated with a Reduced Risk of ASB Relative to No/Low-Stable ASB by Sex

	Odds ratio (ASB profile group)		
Domain / Sample / Predictive variable	Mixed samples	Samples of Boys	Samples of Girls
Socio-environmental level			
Family			
Higher educational level of parents	0.95[a] (AL)	-	-
Parental involvement with child's life	0.94[b], 0.94[c] (HS)	-	-
Living with both mother and father in the house	0.67[d] (HS); 0.65[d] (AO); 0.61[d] (AD)	0.51[e] (HS); 0.44[e] (AO)	-
Having access to alcohol at home	0.65[b] (AD)	-	-
Latino background and speak Spanish at home	0.56[b], 0.35[c] (HS); 0.54[b] (AD); 0.39[c] (ML)	-	-
Married parents	0.32[f], 0.14[f] (AD); 0.14[f] (HS)	-	-
Supportive parenting and quality home environment	-	0.87[g] (AD)	-
School			
Positive school adjustment	0.67[h] (AD); 0.65[h] (HS)	-	-
Child receives free or low-price lunches at school	-	0.47[e] (AO)	-
Peer			
Better understanding of friendship trust	0.16[i] (AO)	-	-
Peer acceptance	0.09[j] (HS)	-	-
Neighbourhood/community			
Higher frequency of religious participation	1.20[a] (LCP)	-	-
Neighbourhood safety and social support	0.95[h] (HS)	-	-
More years in the same out-of-home placement	0.85[l] (LCP)	-	-
Social support	0.47[m] (ML); 0.46[m] (CL)	-	-
Onset city (first offense in Perth/Australia)	-	0.93[n] (ML); 0.90[n] (LCP)	1.19[n] (LCP)
Onset north (first offense in Western Australia)	-	0.91[n] (ML); 0.52[n] (LCP)	0.66[n] (LCP)
Individual-level			
Emotional/personality			
High self-esteem	0.85[m] (AO)	-	-

Domain / Sample / Predictive variable	Odds ratio (ASB profile group)		
	Mixed samples	Samples of Boys	Samples of Girls
High trustfulness	0.61[o] (ML); 0.33[o] (CL), 0.40[o] (HS)	-	-
High concentration	0.21[p] (HS)	-	-
High orderliness	0.19[p] (HS)	-	-
High compliance	0.16[p], 0.05[p] (HS); 0.12[p] (AD)	-	-
High optimism	0.14[p], 0.06[p] (HS)	-	-
High perseverance	0.10[p] (HS)	-	-
High trustworthiness	0.09[o] (ML); 0.03[o] (HS); 0.01[o] (CL)	-	-
(Neuro)Cognition/Academic Achievement			
Low academic achievement	-	0.36[q] (LCP)	-

Note. ASB = antisocial behaviour. Mixed sample = boys and girls combined. CL = Child-limited. AL = Adolescent-limited. LCP = Life-course-persistent. AO = Adolescent-onset. HS = High-stable. AD = Antisocial-desistors. ML = Medium-level.

[a](Petts, 2009). [b](Reingle, Maldonado-Molina, et al., 2012). [c](Maldonado-Molina, Reingle, et al., 2010). [d](Maldonado-Molina, Jennings, et al., 2010). [e](Jennings, Maldonado-Molina, & Komro, 2010). [f](Ehrenreich et al., 2014). [g](Shaw et al., 2012). [h](Sittner & Hautala, 2016). [i](Malti et al., 2015). [j](Silver et al., 2010). [l](Kolivoski et al., 2014). [m](Maldonado-Molina et al., 2009). [n](Ferrante, 2013). [o](Malti et al., 2013). [p](Becht et al., 2016). [q](Day et al., 2012).

and AL ASB trajectories. Therefore, being assets for building resilience within both cultural and context-specific at-risk populations, which is also in line with findings from previous studies (Farrington, 2015; Ttofi et al., 2016).

Unfortunately, comparison of protective factors between samples of boys and girls was not possible because there were only six protective factors significantly associated with membership in distinct ASB profile groups; all six in boys and two of them also in girls (see Table 4.20). More specifically, protective factors in samples of boys were living with both father and mother in the house (OR = 044, AO; 0.51, HS) and receiving free or low-price lunches at school (OR = 0.47, AO), as reported in a community sample of ethnically diverse boys from the USA. Supportive parenting and quality home environment (OR = 0.87, AD), reported in an ethnically diverse sample of low-income boys from the USA. The onset of ASB in the city of Perth (OR = 0.90, LCP; 0.93, ML) and in Western Australia (OR = 0.52, LCP; 0.91, ML), reported in two Australian samples of Aboriginal and non-Aboriginal offender boys. Poor academic achievement (OR = 0.34, LCP), reported in a Canadian sample of offenders (despite its significance, the reliability of this particular variable as a protective factor is questionable as it was not a significant predictor of other three LCP trajectories within the same study). Protective factors in samples of girls were onset of ASB in the city of Perth (OR = 1.19, LCP) and in Western Australia (OR = 0.66, LCP), reported in two Australian samples of Aboriginal and non-Aboriginal offender girls. Overall, these results suggest that living with both parents in the house and receiving free or low-price lunches at school for boys, and being from Perth or Western Australia, for Australian offender boys and girls, may all be promotive or buffering factors, because predicted lower probabilities of ASB in community samples. However, it is unknown whether these same variables would also be protective factors for ASB trajectories because they were not tested in at-risk samples of boys and girls (Farrington, 2015; Ttofi et al., 2016). On this same premise, the results also suggest

that positive parenting quality may be a protective factor in at-risk boys against engaging in LCP ASB trajectories.

Notably, Table 4.20 also shows that protective factors in samples of boys and girls were all reported within the socio-environmental level (one exception in a sample of boys was discussed above). Protective factors within the individual-level were predominantly reported in the emotional/personality domain in mixed samples. For example, the following were four protective factors reported within the individual level as most strongly associated with specific ASB profile groups in mixed samples. High self-esteem (OR = 0.85, AO) was reported in a sample of Latino youth in the general population from Puerto Rico and the USA. Personality traits of high concentration (OR = 0.21, HS), high orderliness (OR = 0.19, HS), and high compliance (OR = 0.16, HS) were reported in a sample of Belgium youth in the general population. These findings confirm that individual characteristics in childhood may be promotive or buffering factors for membership in high-stable ASB trajectories. This is in line with assumptions in recent theories within in developmental psychopathology (Frick & Viding, 2009) and criminology (DeLisi & Vaughn, 2014) fields. Yet, such findings also show that causal links between individual-level variables in childhood and lower likelihood or ameliorative effects for membership in high levels of chronic ASB in at-risk samples are not available in the current prospective longitudinal literature.

In short, these findings uncovered that very little is known about aspects within both socio-environmental and individual levels that could be targeted to build resilience in at-risk boys against engaging in LCP and AL ASB trajectories. This is alarming giving the consensus in the literature that boys compared to girls are at most risk for engaging in LCP and AL ASB trajectories. Also, the findings on protective factors in only one sample of girls showed that factors within both socio-environmental and individual levels that could be identified in childhood and targeted to build resilience in at-risk girls against engaging in

LCP and AL ASB trajectories are mostly unknown in the literature reviewed here. However, the few significant results found also indicate that such protective factors exist and could be identified in childhood. Therefore, much more prospective longitudinal studies are still needed examining socio-environmental but mainly individual-level protective factors in childhood against ASB trajectories in at-risk boys and girls. Importantly, whenever possible, such studies must consider samples of boys and girls separated to inform and enable preventive initiatives that will truly promote, in addition to remedy youth's optimum development from childhood and onwards.

Conclusions and Future Research

This systematic study reviewed findings from 31 prospective longitudinal studies that used multinomial logistic regressions to report on childhood predictors of membership in mutually exclusive ASB profile groups. Overall, the results reported here expand on current literature by describing and comparing temporal, correlational relationships between socio-environmental and individual-level characteristics in childhood and specific ASB profile groups in two different ways. First, to describe childhood predictors associated with an increased likelihood of membership in distinct ASB profile groups, variables predicting groups of no/low-stable ASB in relation to high ASB, as well as groups of child-limited (CL), adolescent-limited (AL), life-course-persistent (LCP), adolescent-onset (AO), high-stable (HS), antisocial-desister (AD), and medium-level (ML) in relation to no/low-stable ASB were summarised and reported in detail when available. In doing this, predictors were also presented separately for samples of boys and girls pooled together and separated, whenever available. Second, to look further into childhood predictors of ASB profile groups, a series of comparisons were reported across both distinct ASB trajectory groups (i.e., Comparison set 1: CL, AL, and LCP. Comparison set 2: HS and ML) and sex-specific samples (i.e., boys and girls mixed and separated). Such comparisons focused on significant childhood predictors

and were reported separately for variables within the socio-environmental and the individual levels to uncover possible similarities and differences in strength and magnitude of specific variables (i.e., qualitative differences) and the number of predictors (i.e., quantitative differences) of ASB profile groups.

Overall, the main findings in this review uncovered the following topics of knowns and unknowns about similarities and differences in number, magnitude, kind, and sex-specificities of childhood predictors of distinct ASB profile groups in the literature. Regarding number and magnitude of childhood predictors of ASB groups, the findings suggested that a greater quantity of significant predictors, of which the effects are also of greater magnitude, are consistently reported for groups of higher levels and chronic ASB (e.g., LCP and HS) in relation to both no/low-stable ASB and groups of non-chronic and moderate levels of ASB (e.g., CL, AL, and ML). Such evidence is consistent with findings in past reviews (Fairchild et al., 2013; Jolliffe, Farrington, Piquero, Loeber, et al., 2017), but also adds to previous research by confirming the danger of cumulative risks in childhood not only for the presence of ASB in youth but especially for its higher levels and chronic developmental patterns. Furthermore, this finding also highlights the relevance of investing in improving risk assessment to be completed over childhood years for the identification of youth most at-risk for LCP or HS.

The relevance of risk assessment has been discussed in a previous review in which researchers highlighted the importance of targeting specific risks that have the greatest impact to increase the chances of successful intervention outcomes (Assink et al., 2015). In this respect, the present review extends on past research by identifying the most relevant risk and protective factors for ASB development in the literature that can be potentially identified in childhood. Most specifically, significant predictors tended to be more numerous within the socio-environmental level rather than the individual-level across all ASB profile groups.

However, qualitative differences in childhood predictors and the strongest effects for the most chronic and severe ASB trajectories (i.e., LCP and HS) tended to be within the individual-level compared to socio-environmental characteristics.

What should be considered here, however, is the greater attention on socio-environmental level variables than on individual-level variables within the included studies, suggesting that individual characteristics may be under-investigated and deserving of more research attention. Across all ASB profiles described here, 368 variables within the socio-environmental level were tested among the eligible studies, with 147 (40%) of these reported as significant, whereas within the individual level, a total of 124 variables were tested with 60 (48%) of them reported as significant. This suggests that more prospective longitudinal studies are needed to improve risk assessment and to better design intervention services for children at risk of ASB. In particular, the results in this review suggest three future directions for longitudinal research and reviews aiming not only at improving childhood risk assessment procedures but also ay better informing intervention services. As a first direction, researchers could replicate in future reviews the prospective analyses of significant individual-level characteristics found in this review, but with a focus on specific ASB types (i.e., aggression, rule-breaking and offending) and across sex-specific subgroups of at-risk and offender youths. As a second direction, researchers could broaden both the number and types of individual-level variables that have been examined as potential correlates of ASB, while also examining interactions of individual-level factors with well-established socio-environmental risks for ASB trajectories (e.g., variables within family and neighbourhood/community domains (for a review that had suggested this first, see Fairchild et al., 2013). As a third direction, although examination of predictors of ASB measured in specific age ranges within childhood (e.g., toddlerhood, early childhood) was beyond of the scope of this review, such a focus also seems relevant in future research, as it could better inform theory and intervention

services on whether the risk assessment for ASB should differ within childhood developmental stages.

Regarding the specific kinds of childhood predictors of ASB groups, almost no domain was left out. Variables in the family, neighbourhood/community, emotional/personality, and (neuro)cognition/academic achievement domains all had significant effects in at least one study. However, it is useful to consider the strength of the effects. The strongest effects were found in the family, emotional/personality, and (neuro)cognition/academic achievement domains. Notably, such stronger effects were not only found to be unique to comparing ASB profile groups to no/low-stable ASB, but variables in these domains also differentiated LCP from AL and CL and HS from ML ASB consistently. More specifically, contrary to the developmental taxonomy theory (Moffitt, 1993) and similar to what has been reported in previous studies (Assink et al., 2015; Fairchild et al., 2013; Jolliffe, Farrington, Piquero, Loeber, et al., 2017), the descriptions of childhood predictors of each ASB group in relation to no/low-stable ASB in this review showed no specific variables predicting specific ASB profile groups (i.e., no qualitative differences). However, findings from the second layer of between-ASB group comparisons reported in this review (i.e., LCP with AL and CL ASB, and HS with ML ASB) suggest that such an absence of qualitative differences in predictors of specific ASB trajectories is probably true about variables within socio-environmental risks, but this may not be the case for individual-level risks. That is, a similar profile of socio-environmental variables may predict all LCP, AL, and HS ASB groups, with a greater quantity of such variables determining higher levels of more chronic ASB. Complimentarily, however, specific individual-level characteristics may also represent a greater risk for higher and chronic ASB compared to lower/milder ASB levels for both boys and girls (i.e., ADHD diagnose and variables in the (neuro)cognition/academic achievement domain).

General implications of this finding concern with the fact that, although it is partially in line with the developmental taxonomy of Moffitt (1993), who had first proposed that differences in predictors of ASB groups would be of qualitative nature, there is currently not yet firm evidence to conclude that there are qualitative differences in the individual-level predictors of different ASB profile groups. However, this could also be a result of less attention to these potential influences relative to socio-environmental influences. In any case, this scenario illustrates again the importance of more extended prospective longitudinal research focused on early childhood individual-level characteristics that may be relevant to the development of ASB into adolescence and adulthood.

The main findings regarding sex-specificities in childhood risks for ASB profile groups in this review showed the significant influence of both socio-environmental and individual level risks. In particular, risk factors in the family and in the emotional/personality domains were most numerous and had the strongest effects for boys and girls. At first sight, these findings seem consistent with the theory that the predictors of ASB trajectories do not differ for boys and girls (Moffitt et al., 2001). However, there was also some evidence that individual-level predictors could be more numerous and of greater magnitude among females compared to males. In practical terms, this result is concerning because was found even though girls are considerably less investigated compared to boys (i.e., studies of girls-only were 50% the number of those of boys-only in this review). This clearly suggests more prospective studies are needed examining ASB in boys and girls separately, but particularly samples of girls-only. Such studies are important to broaden the understanding of origins of ASB development and also to inform assessment and intervention on this matter. For now, the most relevant findings in this regard indicates that mental health-related risk characteristics for the development of ASB may be prospectively identified in childhood for

both sexes, but that such identification of risks may be equally important for assessment and intervention with girls.

There is also a need for future studies that allow for the identification of true LCP and AL trajectories, especially in girls. Such a conclusion is supported by previous studies (Fontaine et al., 2009; Jennings & Reingle, 2012) that found a smaller number of ASB trajectories may exist among girls. Therefore, suggesting less heterogeneity in female-ASB compared to boys-ASB. That is, girls seem to either engage or not in ASB and when they do, a girls-LCP group, starting later compared to boys (in adolescence, not in childhood) is commonly reported. For instance, girls' ASB trajectories in eligible studies were not found within the AL, or the ML ASB profile groups and only one study of girls reported a trajectory within the LCP profile group, as defined here. In contrast, most girls' trajectories were found in both HS and AO profile groups in this review. Logically, given the reduced number of studies that considered samples of girls, this evidence could also be a misrepresentation of female ASB trajectories, but confirmation of any of these hypothesis urges for a closer look at girls' ASB trajectories from childhood with follow-up into adulthood in future longitudinal studies.

Together, the results from such descriptions and comparisons are relevant to broaden understanding of how and what socio-environmental and individual characteristics in childhood represent risks or protective factors for ASB over time in current literature. Essentially, this is vital to help researchers, practitioners, and policymakers with optimizing youth development by identifying children and adolescents most at risk for high and chronic levels of ASB. Hence, allowing for targeting those risk and protective factors that will have the greatest impact on reducing and even preventing ASB alongside its negative consequences in youth's lives, the society, and the economy.

Strengths and Limitations of This Review

This systematic review has several strengths. First, prospective longitudinal studies that identified ASB trajectories and used multinomial logistic regression to predict trajectory membership were located. The search for such studies included research that had been published in three languages (Portuguese, Spanish, and English). Prospective investigation means that all predictors reported in eligible studies were assessed either at the first wave of data collection or earlier. The use of regression in all eligible studies also meant that the associations reported between predictors and the ASB trajectories represent a numerical relationship between them. That is, the impact that a unit of change in each predictor possibly has on belonging to the corresponding ASB trajectory, in relation to a no/low-stable ASB. Therefore, the significant effects examined here are still correlational but provide some confidence in the directional relationship between childhood predictors and later ASB trajectories. Another strength of this review is that socio-environmental and individual-level variables were examined and reported separately, therefore allowing for more in-depth consideration of the number, magnitude, and kind of childhood predictors across ASB trajectories and sex-specificities comparisons. Also, beyond the standard comparisons between LCP and AL ASB, distinct ASB profile groups were compared among themselves after having each of them been compared to a no/low ASB trajectory. This resulted in a broader perspective on whether and which significant predictors could be identified in childhood, in relation to other reviews in this field.

This study also has four limitations that merit discussion. The first three limitations are related to decisions made to select eligible studies and organize their results into this review. The fourth limitation relates to concerns the impact of the variability in included studies' designs/methods on the interpretation of the findings.

First, to be included in this review, the prospective study had to have used GBTM.

Although the use of GBTM is relevant to uncover details about the life-course continuum and distinct levels of ASB over time, some scholars have argued that this approach is also problematic for being just statistical concepts of probabilities, which may present a too simplistic view that does not completely represent the reality of changes in ASB over time for each individual (Skardhamar, 2010). To account for such limitation and more reliably investigate LCP and AL ASB, scholars have suggested methodological precautions that should be taken by researchers before the conduction of the GBTM analyses. These include mainly conducting prospective longitudinal studies that start in childhood and follow participants into adulthood (Jennings & Reingle, 2012; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). In this respect, only prospective studies in which the first assessment of ASB occurred in childhood (age 12 or earlier) were included in this review. However, for a bigger picture of the ASB trajectories reported in prospective studies, having followed participants into adulthood was a condition for an ASB trajectory to be included in the LCP ASB profile group, but not a requirement for a study to be included in the review.

Second, although the definitions of ASB profile groups were established after careful consideration of vast literature, they also only accounted for a limited variation in the age of onset, the duration, and the severity of the continuation of ASB. Therefore, it is possible (although less probable) that the results would have differed from those reported here had such definitions been adjusted for distinct age at onset or either longer or shorter duration of ASB in each profile group. For instance, a previous review testing distinct definitions for ASB profile groups reported that little differences are found in ASB predictors when adjusting the definitions of ASB trajectories to account for a longer duration (Jolliffe, Farrington, Piquero, MacLeod, et al., 2017). Also, despite the popularity of Moffit's descriptions of ASB trajectories in her taxonomy, literature shows no agreement about how many ASB trajectories exist and why, which makes it difficult to standardize their definitions

across different studies. However, the definitions used here seem generally quite similar to those across most longitudinal studies (Jennings & Reingle 2012).

Third, to focus on childhood predictors of a range of ASBs, a broad definition of it was chosen in this review to include studies reporting on trajectories of aggression, rule-breaking behaviour (delinquency), and offending. This is similar to what has been done in previous research reviews (Farrington et al., 2017; Jennings & Reingle, 2012; Ttofi et al., 2016), to report on possible causes of ASB in general, and serve to better inform theory, research, and practice in the field. Importantly, however, evidence also suggests that the specific forms of ASB (aggression or rule-breaking behaviour) may have distinct etiologies (Tremblay, 2010) and also be more relevant than age-of onset to predict ASB continuation into adulthood (Fairchild et al., 2013). Thus, future reviews comparing childhood predictors of ASB trajectories separately for aggression and rule-breaking behaviour are highly desirable. Although such examinations were beyond the scope of this review, they are equally relevant, and their results may potentially suggest a shift in theory and treatment of conduct disorders and ASB development from age-of onset to a behaviour-specific based approach before antisocial youth become offenders. Also, most existent reviews have focused on predictors of offending behaviours (Farrington, 2015; Jolliffe, Farrington, Piquero, Loeber, et al., 2017; Ttofi et al., 2016).

Fourth, regarding limitations that come from the prospective longitudinal studies themselves. The included studies were limited to predictor variables that are relevant in childhood, varied in design (e.g., sample size, age of onset, length of follow-up, measures, and reporters), and varied in attrition rates and probably representativeness. In addition, studies varied in the control variables they did or did not include in their models. The results reported here were from the most comprehensive models (i.e., multivariate) in the original studies, which sometimes or often included controls for static predictors (e.g., sex, SES,

ethnicity) and/or ASB at baseline. All these study differences in design and analyses could impact on the results and conclusions of this review, making it more likely that some differences reported in findings and effect sizes may have been a result of study design differences. However, while methodological differences across included studies could have affected the reliability of comparison analyses reported here, it is worth to highlight that the comparisons across ASB profiles and sex-specificities considered only overlapping variables. This means that most of the time, the confidence of the comparison analyses was ensured because consisted of comparing the same predictive variable tested across distinct ASB profiles or across sexes within the same sample. Still, controlling and reporting on all methodological specificities of included studies was not within the aims of this review, and is usually an enormous challenge in conducting reviews in this field. Also, drawing clear and definitive conclusions about any causal effects is also often challenging and unrealistic in reviewing longitudinal studies.

To conclude, this novel systematic review of prospective longitudinal studies reporting on childhood predictors of ASB assessed from childhood addressed important gaps in current literature. In particular, it may serve as a resource for future research and theory, and as an aid to policymakers and practitioners planning interventions to prevent and/or mitigate the negative impact of high and chronic ASB in children or adolescents to optimize their development.

Summary of Chapter 4

Study 1 was a review that focused on (a) describing childhood socio-environmental and individual-level predictors of antisocial behaviour (ASB) in boys and girls together and separated and (b) providing a closer look into study design differences and quantitative and qualitative differences in such predictors across distinct ASB trajectory profiles. The main findings in Study 1 suggest that the presence of a greater number risks in childhood, at both the social-environmental and individual levels, successfully differentiate youth in trajectory profiles of higher and chronic ASB, relative to youth with a no/low-stable ASB trajectory profile (i.e., quantitative differences). In addition, there was also evidence that the origins of high and chronic ASB profiles (e.g. life-course-persistent and high-stable ASB) may differ of that of other ASB trajectory profiles when compared across specific individual-level risks within both the emotional/personality and (neuro)cognition/academic achievement domains (i.e., qualitative differences), but more research is needed to confirm this finding. Regarding sex differences in predictors, socio-environmental and individual-level factors differentiated ASB profiles in both boys and girls, but predictors of girls' ASB appeared to be slightly more numerous and associations of individual-level factors with ASB appeared to be of greater magnitude in samples of girls compared to boys. However, this conclusion was tentative because studies of individual-level risk factors and girls' ASB profiles are scarce. Given these needs for further research on individual-level risk factors and girls' ASB profiles, the next two chapters are prospective longitudinal studies focusing on early individual-level predictors of separate samples of boys' and girls' aggressive (Study 2) and rule-breaking (Study 3) behaviours into late adolescence.

CHAPTER 5

Study 2

The Roles of Early Childhood Effortful Control Deficits, Negative Emotionality, and Callous-Unemotional Traits in Boys' and Girls' Physical Aggression into Adolescence

Statement of Contribution to Co-authored Paper

This chapter includes a co-authored manuscript that is ready to be submitted for publication. It consists of a version of the paper that is under peer-review process. The bibliographic details of this co-authors paper, including all authors, are:

Pariz, J., Zimmer-Gembeck, M. J., & Modecki, K. (under review). The roles of early childhood effortful control deficits, negative emotionality, and callous-unemotional traits in boys' and girls' physical aggression into adolescence.

My contribution involved: Review of literature, development of hypotheses, co-development of the methodology and design, completion of statistical analyses, lead in writing the paper, corresponding author of the paper.

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Abstract

Developmental theories implicate effortful control deficits and negative emotionality in childhood as risks for severe and chronic physical aggression (PA) into adolescence. Callous-unemotional traits may also add to such risk via both its direct effects and/or by enhancing the increase in ASB associated with other risks. To test these hypotheses, data from a national Australian sample (N = 7460, 51% male) was used. First, patterns of change in mother- (ages 4-17) and self-reported (ages 10-17) PA were estimated. Next, the roles of mother-reports of their child's effortful control deficits, negative emotionality (measured as anger, fear and sadness dysregulation), and callous-unemotional traits (measured as callous-lack of empathic concern for others) at age four on PA level and change over time were investigated. Interactions (i.e., effortful control deficits/negative emotionality × callous-lack of empathic concern for others) and sex-specific physical aggression models were also examined. The results showed a significant quadratic U-shaped pattern in boys' and girls' mother-reported and self-reported physical aggression was found. Furthermore, on average, boys' PA level was significantly higher than girls' only until age 14. All tested risks had unique associations with PA patterns, but significant associations were not always consistent across models of mother- and self-reported PA, or across models for boys and girls. Implications for a focus on assessing and boosting young children's skills to achieve optimum effortful control, regulate specific negative emotions, and increase their empathic concern for others are discussed.

Keywords: Physical aggression; trajectories; negative emotionality; effortful control, callous-unemotional.

The Roles of Early Childhood Effortful Control Deficits, Negative Emotionality, and Callous-Unemotional Traits in Boys' and Girls' Physical Aggression into Adolescence

Multiple developmental theories exist describing an atypical, high level of physical aggression (PA) that starts in childhood and persists into adolescence and/or adulthood (Archer & Côté, 2005; Bussey & Bandura, 1999; Daly & Wilson, 1994; Moffitt, 1993; Wood & Eagly, 2002). One of the most widely disseminated among such theories is Moffit's taxonomic theory (Moffitt, 1993; Moffitt, 2006). Moffitt describes distinct temporal patterns of engagement in antisocial behaviour (which includes PA) that differ according to severity, age-of-onset, and patterns of maintaining or desisting of it from childhood to adulthood. Among such patterns, is a group of roughly 8% to 15% of youth (depending on the study cited), whose high levels of chronic antisocial behaviour begins in childhood and continues into adolescence or adulthood, has consistently been identified (Barnes, 2013; Moffitt, 2006). Notably, these high level and chronic pattern of PA is likely to escalate into a range of antisocial and criminal behaviours which may account for 50% of all the economic costs related to criminality (Cohen, Piquero, & Jennings, 2010; Moffitt, 2006; Smith, Jorna, Sweeney, & Fuller, 2014). According to this taxonomic theory, the onset and growth of high and chronic antisocial behaviour are rooted in adverse socio-environmental conditions (community and family level risks) and neuropsychological deficits (i.e., *individual-level risks)* that are perceptible early in life (Moffitt, 2006).

Scholarly findings consistently support Moffitt's theory, but typically explore early community- and family- level risks, as opposed to individual-level risks for problem PA pathways (DeLisi, Fox, Fully, & Vaughn, 2018; Givens & Reid, 2019; Hay, 2017; Martino, Ellickson, Klein, McCaffrey, & Edelen, 2008; Nigg, 2017). Addressing this gap in understanding is necessary to guide prevention and intervention efforts and more effectively address the needs of children and adolescents most at-risk for PA (Fairchild, Van Goozen,

Calder, & Goodyer, 2013; Jennings & Reingle, 2012; Reingle, Jennings, & Maldonado-Molina, 2012; Thomas, Abell, Webb, Avdagic, & Zimmer-Gembeck, 2017). Thus, the present study uses 7-waves of data from a large national sample to test the direct, and interactive effects of early (age 4) individual-level risks of effortful control deficits and features of both negative emotionality and callous-unemotional traits as predictors of PA growth from early childhood into late adolescence. Although Moffitt's theory is relevant for describing and explaining growth in PA, it does not include a particular focus on specific individual-level predictors. Fortunately, such information is provided in two complimentarily theoretical frameworks, namely the temperament-based theory of antisocial behaviour (e.g., DeLisi & Vaughn, 2014; Eme, 2018) and the developmental psychopathology model of antisocial behaviour (Frick, Ray, Thornton, & Kahn, 2014b; Frick & Viding, 2009).

Early Childhood Individual-Level Risks for PA Patterns Over Time

According to the temperament-based theory of antisocial behaviour, both effortful control deficit and negative emotionality are risks for severe and chronic antisocial behaviour from early childhood and over the life course (DeLisi & Vaughn, 2014; Eme, 2018). Effortful control deficits refer to the poor dispositional (as opposed to automatic) use of self-regulation skills (low persistence, high distractibility, and high impulsivity) which interfere with optimum levels of social competence (DeLisi & Vaughn, 2014). Thus, it indicates a reduced ability to practice prosocial behavioural alternatives to PA (Baglivio, Wolff, Piquero, & Epps, 2015; DeLisi et al., 2018). Negative emotionality refers to a pattern of experiencing environments negatively due to poor emotion regulation (i.e., emotion dysregulation) of anger, fear, and sadness, which may increase the risk for engagement in elevated PA as a way to deal with negative emotions (DeLisi et al., 2018; DeLisi & Vaughn, 2014). Generally, when observed in childhood, deficits in effortful control and high negative emotionality can result in high and chronic PA because they indicate a failure in the optimum development of

executive function skills, which become noticeable from around age four (DeLisi & Vaughn, 2014). Such a failure can result in young children's inability to subordinate dominant impulses (i.e., regulate attention, emotions, and behaviour) in favour of socially appropriate behaviour (DeLisi & Vaughn, 2014) and thus contribute to youths' difficulties in avoiding or even desisting from PA behaviours.

Evidence from longitudinal studies is consistent with these ideas. For instance, regarding effortful control deficits, low inhibitory control in childhood has been shown to predict distinct forms of aggression in adolescence (Sarkisian, Van Hulle, Lemery-Chalfant, & Goldsmith, 2017). Similarly, other longitudinal studies also find that high impulsivity at age two predicts aggression at ages 12 and 15 (Hentges, Shaw, & Wang, 2018) and at age three predicts problem aggression at ages 18 (Caspi & Silva, 1995), and 21 (Caspi, Moffitt, Newman, & Silva, 1996), above and beyond socio-environmental risks. Regarding negative emotionality, although links with concurrent childhood PA have been reported in previous studies (Röll, Koglin, & Petermann, 2012), only a handful of prospective longitudinal studies have reported on its developmental associations with PA in childhood or adolescence. Among these, one study found that negative emotionality in late childhood predicted PA at age 16 (Martino et al., 2008). In contrast, a few more studies exist that have examined features of negative emotionality separately. For example, anger dysregulation is widely recognized as the strongest correlate of high PA across childhood and adolescence (Berkowitz, 2012; Campbell, 2006; Hay, 2017). In addition, both anger and sadness dysregulation in late childhood predict higher aggression in early adolescence (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Furthermore, some evidence shows a negative association between childhood fear dysregulation and childhood and adolescent PA (Campbell, 2006).

In a complementary theoretical perspective that is grounded within developmental psychopathology, indicators of callous-unemotional (CU) traits are also considered particularly relevant risks for high and chronic levels of conduct disorder (Frick et al., 2014b) and PA (Frick & Viding, 2009). By definition, the four traits that are considered indicative of CU are lack of remorse or guilt; callous-lack of empathic concern for others; being unconcerned about performance in important activities, and shallow or deficient affect. The presence of two of these traits is a threshold criterion for youth to qualify under the limited prosocial emotions specifier for the diagnose of conduct disorder within the DSM-5 (American Psychiatric Association, 2013). Thus, the presence of CU traits is often reported as necessary for assessment and intervention in childhood to prevent and/or mitigate high levels of chronic PA linked to conduct disorder presentation (Frick et al., 2014b; Hawes, Byrd, Waller, Lynam, & Pardini, 2017; Waller, Hyde, Baskin-Sommers, & Olson, 2016).

Although solid empirical evidence confirms the association of CU traits with higher levels of PA (Frick et al., 2014b; Frick & White, 2008), only a handful of longitudinal studies have prospectively examined the association of CU traits with aggression in later childhood or into late adolescence. This is because, no study could be found focusing specifically on PA, and most of the current studies included items on verbal and relational aggression in the measures used. Among these, one study found that CU traits at age three predicted higher levels of aggression at age 10 (Waller et al., 2016). Also, in a separate study, CU traits at age 12 predicted aggression one year later (Frick, Cornell, Barry, Bodin, & Dane, 2003), and at age seven predicted more conduct problems in girls at age 11 (Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011). Unfortunately, no prospective longitudinal study examining the unique effect of specific traits indicative of CU in early childhood on aggression into adolescence could be located. Similarly, a focus on the effects of early childhood CU traits in conjunction with other individual-level risks on pathways to PA could not be located within

the literature. Still, few related longitudinal studies have found significant interaction effects of CU traits with both fear dysregulation at age 11 being negatively associated with more antisocial behaviour one year later (Pardini, Lochman, & Powell, 2007) and with anger dysregulation and impulsivity at ages seven to 11 being positively associated with more conduct problems and criminality nine years later (Pardini, Byrd, Hawes, & Docherty, 2018).

Overall, it seems right to say that it has not yet been firmly established whether effortful control deficits and features of both negative emotionality and CU traits assessed in early childhood would explain higher levels and chronic PA patterns through adolescence. Such information is clinically relevant to intervention services aiming at preventing or mitigating aggressive behaviours (Herrenkohl et al., 2000; Pardini et al., 2018), and essential for theory and policymakers as a heuristic to understand pathways to higher and chronic PA over the life course (Pardini, 2006).

Sex Differences in PA pathways

Sex differences in PA are widely reported in longitudinal research. Across most such studies, physically aggressive boys display elevated levels of PA relative to girls in the toddler years (Alink et al., 2006; Tremblay, 2010), and through childhood and adolescence (Baillargeon et al., 2007; Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008; Teymoori et al., 2018). Thus, research on PA has tended to focus predominantly on boys (Barker et al., 2007; Carrasco, Barker, Tremblay, & Vitaro, 2006; Hawes et al., 2017) or boys and girls pooled together (Di Giunta et al., 2010; Van Lier, Vitaro, Barker, Koot, & Tremblay, 2009a), due to the prominence of PA among boys. However, this research focus has been changing with evidence that sex differences in PA are not stable over time, but appear to be higher in childhood and decrease with age (Campbell, 2006), consistently narrowing in adolescence, when levels of PA seem to increase (on average) in adolescent girls (Campbell, 2006; Teymoori et al., 2018). In addition, longitudinal evidence also shows the predicted peaks of

PA in early childhood (around 3.5 years) and again in mid-adolescence (around 15 years) for both sexes (Karriker-Jaffe et al., 2008). Thus, the importance of mapping out possible sex-specific risk factors for the development of boys' and girls' PA has been increasingly discussed, but much is yet to be described in the literature (DeLisi & Vaughn, 2014; Eme, 2018; Fairchild et al., 2013; Ferrante, 2013; Moffitt et al., 2011).

Sex Differences in Individual-Level Correlates of PA Pathways

Moffitt's original taxonomic theory asserted that individual-level correlates of antisocial behaviour (e.g., child's temperament and neurophysiological characteristics) would be similar for boys or girls (Moffitt, Caspi, Rutter, & Silva, 2001). However, both theory (Eme, 2018) and evidence (Fairchild et al., 2013) of the primary role of biological biases/mechanisms for emotional and behavioural tendencies call into question this broad assumption. For example, sex-specificities in effortful control favouring females are often proposed by studies suggesting that the lower number of girls among more severe and chronic physically aggressive youth indicate their lower vulnerability for PA relative to males, due to naturally lower levels of impulsivity and higher levels of fear in females (Campbell, 2013; Chaplin & Aldao, 2013; Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006; Eme, 2018). Regarding CU traits, although evidence suggests that it is a risk in mid-to-late childhood for high and persistent levels of PA in boys (Hawes et al., 2017), girls (Kroneman et al., 2011), and for both sexes (Euler et al., 2015), the generalisability of such evidence to CU traits from early-childhood is unknown for two reasons. First, longitudinal studies assessing CU traits from early childhood are scarce. Second, the latent structure of CU traits seems to differ across developmental stages, being bi-dimensional (e.g., defined as both uncaring and callous traits, instead of the four traits described earlier) when measured in early childhood (Kimonis et al., 2016; Willoughby, Mills-Koonce, Waschbusch, & Gottfredson, 2015). Further, these two dimensions possibly differ in their association with conduct

problems in boys and girls (Dadds, Frost, Fraser, & Hawes, 2005; Kimonis et al., 2016). Therefore, it is still unknown whether specific features of CU measured in early childhood would have similar significance and strength in predicting boys' and girls' PA pathways into adolescence.

The Present Study

To address the gaps in the literature discussed above, the current longitudinal study extracted data from a large national Australian sample to test early childhood individual-level risks for PA patterns extending from childhood into adolescence. The individual-level risks examined were effortful control deficits, three features of negative emotionality (e.g., anger, fear and sadness dysregulation), and the CU trait of callous-lack of empathic concern for others, all reported by mothers at age four. The PA patterns were reported by mothers from age four to 17 and by self from age 10 to 17 (when self-reported PA measures were available). Individual-level risks for mother and self-reported PA patterns were examined separately.

The following hypotheses were tested:

First, consistent with past literature (Campbell, 2006; Teymoori et al., 2018), it was hypothesized that a non-linear PA pathway of higher PA would be found, whereby PA would decline from early childhood to later childhood and rise again beginning in adolescence. Also, PA levels were expected to be consistently higher for boys compared to girls up to mid-adolescence, when negligible sex differences in PA were expected.

Next, based on past research (DeLisi & Vaughn, 2014; Frick, Ray, Thornton, & Kahn, 2014a), the individual-level risk for PA tested here were expected to significantly (and positively) account for interindividual variation in both initial levels and growth of boys' and girls' PA. One exception would be fear dysregulation, which is expected to be negatively associated with PA patterns over time (Campbell, 2006).

Finally, research also suggests that childhood risk factors for PA may not only be additive but may also be multiplicative (Eme, 2018; Ribeiro Da Silva, Rijo, & Salekin, 2015; Röll et al., 2012). In particular, early findings support the possibility for a catalysing effect of CU traits on socio-environmental and individual-level risks to conduct problems (Kroneman et al., 2011; Waller et al., 2016). However, less clarity exists on whether the associations of effortful control deficits and features of negative emotionality in early childhood with PA would differ as a function of CU. These associations were tested here hypothesizing that there would be a catalyst effect of CU, whereby the significant associations of effortful control deficits and negative emotionality with PA would be stronger when CU was high, rather than low. These catalyst effects were expected to hold for both boys and girls.

Method

Participants

Data were drawn from the *Longitudinal Study of Australian Children* (LSAC), an ongoing, multi-reporter, longitudinal study following Australian children (and their caregivers) at two-yearly intervals since they were about ages 0/1 (B cohort, N = 5000, born March 2003 to February 2004), and ages 4/5 (K cohort, N = 5000, born March 1999 to February 2000) (Growing up in Australia: The Longitudinal Study of Australian Children, 2015). For each cohort, seven waves of LSAC data were available as of 2020, with the last wave collected when children were ages 12/13 in the B cohort and ages 16/17 in the K cohort. These waves are referred to as Time 1 (T1) to Time 7 (T7) in this study. Both cohorts include self-reported data from ages 10/11 to 16/17 and parent-reported data from ages 4/5 and onwards.

After identifying children with excessive missing data or families lost to follow-up after only the first wave, 7,460 children were included in this study at baseline (T1, age range 4 to 5, M_{age}= 4.20, *SD*= 0.40, 51% boys), with 3,522 drawn from the B cohort and 3,938

drawn from the K cohort. Most mothers identified their child as having no Indigenous background (97%), and 3% of children were identified as Aboriginal Peoples or Torres Strait Islanders. Mothers ranged in educational attainment; 37% of mothers reported less or equal than 11 years of study and 63% reporting more or equal than 12 years of study. Approximately 34% of families reported their income as equal to or less than $25,999, 54% reported $26,000 to $77,999, and 12% reported a household income of $78,000 or more.

Measures

Physical aggression. One item from the Strengths and Difficulties Questionnaire (SDQ) that was completed by mothers about their children (Goodman, 1997) from T1 to T7 (e.g. *Often fights with other children or bullies them)*, and by children (Goodman, Meltzer, & Bailey, 1998) from T4 to T7 (e.g., *I fight a lot. I can make other people do what I want*) was used to isolate PA from the broader symptomatology of conduct problems (i.e., involving verbal aggression and rule-breaking behaviours). Response options ranged from 0 (*Not true*) to 2 (*Certainly true*). As in previous studies targeting PA that also used one item measure (Baglivio et al., 2015), both mother and self-reported items were carefully selected here to speak to youth's history of using PA and their control of PA (i.e., being capable of practising prosocial behavioural alternatives to PA), which are implicated in both Moffitt's (Moffitt, 1993) and DeLisi's (DeLisi & Vaughn, 2014) theories. The seven-year stability of the mother and child self-reported PA is provided in Table 1 in the results section. Because reporting single-item reliability is not possible, convergent validity was examined. First, correlations between mother and self-reported PA items and the total score for the SDQ conduct disorder scale within each time-point were calculated. Correlations were moderate in size, ranging from .52 to .62, all p <.0001 for mother and self-reported items. In addition, the seven mother-reports and the four self-reports were each averaged, and these scores were correlated

with self-reports of rule-breaking behaviour at T7[8]. Correlations were $r = .21$ for mother-reports and $r = .32$ for self-reports of PA; all $p < .0001$.

Effortful control deficits. Seven items were selected as an indicator of mother-reported children's effortful control deficits at T1. Four items from the persistence subscale of the Short Temperament Scale for Children (STSC) (Sanson, Smart, Prior, Oberklaid, & Pedlow, 1994) and three items from the hyperactivity subscale of the SDQ (Goodman et al., 1998). Items from the STSC focused on child's ability to concentrate (e.g., *in tasks this child stays with an activity (e.g. puzzle, construction, kit, reading) for a long time* – reverse item; *when a toy or game is difficult, this child quickly turns to another activity*) and persist (e.g. *when this child starts a project such as a puzzle or model, he/she works on it without stopping until it is completed, even if it takes a long time* – reverse item; *this child likes to complete one task or activity before going onto the next* – reverse item) in tasks. Items from the SDQ focused on distractibility (e.g., *easily distracted, concentration wanders*) and impulsivity/attention control traits (e.g., *can stop and think things through before acting; sees tasks through the end, good attention span* – reverse items). Response options ranged from 0 (*not true*) to 2 (*very true*). These seven items were considered to tap on child's deficits in effortful control because they relate in content to items in attention focus/shift and inhibitory control scales within previously validated child temperament and effortful control measures (Putnam, Gartstein, & Rothbart, 2006; Rothbart, Ahadi, Hershey, & Fisher, 2001; Verstraeten, Vasey, Claes, & Bijttebier, 2010) which have been used either alone (Eisenberg et al., 2003) or with their items combined (Eisenberg et al., 2010) in past research. In this study, correlations between the seven items were positive and ranged from .11 to .51, all $p < .001$. A composite effortful control deficits score was created by averaging all seven items ($\alpha = .74$).

[8] The score of rule-breaking behaviour was obtained by averaging 15 LSAC items from the Self-Report Delinquency Scale (Moffitt & Silva, 1988). In LSAC data, was $\alpha = .89$ for these 15 items.

Negative emotionality. Items from the emotional symptoms scale in the SDQ (Goodman et al., 1998) were identified as indicators of child anger (*this child often has temper tantrums or hot tempers*), fear (*this child has many fears, is easily scared*), and sadness (*this child is often unhappy, down-hearted or tearful*). Caregivers rated their child's negative emotionality from 0 (*not true*) to 2 (*very true*) at T1, and individual items were used in the analyses.

Callous-unemotional trait (CU). A score of CU corresponding to the trait of callous-lack of empathic concern for others was obtained at T1 using three mother-reported items (0 = *not true to* 2 = *very true*) from the prosocial behaviours scale of the SDQ (Goodman et al., 1998). The three items were: 1 - this child is considerate of other people's feelings, 2 - this child is helpful if someone is feeling hurt, upset or ill, and 3 - this child often volunteers to help others (parents, teachers, other children); all reversed items. These three items were similar in content to parent-reported items in the Inventory of callous-unemotional - preschool version (e.g. "this child is concerned about the feelings of others", "this child does things to make others feel good") (Frick, 2004; Willoughby et al., 2015). Furthermore, the three SDQ items used here are part of the University of New South Wales callous-unemotional scale for young children (UNSW CU) (Dadds et al., 2005), and had shown the highest loading rates in the CU dimension of such measure - .61, .74, .65, respectively for items 1, 2, and 3 above. Thus, in the present study, the three SDQ items were averaged to obtain a baseline score of callous-lack of empathic concern for others (α = .60). Reliability of SDQ and CU scales have been reported within the marginal range (α between .60 and .70) or lower in past studies (see α of SDQ and UNSW CU in Kimonis et al., 2016 and SDQ in Willoughby et al., 2015). Although marginal internal consistency would typically be a source of concern, researchers have argued that this is the case when the scale is measuring a study's

outcome, but not when the items are used to indicate a trait of a broader latent construct (Willoughby et al., 2015), as in the present study.

To further validate the composite score for the CU trait of callous-lack of empathic concern, evidence from previous studies showing positive associations of childhood CU and conduct problems through adolescence (Pardini, 2006; Pardini et al., 2007) and moderate-to-low correlations of specific traits of CU with empathy deficits and conduct problems (Ezpeleta, de la Osa, Granero, Penelo, & Domènech, 2013; Kimonis et al., 2016) were tested with LSAC data. Consistent with this past research, callous-lack of empathic concern for others at T1, as measured in this study, was positively associated with the SDQ conduct problems scale as reported by mothers from T1 to T7 (r's ranged from .12 to .33, all $p <$ .0001) and by self from T4 to T7 (r's ranged from .06 to .11, all $p < .0001$) and negatively associated with youth self-reported empathy[9] at T5 ($r_{Kcohort} = -.11$; $r_{Bcohort} = -.13$, all $p <$ .0001).

Socio-environmental level covariates. These were mother-reported household income Index (in AUD$; 0 = 25,999 or less, 1 = 26,000 to 77,999, 2 = 78,000 or more), which were established following Australian guidelines and definitions of SES status in the Australian population as well as procedures in previous studies (Australian Bureau of Statistics, 2018; Ding, Do, Schmidt, & Bauman, 2015), and child's Indigenous background (0 = no, 1 = yes) at T1.

Overview of Data Analyses

The first set of analyses considered B and K cohort differences and examined missing data and attrition. Next, descriptive statistics and correlations were calculated, and child sex

[9] Assessment of empathy (self-reported) was only available in LSAC cohorts B and K at T5. In K cohort, the empathy score was the average of the 5-item social skills scale of the Social Skills Rating System questionnaire ($\alpha = .77$ in LSAC sample) (Gresham & Elliot, 1990). In B cohort, the empathy score the average of five items ($\alpha = .86$; e.g., "I feel bad when others are sad, I try to make others feel better, I help my friends when they are having a problem, I try to think about how others feel, I am nice to others when they are feeling bad"), but no information about their original scale was found in LSAC documents.

differences in all measures were tested. Finally, a series of latent growth curve models (LGCM) were fit to mother- and child-reports of PA, separately, using Lavaan (Rosseel, 2012) and Foreign (R Core Team, 2018) packages in RStudio 1.1.456, © 2009-2016. All LGCM were estimated using maximum likelihood with standard errors and a chi-square statistic that is robust to non-normality (MLR) and FIML - an optimum approach to manage missing data, which produces unbiased parameter estimates and standard errors for missing random data (Cheung, 2007; Enders & Bandalos, 2001). Model fit was assessed using the comparative fit index (CFI), the root mean square error for estimation (RMSEA), and the standardised root mean square residual (SRMR) (Hu & Bentler, 1999). The following stepwise approach was used to the LGCM: First, to inspect the general pattern of mother and child self-reported PA (i.e., linear and curvilinear), two sets of two unconditional growth models were conducted testing the covariances between the intercepts and slopes of PA for each informant. Next, a series of conditional growth models were estimated to (1) examine sex differences in growth patterns of mother and self-reported PA over time; (2) examine the direct associations of effortful control deficits, anger, fear and sadness dysregulation, and callous-lack of empathic concern for others at age four with growth in PA over time. This was done by entering them all simultaneously as time-invariant covariates (TIC) in two LGCMs with the full sample. In the first model, sex was a TIC, and in the second, sex was a moderator in a two-group (boy/girls) model to allow estimates to differ by sex and test the same effects in boys and girls separated. And to (3) test the moderating effect of the early CU trait of callous-lack of empathic concern for others (i.e., interactions) on the association of effortful control deficits and features of negative emotionality with PA growth. The test of interactions used residual-centred orthogonalized product terms that are uncorrelated with their first-order effects (Little, Bovaird, & Widaman, 2006) to represent each interaction (e.g., effortful control deficit × CU trait, anger dysregulation × CU trait, fear dysregulation ×

CU trait, sadness dysregulation × CU trait). Each interaction was tested in two separate conditional growth models as in (2) above. In each model, each interaction term and direct effects of their corresponding higher-order variables were regressed on PA intercept and slopes. All these models were performed using mother-reported PA and then repeated using child self-reported PA. Socio-environmental risks were entered as TIC in all conditional growth models. Chi-squared difference tests were used to determine the model with the best fit in all unconditional and conditional LGCMs.

Results

B and K Cohort Comparisons, Attrition and Missing Data

B and K cohorts were compared on all study measures using χ^2 test and independent samples *t*-tests. Cohorts did not differ when comparing child sex, Indigenous status, and child self-reported PA at ages 10/11. However, quite small significant cohort differences (e.g., from .03 to .09, $p < .05$) were found for all other variables. Given such small differences, a combined dataset with both cohorts was created by merging variables from the B cohort (T3-T7) and the K cohort (T1-T7) into a single dataset, so that waves aligned to child age, maintaining T1 to T7 (*N*=9,050, 51% male). Of the 9050 participants in the merged dataset, 2176 responded to the mother-reported PA measure across all seven waves of data, and 2578 responded to the child self-reported PA measure across the final four waves of data. Regarding missing data at each wave, 667 (8%) of mothers missed one wave, 2876 (33%) missed two, 1010 (12%) missed three, 348 (4%) missed four, 359 (4%) missed five, 480 (6%) missed six, and 132 (1.5%) missed seven waves of assessment. For this study and after multiple testing of the quality of imputation approaches, mother-reported data were maintained for the primary analyses if mothers participated in T1 or T2 and at least two of the last five time-points. The above criteria resulted in a final dataset with 7460 participants at T1 (representing 83% of participants in T1 of the merged dataset). No differences in baseline

sex, SES, and Indigenous status, were found between participants excluded and those maintained in the final dataset. For child self-reported PA, 652 (7%) of children missed one wave, 3276 (36%) missed two, 990 (11%) missed three, and 1131 (12%) missed four waves of assessment (between T4 to T7). To ensure good estimation of missingness, we maintained only the subset of the 7460 participants in the main dataset, whom themselves provided self-report of PA in at least two out of the four final waves. This produced a subsample of 6551 for analyses of child-reported PA.

A dummy variable (0 = missing, 1 = non-missing) was created and χ^2 tests were performed to determine whether missingness at each wave was a function of groups of sex, Indigenous status, mother's education level, and household income, separately within mother and child self-reported data. Overall, there were many differences found in the amount of missing data for Aboriginal and/or Torres Strait Islander participants, and for low-income families relative to others. Given these differences, T1 Indigenous status and T1 household income were considered as covariates in all analyses - given no differences in PA, and other measures between cohorts B and K were found, the cohort was not a covariate in the analyses. Multiple imputations (15 imputations of missing data) in SPSS v25 were produced with all primary variables to perform descriptive statistics and correlations, and pooled results from such analyses are reported.

Descriptive Statistics, Correlations, and Sex Differences

Correlations among mother (T1 to T7) and child self-reports (T4 to T7) of PA, and sex differences in all PA measures are provided in Table 5.1. Correlations among mother and child reports of PA were predominantly significant and positive for boys (r = .06 to .37) and girls (r = .05 to .30).

Table 5.1

Sex-specific Correlations between Repeated Measures of Physical Aggression (PA), Means (M) and Standard Deviations (SD) of PA for Boys and Girls, and Results of t-tests Comparing PA in Boys and Girls (N = 7,460)

PA	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**
1. T1 MR	-	.23**	.18**	.16**	.11**	.10**	.10**	.06*	.03	.03	.05*
2. T2 MR	.30**	-	.26**	.23**	.18**	.10**	.14**	.10**	.05*	.03	.04
3. T3 MR	.24**	.35**	-	.29**	.19**	.15**	.13**	.10**	.05	.04	.06*
4. T4 MR	.24**	.35**	.37**	-	.21**	.16**	.15**	.10**	.10**	.06**	.10**
5. T5 MR	.17**	.26**	.28**	.30**	-	.20**	.20**	.07	.11**	.10**	.10**
6. T6 MR	.14**	.17**	.21**	.24**	.26**	-	.30**	.06*	.10**	.13**	.11**
7. T7 MR	.13**	.20**	.17**	.20**	.25**	.34**	-	.03	.10**	.10**	.10**
8. T4 SR	.10**	.13**	.14**	.17**	.12**	.10**	.06*	-	.13**	.10**	.10**
9. T5 SR	.04	.11**	.10**	.15**	.11**	.12**	.10**	.20**	-	.16**	.12**
10. T6 SR	.02	.06**	.06*	.10**	.10**	.15**	.11**	.12**	.21**	-	.26**
11. T7 SR	.04	.07**	.10**	.10**	.12**	.12**	.11**	.13**	.16**	.27**	-
Boys *M*	0.21	0.13	0.13	0.12	0.08	0.06	0.05	0.18	0.10	0.10	0.20
Boys *SD*	0.45	0.37	0.37	0.36	0.29	0.26	0.23	0.44	0.33	0.32	0.46
Girls *M*	0.16	0.10	0.08	0.07	0.06	0.06	0.06	0.10	0.06	0.08	0.15
Girls *SD*	0.40	0.32	0.28	0.27	0.25	0.26	0.25	0.34	0.26	0.29	0.39
t(1,7458)	5.18***	4.60***	6.32***	6.21***	3.53***	0.19	-0.50	7.06***	4.95***	1.57	3.13***

*$p < .05$. **$p < .01$. ***$p < .001$.

Note. Pearson correlations reported. Correlations for boys are shown below the diagonal. Correlations for girls are shown above the diagonal. MR = mother-report, SR = child self-report. T1 – T7 = time-points when mothers and children reported children's physical aggression.

Table 5.2

Sex-specific Correlations of Individual-Level Risk Factors with Physical Aggression (PA), and Means (M), Standard Deviations (SD), and Tests of Sex Differences (N = 7,460)

PA	Effortful control deficits	ED Anger	ED Fear	ED Sadness	CU trait
BOYS					
T1 MR	.16***	.29***	.10***	.15**	.19***
T2 MR	.16***	.23***	.10**	.11**	.14***
T3 MR	.17***	.21***	.06**	.10**	.15***
T4 MR	.10***	.22***	.08***	.05**	.12***
T5 MR	.12**	.17***	.06**	.05**	.08***
T6 MR	.12**	.17***	.03	.10***	.11***
T7 MR	.13**	.12***	.04	.10**	.12***
T4 SR	.09***	.10***	.03	.02	.05**
T5 SR	.06**	.10***	-.00	-.00	.05**
T6 SR	.03	.10**	-.00	.01	.06*
T7 SR	.05*	.10**	-.01	.00	.04
M	0.93	0.67	0.38	0.15	0.53
SD	0.42	0.67	0.58	0.41	0.42
GIRLS					
T1 MR	.18***	.29***	.08***	.13***	.18***
T2 MR	.12***	.17***	.06**	.05**	.10***
T3 MR	.12***	.17***	.02	.05**	.07***
T4 MR	.11***	.15***	.02	.02	.10***
T5 MR	.10***	.10**	.04*	.04*	.04*
T6 MR	.11***	.11**	-.01	.08**	.06*
T7 MR	.11***	.10**	.02	.06*	.09**
T4 SR	.07***	.07***	.03	.02	.06**
T5 SR	.05**	.06***	.11	.03	.03
T6 SR	.03	.08**	-.01	.02	.03
T7 SR	.07*	.10***	.01	.01	.02
M	0.82	0.64	0.35	0.13	0.42
SD	0.41	0.66	0.56	0.38	0.39
T(1,7458)	11.11***	2.04*	2.11*	2.74***	11.29***

*$p < .05$. **$p < .01$. ***$p < .001$.

Note. Pearson correlations reported. ED = Emotion dysregulation**.** CU trait = Callous-lack of empathic concern for others. MR = mother-report. SR =child self-report. T1 – T7 = time-points when mothers and children reported children's physical aggression.

Also, the means of PA were significantly higher for boys when compared to girls across mother and child self-reports from T1 to T5, but sex differences in PA levels were not significant both at T6 for both informants and at T7 for mother-reported PA.

Correlations between PA measures and mother-reports of effortful control deficits and features of both negative emotionality and CU are shown in Table 5.2. There were predominantly significant and positive correlations between T1 effortful control deficits, anger dysregulation, and callous-lack of empathic concern for others with PA reports from T1 to T7 for boys (r = .05 to .29) and girls (r = .04 to .29). Correlations of mother-reported fear (r = .06 to .10) and sadness (r = .05 to .15) dysregulation with PA were mostly positive and significantly associated in boys. Boys had significantly higher means for all tested individual-level risks in relation to girls.

General PA Patterns and Sex-Differences in Mother and Self-Reported PA Over Time

Four unconditional LGCMs were conducted to identify the general pattern (i.e., linear vs. curvilinear/quadratic) of mother and child self-reported PA. Table 5.3 shows the model fits and the estimates for all parameters in two of these models, one with mother and the other with self-reported PA. In all models, significant changes over time (linear and quadratic slopes) were found. The quadratic models showed an average significant linear decrease of PA over time but also a U-shaped quadratic growth in PA over time for both mother-report and child-report of PA. Also, the variances in models estimates indicated significant interindividual differences in initial levels and change in PA over time. The results of the χ^2 difference tests comparing the models suggested that the quadratic model had a significantly better fit relative to the linear model in both cases: mother-report $\chi^2_{diff}(4) = 83.88$, $p < .001$; child self-report $\chi^2_{diff}(4) = 159.229$, $p < .001$. In the following analyses, all models tested built on the unconditional quadratic model.

Table 5.3

Results of Unconditional Growth Models of Mother-Reported (T1 to T7) and Self-Reported (T4 to T7) Physical Aggression (PA, N = 7,460)

	Mother-report PA[1] *B* (*SE*)	Child self-report PA[2] *B* (*SE*)
Linear model[a]		
Intercept		
Mean	0.133 (0.003)***	0.091 (0.003)***
Variance	0.043 (0.003)***	0.016 (0.003)***
Slope		
Mean	-0.016 (0.001)***	0.000 (0.002)
Variance	0.001 (0.001)***	0.002 (0.001)**
Quadratic model[b]		
Intercept		
Mean	0.152 (0.004)***	0.123 (0.004)***
Variance	0.044 (0.003)***	0.034 (0.007)***
Linear slope		
Mean	-0.035 (0.002)***	-0.087 (0.006)***
Variance	0.007 (0.001)***	0.032 (0.010)**
Quadratic shape		
Mean	0.003 (0.000)***	0.032 (0.002)***
Variance	0.000 (0.000)***	0.002 (0.001)*

*$p < .05$. **$p < .001$. ***$p < .0001$.

Model Fit: [1a]χ^2 (23) = 144.02, $p < .0001$; CFI=.95, RMSEA=.043, SRMR= .048

[1b]χ^2 (19) = 60.14, $p < .0001$; CFI=.98, RMSEA=.027, SRMR= .033

[2a]χ^2 (5) = 161.02, $p < .0001$; CFI=.64, RMSEA=.089, SRMR= .064

[2b]χ^2 (1) = 1.80, $p < .0001$; CFI=1.00, RMSEA=.011, SRMR= .005

Next, two conditional LGCMs were estimated to examine sex differences in growth patterns of mother and self-reported PA over time, while also accounting for socio-environmental covariates. As shown in Table 5.4, these two models had an excellent fit to the data. As reported by their mothers, girls were significantly lower in PA at T1, but also PA declined less for girls from T1 to T7, and the U-shaped pattern was more prominent for girls compared to boys, as girls showed a more of an upturn in PA at about age 14/15 years. For self-report of PA, girls reported a significantly lower initial level of PA at T4, less decline in PA from T4 to T7, but a less prominent U-shape PA growth than boys. Figure 5.1 illustrates these results by showing an overall decline in PA from age 4/5 and from ages 10/11 to 12/13

for both boys and girls, but then an increase in PA beginning at age 14/15 years for boys and age 12/13 for girls.

Table 5.4

Results of Quadratic Growth Curve Models Testing Child Sex and Socio-environmental Factors as Covariates of Mother-Reported (T1 to T7) and Self-Reported (T4 to T7) Physical Aggression (PA, N = 7,460)

	Mother-report PA *B* (*SE*)	Child self-report PA *B* (*SE*)
Intercept		
Mean	0.195 (0.008)***	0.163 (0.008)***
Variance	0.043 (0.003)***	0.030 (0.007)***
Sex (girl)	-0.044 (0.008)***	-0.059 (0.008)***
Indigenous background (yes)	0.061 (0.029)*	0.093 (0.033)**
Household income	-0.032 (0.006)***	-0.018 (0.006)**
Linear slope		
Mean	-0.038 (0.005)***	-0.123 (0.012)***
Variance	0.007 (0.001)***	0.023 (0.011)*
Sex (girl)	-0.002 (0.005)	0.044 (0.012)***
Indigenous background (yes)	0.037 (0.018)*	-0.065 (0.052)
Household income	0.006 (0.004)	0.022 (0.010)*
Quadratic shape		
Mean	0.003 (0.001)**	0.043 (0.004)***
Variance	0.000 (0.000)***	0.001 (0.001)
Sex (girl)	0.002 (0.001)*	-0.012 (0.004)**
Indigenous background (yes)	-0.008 (0.003)**	0.018 (0.017)
Household income	-0.001 (0.001)	-0.006 (0.003)*

*p <.01. **p <.001. ***p < .0001.

Note. Indigenous status was coded 0=Non-Indigenous, 1=Aboriginal, and/or Torres Strait Islander. Household income was coded 0=low, 1=mid, 2=high income. Mother-report model fit (T1 to T7): χ^2 (31) = 89.845, p < .0001; CFI=.98, RMSEA=.023, SRMR= .027 Child report model fit (T4 to T7): χ^2 (4) = 3.372, p < .0001; CFI=1.00, RMSEA=.000, SRMR= .005.

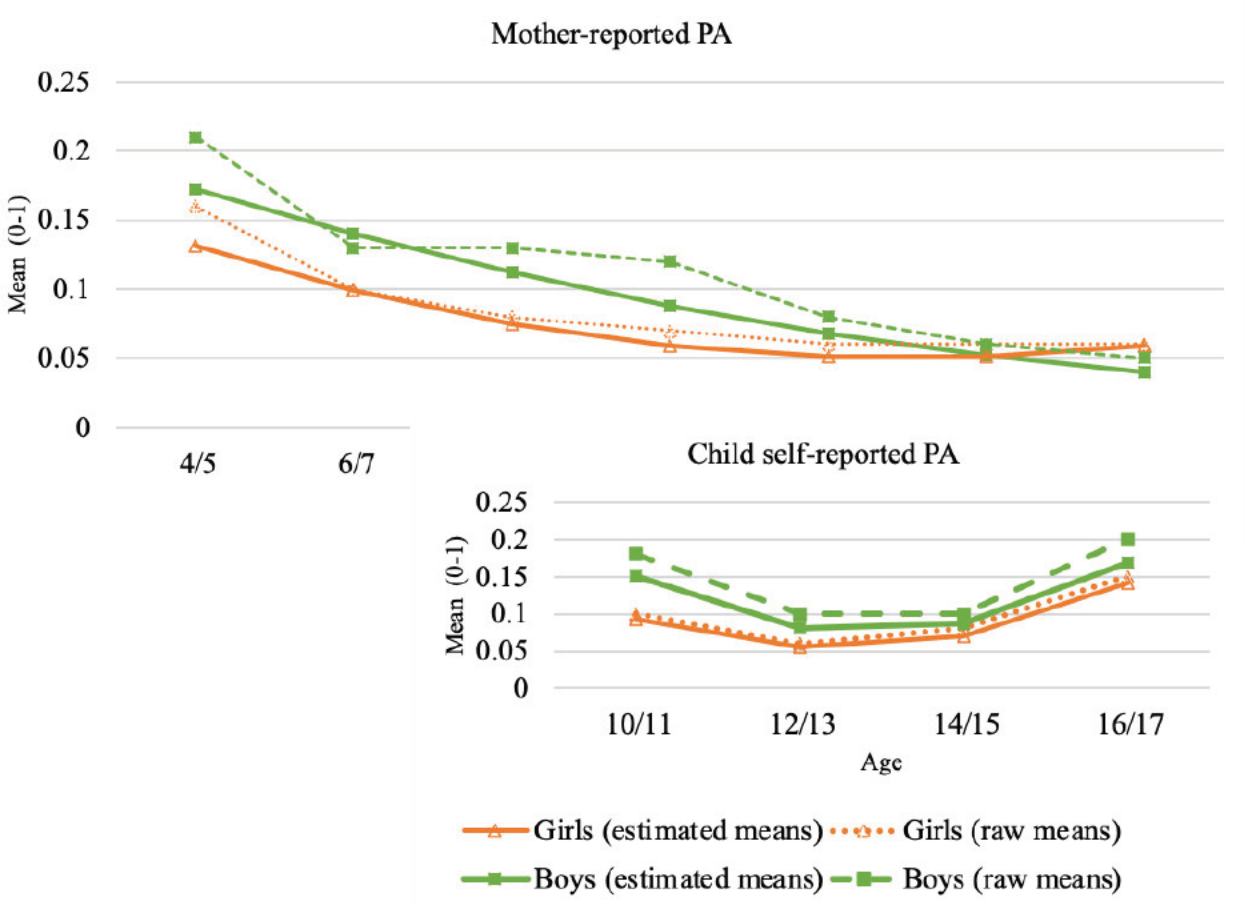

Figure 5.1. Boys' (N=3,816) and girls' (N=3,644) observed (raw means) and predicted (estimated means) mother-reported PA (coded 0 = no PA, 1 = else) from T1 to T7 and child self-reported PA per age from T1 to T7. PA = physical aggression.

Direct Effects of Early Childhood Individual-level Risks on Growth in PA

Two sets of two LCGMs were performed to test the direct associations of effortful control deficits, anger, fear and sadness dysregulation, and callous-lack of empathic concern for others at age four with growth in mother and self-reported PA. In each set of analyses, two models with the full sample were tested, one with estimates for all parameters and another with the estimates freed to differ for boys and girls. These models were tested considering the full LSAC sample, and boys and girls separated. Tables 5.5 and 5.6 show the model fits and the estimates for the tested models in each set.

Mother-reported PA. As shown in Table 5.5, all individual-level risk factors were significantly associated with a higher initial level of PA (full sample model). Regarding PA slopes, higher levels of anger and sadness dysregulation and the CU trait of callous-lack of empathic concern for others at T1 were significantly associated with a steeper decline but a more prominent U-shaped trajectory of PA from T1 to T7.

When the model estimates were allowed to differ for boys and girls, no significant sex differences in associations of predictors with PA patterns (intercepts, slopes or quadratic pattern) were found. The χ^2 difference test comparing the 2-group model to a model with all estimates constrained to be equal for boys and girls did not differ in fits, χ^2_{diff} (55) = 63.117, $p < .20$. Also, in general, the results were rather similar for boys and girls. However, two differences in significance across the models for boys and girls were found. First, boys', but not girls', fear dysregulation was significantly associated with a higher initial level of PA. Second, girls', but not boys', anger dysregulation was associated with a sharper quadratic upturn of PA in later adolescence.

Child self-reported PA. As presented in Table 5.6, the first LGCM with the full LSAC sample showed significant associations of mother-reported anger dysregulation and effortful control at T1 with higher initial levels of PA at T4. Also, effortful control deficits were associated with more linear decline, and anger dysregulation was associated with a more prominent U-shaped curve of PA from T4 to T7. When the model estimates were allowed to differ for boys and girls, no significant sex differences in associations of predictors with PA patterns (intercepts, slopes or quadratic pattern) were found. The results of the χ^2 difference tests comparing this 2-group model to a model with all estimates constrained to be equal for boys and girls suggest both models fitted the data equally well χ^2_{diff} (10) = 5.750, $p < .20$.

Table 5.5

Results of the Full Quadratic Growth Curve Model for the Combined Sample and for the Two-Group (Boy/Girl) Quadratic Model of Individual-Level risks for Mother-Reported Physical Aggression (N = 7,460)

	Full sample	Boys	Girls
Effects	*B* (*SE*)	*B* (*SE*)	*B* (*SE*)
Intercept			
Mean	-0.021 (0.011)	-0.026 (0.015)	-0.039 (0.013)**
Variance	0.033 (0.003)***	0.035 (0.004)***	0.031 (0.004)***
Anger dysregulation	0.113 (0.007)***	0.117 (0.010)***	0.110 (0.009)***
Fear dysregulation	0.022 (0.008)**	0.024 (0.011)*	0.019 (0.010)
Sadness dysregulation	0.072 (0.012)***	0.081 (0.018)***	0.062 (0.016)***
Effortful control deficits	0.068 (0.010)***	0.069 (0.014)***	0.069 (0.014)***
CU trait	0.100 (0.011)***	0.097 (0.016)***	0.103 (0.016)***
Linear slope			
Mean	0.017 (0.007)*	0.007 (0.010)	0.019 (0.009)*
Variance	0.007 (0.001)***	0.005 (0.002)*	0.008 (0.002)***
Anger dysregulation	-0.025 (0.004)***	-0.016 (0.006)*	-0.035 (0.006)***
Fear dysregulation	-0.005 (0.005)	-0.002 (0.007)	-0.008 (0.006)
Sadness dysregulation	-0.035 (0.008)***	-0.038 (0.011)**	-0.033 (0.010)**
Effortful control deficits	-0.009 (0.007)	-0.007 (0.009)	-0.012 (0.009)
CU trait	-0.039 (0.007)***	-0.029 (0.010)**	-0.054 (0.010)***
Quadratic shape			
Mean	-0.003 (0.001)*	-0.001 (0.002)	-0.002 (0.001)
Variance	0.000 (0.000)***	0.000 (0.000)*	0.000 (0.000)
Anger dysregulation	0.002 (0.001)**	0.000 (0.001)	0.004 (0.001)***
Fear dysregulation	0.000 (0.001)	-0.000 (0.001)	0.001 (0.001)
Sadness dysregulation	0.005 (0.001)***	0.005 (0.002)**	0.005 (0.002)**
Effortful control deficits	0.001 (0.001)	0.000 (0.002)	0.001 (0.002)
CU trait	0.005 (0.001)***	0.003 (0.002)*	0.007 (0.002)***

* $p < .05$. **$p < .001$. *** $p < .0001$.

Note. Mothers reported on their children's PA seven times from age 4 to 17. Both models were adjusted for covariates: Indigenous status, and household income (see Table 4). Sex was included as a covariate in the full sample model. CU trait = Callous-lack of empathic concern for others. Model fit: Full sample model: $\chi 2$ (51) = 150.378, $p < .0001$, CFI=.976, RMSEA=.018, SRMR= .020. Two-group model: $\chi 2(94) = 195.786$, $p < .0001$; CFI=.973, RMSEA=.019, SRMR= .023.

Table 5.6

Results of the Full Quadratic Growth Curve Model for the Combined Sample and for the Two-Group (Boy/Girl) Quadratic Model of Early Individual-Level risks for Child Self-Reported Physical Aggression (N = 7,460)

	Full sample	Boys	Girls
Effects	*B* (*SE*)	*B* (*SE*)	*B* (*SE*)
Intercept			
Mean	0.104 (0.014)***	0.103 (0.020)***	0.049 (0.015)**
Variance	0.028 (0.007)***	0.044 (0.012)***	0.012 (0.007)
Anger dysregulation	0.032 (0.007)***	0.039 (0.011)***	0.023 (0.009)*
Fear dysregulation	0.010 (0.008)	0.006 (0.012)	0.014 (0.011)
Sadness dysregulation	-0.011 (0.011)	-0.023 (0.016)	0.002 (0.014)
Effortful control deficits	0.035 (0.011)**	0.045 (0.017)*	0.026 (0.014)
CU trait	0.014 (0.012)	0.011 (0.017)	0.016 (0.016)
Linear slope			
Mean	-0.082 (0.020)***	-0.097 (0.028)***	-0.025 (0.022)
Variance	0.022 (0.011)*	0.026 (0.018)	0.017 (0.012)
Anger dysregulation	-0.019 (0.011)	-0.028 (0.015)	-0.010 (0.015)
Fear dysregulation	-0.013 (0.012)	-0.017 (0.016)	-0.008 (0.017)
Sadness dysregulation	0.022 (0.016)	0.019 (0.023)	0.025 (0.023)
Effortful control deficits	-0.033 (0.016)*	-0.032 (0.024)	-0.034 (0.022)
CU trait	-0.002 (0.017)	0.021 (0.024)	-0.029 (0.023)
Quadratic shape			
Mean	0.029 (0.007)***	0.038 (0.009)***	0.010 (0.007)
Variance	0.001 (0.001)	0.001 (0.002)	0.002 (0.001)
Anger dysregulation	0.008 (0.004)*	0.009 (0.005)	0.006 (0.005)
Fear dysregulation	0.003 (0.004)	0.003 (0.006)	0.002 (0.006)
Sadness dysregulation	-0.007 (0.005)	-0.003 (0.008)	-0.011 (0.008)
Effortful control deficits	0.010 (0.006)	0.005 (0.008)	0.014 (0.007)*
CU trait	0.001 (0.006)	-0.005 (0.008)	0.007 (0.008)

* $p < .05$. **$p < .001$. *** $p < .0001$.

Note. Adolescents reported on their PA four times from age 10 to 17. Both models were adjusted for covariates: Indigenous status, and household income (see Table 5.4). Sex was included as a covariate in the full sample model. CU trait = Callous-lack of empathic concern for others. Model fit: Full sample model: $\chi 2\ (9) = 5.575$, $p = .782$, CFI= 1.000, RMSEA=.000, SRMR= .004. Two-group model: $\chi 2(16) = 10.406$, $p = .845$; CFI = 1.000, RMSEA=.000, SRMR= .005.

Common to both sexes, anger dysregulation was significantly and positively associated with PA intercept at T4. In contrast, the risk factor of effortful control deficits was significantly associated with both a higher PA intercept for boys, but not for girls and with a more prominent U-shaped curve of PA from T4 to T7 for girls, but not for boys.

Testing Interaction Effects at T1 Predicting Growth in PA

Two sets of eight LCGMs were conducted to test whether each of the four interactions of effortful control deficits and anger, fear and sadness dysregulation with the CU trait of callous-lack of empathic concern for others would help explain interindividual variation in growth in mother and child self-reported PA considering the full LSAC sample and boys and girls separately. All models fitted the data adequately, and the estimates for all interaction terms are shown in Tables 5.7 and 5.8. All significant effects were observed above and beyond the main effects of the corresponding first-order variables and the covariates in the model (Indigenous status, household income).

Mother-reported PA. As seen in Table 5.7, the LCGMs examining each interaction in the full sample showed higher levels of callous-lack of empathic concern for others increased child's risk for engagement in PA at higher levels of both anger and sadness dysregulation, all at T1. Also seen in Table 5.7, when the model estimates were allowed to differ for boys and girls, one interaction was associated with PA growth in boys and girls and another one only with PA growth in girls. In particular, the presence of higher levels of both anger dysregulation and callous-lack of empathic concern for others (anger dysregulation × CU trait) was significantly associated with a higher PA intercept for both boys and girls. Also, the combination of higher levels of both sadness dysregulation and callous-lack of empathic concern for others (sadness dysregulation × CU trait) was significantly associated with both a higher intercept and a steeper linear decline in PA in girls, but not in boys.

Table 5.7

Summary of the Interaction Effects in Growth Curve Models of Mother-reported Physical Aggression (N = 7,460)

Effects	Full sample *B* (*SE*)	Boys *B* (*SE*)	Girls *B* (*SE*)
Intercept			
Anger dysregulation × CU trait	0.061 (0.016)***	0.050 (0.022)*	0.081 (0.024)**
Fear dysregulation × CU trait	0.030 (0.019)	0.028 (0.027)	0.030 (0.028)
Sadness dysregulation × CU trait	0.060 (0.031)*	0.017 (0.044)	0.117 (0.044)**
Effortful control deficits × CU trait	0.044 (0.025)	0.027 (0.034)	0.070 (0.040)
Linear slope			
Anger dysregulation × CU trait	-0.013 (0.011)	-0.013 (0.014)	-0.025 (0.016)
Fear dysregulation × CU trait	-0.007 (0.012)	-0.004 (0.016)	-0.016 (0.018)
Sadness dysregulation × CU trait	-0.026 (0.020)	-0.008 (0.028)	-0.058 (0.026)*
Effortful control deficits × CU trait	0.014 (0.016)	0.004 (0.022)	0.008 (0.026)
Quadratic shape			
Anger dysregulation × CU trait	0.001 (0.002)	0.001 (0.002)	0.002 (0.003)
Fear dysregulation × CU trait	0.001 (0.002)	0.000 (0.003)	0.002 (0.003)
Sadness dysregulation × CU trait	0.005 (0.003)	0.004 (0.005)	0.007 (0.004)
Effortful control deficits × CU trait	-0.001 (0.003)	0.001 (0.004)	-0.001 (0.004)

*$p < .05$. **$p < .001$. ***$p < .001$.

Note. Mothers reported on their children's PA seven times from age 4 to 17. Each interaction is a residual-centred term that was tested in a separate model. The direct effects of each of the two variables in the interaction, as well as covariates of Indigenous status and household income (see Table 5.4) (and sex in the model with the full sample), were all entered simultaneously. Model fit statistics were adequate for all models tested with the full sample and the two-group (boy/girl) models. CU trait = Callous-lack of empathic concern for others.

Child self-reported PA. As presented in Table 5.8, the LCGMs examining each interaction in the full sample indicate that the interactions of effortful control deficits and fear dysregulation with callous-lack of empathic concern for others were significantly associated with PA. The combination of higher anger and fear dysregulation with high CU, and the combination of high effortful control deficits and high CU were all significantly associated with a higher initial level of PA. Also, the combinations of fear dysregulation and effortful control deficits with CU were each associated with a steeper decrease in PA from T4 to T7, but also a more prominent U-shaped trajectory of PA, with a more prominent upturn in PA in later adolescence. Table 5.8 also shows that, when model estimates were allowed to differ for boys and girls, significant associations were found only for boys. In particular, among boys, two interactions (anger dysregulation × CU trait; effortful control deficits × CU trait) remained significantly associated with the PA intercepts and one interaction (Fear dysregulation × CU trait) remained associated with both the linear slope and the quadratic pattern of PA.

Discussion

This multi-informant thirteen-year longitudinal study addressed early childhood risks of low effortful control deficits and features of both negative emotionality (i.e., anger, fear and sadness dysregulation) and CU traits (i.e., callous-lack of empathic concern for others) to identify boys and girls most at risk for engaging in problematic PA through late adolescence. More specifically, both growth and sex differences in mother- (ages 4 to 17) and self- (ages 10 to 17) reported PA trajectories over time were mapped first. Next, the direct associations of specific individual-level risks were tested. Finally, a possible catalyst effect (i.e., interactions) of CU on the associations found for effortful control deficits and features of negative emotionality on boys' and girls' growth in PA over time was examined.

Table 5.8

Summary of the Interaction Effects in Growth Curve Models of Child Self-reported Physical Aggression (N = 7,460)

Effects	Full sample *B* (*SE*)	Boys *B* (*SE*)	Girls *B* (*SE*)
Intercept			
Anger dysregulation × CU trait	0.036 (0.017)*	0.046 (0.024)*	0.015 (0.022)
Fear dysregulation × CU trait	0.038 (0.020)*	0.045 (0.028)	0.033 (0.029)
Sadness dysregulation × CU trait	0.026 (0.025)	0.051 (0.035)	0.003 (0.036)
Effortful control deficits × CU trait	0.069 (0.027)*	0.074 (0.038)*	0.056 (0.037)
Linear slope			
Anger dysregulation × CU trait	-0.042 (0.024)	-0.033 (0.034)	-0.049 (0.034)
Fear dysregulation × CU trait	-0.067 (0.028)*	-0.088 (0.037)*	-0.040 (0.043)
Sadness dysregulation × CU trait	-0.038 (0.041)	-0.072 (0.057)	0.009 (0.057)
Effortful control deficits × CU trait	-0.076 (0.038)*	-0.086 (0.052)	-0.078 (0.057)
Quadratic shape			
Anger dysregulation × CU trait	0.011 (0.008)	0.005 (0.011)	0.017 (0.011)
Fear dysregulation × CU trait	0.020 (0.009)*	0.029 (0.013)*	0.008 (0.014)
Sadness dysregulation × CU trait	0.019 (0.013)	0.029 (0.019)	0.004 (0.017)
Effortful control deficits × CU trait	0.026 (0.012)*	0.029 (0.017)	0.030 (0.019)

*$p < .05$.

Note. Adolescents reported on their PA four times from age 10 to 17. Each interaction is a residual-centred term that was tested in a separate model. The direct effects of each of the two variables in the interaction, as well as covariates of Indigenous status and household income (see Table 5.4) (and sex in the model with the full sample), were all entered simultaneously. Model fit statistics were adequate for all models tested with the full sample and the two-group (boys/girls) models. CU trait = Callous-lack of empathic concern for others.

Overall, study findings indicated that the average growth pattern of mother-reported and self-reported PA across the seven waves and four waves of data, respectively, was best represented by a quadratic shape for boys and girls. This average pattern held for boys and girls, but sex differences in level (boys higher) and stability (girls more stable) were also found. Moreover, both the direct and interaction effects of effortful control deficits, anger, fear, and sadness dysregulation, and callous-lack of empathic concern for others predicted growth in PA for both boys and girls up to 13 years later. This pattern was especially robust when PA was reported by mothers but also when children themselves reported PA. In particular, the results showed the unique role of all tested individual-level risks in predicting higher initial levels of mother-reported PA at age four and the role of anger and sadness dysregulation and callous-lack of empathic concern for others in predicting less decline and a more substantial upturn in mother-reported PA into adolescence. For child-self-reported PA, the unique role of effortful control deficits and anger dysregulation on initial levels and change in PA over time were found. Furthermore, the combination of effortful control deficits and anger dysregulation with the tested CU trait in boys and anger and sadness dysregulation with the tested CU trait in girls were particularly risky for their engagement in higher PA pathways.

Growth Patterns of PA: Full Sample and by Sex

Consistent with the literature (Campbell, 2006; Teymoori et al., 2018) and hypothesis in this study, the average child exhibits a curvilinear pattern of PA that declines from age four and swings upward again from about age 14. The focus here on mapping PA based on reports from mothers and, separately, from self-report showed some similarities, but also some differences (see Figure 1). First, regarding similarities, initial levels of PA were reported higher for boys relative to girls by both mother and self. Also, a more pronounced upswing in girls' PA (relative to boys) at around the age of 12 was reported by both informants. Taken

together, these two pieces of evidence may help explain the widely reported lack of stability in sex differences in PA development (Campbell, 2006; Piquero, Carriaga, Diamond, Kazemian, & Farrington, 2012; Pitzer, Esser, Schmidt, & Laucht, 2010; Teymoori et al., 2018).

In terms of differences, the average girl (relative to the average boy) had a more pronounced U-shaped pattern when mothers reported their child's PA, but a slightly less pronounced U-shaped pattern when PA was self-reported. In contrast, the average boy reported a more prominent U-shaped pattern to their own PA; mothers did not report this same increase in their adolescent sons' PA. That is, according to their mothers, girls may exhibit less PA in early childhood (and perhaps at any point in childhood) than boys, but also seem more vulnerable to persistence in PA and an upswing in PA in adolescence. However, when PA is self-reported, an average upswing in PA occurs for boys and girls from around the age of 12. Although comparing PA across different informants is beyond the scope of this paper, previous longitudinal studies (Campbell, 2006; Teymoori et al., 2018) have reported similar results of differences in sex-specific patterns of PA when these are reported by distinct informants from childhood to adolescence. Further, the differences in PA growth across mother and child-reported PA in this study are also consistent with empirical research examining and explaining discrepancies in either (or both) parent- or child- reports of PA (De Los Reyes, Goodman, Kliewer, & Reid-Quiñones, 2010; De Los Reyes, 2013; Ksinan & Vazsonyi, 2016). That said, findings speak to the utility of considering multiple informants when mapping childhood behaviour, and more specifically, suggests that mother-reported PA is tapping behaviour that overlaps, but is distinct from youth self-reports.

The Role of Individual-Level Risks and Their Interactions on Growth Patterns of PA

Overall, the direct and interactive effects of effortful control deficits, anger, fear and sadness dysregulation, and callous-lack of empathic concern for others with growth in boys'

and girls' PA were partially consistent with this study's hypotheses. Consistent with theory (DeLisi & Vaughn, 2014; Frick et al., 2014a), the direct associations found here were especially robust when PA was reported by mothers, but many associations were also found when children themselves reported PA. In particular, the results showed the unique role of all tested individual-level risks in predicting higher initial levels of mother-reported PA at age four and the role of anger and sadness dysregulation and callous-lack of empathic concern for others in predicting less decline and a more substantial upturn in mother-reported PA into adolescence. For self-reported PA, the unique role of effortful control deficits and anger dysregulation on initial levels and change in PA over time were found. Furthermore, the combination of effortful control deficits and anger dysregulation with the tested CU trait in boys and anger and sadness dysregulation with the tested CU trait in girls were particularly risky for their engagement in higher PA pathways.

Direct effects of effortful control deficits. Children who had more deficient effortful control skills at age four were also reported by both their mothers and by themselves to exhibit higher PA at ages four and 10, respectively. In contrast, less evidence was found for a significant role of early effortful control deficits on change in PA patterns over time. These findings are consistent with evidence that engagement in high levels of PA may reflect youths' current deficits in the modulation of cognition, emotions, and behaviours, which leads to a failure in achieving an optimum level of social and personal competence (DeLisi et al., 2018; DeLisi & Vaughn, 2014; Nigg, 2017). However, the lack of significant associations of deficits in effortful control with PA growth into adolescence (i.e., the slope and the upward curve in adolescence) seems to suggest its primary role in child's decision/control upon their engagement in PA behaviours – as opposed to the continuation of PA. Interestingly, this could indicate that assessment and treatment of effortful control deficits in early childhood may be more relevant in preventing than in mitigating PA patterns into adolescence.

Direct effects of features of negative emotionality. The significant role of anger dysregulation at age four on both initial levels and change in PA into late adolescence found across both informants and boys and girls was notable. Such findings are consistent with evidence that difficulty regulating anger in childhood is the strongest correlate of high PA across childhood and adolescence (Berkowitz, 2012; Campbell, 2006; Hay, 2017; Hay et al., 2014; McLaughlin et al., 2011). In addition, these findings also add to current research by showing the relevance of anger dysregulation in early childhood for escalating use of PA, especially during its upswing time (post-age 12).

In contrast, the direct associations of early childhood fear and sadness dysregulation had less consistent associations with PA but were not unimportant. Associations of fear dysregulation and PA patterns over time were generally not significant and seemed to be more relevant in the presence of the CU trait tested here; hence, it will be further discussed below. Moreover, early sadness dysregulation was associated with higher initial levels and continuation of mother-reported PA, but these results were not replicated in child self-reported PA. These findings are somewhat consistent with other longitudinal work indicating that sadness dysregulation in late childhood predicts elevated self and peer-reports of early adolescent PA (McLaughlin et al., 2011). However, longitudinal studies reporting on the role of sadness dysregulation on aggression are scarce in the literature, and more research with measures with good internal consistency is needed to access more reliable and informative results in this arena.

Direct effects of callous-lack of empathic concern. Overall, findings clearly showed that children higher in CU trait were also higher in mother-reported PA at age four and showed a more prominent upswing in PA in adolescence. In addition, these associations held for both boys and girls. Complementarily to supporting this study's hypotheses, these findings are also consistent with past evidence confirming the association of CU traits with

higher levels of PA (Frick et al., 2014b; Frick & White, 2008). However, these same results were not found in the analyses of child self-reported PA, where the CU trait was not associated with PA. At least three possible explanations exist for why this was the case. First, the items measuring the CU trait investigated here are similar in content to those assessing cognitive empathy (Dadds et al., 2009). The stability of cognitive empathy has shown variations from childhood to adolescence, as youth who lack cognitive empathy may learn to compensate for it in adolescence, as they became more skilled in social interactions/expectations (Dadds et al., 2009). Second, the latent structure of CU traits seems to become more complex as the youth approach adolescence. Some argue that it is possibly bi-dimensional in childhood (Kimonis et al., 2016; Willoughby et al., 2015) but may include four distinct traits by adolescence or later (American Psychiatric Association, 2013; Frick et al., 2014a), of which callous-lack of empathic concern for others is just another one. Third, our mother-reported and child-reported PA items tapped on somewhat different dimensions of aggression (the former more overt and measured from age 4, the later experienced internally and measured beginning in mid-childhood). Thus, the CU trait measured here in childhood may change over time and need to be considered alongside other traits that emerge later to fully identify its role in PA that is outside the awareness of a parent (i.e., self-reported PA). This more complex pattern of associations should be tested in future studies examining longitudinal associations of distinct CU traits with problem PA pathways. However, it should be noted that the CU trait measured here did work in concert with other individual risk factors to have a significant effect on PA level and growth, even in the models of self-reported PA (discussed below). In fact, although small in magnitude, significant interactions effects were more numerous in the models of self-report PA relative to the models of mother-reported PA.

Interaction effects. The expected catalyst effects of callous-lack of empathic concern for others on associations of all tested risks with PA pathways were also found, but the significance of interactions of effortful control deficits and anger, fear, and sadness dysregulation with the CU trait varied across mother and child self-reports of PA. These results are consistent with evidence that childhood risk factors for PA may not only be additive but may also be multiplicative (Eme, 2018; Ribeiro Da Silva et al., 2015; Röll et al., 2012). However, they also expand on current research by suggesting that a specific dimension of CU (i.e., callous-lack of empathic concern for others) could be successfully assessed in early childhood and place children that have deficits in effortful control or difficulties in regulating anger or fear at an increased risk for higher initial levels and continuation of PA into adolescence. Previous studies had only reported such catalyst effect for CU traits in childhood (but not early childhood) and with socio-environmental risks and executive functions (Kroneman et al., 2011; Waller et al., 2016). In line with the temperament-based theory of antisocial behaviour, the few significant catalyst effects in this study indicated that the presence of callous-lack of empathic concern for others at age four, coinciding with poor effortful control skills, may reduce children's willingness or ability to learn and apply a broader repertoire of more socially appropriate behaviours due to lower levels of empathic concern for others (DeLisi & Vaughn, 2014; Eme, 2018). These combined deficits may result in further difficulties for children when they need to control impulses and/or strong emotions of anger or fear in daily life. Such difficulties would, in turn, either maintain or increase children's negative emotionality when interacting with other individuals (i.e., peers, relatives, and teachers), therefore perpetuating emotion dysregulation tendencies, as well as PA.

Sex-specificities in individual-level risks for PA pathways. Generally, only a few sex specificities in both main and interaction effects of individual-level risks for PA emerge

when estimates were allowed to vary between boys and girls. These include the significant effects of fear dysregulation and effortful control on initial levels of mother and self-reported PA, respectively, for boys-only; as well as the significant combination of both effortful control deficits and fear dysregulation with the CU trait in boys and sadness dysregulation with the CU trait in girls. Consistent with both theory (Eme, 2018) and previous evidence (Campbell, 2013; Chaplin & Aldao, 2013; Else-Quest et al., 2006; Eme, 2018), these results seem to support the lower vulnerability for PA in females, relative to males, as a result of lower impulsivity and higher levels of fear in females. Such sex-specificities may be attributable to biological biases/mechanisms associated emotional and behavioural tendencies (Archer, 2009) but also to socio-cultural aspects, such as parenting different conventions in relation to gender (e.g. more careful and danger aversion with girls; for a more detailed explanation, see Wood & Eagly, 2002). In any case, practical implications of these sex-specific findings concern with interventions designed to target problem PA in boys and girls. These should focus mainly on strategies to boost and develop regulation of anger in both sexes, while also considering that children high in the trait of callous-lack of empathic concern for others are at most risk for PA.

Strengths, Limitations, and Directions for Future Research

This study has several strengths, including using data from a large national sample, across multiple informants (mother and self), and over 13 years from baseline measures completed at age four. These features enhance the generalizability of findings, speak to differences in outcomes across reporters, provide ample power to test effects, and characterize a long-term PA developmental progression. This study also has several limitations related to the measurements used that merit discussion. First, three single-items were employed to assess traits of anger, fear, and sadness dysregulation as features of negative emotionality. Although the use of single items meant that measurement reliability

could not be calculated, cross-informant and significant associations of these items at baseline with PA 13 years later were still found, which were also in line with the literature. Second, baseline CU focused on the trait of callous-lack of empathic concern for others assessed by a composite of three items from the prosocial behaviour scale of the SDQ. This may leave room for questions as to which extend the operationalisation of the CU maps onto the full construct of CU traits. It is worth noting that many other studies have likewise used items in a pre-existent measure to create a proxy-measure of CU traits in early childhood (Kimonis et al., 2016; Willoughby, Waschbusch, Moore, & Propper, 2011), and the three items used here are a third of the items in the UNSW CU scale (Dadds et al., 2005). In addition, more confidence in the measurement of callous-lack of empathic concern for others in the present study can be drawn from its significant correlations with conduct problems and empathy up to eight years later and across distinct informants in the LSAC sample (see details in the method section). In addition, the role of empathic traits in inhibiting antisocial behaviours through motivating/eliciting prosocial emotions is well described in the literature (Frick et al., 2014a) and some have argued that empathy is one of the best indicators of the overall construct of CU traits (Frick & Viding, 2009). Thus, in relation to the assessment of callous-lack of empathic concern for others, this study provides a useful proof of concept, in that a particular indicator of the broader latent construct of CU traits is related to PA growth over time.

Third, single items were used to assess PA. Although the use of one (Baglivio et al., 2015) or a maximum of three-items (Petts, 2009; Reingle, Maldonado-Molina, Jennings, & Komro, 2012) is common in longitudinal studies of PA, the use of single items here meant that measurement reliability could not be calculated. Thus, special attention was given to checking stability over time and validating the PA items with measures of conduct disorder and rule-breaking behaviour over the seven LSAC waves (see details in the method section).

Nonetheless, the identified PA growth patterns were consistent with developmental theories, and evidence from previous empirical studies. Notably, there was a focus here on PA as a specific outcome of theory-based predictors from early childhood, and results may not be generalizable to the development of other forms of antisocial behaviour (e.g., rule-breaking behaviour). Future studies could also extend on this work by focusing on other forms of aggression (i.e., relational, verbal) across childhood and adolescence. Fourth, the somewhat low internal consistency of some measures may have hampered our ability to detect effects. Although we employed a large sample, many of the associations found here were small. That said, small effect sizes are usually reported and even expected in long-term longitudinal studies (over ten years from T1) with large sample sizes (Nivette, Eisner, Malti, & Ribeaud, 2014; Verhulst et al., 2003). Finally, on the discrepancies found between reports of PA from mothers and children, only a brief mention and some background literature were provided here, but this matter is of use and interest to the field, and worthy of deeper investigation. Overall, fully addressing this topic is beyond the scope of this study, and it is a compelling arena in and of itself. Future research might examine this more directly (see De Los Reyes, 2013, for an overview).

Conclusion

By mapping the development of PA and tying deficits in early childhood (age 4) to steeper trajectories across 13 years, this study widens the avenue for assessment and preventive initiatives beginning in early childhood. Thus, findings in this study are relevant to clinicians, developmental researchers, policymakers, schools, and families, each of whom has responsibility for working towards optimizing prevention and intervention efforts to deal with PA in children and adolescents. Findings also corroborate with evidence that early childhood interventions may be most effective by focusing on boosting children's emotion regulation skills by working directly with children or assisting parents in supporting their children's

learning of how to regulate negative emotions of anger and sadness (Hay, 2017; Hay et al., 2014; Jennings & Reingle, 2012; Reingle, Jennings, et al., 2012; Thomas et al., 2017). Moreover, findings suggest that, beyond emotion regulation, a second focus should be on assisting children, especially boys, in developing optimum effortful control skills. In practical terms, this might include providing children with opportunities to confront just-manageable challenges while sustaining attention and providing feedback and rewards to build confidence (Piquero, Jennings, Farrington, Diamond, & Gonzalez, 2016). Not to be overlooked, the trait of callous-lack of empathic concern for others could also be addressed early in childhood, which may help mitigate the impact of other risk factors, and some early work is already showing success in this arena (e.g. Kimonis et al., 2019).

Summary of Chapter 5

Study 2 addressed the need for more research on individual-level risk factors for ASB in boys and girls, which was identified in the review (Study 1, Chapter 4). The aim of Study 2 was to prospectively examine early individual-level predictors of boys' and girls' physical aggression (PA) in a 7-wave national data from ages four to 17. Direct effects of early effortful control deficits, negative emotionality (measured separately as anger, fear, and sadness dysregulation), and callous-lack of empathic concern for others on both mother and self-reports of boys' and girls' use and control over their PA were found. In addition, high callous-lack of empathic concern for others was expected to produce even more elevated and steeper increases in PA when combined with effortful control deficits and features of negative emotionality.

In general, PA patterns were similar for boys and girls, but (on average) boys had a higher PA level relative to girls up to mid-adolescence. Considerably fewer sex differences in PA were found in adolescence (up to age 17). All tested individual-level risks predicted higher initial levels of mother-reported PA at age four. Also, anger and sadness dysregulation and callous-lack of empathic concern for others predicted less decline in PA and a more substantial upturn in mother-reported PA into adolescence. There was also a unique risk imparted by early childhood effortful control deficits and anger dysregulation for self-reported PA. Higher PA levels were particularly problematic for boys that had effortful control deficits or higher anger dysregulation and for girls high in anger or sadness dysregulation when these traits were combined with higher lack of empathic concern for others at age four. To complement the findings of Study 2, the next study (Study 3, Chapter 6) presents a prospective examination of the same age four individual-level risk factors (effortful control deficits, negative emotionality, and CU) as correlates of boys' and girls' rule-breaking behaviour into late adolescence.

CHAPTER 6

Study 3

The Roles of Early Childhood Effortful Control Deficits, Negative Emotionality, and Callous-Unemotional Traits in Adolescent Boys' and Girls' Rule-breaking Behaviour

Statement of Contribution to Co-authored Paper

This chapter includes a co-authored manuscript that is ready to be submitted for publication. It consists of a version of the paper that is under peer-review process. The bibliographic details of this co-authors paper, including all authors, are:

Pariz, J., Zimmer-Gembeck, M. J., Modecki, K. (under review). The roles of early childhood effortful control deficits, negative emotionality, and callous-unemotional traits in adolescent boys' and girls' rule-breaking behaviour.

My contribution involved: Review of literature, development of hypotheses, co-development of methodology and design, completion of statistical analyses, lead in writing of the paper, corresponding author of the paper.

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Supervisor: Prof Melanie Zimmer-Gembeck

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Abstract

Early childhood effortful control deficits, features of negative emotionality (measured as anger, fear and sadness dysregulation), and callous-unemotional traits (measured as callous-lack of empathic concern for others) were investigated as independent and interactive predictors of boys' and girls' rule-breaking behaviours (RBB), in a 7-wave Australian national Australian sample (N = 3,089; 51% boys). Caregivers reported children's effortful control, anger, fear, and sadness dysregulation, and callous-lack of empathic concern for others at T1 (Mage = 4.17, SD = 0.37). Adolescents reported their RBB three times from age 12 to 17 years. The results showed that anger dysregulation at age four was a risk factor for boys' and girls' RBB. In addition, RBB was higher in boys that had more effortful control deficits and lower fear dysregulation at age four and in girls that had a higher level of callous-lack of empathic concern for others, also at age four. Overall, no significant interactions between individual-level risk factors were found, and there was no support for sex as a moderator of associations between the early risk factors testes and higher RBB in adolescence. The findings have implications for theory and future interventions targeting adolescent RBB. Future longitudinal studies are needed to investigate the role of early fear and sadness dysregulation as well as other dimensions of callous-unemotional traits on RBB development.

Keywords: Rule-breaking behaviour, longitudinal, effortful control, negative emotionality, callous-unemotional traits.

The Roles of Early Childhood Effortful Control Deficits, Negative Emotionality, and Callous-Unemotional Traits in Adolescent Boys' and Girls' Rule-breaking Behaviour

Adolescents have the highest prevalence rates of rule-breaking behaviour (RBB), relative to any other age group (Frick & Viding, 2009; Jolliffe, Farrington, Piquero, MacLeod, & Van de Weijer, 2017). Adolescent RBB (also referred to as delinquency) is a pattern of disregard for the law and social rules that are characteristic of conduct disorders and general behavioural problems (Lanza, Cooper, & Bray, 2014; Loeber, 1996). RBB often has substantial negative consequences for adolescents, including poor educational outcomes, substance use, psychiatric symptoms and disorders, and general health problems (Bor, McGee, Hayatbakhsh, Dean, & Najman, 2010; Odgers et al., 2008). Also, adolescents who engage in frequent RBB usually become offenders. These offenders account for 50% of the criminality in any society (Hemphill, Heerde, Herrenkohl, & Farrington, 2015; Jolliffe et al., 2017; Moffitt, 2006). Hence, the social impact and associated economic costs related to preventing, intervening, rehabilitating, and detaining youth, place RBB in adolescence as major social, economic, and public health problems (Hair, Park, Ling, & Moore, 2009; Herrenkohl et al., 2000; Rosenberg, O'Carroll, & Power, 1992).

Childhood Individual-Level Risks for RBB

Because of the considerable negative impact of RBB in adolescence, the accurate identification of its early risk factors to inform interventive strategies has attracted a great deal of research attention. Particularly regarding individual-level risks for RBB that can be identified in early childhood, self-control theories suggest that adolescents' engagement in RBB is rooted in deficits in their self-control skills development from early in life (DeLisi, 2011; Gottfredson & Hirschi, 2003). That is, such deficits would have triggered in children a pattern of experiencing exciting and gratifying behaviours as difficult to resist that is stable from childhood onwards. Building on such self-control hypotheses, recent theories describe

specific individual characteristics in early childhood that may contribute to youth's engagement in RBB later in life. In particular, the temperament-based theory of antisocial behaviour (DeLisi & Vaughn, 2014; Eme, 2018) has identified two key characteristics: (a) effortful control deficits (e.g., poor dispositional - as opposed to automatic - use of one's self-regulation skills (persistence, low distractibility, and low impulsivity) to achieve optimum levels of social competence and (b) negative emotionality (e.g., experiencing the environment negatively due to emotion dysregulation of anger, fear, and sadness). In a complementary theory addressing antisocial behaviour from a developmental psychopathology perspective, callous-unemotional traits (CU; e.g., defined as traits of lack of remorse or guilt; callous-lack of empathic concern for others; being unconcerned about performance in important activities, and shallow or deficient affect) has also been identified as an essential individual characteristic that is a risk for more serious RBB (Frick, Ray, Thornton, & Kahn, 2014; Frick & Viding, 2009) and severe levels of conduct problems (Kroneman, Hipwell, Loeber, Koot, & Pardini, 2011) during adolescence and early adulthood (Docherty, Beardslee, Byrd, Yang, & Pardini, 2019). The four traits that are indicative of CU have been recently included, under a specifying criterion of limited prosocial emotions, as criteria for the diagnose of conduct disorder within the DSM-5 (American Psychiatric Association, 2013). To qualify for such specifier in the DSM-5, youth must display at least two of the four traits. Such criteria followed evidence that the traits of CU predict RBB even after accounting for conduct disorder symptoms (Frick et al., 2014).

Evidence from a handful of longitudinal studies supports the notion that childhood effortful control deficits, features of negative emotionality, and CU may independently predict RBB in adolescence and/or early adulthood. In relation to effortful control deficits, impulsivity at age two predicted RBB at age 22 (Hentges, Shaw, & Wang, 2018), and, in separate studies, hyperactivity at age seven and low impulse control at ages seven and 10

predicted delinquency during late adolescence (Herrenkohl et al., 2000; Pardini, Byrd, Hawes, & Docherty, 2018). Regarding features of negative emotionality, anger dysregulation at age one predicted conduct problems from ages four to 13 (Lahey et al., 2008) and higher levels of fear predicted less conduct disorder in childhood (Shaw, Gilliom, Ingoldsby, & Nagin, 2003) and criminal offending in adolescence and adulthood (Pardini et al., 2018). However, longitudinal examinations of the effect of early sadness dysregulation on adolescent RBB are scarce, and only a handful of studies could be located that suggested its positive associations with aggression in adolescence (Campbell, 2006; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011).

Similar associations with RBB in adolescence have been found for childhood CU. For example, higher CU at age seven predicted more conduct problems in girls at age 11 (Kroneman et al., 2011), and in a separate study, CU at ages 8-15 was associated with more serious offending from ages 16-29 in boys and girls (Docherty et al., 2019). However, only a few studies have focused on the unique associations of CU traits with antisocial behaviour simultaneously with other known individual-level risks for RBB (Pardini, 2006; Pardini, Lochman, & Powell, 2007; Pardini et al., 2018) and even fewer have reported associations of specific dimensions/traits of CU with behavioural problems in early childhood (Kimonis et al., 2016). In addition, only one study could be located that had investigated childhood effortful control deficits and features of negative emotionality alongside CU traits as risks for antisocial behaviour in adolescence (Pardini et al., 2018). Thus, despite the relative stability over time of deficits in effortful control, negative emotionality, and CU and previous evidence of their associations with RBB, it is still unclear whether and how, when assessed in early childhood, features of these three individual-level risks may help understand high levels of RBB in adolescent years. This is the first study to conduct such an examination in early childhood (age 4) to investigate outcomes in RBB across adolescence (ages 12 to 17). Such

information is clinically relevant to interventive services aiming at preventing or mitigating such behaviours (Herrenkohl et al., 2000; Pardini et al., 2018), as it is to theory pinpointing and explaining individual characteristics at the basis of the development of RBB in adolescence.

Sex Differences and Sex-Specific Risks for RBB

In the present study, sex was particularly important to consider. Overall, although a range of predictive characteristics associated with RBB in adolescence has been identified in previous research, much is yet to be uncovered about whether or which specific risks could be targeted and the importance of sex-specific interventions (Assink et al., 2015; Jordan, 2011; Ttofi, Farrington, Piquero, & DeLisi, 2016). In this respect, it is widely known that RBB is more characteristic of males than females, which has long justified an empirical focus on boys' problem behaviours or cohorts of boys and girls together (Ellickson & McGuigan, 2000). However, some scholars have also argued that the severity of rule-breaking in adolescent girls may match or exceed that found in boys, especially when assessed in adolescence, and across multiple behavioural domains (Pepler et al., 2010; Tiet, Wasserman, Loeber, McReynolds, & Miller, 2001). These sex-based similarities raise the question about possible sex-specificities on the basis of male and female propensity to RBB. In particular, research is needed to examine whether a threshold-variation in distinct levels of risks early in childhood (e.g., socio-environmental or individual-level risks) explains the sex-specific propensity to RBB later in life (Fairchild, Van Goozen, Calder, & Goodyer, 2013; Jordan, 2011; Lanza et al., 2014; Tiet et al., 2001).

The Present Study

This novel study expands on past research by using a large national longitudinal sample to test three hypotheses. First, consistent with theory (DeLisi & Vaughn, 2014; Frick & Viding, 2009), age-four deficits in effortful control and features of both negative

emotionality (measured here as anger, fear, and sadness dysregulation) and CU traits (measure here as callous-lack of empathic concern for others) are expected to be positive and significant risks for RBB at ages 12 to 17. However, fear dysregulation was expected to show a negative association with RBB, consistent with what was reported in one previous study (Pardini, 2006). Second, research also suggests that childhood risk factors for antisocial behaviour may be not only additive but also be multiplicative (Eme, 2018; Ribeiro Da Silva, Rijo, & Salekin, 2015; Urben et al., 2017). Thus, consistent with the evidence of CU catalysing the effect of other individual-level risks on RBB (Baskin-Sommers, Waller, Fish, & Hyde, 2015; Frick, 2012) the catalysing effect of the CU trait of callous-lack of empathic concern for others on the detrimental effects of effortful control deficits and features of negative emotionality on RBB is also expected here. Importantly, because current developmental literature on RBB has predominantly examined samples of boys-only or boys and girls pooled together (Ellickson & McGuigan, 2000), little is known about whether early effortful control deficits and features of negative emotionality and the CU trait of callous-lack of empathic concern for others would have the same significance and strength in predicting RBB in adolescent boys and girls. Thus, in the present study, the two hypotheses above are tested, reported, and discussed considering boys and girls together and separated.

To the test of the third hypothesis, assumptions from theory (DeLisi & Vaughn, 2014; Eme, 2018) and other research (Fairchild et al., 2013) underpinned examinations of a moderating effect of sex on the associations of early childhood individual-level risks with RBB in adolescence. Consistent with these studies, it was expected that anger and sadness dysregulation, and the CU trait of callous-lack of empathic concern for others would be associated with higher levels of RBB for both sexes. However, considering the evidence in additional studies, sex differences in the associations of effortful control deficits and fear dysregulation favouring females (Cross, Copping, & Campbell, 2011; Else-Quest, Hyde,

Goldsmith, & Van Hulle, 2006) were also expected to show male-specific vulnerabilities to the development of RBB in adolescence.

Method

Participants and Procedure

Data were drawn from the *Longitudinal Study of Australian Children* (LSAC), an ongoing, multi-reporter, longitudinal study following Australian children (and their caregivers) at two-yearly intervals from 2004 (Growing up in Australia: The Longitudinal Study of Australian Children, 2015). Seven waves of LSAC data were available as of 2020, and Time 1 (T1), Time 5 (T5), Time 6 (T6), and Time 7 (T7) data were used in this study (N = 3,089, 51% males).

Participants were aged 4/5 at T1 (M_{age} = 4.17 years old, SD = 0.37, 83% age 4), ages 12/13 at T5 (M_{age} = 12.40 years old, SD=0.49, 60% age 12), ages 14/15 at T6 (M_{age} = 14.40 years old, SD = 0.49, 60% age 14), and ages 16/17 at T7 (M_{age} = 16.46 years old, SD = 0.50, 55% age 16). Most participants (96%) were born in Australia. The next most common birth nations were the UK (0.9%) and New Zealand (0.6%). The sum of participants born in South Africa, USA, India, Philippines, China, Japan, and Kenya was 1% of the LSAC sample in this study. Of the T1 caregiver respondents, 97% were the biological mother and 3% the biological father. Also, 2.3% endorsed the child's ethnic background as Aboriginal Peoples and/or Torres Strait Islanders. Most families (90%) were dual-parent households, and 65% reported 12 years of formal school education or more. Approximately 64% of families lived in urban areas (population $\geq$ 100,000), and 8% of families reported their yearly income as equal to or less than $25,999, 53% reported it from $26,000 to $77,999, and 39% of families reported a yearly household income of $78,000 or more. LSAC participants also reported on whether they had had contact with the justice system at T6 and/or T7. Around 4% of adolescents reported having attended to a youth justice conference, been charged with an

offence by the police, appeared in court as a defendant, been convicted of an offence, and/or been in youth detention at least once in T6 and/or T7.

Measures

Rule-breaking behaviour (RBB). Adolescents completed 15 items on their RBB (e.g. "carried a weapon like a knife, gun, or piece of wood", "purposely damaged or destroyed others' property", "got into physical fights in public") in the past 12 months at T5, T6, and T7 using a six-point scale from 0 (*not at all*) to 5 (*5 or more times*). Self-reported data on RBB was not available in other LSAC waves. In LSAC, RBB items were drawn from the Self-Report Delinquency Scale (Moffitt & Silva, 1988). The five-year stability of the RBB scale was moderate from T5 to T7 and correlations were significant between T5 and T6 ($r = .40$, $p < .001$) and T6-T7 ($r = .60$, $p < .001$). The 15 RBB items also showed good reliability for the total LSAC sample and for boys, and girls, respectively, at T5 ($\alpha = .90, .86, .95$), T6 ($\alpha = .86, .83, .76$), and T7 ($\alpha = .89, .90, .84$). Preliminary analyses indicated that results based only on one time-point (wave 5, 6, or 7 RBB) were similar across T5 to T7 for the total LSAC sample, boys, and girls. Hence, adolescents' RBB scores across T5 to T7 were averaged to test whether baseline independent variables predicted greater frequency of RBB over time in the analyses reported here.

Effortful control deficits. Seven parent-reported items were selected to indicate children's effortful control at T1; four items from the persistence subscale of the Short Temperament Scale for Children (STSC) (Sanson, Smart, Prior, Oberklaid, & Pedlow, 1994) and three items from the hyperactivity subscale of the Strengths and Difficulties Questionnaire (SDQ) (Goodman, Meltzer, & Bailey, 1998). Items from the STSC focused on child's ability to concentrate (e.g., *in tasks this child stays with an activity (e.g. puzzle, construction, kit, reading) for a long time* – reverse item; *when a toy or game is difficult, this child quickly turns to another activity*) and persist (e.g. *when this child starts a project such*

as a puzzle or model, he/she works on it without stopping until it is completed, even if it takes a long time – reverse item; *this child likes to complete one task or activity before going onto the next* – reverse item) in tasks. Items from the SDQ focused on distractibility (e.g., *easily distracted, concentration wanders*) and impulsivity/attention control traits (e.g., *can stop and think things through before acting; sees tasks through the end, good attention span* – reverse items). These seven items were considered to tap on child's deficits in effortful control because they relate in content to items in attention focus/shift and inhibitory control scales within previously validated child temperament and effortful control measures (Putnam, Gartstein, & Rothbart, 2006; Rothbart, Ahadi, Hershey, & Fisher, 2001; Verstraeten, Vasey, Claes, & Bijttebier, 2010) which have been used either alone (Eisenberg et al., 2003) or with their items combined (Eisenberg et al., 2010) in past research. Items had response options that ranged from 0 (*not true*) to 2 (*very true*). In this study, correlations between items were positive and significant, *r*'s ranged from .20 to .50, all $p < .001$. Thus, a composite effortful control deficits score was created by averaging all seven items (full sample $\alpha = .78$, boys $\alpha = .79$, girls $\alpha = .76$).

Negative emotionality. Items from the emotional symptoms scale in the SDQ (Goodman et al., 1998) were identified as indicators of child anger (*this child often has temper tantrums or hot tempers*), fear (*this child has many fears, is easily scared*), and sadness (*this child is often unhappy, down-hearted or tearful*). Caregivers rated their child's negative emotionality from 0 (*not true*) to 2 (*very true*) at T1, and individual items were used in the analyses.

Callous-unemotional trait (CU). A score of CU corresponding to the trait of callous-lack of empathic concern for others was obtained at T1 using three caregiver-reported reversed items (0 = *not true to* 2 = *very true*) from the prosocial behaviours scale of the SDQ (Goodman et al., 1998). The three items were: 1 - this child is considerate of other people's

feelings, 2 - this child is helpful if someone is feeling hurt, upset or ill, and 3 - this child often volunteers to help others (parents, teachers, other children). The three items used here were similar in content to those in the Inventory of callous-unemotional - preschool version (e.g. “this child is concerned about the feelings of others”, “this child does things to make others feel good”) (Frick, 2004; Willoughby, Mills-Koonce, Waschbusch, & Gottfredson, 2015). Furthermore, the three SDQ parent-reported items used here are part of the University of New South Wales callous-unemotional scale for young children (UNSW CU) (Dadds, Frost, Fraser, & Hawes, 2005) and had shown the highest loading rates in the CU dimension in the UNSW CU scale validation study (Dadds et al., 2005) - .61, .74, .65, respectively for items 1, 2, and 3 above. In the present study, the three SDQ items were averaged to obtain a baseline score of callous-lack of empathic concern for others for the total LSAC sample and boys, and girls (α = .60, α = .61, α = .60, respectively). Reliability of SDQ and CU scales have been reported within the marginal range (α between .60 and .70) or lower in past studies (see α of SDQ in Kimonis et al., 2016 and Willoughby et al., 2015; α of APSD in Dadds et al., 2005 and Pardini, 2006; α of UNSW CU in Kimonis et al., 2016). Although marginal internal consistency would typically be a source of concern, researchers have argued that this is the case when the scale is measuring a study’s outcome, but not when a scale is used as an indicator of a broader latent construct (Willoughby et al., 2015), as in the present study.

To validate the composite score for the CU trait of callous-lack of empathic concern, its associations with caregiver-reported conduct problems and prospective associations with both caregiver and self-reported conduct problems and self-reported empathy in adolescence (T5, T6, and T7) were examined. Previous studies have found that CU in childhood is positively associated with higher levels and persistent conduct problems through adolescence (Pardini, 2006; Pardini et al., 2007). Also, when assessed in childhood, specific dimensions of CU scales show moderate-to-low correlations with empathy deficits and conduct problems

(Ezpeleta, de la Osa, Granero, Penelo, & Domènech, 2013; Kimonis et al., 2016). Consistent with this past research, callous-lack of empathic concern for others at T1, as measured in this study, was positively and significantly ($p < .001$) associated with the SDQ conduct problems scale as reported by LSAC caregivers at T1 ($r = .11$), T2 ($r = .25$), and T3 ($r = .22$), and by both caregivers and self, respectively, at T4 ($r = .18$ and $r = .08$), T5 ($r = .17$ and $r = .07$), T6 ($r = .15$ and $r = .08$), and T7 ($r = .13$ and $r = .07$). Also in line with the literature, negative and significant ($p < .001$) cross-informant correlations were found for caregiver-reported callous-lack of empathic concern for others at T1 and youth self-reported empathy[10] at T4 ($r = -.12$) and T5 ($r = -.10$).

Socio-environmental level covariates. Socio-environmental variables at baseline were included in the analyses to account for the effect of well-known external risks to youth's RBB and increase the robustness of the predictive models tested. These included caregiver' educational level (0 = year 11 or less, 1 = year 12 or equivalent), yearly household income (0 = 25,999 or less, 1 = 26,000 to 77,999, 2 = 78,000 or more), child's Indigenous background (0 = no, 1 = yes), and neighbourhood safety at T1. Considering data available in LSAC, two items were averaged to obtain a neighbourhood safety score (*This is a safe neighbourhood,* and there *is good street lighting in this neighbourhood*, 1 = Strongly agree to 4 = Strongly disagree).

Data Analyses

All analyses were completed in SPSS v.25. First, an inspection of missing data was completed. Missingness within T1 was 5% for household income variable and 0.2% or less for all other covariates and independent variables. Missingness from T5 to T7 was 8% or less. Missing items in all variables included in this study were replaced via multiple imputation, and pooled results from 15 complete datasets are reported in all analyses. Next, descriptive

[10] Assessment of empathy was only available in LSAC in T4 and T5.

statistics and bivariate correlations were calculated, and sex differences in all measures were tested using independent samples *t*-tests. Finally, after assuring that multicollinearity among the variables was not present, three sets of regression analyses were conducted to test this study's hypotheses. In the first set of analyses, three multiple regression models were conducted with the total LSAC sample, and boys and girls separated, to examine whether deficits in effortful control, features of negative emotionality (anger, fear, and sadness dysregulation) and the CU trait of callous-lack of empathic concern for others at age four were associated with boys' and girls' RBB in adolescence. The main effect of sex (only for analysis with the total LSAC sample) and all socio-environmental covariates were entered simultaneously with all individual-level risks in these three models.

The second set of regression analyses was conducted to test whether, when assessed in early childhood, the CU trait of callous-lack of empathic concern for others acts in combination with effortful control deficits and features of negative emotionality to increase risk of boys' and girls' RBB in adolescence. With this purpose, four two-step hierarchical multiple regression models were conducted by entering each residual-centred interaction term (effortful control deficit × the CU trait, anger dysregulation × the CU trait, fear dysregulation × the CU trait, sadness dysregulation × the CU trait) individually focusing on the total LSAC sample. These same four models were then repeated, focusing on boys only and then girls only. The residual-centring approach (Little, Bovaird, & Widaman, 2006) was followed before estimating the interaction effects to produce six orthogonalised product terms (i.e., interaction terms that are uncorrelated with their first-order effects) better representing each interaction.

Finally, the third set of analyses aimed at investigating a possible moderating role of sex in the predictive role of early effortful control deficits, features of negative emotionality, and callous-lack of empathic concern for others on the higher frequency of RBB over

adolescent years. With this purpose, five two-step hierarchical multiple regression models were conducted by adding each interaction term (effortful control deficit × sex, anger dysregulation × sex, fear dysregulation × sex, sadness dysregulation × sex, callous-lack of empathic concern for others × sex) individually, building on the first multiple regression model.

Results

Descriptive Data, Correlations, and Comparisons of Boys and Girls

Table 6.1 presents descriptive data (means and standard deviations) for the total LSAC sample and correlations between all variables for all participants. All early childhood measures were positively and significantly correlated with more frequent RBB, with the exceptions of dysregulation of fear and sadness. Significant associations ranged from $r = .07$ to $r = .31$. However, the large sample size may have favoured the significance of small effect sizes, which indicates caution in interpreting significant and small correlations (such as $r = .07$).

Table 6.1

Bivariate Correlations between Individual Child-Level Risks at Age 4 and Rule-breaking Behaviour in Adolescence (N = 3,089)

	1	2	3	4	5	6
1. Rule-breaking behaviour[1]	-					
2. Effortful control deficit	.12***	-				
3. Anger dysregulation	.09***	.24***	-			
4. Fear dysregulation	-.02	.13***	.13***	-		
5. Sadness dysregulation	.03	.11***	.15**	.24***	-	
6. CU trait	.07***	.31***	.21***	.08***	.07***	-
Mean	.08	.78	.67	.40	.15	.47
SD	(.21)	(.42)	(.68)	(.59)	(.41)	(.41)

**$p < .001$. ***$p < .0001$.

Note. CU trait refers to callous-lack of empathic concern for others. [1]Averaged frequency of self-reported rule-breaking behaviour, available in LSAC data at T5 (ages 12/13), T6 (ages 14/15) and T7 (ages 16/17).

Table 6.2 shows descriptive data for boys and girls, *t*-tests comparing boys to girls, and correlations between all measures for boys separated from girls. Correlations were similar in size to those for the total sample. Early deficits in effortful control and anger dysregulation were positively and significantly associated with RBB for both boys and girls.

Table 6.2

Bivariate Correlations and Means of Individual-Level Risks at Age 4 and Rule-breaking Behaviour in Adolescent Boys (n = 1,576) and Girls (n = 1,513)

	1	**2**	**3**	**4**	**5**	**6**
1. Rule-breaking behaviour[1]	-	.09***	.11***	.04	.04	.09***
2. Effortful control deficit	.10***	-	.21***	.12***	.08**	.31***
3. Anger dysregulation	.08**	.26***	-	.11***	.14***	.18***
4. Fear dysregulation	-.06*	.12***	.14***	-	.22***	.09***
5. Sadness dysregulation	.02	.13***	.16***	.25***	-	.07**
6. CU trait	.04	.29***	.22***	.07**	.07**	-
Girls						
Mean	.05	.72	.64	.37	.14	.43
SD	(.14)	(.40)	(.68)	(.57)	(.39)	(.39)
Boys						
Mean	.11	.83	.69	.43	.17	.52
SD	(.25)	(.44)	(.69)	(.61)	(.42)	(.43)

**$p < .001$. ***$p < .0001$.
Note. Girls' correlations are reported above the diagonal; boys' correlations are reported below the diagonal. CU trait refers to callous-lack of empathic concern for others. [1]Averaged frequency of self-reported rule-breaking behaviour, available in LSAC data at T5 (ages 12/13), T6 (ages 14/15) and T7 (ages 16/17).

There was a significant, but negative association of boys' fear dysregulation with RBB, but this association was not significant in girls. Conversely, the association of early callous-lack of empathic concern for others and RBB was positive and significant for girls, but not for boys. As also shown in Table 6.2 and using an adjusted p-value of .008 (.05/6), relative to girls, boys reported more RBB, $t(3087) = 8.11$, $p < .0001$; had more effortful control deficits, $t(3087) = 7.75$, $p < .0001$; were higher in fear dysregulation, $t(3087) = 2.63$, $p < .009$; and were higher in callous-lack of empathic concern for others, $t(3087) = 6.07$, $p < .0001$.

Predicting RBB across Adolescence from Individual-level Risks at Age Four

In the next analyses, RBB was regressed on measures of early (age 4) effortful control deficits; anger, fear, and sadness dysregulation; and the CU trait of callous-lack of empathic concern for others. A first model included the full LSAC sample, with follow-up models for boys and girls. Model diagnostics showed no problem with multicollinearity (tolerance = from .79 to .98 and VIF = from 1.01 to 1.40 for all independent variables). As can be seen in Table 6.3, after controlling for household income, Indigenous status, caregiver education and neighbourhood safety, high anger dysregulation at age four was associated with more RBB in the total sample, among boys only, and among girls only. In addition, both effortful control (positively) and fear dysregulation (negatively) were also significantly associated with RBB, but this was observed only in the full sample and in boys-only. Only in the model of girls' RBB was the CU trait of callous-lack of empathic concern for others significantly associated with more RBB. Overall significant effect sizes for individual-level risks were small, ranging from .05 to .08.

Interactions of Effortful Control Deficits and Negative Emotionality with CU Traits

After testing each of four interactions, one at a time, in four separate models with the full sample (i.e., effort control deficits × CU trait and each form of dysregulation × CU traits), the same interactions were also tested in eight sex-specific models (four models for boys and four for girls). This resulted in testing a total of twelve interaction models. Across these models, one interaction - anger dysregulation × callous-lack of empathic concern for others - was significant at $p < .05$ for the sample of girls ($R^2_{\text{change}} = .003$, $p < .03$). This association was very small and did not meet the adjusted p-value of .004 (.05/12 to adjust for 12 models).

Table 6.3

Results of Regressing Rule-breaking Behaviour on Age-Four Risks in the Total LSAC Sample (N = 3,089) and by Sex (n_{boys} = 1,576; n_{girls} = 1,513)

	Total sample	Boys	Girls
Independent variables	β	β	β
Sex[1]	-.13***	-	-
Socio-environmental covariates			
Household income	-.02	-.02	-.01
Indigenous status	.07***	.07**	.10***
Primary caregiver's education	-.06**	-.06**	-.05
Neighbourhood safety	.03	.06*	-.03
Individual-level risks			
Effortful control deficit	.07***	.08**	.04
Anger dysregulation	.06**	.05*	.08**
Fear dysregulation	-.05**	-.08**	.00
Sadness dysregulation	.01	.01	.01
CU trait	.02	.00	.06*

* $p < .01$. ** $p < .001$. *** $p < .001$.
Note. CU traits refer to callous-lack of empathic concern for others. Direct associations were obtained by entering all independent variables simultaneously in the model. [1]Sex was coded 0 = boys and 1 = girls.
Total sample: $R^2 = .05$, F (10, 3078) = 16.048, $p<.0001$.
Boys: $R^2 = .04$, F (9, 1566) = 6.617, $p<.0001$.
Girls: $R^2 = .04$, F (9, 1503) = 6.028, $p<.0001$.

Testing Interactions of Early Individual-Level Risks with Sex

Building on the model in Table 6.3, interactions with gender were entered, one at a time (five models), to test whether associations of effortful control deficits, the three measures of negative emotionality, and callous-lack of empathic concern for others (e.g., gender × CU trait) with adolescent RBB differed for boys and girls. No interactions were significant.

Discussion

This novel study expanded on previous research by using seven waves of a large national sample to examine whether/how effortful control deficits, features of negative emotionality and the CU trait of callous-lack of empathic concern for others at age four may directly and/or interactively predict higher levels of boys' and girls' RBB across adolescence (ages 12 to 17). The main findings suggest the consistent role of anger dysregulation at age

four on both boys' and girls' higher frequency of RBB across adolescence. In addition, the three models considering the total LSAC sample and boys and girls separated suggest early childhood effortful control deficits and low fear dysregulation significantly predicted higher frequency of RBB in adolescent boys, but not in girls. In contrast, the CU trait of callous-lack of empathic concern for others at age four was a significant predictor of higher frequency of RBB only in adolescent girls. Furthermore, the test of the catalysing effect of CU trait of callous-lack of empathic concern for others on the longitudinal associations of effortful control deficits and features of negative emotionality with RBB in adolescence was only significant for anger dysregulation and in the sample of girls. Notably, all such significant effects were observed above and beyond the direct effect of sex and baseline socio-environmental and individual-level risks in the models.

In line with theory (DeLisi & Vaughn, 2014; Frick et al., 2014; Frick & Viding, 2009) the direct associations of effortful control deficits, features of negative emotionality, and callous-lack of empathic concern for others at age four were expected to be all positive and significantly predict higher levels of RBB in adolescence. One exception was fear dysregulation, which was expected to correlate negatively and significantly with RBB. Overall, the results of testing such associations have only partially confirmed this study's hypotheses. For example, as expected, anger dysregulation emerged as an especially relevant early childhood risk, as it predicted RBB over eight years later and across distinct informants (mother and self) in the total LSAC sample and samples of boys and girls separated. This finding supports theory (DeLisi & Vaughn, 2014; Eme, 2018) and empirical evidence (Garofalo & Velotti, 2017; Kemp et al., 2017; Urben et al., 2017) that early childhood negative emotionality is especially relevant and reliable to predict conduct disorder and later antisocial behaviour. Therefore, the results found for anger dysregulation in the present study expand on current literature (Stringaris, 2011; Urben et al., 2017) by showing it as an

important risk factor identifiable in early childhood for predicting and specifying RBB across adolescence. In contrast, opposite to this study's hypothesis, the effect of sadness dysregulation was not significant in any of the three tested samples. Although evidence of the role of sadness dysregulation is scarce in the literature, findings in this study seem consistent with few studies that had found its associations with aggression, rather than the broader construct of RBB (Campbell, 2006; McLaughlin et al., 2011). Also, contrary to what had been anticipated, the roles of both fear dysregulation and effortful control deficits were not significant in the sample of girls. Similarly, the role of callous-lack of empathic concern for others was not significant in the total LSAC sample and in the sample of boys. Possible explanations for these findings in light of previous research on effortful control and CU traits are discussed next.

Regarding the role of early effortful control deficits on RBB in adolescence, the results obtained for the total LSAC sample and boys are consistent with theory (DeLisi & Vaughn, 2014) and longitudinal evidence (DeLisi, Fox, Fully, & Vaughn, 2018) placing it as a risk in childhood for increased antisocial behaviour over the life course. However, the non-significant role of effortful control deficits in adolescent girls' RBB seems consistent with research considering the primary role of biological biases/mechanisms on rooting emotional and behavioural tendencies, which posit natural sex-specificities in effortful control favouring females (Cross, Copping, & Campbell, 2011; Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006). This evidence would explain the consistent lower levels of not only effortful control deficits but also features of negative emotionality and especially RBB in females relative to males, which were also found in this research. In this respect, previous research did suggest that, compared to young boys, young girls seem to have naturally lower levels of impulsivity and higher levels of fear (Campbell, 2013; Chaplin & Aldao, 2013; Else-Quest et al., 2006; Eme, 2018), which taken together, may act as a protective shield against both female

involvement in RBB and rule-breaker girls reaching male-equivalent high levels of RBB. Further support for such a hypothesis in this study is that fear dysregulation was negatively associated with boys RBB in adolescence, but the same association was not significant in girls. Complimentarily to such biological explanations, biosocial analyses of male and female behaviour also consider the effect of the interaction of physical differences between sexes (i.e., male's higher physical strength and female reproductive capacity) with both economic and social aspects of families/societies in exacerbating or mitigating the risk vs. protective effects of effortful control deficits and fear dysregulation on human behaviour over time (Wood & Eagly, 2002). Altogether, this may confirm, as others have already proposed (Cross et al., 2011; Else-Quest et al., 2006; Eme, 2018), sex-specificities in pathways from early childhood individual characteristics to antisocial behaviour in adolescence. However, such specificities should not be interpreted as sex-differences per se, as they seemed to be shaped by an equation between sex-specific biological characteristics and the social interactions/expectations surrounding boys and girls since early childhood. Newer to the literature, therefore, is cross-informant evidence presented in this study that, when examining samples of boys and girls separated, the predictive role of effortful control deficits can be successfully tested in early childhood in relation to RBB from ages 12 to 17.

The results for the CU trait of callous-lack of empathic concern for others, which were not significant for the total LSAC sample and boys, but showed significant direct and interactive (anger dysregulation × the CU trait) effects in the sample of girls were somewhat surprising because were contrary to this study's hypothesis. Two aspects concerning the assessment of RBB in this study and previous evidence of sex-specificities for the CU trait of callous-lack of empathic concern for others are relevant to interpret such findings. Firstly, the non-significant association of the CU trait with RBB in the total LSAC sample and boys could be explained by evidence that childhood CU traits predict RBB only in a particularly

severe and violent group of antisocial adolescents who have engaged in conduct problems since childhood (Docherty et al., 2019; Frick et al., 2014; Frick & Viding, 2009). In contrast, the adolescent-onset of RBB would generally not have its roots in early childhood CU traits (Frick & Viding, 2009). In the present study, both violent vs. non-violent and childhood-onset vs. adolescent-onset rule-breakers were not discriminated or controlled for because the focus was on the linear effects of early individual-level predictors on higher RBB over adolescent years.

Conversely, the significant direct and interaction effects of callous-lack of empathic concern for others in LSAC girls are in line with evidence suggesting that considerably less heterogeneity exists in the development of antisocial behaviour (Tiet et al., 2001; Waschbusch, 2002) and CU traits (Euler et al., 2015) in the female population, which result in considerably less variability in their scores of both constructs. Implications of such body of evidence have been described as a paradox in that rather than differing from boys, girls are generally less affected (in both numerous and levels) by conduct problems or CU traits, but for those who do, the severity and comorbidity of these two characteristics and other symptoms (e.g., anger dysregulation) are much higher in girls (Tiet et al., 2001; Waschbusch, 2002; Wasserman, McReynolds, Ko, Katz, & Carpenter, 2005). Such paradox would also explain that even in the absence of sex differences, the prognosis is much less positive for young girls than young boys with conduct problems and CU traits (Euler et al., 2015). Further support for such ideas comes from the non-significant moderation effect of sex in the associations of any individual-level risk with RBB across adolescence in this study. Thus, the effect of less heterogeneity in girls' CU traits and RBB posed by such a paradox could account for the significance of both the direct and the interaction effects of the CU trait in the LSAC sample of girls.

Study Strengths, Limitations and Directions for Future Research

There were several key strengths in this study. First, it made use of a large national longitudinal sample to assess the development of RBB across multiple informants (mother and self) and over eight years after baseline measures had been completed. This provided power to test whether sex moderated effects of early childhood individual-level risks on RBB. Second, the outcome measure of RBB was self-reported over three time-points and had high internal consistency for the total LSAC sample and subsamples of boys and girls from T5 to T7. Self-reports of RBB are likely to capture more accurate and detailed information than parent-reports or criminal records only (Moffitt, 2006),

Findings in this study must also be considered in the context of four limitations that relate to the assessment of negative emotionality, the CU trait, and the predictive models tested. First, features of negative emotionality were three single-item measures assessing traits of anger, fear, and sadness dysregulation. Although the three items were part of the 5-item scale of emotional symptoms in the SDQ, the use of single items (instead of the total scale score) meant that measurement reliability could not be calculated. Nonetheless, cross-informant and significant associations of such items with a robust measure of RBB were still found which were also in line with the literature. Second, the examination of the predictive role of CU trait on RBB focused on callous-lack of empathic concern for others assessed by a composite of three items from the prosocial behaviour scale of the SDQ. Although other studies have also used items in a pre-existent measure to create a proxy-measure of CU traits in early childhood (Kimonis et al., 2016; Willoughby, Waschbusch, Moore, & Propper, 2011), and the three items used here are part of the UNSW CU scale (Dadds et al., 2005), one could still argue that it is unclear to what extend the operationalisation of the CU trait in this study maps onto the full construct of CU. In this respect, more confidence to the measurement of callous-lack of empathic concern for others in the present study was drawn

first from its significant correlations both with conduct problems and empathy up to 10 years later and across distinct informants in the LSAC sample. All such correlations were also consistent with the literature (see details in the method section). Furthermore, the non-significant association in boys and the significant associations in girls of early callous-lack of empathic concern for others with RBB were similar to previous evidence of the association of cognitive empathy (i.e., traits of empathic concern for others) in childhood with the development of psychopathy in boys and girls (Dadds et al., 2009). Also, the role of empathic traits in inhibiting antisocial behaviours through motivating/eliciting prosocial emotions is well described in the literature (Frick et al., 2014) and some have argued that empathy is one of the best indicators of the overall construct of CU traits (Frick & Viding, 2009). Thus, in relation to the assessment of callous-lack of empathic concern for others, this study makes an important contribution in that it represents a valuable test of a particular indicator of the broader latent construct of CU trait on RBB measure eight years later and across adolescence.

Third, because only one dimension of CU traits was addressed in this research, the non-significant results obtained for the interactions of effortful control deficits and fear dysregulation with callous-lack of empathic concern for others should be interpreted carefully. That is, findings in this study may have failed to replicate significant associations reported for the broad construct of CU traits with both early effortful control deficits and fear dysregulation on adolescent conduct problems because only the CU dimension of callous-lack of empathic concern for others was considered. Notably, previous examinations of parent-reported measures of CU traits in pre-schoolers have suggested its bi-factorial solution (e.g., uncaring and callous) in early childhood population (Kimonis et al., 2016; Willoughby et al., 2015). Thus, future longitudinal examinations of other dimensions of CU traits (e.g., uncaring/lack of guilt) could still successfully find their catalyst effect on the associations of effortful control deficits and fear dysregulation on conduct problems in adolescence.

Fourth and last, the effects of baseline individual-level risks on RBB across adolescence were assessed independently and in interaction with the CU trait, but not in interaction with each other or with baseline conduct problems. Although such examinations were beyond the scope of this research, evidence suggests that the combination of CU traits and conduct problems usually identifies with more confidence youth who will show adult antisocial and violent behaviour (Frick et al., 2014). Thus, it is possible that accounting for the interaction of callous-lack of empathic concern for others with baseline conduct problems might have improved the significance and/or the strength of its associations with boys' and girls' RBB in adolescence. Although a particular focus on the role CU traits was also beyond the scope of the present study, which focused on one of its features in conjunction with other individual-level risks, such a particular focus is relevant to uncover whether/how specific dimensions of CU traits in early childhood may associate with RBB in adolescence. Such investigations are scarce in the current literature and should be targeted in future studies.

Conclusion

In summary, this multi-reporter, national longitudinal study uniquely characterises key early childhood individual-level risks for the development of RBB in adolescence while also controlling for well-established socio-environmental risks. Findings point to two main implications for researchers, intervention services, and policymakers. First, investigation of sex-specificities in early childhood individual-level risks for the development of RBB across adolescence are still scarce in the literature, and while findings in this studies failed to confirm such hypothesis, they seemed to support a growing body of evidence on the paradox in the development of conduct problems (Wasserman et al., 2005) and CU traits (Euler et al., 2015) in adolescence. That is, although little evidence for broad sex differences in early childhood individual-level risks for RBB was found, selected findings did suggest sex-specific preventive initiatives could be useful, mainly concerning girls' lack of empathic

concern for others and later RBB. Yet, on balance, results indicate that young children, both boys, and girls, will benefit from programs that focus on improving anger regulation. Second, anger regulation was especially relevant to the higher frequency of subsequent adolescent behavioural problems from age 12 to 17, even having been assessed by a single-item measure and across informants. This confirms the theoretical relevance of anger dysregulation, under the umbrella of negative emotionality, as a risk for later RBB and may also indicate that assessment and intervention as early as age four could conceivably cascade to mitigated RBB during the high-risk behavioural period of adolescence. All told, anger dysregulation during early childhood represents a marked risk for problem behaviour more than a decade later and might be a useful specifier for youth's problem and disordered behaviours.

Summary of Chapter 6

Study 3 (Chapter 6) expanded on Studies 1 and 2 by prospectively examining effortful control deficits and features of both negative emotionality (measured as anger, fear and sadness dysregulation) and callous-unemotional traits (measured as callous-lack of empathic concern for others) at age four as risks for boys' and girls' rule-breaking behaviour (RBB) from ages 12 to 17 in a 7-wave national sample. Well-established socio-environmental risks for RBB (income, Indigenous status, mother's educational level, and neighbourhood safety) were also considered as control variables in all analyses. The large sample also provided power to test sex differences in RBB and whether sex moderated effects of all tested risks for higher RBB. Overall, the findings highlight the robust and particular role of boys' and girls' anger dysregulation, boys' effortful control deficits and low fear dysregulation, and girls' callous-lack of empathic concern for others as early childhood predictors of RBB measured up to 12 years later. In opposition to what was expected, callous-lack of empathic concern for others was not a catalyst boosting the negative impacts of effortful control deficits and features of negative emotionality on later RBB, and there was no evidence that sex moderated effects of early risk factors on RBB. In the next chapter (Chapter 7), the main findings from Chapters 4, 5, and 6 will be discussed in the context of this thesis' general aims and their implications for the developmental study of the childhood origins of antisocial behaviour in boys and girls. Furthermore, the implications of such findings to theory and research on this field are also discussed in the context of this thesis' studies' limitations. Directions for future research are also presented in Chapter 7.

CHAPTER 7

General Discussion

In the present doctoral thesis, the childhood origins of boys' and girls' ASB development were reviewed and investigated across three distinct and complementary studies (Chapters 4, 5, and 6). Chapter 1 in this thesis provided an overview of definitions and prevalence rates of ASB from childhood onwards, as well as a broad consideration of the impact of ASB for youth, their victims, society, and the economy. The developmental study of human behaviour, multiple developmental theories of ASB, and current research on boys' and girls' ASB origins and development were introduced and discussed in Chapter 2. A summary of ASB development according to the developmental taxonomic theory (Moffitt, 1993, 2006), followed by a presentation of the individual-level risks for ASB, as proposed by both the temperament-based theory of ASB (DeLisi & Vaughn, 2014) and a developmental psychopathology model of ASB (Frick, Ray, Thornton, & Kahn, 2014a; Frick & Viding, 2009), was also provided in Chapter 2. Chapter 3 gave an overview of the three studies presented in Chapters 4, 5, and 6.

Study 1 (Chapter 4) was a systematic review of prospective longitudinal studies published between 2008 and 2018 that had 1- used group-based trajectory model analyses to identify multiple ASB trajectory profiles and 2- compared such profiles to identify risk and protective factors for ASB. This review focused on (a) describing childhood socio-environmental and individual-level predictors of antisocial behaviour (ASB) in boys and girls together and separated and (b) providing a closer look into quantitative and qualitative differences in such predictors across distinct ASB trajectory profile groups and participant sex. Studies 2 and 3 (Chapters 5 and 6) accessed data from a 7-wave national Australian sample (the Longitudinal Study of Australian Children) to investigate individual-level risks in early childhood for boys' and girls' aggressive (Study 2) and rule-breaking (Study 3)

behaviours into late adolescence. These two studies were designed to further address gaps in theory and research and also to expand on Study 1's findings by attending to a particular need for more prospective longitudinal studies of early childhood individual-level variables as predictors of the development of specific types of ASB into late adolescence in samples of boys and girls separately. In particular, the investigation of individual-level risks in Studies 2 and 3 focused on the direct and interactive effects of effortful control deficits and features of negative emotionality (emotion dysregulation of anger, fear, and sadness) with a specific trait of callous-unemotional (callous-lack of empathic concern for others) at age four on the development of boys' and girls' physical aggression and rule-breaking behaviours into late adolescence.

General Conclusions from Findings in Studies 1, 2, and 3

Overall, all findings from the Review (Study 1) and the two longitudinal studies (Studies 2 and 3) were described and discussed in detail in Chapters 4, 5, and 6 in this thesis. However, when all findings are considered, two general conclusions can be drawn. The first conclusion concerns the descriptions of ASB development in the literature and its implications for the study of the origins of ASB. The second conclusion concerns whether there are specific early childhood socio-environmental and individual-level risks that might explain sex differences in ASB level or development.

Implications of distinct descriptions of ASB development for the developmental study of ASB origins. As first proposed by Moffitt in the developmental taxonomic theory (Moffitt, 1993, 2006), three developmental patterns of ASB (i.e., ASB trajectories) were described, including child-limited (CL), adolescent-limited (AL), and life-course-persistent (LCP). Also, in this theory, the existence of different ASB trajectories was not simply identified, but it was also argued that each ASB trajectory profile would be predicted by a specific set of risks (i.e., socio-environmental vs. individual-level) or circumstances

(normative vs. non-normative development). To date, and beyond the original three ASB trajectory patterns that had been firstly proposed by Moffitt in the developmental taxonomy, up to eight ASB trajectory profiles have been reported in longitudinal studies considering distinct samples and over different timeframes (Jolliffe & Reingle, 2012). Reviewing the studies published between 2008-2018 that had reported on predictors of distinct ASB trajectory profiles (Study 1) confirmed that the number of ASB trajectories in the literature often diverge from the original three (i.e., CL, AL, LCP). In total, nine ASB categories were found in this review. Thus, there is little consensus in replicating the CL, AL, and LCP in the literature, and there does appear to be profiles that do not conform to these three originally proposed trajectories. This continues to pose a challenge to the original assumptions in the developmental taxonomy which proposed specific risks explaining each ASB trajectory profile, especially because these assumptions focused only on the origins of CL, AL, and LCP. Hence, it seems reasonable to think that each study reporting trajectories of ASB beyond those within the taxonomic theory would be proposing a potential additional description of ASB development that would possibly need a new explanation of its origins. In this respect, it may be of benefit to the field to consider whether other identified trajectory profiles, which are in addition to LCP, AL, and CL, would be simply minor extensions on one of these three ASB profiles because of different participants and/or methods, or whether some emerging ASB trajectory profiles do have theoretical or practical meaning because of their unique origins, correlates or outcomes. The problem here seems to be that these considerations are not always provided in published studies. Thus, although there has been an enormous amount of attention on the study of ASB development, there is still much to do to build a comprehensive theory on its origins and to test ideas to the extent needed to clearly direct intervention, prevention or policies towards the best strategies to prevent and/or mitigate ASB and its negative consequences.

The evidence summarised in Study 1 also showed that, overall, there were few differences in predictors of the distinct ASB trajectory profiles when considered relative to those classified into a trajectory profile group with continuous no/low ASB. Still, the presence of a greater number of risks was the most noticeable difference in childhood risks for higher and more chronic ASB (LCP and high-stable ASB) in relation to lower and/or non-chronic ASB (CL, AL, medium-stable trajectories). Such findings are consistent with the emerging consensus that ASB developmental patterns are better described as on a continuum of the same trait instead of mutually exclusive categories (Fairchild et al., 2013; Raine et al., 2005; Walters, 2011; Walters & Ruscio, 2013). Also, this finding prompted the focus of Study 2 on considering growth in intraindividual changes in ASB, rather than on trajectory profile groups. Describing ASB trajectories on a continuum with interindividual variation in level at one time or over time (instead of categories) has great advantages to the prospective longitudinal examination of its origins. This approach allows for the identification of factors that are more significantly associated with higher and chronic ASB levels in relation to low or less chronic ASB. In addition, such description of ASB as a continuum could also be much more reliably applied to and relevant across a variety of sample characteristics (e.g., community or clinical samples, samples of offenders, and distinct age or sex groups), the number of follow-ups available in the dataset, and developmental stage investigated, regardless of the age range of participants (e.g., early or late childhood, adolescence, or adulthood).

Although considering ASB as falling along a continuum has its advantages, the results across Studies 1, 2, and 3 in this thesis also suggest that Moffitt's (1993, 2006) descriptions of CL, AL, and, LCP ASB trajectory profiles as distinct categories should not be rejected completely for two reasons. First, the results in Study 1 showed that, although many studies examining ASB trajectories exist, only about a third of them (31 studies included in the

review out of 96 that made it to the last inclusion/exclusion criteria) have examined predictors of the ASB trajectory profiles that had been found. In addition, of the 31 studies included in the review, only seven had followed participants until adulthood. The challenges of conducting longitudinal studies from childhood into adulthood are many (e.g., high financial costs, dropping out of participants at each data collection point). Unfortunately, however, it seems that follow-up into adulthood is needed to provide full and accurate documentation of ASB development. Although more attention to this issue of profile groups for modelling of ASB on a continuum is needed, it appears to be the case that, without follow-up from childhood to adulthood, a methodological design that considers ASB development as a continuum would be more appropriate and even more meaningful because would be a report of the predictors of the highest and most chronic pattern of ASB in the tested sample.

The second reason why a focus on trajectory profiles should not be rejected completely is that they are relevant to the interpretation of findings in studies of ASB development, regardless of whether researchers have considered ASB as categories or on a continuum. For example, in Study 2, there was significant variance in the growth pattern intercepts and slopes (and quadratic pattern), indicating that children do significantly differ in their initial levels and patterns of change in aggression over time. This was consistent and held for physical aggression as reported by both mothers from ages four to 17 and self from ages 10 to 17. In this case, knowledge of the distinct ASB trajectories expected for the participants' age range within mother and self-reported data may help explain the significant variance among individual ASB trajectories. However, because mother and self-reported data into adulthood were not available, it means that the LCP ASB trajectory could not have been reliably identified and would be mixed with AL ASB. Thus, it seems like the descriptions of profiles of ASB development focusing on both or either intraindividual change and/or

interindividual differences in change in ASB are complimentary, and providing a richer database of findings and, perhaps, a more accurate interpretation of their practical implications for the developmental study of ASB. Hence, where the right conditions for the examination of ASB developmental categories are not met, the use of methodological designs focusing on the origins of the highest and most chronic levels of ASB in a sample seems the best choice. That said, even then, knowledge of the expected ASB trajectories for the age range of the studied sample will be useful for interpreting findings (e.g., some of the variances in the analyses).

Early childhood socio-environmental and individual-level risks for ASB by sex. Evidence from the review and the two empirical studies included in this thesis seems to confirm that most of what is known about ASB development and its origins currently is based on longitudinal investigations considering samples of boys-only that have focused predominantly (but not always exclusively) on a variety of socio-environmental risks. Although this may have been driven by the higher prevalence of higher levels of ASB in boys (Baillargeon et al., 2007; Karriker-Jaffe, Foshee, Ennett, & Suchindran, 2008; Jolliffe, Farrington, Piquero, MacLeod, et al., 2017; Moffitt et al., 2001; Teymoori et al., 2018) and because socio-environmental risks may be most amenable to intervention and change, this is also a limitation that deserves attention in future research for two reasons. First, most of what is known about ASB trajectories (and overall ASB development) over the lifespan may not be entirely generalisable to girls and young women. Second, although both socio-environmental and individual-level risks are described at the origins of ASB in the developmental taxonomic theory, individual-level predictors seem underinvestigated when compared to a great deal of research focusing on socio-environmental risks, at least in the studies reviewed that have focused on trajectory profiles (Study 1). Assuming this is reflective of the larger body of longitudinal research on ASB, what is currently known about

the origins of boys' compared to girls' ASB is likely to still be limited. Although this thesis made an attempt to consider these questions, much more research is needed focused on sex differences and sex moderation effects in early childhood predictors of ASB development.

Findings from Study 1 suggest that girls are particularly underrepresented in studies of individual-level risk for ASB development. Yet, some findings here were revealing of possible sex differences. For example, findings in the systematic review (Study 1) suggest that there were more differences in the identification of significant individual-level factors when models were estimated separately for boys and girls, then were observed for socio-environmental factors. This is particularly relevant because this finding was observed even though only five of the 31 studies included in the systematic review had examined samples of girls-only. Hence, much is yet to be known about sex differences in the individual-level risks for ASB development.

Another important consideration from the findings in the current thesis is that the term sex-specificities used in this thesis to refer to predictors of boys' but not girls' ASB (or vice versa) was used to not infer significant sex differences in ASB origins. Overall, the results across the three studies did not confirm sex as a significant moderator of the origins of ASB, as studies reviewed either did not examine or did not find sex moderation, and sex moderation was not supported in either Study 2 or 3 in this thesis. Therefore, sex-specificities here referred to overall tendencies for one or another predictor to be associated with ASB development in boys or girls. In this thesis, sex-specificities were found when summarising similarities and differences in the predictive models of ASB trajectories (Study 1), at physical aggression growth models allowing estimates to differ between boys and girls (Study 2), and at rule-breaking behaviour models with samples of boys and girls separated (Study 3). Generally, the sex-specificities found in all such models seemed to be best explained by the biological biases/mechanisms, which were discussed within each of these Studies. An

interesting conclusion from such explanation is that girls (but not boys) seem to benefit from the protection of their temperamental characteristics (i.e., better effortful control skills and higher levels of fear dysregulation early in life, in relation to boys (Campbell, 2013; Chaplin & Aldao, 2013; Else-Quest et al., 2006; Eme, 2018). Nevertheless, it is also important to consider that, beyond the biological bases of ASB, which is argued here to include early temperamental and emotion regulation tendencies, the role of society and culture in teaching, monitoring, and reinforcing girl-like appropriate behaviours should continue to be considered as a possible explanation for sex differences and development of ASB (Archer, 2009; Wood & Eagly, 2002). Overall, the teaching of girls-like appropriate behaviours across western cultures includes more intense impulse-control training in everyday tasks for girls in relation to boys, such as sitting, talking, laughing, smiling, and more access to toys and games that role-play caring for and listening to other's needs, such as baby dolls, cooking, and cleaning (Wood & Eagly, 2002). Further examination and discussion of the interplay between biological bases and the role of society and culture in shaping boys' and girls' behaviour and the impact of it in children's temperament and effortful control and emotion regulation skills were beyond the scope of this thesis. This has been discussed and researched in the past (Archer, 2009; Wood & Eagly, 2002) and should be further addressed in future longitudinal studies of ASB development.

Strengths, Limitations, and Future Research Needs

This thesis has a few strengths that warrant mention. First, the focus of this thesis was on sex-specificities in ASB development by examining boys and girls separately across the three studies. This was undertaken to produce a fuller understanding not only of girls' ASB but also of girls' in relation to boys' ASB development. Such understanding is relevant to optimise youth development by informing interventive services that have the best chance of being effective with boys and girls. Second, the systematic review (Study 1) was designed to

provide a more in-depth consideration of the number, magnitude, kind, and sex-specificities in childhood predictors across the distinct ASB trajectory profiles that have been reported in recently published (2008-2018), prospective longitudinal studies. Third, the two empirical studies (Studies 2 and 3) used a large 7-wave national sample. The national, large sample enhances the generalisability of findings. Fourth, individual child-level risks for mother (in Study 2) and self-reported (in Studies 2 & 3) adolescent ASB were investigated, while also controlling for some well-known socio-environmental risks, which and provided insight into the developmental origins of two different forms of ASB (i.e., aggression in Study 2 and rule-breaking in Study 3). Fifth, Studies 2 and 3 had ample power to test effects. Sixth and finally, early childhood risks measured at age four were examined as correlates of ASB development of SB outcomes in adolescence, spanning up to a 13-year lag of time. Despite these six strengths of the studies reported here, the studies in this thesis have four limitations that are worth discussing here. All four limitations relate to the specifics of the methodology. The first two such limitations are relevant to the systematic review (Study 1):

(1) The search period was 2008 to 2018 because of the PhD timeline. This means that the search of studies needs to be updated to include the years 2019 and 2020 before the review is ready for submission for publication. The main outcome of such an update could be returning a few more prospective longitudinal studies on the predictive role of individual-level characteristics in childhood for girls' ASB trajectories. Although this is possible, there is a small probability that some of such studies could make it through the inclusion/exclusion criteria, and consideration of their results could change some of the results reported here.

(2) The report of childhood predictors of boys and girls ASB trajectories was a main purpose of the review. Essentially, this justified that controlling for both the specific sub-dimensions of ASB (aggression and rule-breaking behaviours) and the type of sample investigated (community sample, offenders, at-risk youth) in the study was secondary and

beyond the scope of the review, hence was not done. However, controlling for distinct ASB sub-dimensions and sample type could also have made a great contribution to characterising the heterogeneity of ASB development in terms of the origins of its distinct manifestations (e.g. distinct aggression types, and rule-breaking and offending behaviours) in distinct samples. Future studies should invest in such an examination, as it can provide useful information to those involved with optimising youth development. This could also be done with this very review in the future by rearranging data used in Study 1 to look at childhood predictors of trajectories of aggression and rule-breaking it the distinct sample types in the studies included in the review.

The last two limitations in this thesis relate to the predictors investigated, and measurement reliability reported in Studies 2 and 3.

(3) Although effortful control deficits and features of both negative emotionality and callous-lack of empathic concern for others are relevant risks for ASB development, many other individual child-level characteristics still need to be tested for their role in ASB into late adolescence. For example, the full range of callous-unemotional traits (i.e., shallow emotions, uncaringness, and lack of guilt) seems particularly important to continue to examine in future research. Longitudinal examinations of the distinct CU traits from early childhood could help with the design of early assessment and preventive interventions focused on the most severe forms of ASB, therefore being relevant to inform optimisation of youth development. Unfortunately, data on the full range of CU traits were not available in LSAC, but the findings of the studies reported here suggest that is important to include a full measure of CU traits when designing future studies that are designed with the aim of understanding the development of externalising symptoms, ASB, or criminal behaviour. This will allow for a broader understanding of youth's behavioural development in a much larger and more realistic scale.

(4) The items that were available and selected to tap the constructs of interest in Studies 2 and 3 may have impacted the findings. Overall, implications of the use of single items to assess both physical aggression in Study 2 and features of negative emotionality in Studies 2 and 3, as well as the marginal significance of the measure of callous-lack of empathic concern for others in Studies 2 and 3 were previously discussed within each study. However, regarding the measures of negative emotionality and callous-lack of empathic concern for others, they were significantly associated with more ASB in Studies 2 and 3, indicating that assessing them via mother-reports in early childhood could be useful to flag young children most at-risk for problem ASB development into adolescence. Ideally, these findings will need replication, however. Hopefully, also, the measures used here can also be improved upon in replication studies (e.g., selection of measures with higher reliability). However, conducting large national studies with follow-ups from early childhood into late adolescence is an extremely difficult, expensive, and time-consuming task. In this respect, the opportunity of using LSAC data in this thesis was unique, and the results obtained here for the associations of early childhood indicators of negative emotionality and callous-unemotional traits with ASB development into late adolescence open promising avenues for developmental theory and research aiming at describing, explaining, and optimising youth development.

Conclusion

The present doctoral thesis aimed at examining the childhood origins of boys' and girls' ASB development by three distinct and complimentarily studies. Study 1 was a systematic review of prospective longitudinal studies published between 2008 and 2018 that summarised childhood socio-environmental and individual characteristics that have been reported as predictors of distinct ASB trajectory patterns. Building on findings in this review, Studies 2 and 3 were two longitudinal studies that focused on the developmental origins of

adolescent ASB, focusing on associations of multiple individual-level characteristics with two distinct types of ASB in boys and girls: physical aggression (Study 2) and rule-breaking-behaviour (Study 3).

Overall, two general conclusions could be drawn from the findings. First, although many risk factors were identified across all three studies, the lack of consensus in the literature about the distinct descriptions of ASB development posits challenges to the examination and interpretation of such findings across distinct studies. Second, risks for ASB development can be identified in childhood, and risks for physical aggression and rule-breaking into adolescence can be identified as early as age four. However, the degree to which the considerable sex differences in ASB levels could be explained/predicted by socio-environmental and individual-level risks measured in childhood is still not clear.

In general, future longitudinal studies on ASB development would benefit from a more consistent consideration of how study design (i.e., participants sex and age range and/or methods) aligns with the theoretical/expected description of ASB development; hence making their findings of ASB's unique origins, correlates or outcomes much more informative and practically meaningful. In addition, building on findings across Studies 1, 2, and 3, future research addressing a broader range of individual characteristics that can be assessed in childhood to predict boys', but especially girls', ASB into adolescence is urgently needed; new studies examining both boys and girls appear particularly promising for identifying differences and similarities in patterns and developmental origins. In particular, much more research is still needed to consider how similar individual child-level characteristics explain ASB in both boys and girls, and to identify whether/which of such factors might explain sex differences in ASB trajectories. The main relevance for future research to invest in all such examinations is that it can provide precious information to those involved with optimising youth development. More specifically, such information is

important to continue building comprehensive theories on the origins of high levels of and chronic ASB and to test their ideas to the extent needed to clearly direct intervention, prevention, or public policies towards the best strategies to prevent and/or mitigate ASB and its negative consequences to children and youth. In this respect, all findings in this thesis also called attention to the importance of further understanding and differentiating girls' in relation to boys' ASB development in future studies. Such understanding is relevant to optimise youth development by informing assessment and interventive services that have the best chance of being effective with boys and girls.

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APPENDIX A

Systematic Review Summary Table A - Study 1

Results from Multinomial Regression Testing Static Background Factors as Predictors of distinct ASB Profile Groups Relative to No/Low-Stable ASB

Profile group / Sample / Predictive variable		Odds ratio: Non-significant	Odds ratio: Significant (p <.05)
ASB Profile Group 1: No /low stable			
Mixed sample	Being female	-	8.94[a]
	Being male	-	0.08[b] [LR]/ 0.11[b] [LR]
	High SES	-	1.03[b] / 1.04[b]
	Low income	1.00[a]	-
ASB Profile Group 2: Child-limited			
Mixed sample	High SES	-	0.96[b] [LR] / 0.96[b] [LR]
	Being male	0.82[k]	3.86[b] / 2.56[b] / 0.34[k] / 0.24[j]
	Low income	1.00[j]	-
ASB Profile Group 3: Adolescent-limited			
Mixed sample	Being female	-	0.47[n] [LR]
	Being male	1.59[e] / 0.54[j]	3.26[s] / 2.17[s]
	Black ethnicity	1.32[s]	3.33[s] / 1.39[n]
	Hispanic/Latin ethnicity	1.24[n]	-
	Low income	0.95[j]	-
Boys	Black ethnicity	-	2.66[p]
	Low SES	1.79[p]	-
ASB Profile Group 4: Life-course persistent			
Mixed sample	Being male	-	27.56[s] / 7.75[s]
	Hispanic/Latin ethnicity	-	0.54[n] [LR]
	Being female	-	0.39[n] [LR]
	Black ethnicity	1.37[s] / 0.96[n]	5.26[s]
Boys	Aboriginal peoples	-	11.60[u]
	Low SES	-	4.40[q] / 2.00[q]
Girls	Aboriginal peoples	-	6.68[u]
ASB Profile Group 5: Adolescent- onset			
Mixed sample	Being male	1.91[h]	1.52[g] / 0.50[k] [LR]
	Low SES/income	0.78[g]	0.52[m]
	Black ethnicity	1.81[g] / 1.56[h]	-
	Hispanic/Latin ethnicity	1.15[g]	-
	Being female	1.06[t]	-
	Latin origin born in the USA	0.98[g]	-
Boys	Black ethnicity	1.59[r] / 1.19[r]	-
	Low SES	1.25[o]	-
	Hispanic ethnicity	1.06[r] / 1.02[r]	-
	Latin origin born in the USA	0.92[r] / 1.46[r]	-
Girls	Black ethnicity	2.27[r]	-
	Low SES	1.70[q] / 0.43[r]	-
	Latin origin born in the USA	1.69[r] / 1.10[r]	-
	Hispanic ethnicity	0.90[r] / 0.50[r]	-

Profile group / Sample / Predictive variable		Odds ratio	
		Non-significant	Significant (p <.05)
ASB Profile Group 6: High - stable			
Mixed sample	High SES	-	0.98[b] [LR] / 0.98[b] [LR]
	Being Male	21.97[c] / 2.29[d] / 1.47[d] / 1.20[d] / 1.14[h] / 0.77[d] / 0.77[d] / 0.55[i] / 0.49[d] 0.38[k]	5.05[b] / 3.78[b] / 2.06[e] / 1.80[f] / 1.53[g] 0.34[i] [LR] / 0.16[j] [LR]
	Low income	3.35[c] / 2.06[l] / 1.15[f] / 1.11[i]/ 0.83[g]	0.25[m] / 0.90[j] [LR]
	Black ethnicity	0.87[i] / 0.57[i]	2.52[g]
	Latin origin born in the USA	1.26[g]	-
	Ethnicity other than Caucasian or African American	0.98[i] / 0.62[i]	-
	Hispanic/Latino ethnicity	0.95[g]	-
	Caucasian	0.44[l]	-
	Being Female	0.43[l]	-
Boys	Low SES	1.25[o] / 0.50[p]	-
	Black ethnicity	2.23[r] / 1.43[r] / 0.44[p]	-
	Latin origin born in the USA	1.37[r] / 1.26[r]	-
	Hispanic ethnicity	0.80[r] / 0.57[r]	-
Girls	Low SES	-	7.10[q]
	Black ethnicity	2.96[r] / 2.58[r]	-
	Latin origin born in the USA	2.06[r] / 1.72[r]	-
	Hispanic ethnicity	0.71[r] / 0.53[r]	-
ASB Profile Group 8: Antisocial - desistors			
Mixed sample	Latin origin born in the USA	-	1.68[g]
	Being male	2.42[d] / 2.09[d] / 1.22[g]	3.64 / 0.41[j] [LR]
	Low SES/income	2.80[l] / 2.41[l] / 0.96[j] / 0.77[g]	1.45[m]
	Being female	0.72[t] / 0.43[t]	0.47[l] [LR] / 0.26[l] [LR]
	Black ethnicity	1.02[g]	-
	Hispanic/Latino ethnicity	0.93[g]	-
	Caucasian	0.55[l] / 0.55[l]	-
Boys	Low SES	1.19[o] / 1.39[p]	1.80[q]
	Black ethnicity	0.77[p]	-
Girls	Black ethnicity	1.58[r] / 0.88[r]	-
	Low SES	1.50[q]	-
	Latin origin born in the USA	1.42[r] / 1.31[r]	-
	Hispanic ethnicity	0.97[r] / 0.49[r]	-
ASB Profile Group 9: Medium - level			
Mixed sample	High SES	-	0.98[b] [LR] / 0.98[b] [LR]
	Being male	0.87[k] / 1.70[c]	3.92[h] / 1.87[e] / 1.80[b] / 2.00[b]
	Black ethnicity	1.34[h]	1.97[p]
	Low SES	0.64[c]	2.41[p]
Boys	Aboriginal peoples	-	3.97[u]
	Black ethnicity	1.85[r] / 0.90[r]	-
	Latin origin born in the USA	1.36[r] / 1.07[r]	-
	Hispanic ethnicity	0.97[r]/ 0.73[r]	-

Note. Mixed sample = boys and girls combined. LR = a variable associated with a reduced risk of membership in the focal group. [a](Bowers et al., 2011). [b](Malti et al., 2013). [c](Silver et al., 2010). [d](Becht et al., 2016). [e](Maldonado-Molina, Reingle, et al., 2010). [f](Petitclerc et al., 2009). [g](Maldonado-Molina, Jennings, et al., 2010); [h](Higgins et al., 2010). [i](Tabone et al., 2011). [j](Sittner & Hautala, 2016). [k](Maldonado-Molina et al., 2009). [l](Ehrenreich et al., 2014). [m](Roisman et al., 2010). [n](Petts, 2009). [o](Evans et al., 2016). [p](Hoeve et al., 2008). [q](Odgers et al., 2008). [r](Jennings, Maldonado-Molina, & Komro, 2010). [s](Kolivoski et al., 2014). [t](Malti et al., 2015). [u](Ferrante, 2013).

APPENDIX B

Systematic Review Summary Table B - Study 1

Summary of Significant Results ($p < .05$) from 31 Studies using Multinomial Regression to Test Socio-Environmental and Individual-level Predictors of Specific ASB Profiles Groups in Relation to No/Low-Stable ASB

	Total No of predictors tested in the corresponding level or domain / Odds ratio						
Predictor level / Domain / Variables	Child-limited	Adolescent-Limited	Life-course-Persistent	Adolescent-Onset	High-Stable	Antisocial-Decreasing	Medium-stable
All variables tested	**9**	**10**	**28**	**16**	**58**	**38**	**18**
Socio-environmental level:	6	9	22	13	35	27	13
Family	1	4	8	6	21	18	3
Child lives with single caregiver	-	1.84[a]	3.14[a]	-	-	0.38[b]	-
More hours without adult supervision	-	-	-	-	1.21[c] / 1.29[d]	1.69[e]	-
Parent-child arguments over rules	-	1.40[a]	-	-	-	-	-
Caregiver's alcoholism	-	-	-	-	1.42[f]	-	-
Volatile family	-	-	2.53[g]	-	-	-	-
Overreactive parenting	-	-	-	-	18.45[h] / 3.79[h]	7.90[h] / 6.76[h]	-
Paternal depressive symptoms	-	-	-	-	1.90[i] / 1.10[f]	-	-
Low maternal sensitivity to child's needs	-	-	-	0.59[b]	0.38[b]	0.45[b]	-
Child's perceived parental rejection	-	-	-	-	1.36[j]	-	-
Maternal permissive parenting style	-	-	-	-	4.54[k]	-	-
Poor parental monitoring of child's behaviour	-	-	-	-	-	1.30[l]	-
Parent Coercive discipline	2.01[m] / 1.91[m] / 4.41[n]	-	-	-	1.86[m]	-	2.18[n]
Neglectful parenting style	-	7.10°	-	-	2.03°	11.47°	-
Authoritarian parenting style	-	-	-	-	1.62°	5.47°	-
More quantity of maltreatment indicators	-	-	14.50[p] / 4.00[p]	3.90[p]	7.80[p]	6.70[p] / 2.80[p]	-
Family's physical and verbal conflict	-	-	NR[p]	NR[p]	NR[p]	NR[p]	-
Mother's depressive symptoms	-	-	-	-	1.07[q]	-	-
Inconsistent discipline (child vs. mother)	-	-	NR[p]	-	NR[p]	NR[p] / NR[p]	-

Predictor level / Domain / Variables	Total No of predictors tested in the corresponding level or domain / Odds ratio						
	Child-limited	Adolescent-Limited	Life-course-Persistent	Adolescent-Onset	High-Stable	Antisocial-Decreasing	Medium-stable
Mother's affective distress and somatic response	-	-	NR[p]	NR[p]	NR[p]	NR[p], NR[p]	-
Broken home or family transitions	-	-	-	-	-	-	1.82[r]
Mother's low IQ	-	-	2.50[p]	2.40[p]	3.10[p]	1.80[p]	-
Parents' self-report of criminal conviction	-	-	3.40[p]	-	3.90[p]	-	-
Parental hostility	-	-	-	-	-	4.05[s]	-
Higher educational level of parents [LR]	-	0.95[a]	-	-	-	-	-
Parental involvement with child's life [LR]	-	-	-	-	0.95[e] / 0.94[c]	-	-
Living with both mother and father in the house [LR]	-	-	-	0.65[t] / 0.44[d]	0.67[t] / 0.51[d]	0.61[t]	-
Having access to alcohol at home[†]	-	-	-	-	-	0.65[e]	-
Latino background and speak Spanish at home [LR]	-	-	-	-	0.56[e] / 0.35[c]	0.54[e]	0.39[c]
Married parents [LR]	-	-	-	-	0.14[k]	0.32[n] / 0.14[n]	-
Supportive parenting and quality home environment [LR]	-	-	-	-	-	0.87[q]	-
School	1	0	0	3	2	2	2
School suspension	-	-	-	2.37[l]	-	3.56[l]	-
Negative school environment	1.11[n]	-	-	1.12[u]	-	-	1.11[n]
Teacher-child interpersonal conflict	-	-	-	-	12.95[v]	-	2.17[v]
Positive school adjustment[†]	-	-	-	-	0.65[j]	0.67[j]	-
Child receives free or low-price lunches at school [LR]	-	-	-	2.00[d], 0.47[d]	-	-	-
Peer	1	2	1	3	6	6	2
More number of friends who drink alcohol	-	2.47[c]	-	-	3.43[c] / 1.73[t]	1.90[t]	2.23[c]
Having friends who drink alcohol	-	-	-	-	2.28[e]	2.57[e]	-
Delinquent peer association	2.27[n]	1.83[c]	NR[p] / 1.78[a] / 4.63[g]	NR[p] / 1.34[u]	NR[p] / 2.94[j] / 1.51[u]	NR[p] / 2.05[j]	1.44[u]
Gang membership	-	-	-	-	2.94[e] / 2.00[e]	-	-
More sophisticated understanding of	-	-	-	-	-	4.39[x]	-

Predictor level / Domain / Variables	Total No of predictors tested in the corresponding level or domain / Odds ratio						
	Child-limited	Adolescent-Limited	Life-course-Persistent	Adolescent-Onset	High-Stable	Antisocial-Decreasing	Medium-stable
reasons for having friends							
Child's understanding of friendship closeness	-	-	-	-	-	2.84[x]	-
Greater number of friends who engage in delinquent behaviour	-	-	-	1.16[z]	1.20[z]	1.10[z]	-
More mature understanding of friendship trust [LR]	-	-	-	0.16[x]	-	-	-
Peer acceptance [LR]	-	-	-	-	0.09[v]	-	-
Neighbourhood/Community	3	3	13	1	5	0	6
Youth exposure to violence	1.46[n] / 1.21[n] / 1.74[m] / 1.19[m] / 1.34[m] / 1.11[m]	-	-	1.16[n] / 1.07[m]	1.20[m] / 1.16[m] / 1.21[n]	-	1.18[n]
Acculturation into English language and culture	2.26[n] / 5.08[m]	-	-	-	-	-	-
More number of child welfare placement	-	1.07[w]	1.12[w]	-	-	-	-
Adult's alcohol use in the neighbourhood	-	1.49[c]	-	-	1.87[c] / 1.75[e] / 1.55[e]	-	1.41[c]
Child was ever involved with the drug and alcohol treatment system	-	3.06[w]	2.33[w] / 2.17[w]	-	-	-	-
Child has utilized mental health care services	-	-	0.44[w]	-	4.78[f] / 2.72[f]	-	-
Onset resource	-	-	1.16[y] / 1.33[y]	-	-	-	-
Onset edocc	-	-	-	-	-	-	1.32[y] / 1.83[y]
Onset diversion	-	-	1.50[y]	-	-	-	-
High risk in PRA-V1	-	-	7.96[g], 4.07[g]	-	-	-	-
Involvement with alternative care (institutional, foster, child welfare care)	-	-	3.82[r], 3.14[r]	-	-	-	-
Ever in group home and/or regular residential facilities	-	-	2.78[w]	-	-	-	-
Unstable living arrangements	-	-	3.48[g]	-	-	-	-
Frequency of experiencing racial	-	-	-	-	1.07[z]	-	-

Predictor level / Domain / Variables	Total No of predictors tested in the corresponding level or domain / Odds ratio						
	Child-limited	Adolescent-Limited	Life-course-Persistent	Adolescent-Onset	High-Stable	Antisocial-Decreasing	Medium-stable
discrimination							
Higher frequency of religious participation [LR]	-	-	1.20[a]	-	-	-	-
Neighbourhood safety and social support [LR]	-	-	-	-	0.95[f]	-	-
More years in the same out-of-home placement [LR]	-	-	0.85[w]	-	-	-	-
Social support [LR]	0.46[n]	-	-	-	-	-	0.47[n]
Onset city (Perth/Australia) [LR]	-	-	0.90[y] / 1.19[y]	-	-	-	0.93[y]
Onset north (Western Australia) [LR]	-	-	0.52[y] / 0.66[y]	-	-	-	0.91[y]
Sexual Behaviour	0	0	0	0	1	1	0
Child has/had a steady boy/girlfriend	-	-	-	-	2.65[j]	2.76[j]	-
Individual level	3	1	6	3	23	11	5
Physical/Physiological	0	0	1	1	2	1	0
Worse physical health	-	-	-	0.63[b]	0.43[b]	0.39[b]	-
Developmental disability	-	-	-	-	3.28[f] / 2.39[f]	-	-
Low resting heartrate	-	-	NR[p]	-	-	-	-
Emotional/personality	3	1	2	2	18	6	5
Sensation-seeking	1.28[n] / 1.34[n] / 1.78[m] / 1.45[m] / 1.12[m]	-	-	1.22[n] / 1.11[m]	1.37[m] / 1.81[n] / 1.62[m]	-	1.46[n]
Depressive symptoms	-	2.28[c]	-	-	2.68[c] / 1.63[e]	-	1.91[c]
Behaviour psychopathy	-	-	-	-	1.50[aa]	1.72[aa]	-
Risk-taking	-	-	-	-	1.40[u]	-	1.22[u]
Hopelessness	-	-	-	-	-	1.54[l]	-
High energy	-	-	-	-	4.70[h]	8.69[h]	-
High impulsivity	-	-	-	-	1.28[aa]	-	-
High irritability	-	-	-	-	8.92[h]	-	-
High expressiveness	-	-	-	-	17.08[h]	-	-

Predictor level / Domain / Variables	Total No of predictors tested in the corresponding level or domain / Odds ratio						
	Child-limited	Adolescent-Limited	Life-course-Persistent	Adolescent-Onset	High-Stable	Antisocial-Decreasing	Medium-stable
Uncontrolled temperament	-	-	3.50[p] / 3.20[p]	-	2.40[p]	4.70[p]	-
ADHD diagnoses	-	-	18.70[p]	-	51.80[p]	4.30[p] / 14.14[p]	-
High surgency/extraversion	-	-	-	-	1.67[s]	-	-
High trustfulness [LR]	0.33[ab]	-	-	-	0.40[ab]	-	0.61[ab]
High trustworthiness [LR]	0.01[ab]	-	-	-	0.03[ab]	-	0.09[ab]
High self-esteem [LR]	-	-	-	0.85[n]	-	-	-
High concentration [LR]	-	-	-	-	0.21[h]	-	-
High orderliness [LR]	-	-	-	-	0.19[h]	-	-
High compliance [LR]	-	-	-	-	0.16[h] / 0.05[h]	0.12[h]	-
High optimism [LR]	-	-	-	-	0.14[h] / 0.06[h]	-	-
High perseverance [LR]	-	-	-	-	0.10[h]	-	-
(Neuro)Cognition/Academic Achievement	0	0	3	0	3	4	0
Low academic achievement	-	-	-	-	1.59[e]	1.82[e]	-
Low cognitive functioning	-	-	-	-	-	0.42[b]	-
Low IQ	-	-	5.80[p] / 2.30[p]	-	5.10[p]	4.00[p] / 2.90[p]	-
Low reading achievement	-	-	NR[p] / NR[p]	-	NR[p]	NR[p] / NR[p]	-
Low academic achievement [LR]	-	-	0.36[ac]	-	-	-	-

Note. ASB = antisocial behaviour. LR = a variable associated with a reduced risk of membership in the focal group. NR = Estimate not reported.
[a](Petts, 2009). [b](Roisman et al., 2010). [c](Maldonado-Molina, Reingle, et al., 2010). [d](Jennings, Maldonado-Molina, & Komro, 2010). [e](Reingle, Maldonado-Molina, et al., 2012). [f](Tabone et al., 2011). [g](Yessine & Bonta, 2009). [h](Becht et al., 2016). [i](Petitclerc et al., 2009). [j](Sittner & Hautala, 2016). [k](Ehrenreich et al., 2014). [l](Park et al., 2010). [m](Jennings, Maldonado-Molina, Piquero, et al., 2010). [n] (Maldonado-Molina et al., 2009). [o](Hoeve et al., 2008). [p](Odgers et al., 2008). [q](Shaw et al., 2012). [r](Ward et al., 2010). [s](Boutin et al., 2017). [t](Maldonado-Molina, Jennings, et al., 2010). [u](Higgins et al., 2010). [v](Silver et al., 2010). [x](Malti et al., 2015). [z](Evans et al., 2016). [w](Kolivoski et al., 2014). [y](Ferrante