Understanding delinquent development from childhood into early adulthood in early onset offenders

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1 General introduction

1.1 Background

It has long been recognized that offending behavior over time is characterized by both a striking degree of continuity, as well as a considerable amount of change (Paternoster, Dean, Piquero, Mazerolle, & Brame, 1997). A first generation of nowadays classic longitudinal studies showed that a small group of people exhibits frequent offending behavior over a long period of time (West & Farrington, 1977; Wolfgang, Figlio, & Sellin, 1972). In spite of significant evidence for stability of offending, many offenders display decreasing offense rates with age and desist from active delinquent involvement in early adulthood (Hirschi & Gottfredson, 1983; Sampson & Laub, 2003).

Even though a fairly consistent collection of risk factors of offending has been identified in prior work (for an overview see Farrington, 2003; Thornberry & Krohn, 2003), explaining variation in offending behavior over an extended period of time remains difficult (Day et al., 2012; Jolliffe, Farrington, Piquero, Loeber, & Hill, 2017; Laub, Nagin, & Sampson, 1998; Sampson & Laub, 2003). While chronic high-rate offenders are generally exposed to highest levels of risk, offenders showing other developmental types of offending are – to some extent – exposed to the same types of risk (e.g., Assink et al., 2015; Baglivio, Wolff, Piquero, & Epps, 2015; Jennings et al., 2019; Jolliffe et al., 2017). As a result, there is a lack of scientific knowledge on which types of offenders are likely to display either stable high or decreasing offending rates with age.

This knowledge-gap is problematic, as the advantages of differentiating between offenders who are about to stop offending and those who are likely to display a long criminal career have been widely acknowledged by criminological theorists and policymakers. It is of theoretical importance to study variation in long-term offending behavior and its correlates in order to confirm or challenge theoretical assumptions on these issues, which constitute an important cornerstone of criminological theory (Moffitt, 1993, 2006). From a policy perspective, knowledge on correlates of distinct long-term offense patterns may strengthen our ability to identify persistent offenders at an early stage of their criminal career, as well as help develop appropriate approaches to intervene to prevent further continuity of offending behavior.

In order to improve our understanding of variation in long-term offending behavior, theory (Moffitt, 1993, 2006; Patterson, Debaryshe, & Ramsey, 1989), and prior studies (Assink et al., 2015; Jolliffe et al., 2017; Mulvey et al., 2010)

emphasize the importance of adopting an integrated approach to risk exposure by examining the collective impact of (the absence of) risk in several life domains (i.e., individual, familial, the peer group, school, and neighborhood). Theoretical approaches and empirical findings from many (inter)national studies suggest that offending behavior is explained by risk factors of offending originating from all life domains, and that they tend to cluster and have mutually reinforcing effects (Blokland & Nieuwbeerta, 2010; Farrington, 2003; Loeber, Stouthamer-Loeber, Slot, van der Laan, & Hoeve, 2008; Moffitt, 1993, 2006; Monroe & Simons, 1991; Zuckerman, 1999). As a result, there has been an increasing acceptance of the notion that individual factors and social/environmental context both contribute to variation in offending behavior (Ousey & Wilcox, 2007). While (biologically based) individual factors may be related to the initial onset of offending behavior, social and environmental factors related to the family, peer group, school, and neighborhood may bring about offending behavior in some individuals, while they contribute to the escalation or stabilization of offending in others (Moffitt, 1993). It is therefore important to account for the complex interplay between risk factors of offending to understand processes of change and continuity in offending behavior (Morizot, 2019).

Studying delinquent development and its correlates in early onset offenders may represent an important opportunity to increase our understanding of variation in long-term offending behavior. Especially a police contact/arrest at an early age (i.e., below age 12, see Loeber & Farrington, 2001; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996) has emerged as an important indicator for persistence in offending (DeLisi, Neppl, Lohman, Vaughn, & Shook, 2013). Not only are offenders with an early onset at higher risk of displaying persistent offending behavior than those who start after age 12 during adolescence (Farrington et al., 1990; Glueck & Glueck, 1950; Moffitt, 1993; Tolan, 1987), they are also likely to commit more serious and violent offenses (Snyder, 2001). On the other hand, most delinquent children do not display persistent offending behavior (Robins, 1966, 1978), and even the most troubled children may desist from crime (Wilson, 1991). Early onset offenders thus constitute an important offender population displaying both stability and change in their long-term offense patterns. Studying (heterogeneity in) their delinquent development provides the opportunity to follow a high-risk offender population during an extended period of the life-course, during which changes occur in several life domains (Berndt, 1982; Larson & Richards, 1991), and possibly identify early origins and contributing social/environmental factors of chronic and violent offending (Loeber & Farrington, 2001). To date, insight into long-term delinquent development and its correlates in early onset offenders with a police contact/arrest (i.e., childhood arrestees) is however largely lacking, because of a lack of suitable longitudinal data on offenders in contact with the law below the age of criminal responsibility in many Western countries

(e.g., 12 years in the Netherlands) (Farrington, Loeber, Yin, & Anderson, 2002; Hemphill, Heerde, Herrenkohl, & Farrington, 2015; Jolliffe et al., 2019).

The current thesis therefore intends to provide insight into variation in offending behavior over an extended period of time by addressing the following two general aims. First, this thesis aims to provide empirical insight into (variation in) long-term development of offending behavior in early onset offenders known to the police, and its associated singular identified risk factors. Second, the current thesis aims to improve our understanding of variation in long-term offense patterns by accounting for risk exposure across life domains (i.e., individual, familial, the peer group, school, and neighborhood). In order to do so, this thesis adopts an interdisciplinary approach to risk exposure by accounting for interaction effects between and possible clustering of risk factors previously identified in the fields of sociological, biosocial, and developmental criminology.

In order to address its aims, the current thesis uses unique data from the *Dutch Childhood Arrestees Study*, containing information on offenders who were first registered by the police between 2000 and 2006 for showing offending behavior below age 12. Longitudinal data on offending, and incarceration on over 700 childhood arrestees were retrieved from official registers in the Netherlands, and merged with rich survey data on theoretically important risk factors from individual, familial, peer, school, and neighborhood domains. In this thesis, using advanced statistical techniques, several ways are adopted to take interaction effects between and possible clustering of risk factors of offending into account.

Before turning to the empirical chapters of the current thesis, the remainder of this introduction is organized as follows. The general theoretical background of this thesis is discussed in Section 1.2. Section 1.3 provides an overview of earlier empirical studies on delinquent development and its correlates. Contributions of the current thesis are specified in Section 1.4. And lastly, the outline of the current thesis is described in Section 1.5.

1.2 Theoretical framework

While criminological theory overall states that offenders with an onset in childhood are likely to display persistent offending behavior across the life course (i.e., Gottfredson & Hirshi, 1990; Moffitt, 1993), it offers divergent explanations for the overall expected continuity in offending. Theory aimed at explaining the likelihood and development of offending behavior can been anchored by three broad explanatory paradigms.

First, psychological criminology suggests that individuals develop a static antisocial propensity – from both biological and social origins – in early childhood, which determines the risk of offending during the entire life course (Gottfredson & Hirshi, 1990; Wilson & Herrnstein, 1985). According to this

line of reasoning, stable antisocial propensity is reflected by personality characteristics – such as impulsiveness, hyperactivity, and low self-control – which are expected to determine whether individuals are highly involved in offending behavior or display little delinquent involvement. In the case of an early onset of offending, the relatively high criminal propensity that caused the early onset is thought to also result in continuous delinquent involvement across the lifespan.

Second, sociological theories of offending have traditionally focused on neighborhood and environmental characteristics (Merton, 1938; Shaw & McKay, 1969), and social relationships with family, peers, and school (Akers, 1973; Akers & Jennings, 2016; Hirschi, 1969; Hoeben, Meldrum, Walker, & Young, 2016; Sutherland, 1947). Regarding neighborhood characteristics, it has been suggested that individuals residing in deprived neighborhoods are more likely to display offending behavior, because they lack the legitimate means to achieve their desired financial or economic goals (Merton, 1938), and community relationships and local institutions fail to exert informal social control (Shaw & McKay, 1969). Sociological theories focused on social relationships continue to argue that informal social control resulting from close relationships with conventional others restrains individuals engaging in offending behavior (Hirschi, 1969), while continued social interaction with delinquent others – especially delinquent peers – increase the likelihood of offending behavior (Akers, 1973; Sutherland, 1947). To the extent that neighborhood characteristics and social relationships lead to an early onset of offending, these social influences are also thought to result in the continuation of the offending behavior.[1]

A third framework combines ideas from psychological and sociological criminology to explain the likelihood (Monroe & Simons, 1991; Zuckerman, 1999) and development (Moffitt, 1993, 2006) of offending behavior. Both biosocial (Monroe & Simons, 1991; Zuckerman, 1999) and developmental (Moffitt, 1993, 2006) theorists suggest that stable antisocial propensities and (changes in) social influences may interact, and together explain within- and between-individual change in offending over time. Specifically, Moffitt (1993, 2006) suggests that early onset offenders – as opposed to adolescent onset offenders – develop relatively high levels of antisocial propensity, based on inherited or acquired (through mechanisms such as maternal drugs use or pregnancy complications) biological vulnerability. Children suffering from high levels of antisocial propensity are also thought to particularly experience, as well as be more susceptible to, adverse social interactions in several life

1 Chapter 6 offers a more elaborate theoretical discussion of the way social influences are theorized to influence the likelihood of offending behavior.

domains (i.e., family, peers, school, and neighborhood).[2] The combination of biological vulnerability and social/environmental disadvantage is assumed to exponentially increase the likelihood of persistent, versatile, and increasingly violent offending behavior in a large share of early onset offenders (i.e., high level chronics). When early onset offenders additionally suffer from social isolation (i.e., heightened depression and anxiety), or reside in more adaptive social environments, the escalation of offending may be prevented, resulting in persistent yet low offending rates across adolescence (i.e., low level chronics) (Moffitt, 2006).[3]

In sum, criminological theory assumes that offenders with an onset in childhood will typically display persistent offending behavior throughout the life-course. It follows from biosocial and developmental criminological theory that continuity in offending results from a process of reciprocal interactions between individual, familial, school, peer, and neighborhood characteristics. To increase our understanding of the development of offending in offenders with an onset in childhood, it is thus of theoretical importance to study risk exposure in multiple life domains, and account for their mutually reinforcing effects. In the current thesis, different approaches are therefore used to account for the combined effects of (the absence of) risk exposure across life domains.

1.3 Prior research

The following section provides an overview of research on the development of offending with age and associated singular identified risk factors, as well as of (ways to conduct) research on associated risk exposure across life domains. Subsequently, limitations of prior work and underexplored research areas are stipulated.

1.3.1 Delinquent development and associated singular identified risk factors

With the aim of providing extensive insight into longitudinal patterns of offending, and based on criminological theory assuming that many individual

2 If the biological vulnerability were inherited, this implies that at least one parent also suffers from neuropsychological, temperamental, or cognitive deficits. These inherent deficits in the parents then have a direct negative impact on the child's social environment. Alternatively, a deficient social environment might actually cause prenatal problems (e.g., such as prenatal substance – alcohol, drugs, cigarette – use) that, in turn, result in biological vulnerability in the child.

3 A more extensive overview of dual taxonomic theory on delinquent development is provided in Chapters 3 and 4. Additionally, Chapter 2 discusses biosocial theory useful for understanding why individuals exposed to a combination of biological and social risk factors are at increased risk of displaying offending behavior.

offense patterns will be similar (Moffitt, 1993), a rapidly accumulating number of empirical studies has focused on grouping individuals with homogeneous offending patterns (Nagin, 2005; Nagin & Land, 1993). An important advantage of identifying subgroups displaying distinct developmental trajectories of offending behavior is that it allows researchers to explore varying levels of continuity and change in offending behavior in a given sample.

While prior work aimed at identifying distinct offending trajectories has not yet reached consensus about the number or shape of distinct offending trajectories, it is well supported that substantial variation in the development of offending with age exist (for narrative reviews, see Jennings & Reingle, 2012; Piquero, 2008; van Dulmen, Goncy, Vest, & Flannery, 2009). Most prior studies identified between two and seven trajectory subgroups, with three or four being the most common. Several studies identifying four groups report a similar model: stable-low, stable-high, increasing, and decreasing trajectories (Lynne-Landsman, Graber, Nichols, & Botvin, 2011; Miller, Malone, Dodge, & Conduct Problems Prevention Research Group, 2010; Odgers et al., 2008; White, Bates, & Buyske, 2001). In the Netherlands, strong support for distinct offending trajectories was found in the Criminal Career and Life Course Study based on a Dutch conviction cohort (Blokland, Nagin, & Nieuwbeerta, 2005). Four trajectory subgroups were identified, of which the high-rate persisters continued offending even after age 50. The few prior studies that were able to explore which trajectories were populated by early onset offenders confirmed taxonomic assumptions, by showing that early onset offenders generally populate the most chronic trajectory subgroup, and commit the highest amount and most diverse types of offenses (Allard, Chrzanowski, & Stewart, 2017; Broidy et al., 2015; Day et al., 2012).

Various studies have shown that singular identified risk factors can be used to distinguish between high-rate chronic offenders and non- or sporadic offenders, with high-rate chronic offenders being exposed to overall heightened levels of risk in several life domains. For example, risk factors characterizing high-level trajectories include increased levels of impulsivity (Baglivio et al., 2015), low parental supervision/neglectful parenting (Hoeve et al., 2008; Monahan & Piquero, 2009; Wiesner & Capaldi, 2003), and deviant peers (Baglivio et al., 2015; Chung, Hill, Hawkins, Gilchrist, & Nagin, 2002; Monahan & Piquero, 2009; van der Geest, Blokland, & Bijleveld, 2009). Unfortunately however, singular risk factors are less helpful in differentiating between distinct offending trajectories (Day et al., 2012; Laub et al., 1998; Mulvey et al., 2010; Sampson & Laub, 2003; Wiesner, Kim, & Capaldi, 2005).

1.3.2 Delinquent development and associated risk exposure across life domains

As previously mentioned, scholars have highlighted that accounting for exposure to combinations of risk factors across life domains, as well as their mutually reinforcing effects (Loeber, Stouthamer-Loeber, et al., 2008; Moffitt, 1993, 2006), might help improve our understanding of variation in (long-term) delinquent development (Morizot, 2019). Three ways of taking the interaction effects and clustering of risk factors of offending into account are described below.

Biosocial interaction
One way of accounting for possible mutually reinforcing effects of risk factors of offending is by studying their interaction. As previously discussed, biosocial and developmental criminological theory emphasize the importance of combining biological and social/environmental explanatory factors into a multidisciplinary (i.e., biosocial) perspective on adverse behavioral outcomes. In response, researchers have addressed how social (the family and peers), and environmental (the neighborhood) correlates of offending may exert diverse effects on individuals with different biological wiring. As the body of literature on biosocial interactions and delinquency is rapidly growing, it is important to synthesize this research in order to offer new interpretations that transcend findings from individual studies.

Risk profiles
Building on studies highlighting the importance of risk exposure in distinct life domains, some prior work has aimed to identify subgroups of individuals exposed to similar levels or combinations of risk factors in multiple life domains (i.e., risk profiles). This approach allows researchers to simultaneously examine numerous risk factors of offending, while accounting for interaction effects between and possible confounding of singular risk factors. Findings from the limited number of studies identifying risk profiles within offender populations support the assumption that there are subgroups of individuals exposed to distinct patterns of risk (i.e., Dembo, Wareham, Poythress, Meyers, & Schmeidler, 2008; Lopez-Romero et al., 2019; Schwalbe, Macy, Day, & Fraser, 2008). Importantly, prior work highlights the utility of risk profile identification as they revealed associations between specific combinations of risk and variation in delinquent involvement (Onifade et al., 2008).

Within-individual change in risk exposure
Finally, scholars have accounted for risk exposure across life domains by studying the association between developmental changes in risk exposure and variability in individual offending behavior over time (Thornberry, 1996). By focusing on associations between within-individual change in risk exposure

and behavioral outcomes, pre-existing differences between individuals are held constant and are therefore accounted for (Allison, 2009). The few studies that used the within-individual methodology to explain variation in offending across adolescence have generated mixed findings. While some work showed that change in social influences in familial, peer, and school domains had the expected effect on changes in individual offending behavior (see for example Beardslee et al., 2018; Craig, 2016; Rokven, de Boer, Tolsma, & Ruiter, 2017), other studies failed to find associations between changes in time-varying social influences and individual's own delinquent behavior (Farrington et al., 2002; Unnever & Chouhy, 2019).

1.3.3 Shortcomings of previous research

While recognizing the clear value of prior work on delinquent development and its correlates, there are three main limitations that should be mentioned, justifying the need for further research. First and foremost, on the basis of reviewing previous studies it can be concluded that, despite the apparent theoretical and practical importance of studying delinquent development in the population of offenders in contact with the law below age 12, this has rarely been done (but see van Domburgh, Vermeiren, Blokland, & Doreleijers, 2009). International and national longitudinal studies on the development of offending over time are typically based on general population and general offender samples. Unfortunately, it is not sufficient to simply generalize previous findings to the specific offender population of early onset offenders, as early onset offenders without an arrest during adolescence are not included in general population or general offender samples. Furthermore, variation in offending among children that do re-offend might be overshadowed by offending behavior of the more common adolescent onset offender. Because of a lack of available longitudinal studies focused on early onset offenders known to the police, it is currently unknown how many children in contact with the law are continuously registered by the police for offending behavior into early adulthood, and what long-term re-offense patterns in childhood arrestees might look like.

Second, most prior studies aimed at providing insight into the etiology of variation in offending pathways used singular identified risk factors, which were largely incapable of distinguishing between offenders populating distinct offending trajectories (e.g., Assink et al., 2015; Day et al., 2012; Ferrante, 2013; Jolliffe et al., 2017; Ward et al., 2010). While accounting for risk exposure across life domains seems like a promising avenue to further our understanding of variation in delinquent behavior, the limited number of studies that adopted a holistic view on risk exposure (see, among others, Craig, 2016; Lopez-Romero et al., 2019; Na, 2017; Schwalbe et al., 2008) unfortunately measured delinquent outcomes across a short period of the lifespan. As a result, our understanding

of variation in longitudinal offense patterns remains limited, and theoretically-relevant issues regarding the etiology of distinct long-term developmental patterns of offending remain understudied.

And finally, although prior work has progressed our understanding of within-individual change in offending behavior over time, empirical studies have yet to address the biosocial and developmental theoretical assumption that the effects of time-varying social influences on offending depend on individual's antisocial propensity. Consequently, we do not know whether variability in individual offending behavior over time can be explained by interaction effects between antisocial disposition and changes in social influences, like developmental taxonomic theory suggests.

1.4 The current thesis

The current thesis aims to address above-mentioned matters, by studying the extent to which risk factors across life domains can help explain variation in both between- and within-individual offending behavior over time in early onset offenders. As mentioned earlier, its two central aims are to (1) provide insight into (variation in) the long-term development of offending behavior in early onset offenders with a police contact/arrest, and associated singular identified risk factors, and (2) improve our understanding of variation in long-term offense patterns by combining theoretical insights stemming from different scholarly traditions (i.e., sociological, biosocial, and developmental criminology) on risk exposure in multiple life domains (i.e., individual, familial, peers, school, and neighborhood).

In doing so, the current thesis adds to prior research in three important ways. First, given the paucity of studies focused on long-term re-offense patterns in early onset offenders, the current thesis follows children in contact with the law across a lengthier follow-up period than all of the previous studies, making use of the rare opportunity to explore meaningful variation in long-term delinquent pathways within this high-risk offender population. Importantly, early onset offenders were followed beyond adolescence (Jennings & Reingle, 2012), during which delinquent behavior is theorized to peak for all individuals regardless of age of onset (Moffitt, 1993). As such, the current thesis represents an important contribution to our understanding of distinct delinquent pathways in offenders with an onset in childhood, who cause so much harm to society. Second, this thesis incorporates a large set of correlates of offending from a variety of scholarly traditions, including sociological, biosocial, and developmental criminology. This is important, as all three research fields have found associations between singular identified risk factors and future offending behavior. Furthermore, criminological theory and prior studies suggest that risk factors of offending do not operate in isolation but tend to cluster and are mutually reinforcing (Caspi et al., 2014; Farrington &

Welsh, 2008; Loeber, Stouthamer-Loeber, et al., 2008; Moffitt, 1993, 2006; Monroe & Simons, 1991; Zuckerman, 1999). By adopting an interdisciplinary perspective on offending, the current thesis is therefore able to empirically address several theoretical assumptions on associations between risk exposure across life domains and development of offending behavior over an extended period of the lifespan. Third, state of the art methods are used to account for possible clustering of and cumulative effects between risk factors of offending.

In order to address its aims, the current thesis builds upon the work of van Domburgh (2009), Geluk (2014), and Cohn (2017), by using and extending data from the *Dutch Childhood Arrestees Study* – a prospective longitudinal research project focused on children in contact with the police for the first time because of an alleged offense under the age of 12.,[45] As offending behavior displayed under the age of 12 is not recorded in national registration systems, three local police registration systems (Rotterdam-Rijnmond, Gelderland-Midden and Utrecht) were used to select children registered for displaying behavior that could be prosecuted when displayed from age 12 onward, excluding status offenses (i.e., behavior that is only prosecutable for certain (age) groups, such as truancy) as these are generally not dealt with by the Dutch police. In order to address the first aim, the delinquent development of children in contact with the law was reconstructed using official registration data, containing information on police registrations, mortality, and criminal sanctions from age 12 into early adulthood. The second aim was addressed by combining official registration data with information on a large number of individual, familial, peer, school, and neighborhood characteristics, derived from standardized instruments – interviews as well as questionnaires – administered to parents and children during three assessment occasions across adolescence.

4 This study was carried out by VU University Medical Centre, Department of Child and Adolescent Psychiatry and approved by the Dutch Ministry of Justice. The *Dutch Childhood Arrestees Study* was supported by the municipalities of Utrecht and Amersfoort, the Rotterdam metropolitan region, and the province of Utrecht; the Kinderpostzegels Nederland Foundation, and Leiden University.

5 It is important to note that, by using data from the *Dutch Childhood Arrestees Study*, the current thesis used a first police registration below age 12 as a proxy for early onset offending. While a police registration in childhood is an important risk factor for persistent delinquent behavior (DeLisi et al., 2013), our sample of early onset offenders may include children who only displayed offending behavior that one time they were registered by the police. The proxy for early onset offending used in the current thesis therefore differs from the one used in studies conducted by Moffitt and colleagues (i.e., Moffitt & Caspi, 2001; Moffitt et al., 1996; Moffitt, Caspi, Harrington, & Milne, 2002), which defined early onset offenders as stable and pervasive antisocial behavior problems across situations below age 12 (i.e., rated one standard deviation above the sample mean by parents and teachers on at least three of four assessment occasions).

1.5 Outline of the thesis

Empirical findings addressing the two central aims are presented in the following four chapters of this thesis (an overview of which is presented in Table 1.1).[6]

As questions surrounding the interaction between antisocial disposition and social/environmental influences on offending behavior are central to the current thesis, a review of prior literature on interactions between biological and social/environmental correlates of offending is offered in Chapter 2. The current thesis is especially focused on antisocial disposition resulting from biological vulnerability, as both theory (Moffitt, 1993) and prior research (for a review see Yang et al., 2014) have identified biological vulnerability as an important indicator of antisocial disposition interacting with social risk. As crime is a relatively rare phenomenon, Chapter 2 is based on the related, but more general phenomenon of antisocial behavior, in order to learn more about ways in which biosocial interaction is associated with adverse behavioral outcomes.

Chapter 3 addresses the first general aim of this thesis, by empirically evaluating hypotheses on the delinquent development in early onset offenders and associated singular identified risk factors. Specifically, Chapter 3 studies whether early onset offenders have distinctive long-term re-offense patterns from age 12 to age 25 across several types of offenses. In order to address the assumption from taxonomic theory that males, minorities, and children from disadvantages neighborhoods are at increased risk of following chronic offending pathways, Chapter 3 also examines whether offenders following distinct trajectories can be characterized based on gender, ethnicity, and neighborhood socioeconomic status and urbanization levels.

Building on findings from the systematic review described in Chapter 2, Chapter 4 continues to address the second aim of the thesis by examining whether clusters of risk in childhood can help explain variation in long-term variation in offending behavior. Specifically, it investigates whether subgroups of early onset offenders are identifiable based on re-offense patterns into early adulthood (i.e., trajectory subgroups), as well as based on similarity in risk exposure across life domains (i.e., risk profiles). Subsequently, Chapter 4 compares risk profiles on placement across distinct offending trajectories up to age 20.

Chapter 5 presents a study that uses the interaction between time-stable biological characteristics with time-varying social variables that reflect elements of social bonding and social learning to explain within-individual variation in offending behavior over time. This chapter first provides insight into the extent to which change in social bonds with family, peers, and school can help

6 It should be noted that Chapters 2 to 5 were originally written as separate manuscripts, resulting in a degree of overlap between the chapters in this dissertation.

explain variability in individual offending behavior in early onset offenders. In addition, it is investigated whether the social bonds-offending relationship varies across early onset offenders who suffered from biological vulnerability as opposed to the early onset offenders who did not.

Finally, Chapter 6 provides a summary of the main findings from this thesis and recapitulates answers to its general aims. After evaluating the current thesis' strengths and weaknesses, recommendations are made for future research and intervention efforts aimed at curbing the delinquent development in early onset offenders in contact with the law.

Table 1.1 Outline of the thesis

Chapter	Research question(s)	Data	Dependent variable	Independent variable	Analytical strategy
2	– To what extent are interactions between biological and social/ environmental risk factors associated with antisocial and delinquent behavior?	– Prior empirical work	– Antisocial behavior	– Peri/prenatal problems or psycho-physiological risk – Social/envi-ronmental risk	– Literature review
3	– To what extent can distinct offending trajectories be identified based on frequency and type of offending from childhood into adulthood in early onset offenders? – And to what extent can sex, ethnicity, and childhood neighborhood factors characterize subgroups following distinct offending trajectories?	– Registration data S1 & S2, *N* = 708	– Offense frequency across age and offense types	– Sex – Ethnic origin – Neighborhood SES – Neighborhood urbanization	– Multi-trajectory modeling – Multinomial regression
4	– To what extent are early onset offenders assigned to specific risk profiles identified in childhood differentially at risk of following specific offending trajectories into early adulthood?	– Registration data – Survey data S2, W1, *N* = 348	– Offense frequency across age	– Risk profiles, based on risk exposure in individual, familial, peer, school, and neighborhood domains	– Trajectory modeling – Latent profile analysis – Analysis of variance – Chi-squared test – Multinomial regression
5	– To what extent are changes in social bonds with parents, peers, and school associated with changes in offense frequency in early onset offenders, during the transition from childhood into early adolescence? – And to what extent do such associations depend on biological vulnerability resulting from peri/prenatal problems?	– Registration data – Survey data S2, W1-3, *N* = 348	– Offense frequency across time	– Changes in social bonds with family, peers, and school – Peri/prenatal problems	– Hybrid random effects models

Note. S1 = Sample 1; S2 = Sample 2; W1 = wave 1; W1-3 = wave 1 through 3, SES = socioeconomic status.

2 Biosocial studies of antisocial behavior

A systematic review of interactions between peri/prenatal complications, psychophysiological parameters, and social risk factors ▪

ABSTRACT

In order to reduce antisocial behavior (ASB) and associated individual and societal problems, insight into determinants of ASB is warranted. Increasing efforts have been made to combine biological and social factors in explaining antisocial development. Two types of biological parameters have been studied vastly and provide the most compelling evidence for associations between biosocial interaction and ASB: peri/prenatal complications and psychophysiological parameters. A systematic review was conducted to synthesize empirical evidence on interactions between these biological measures and social risk factors in predicting ASB. In doing so, we aimed to (1) examine whether *specific* peri/prenatal and psychophysiological measures composite a vulnerability to social risk and increase risk for *specific* types of ASB, and (2) evaluate the application of divergent biosocial theoretical models. Based on a total of 50 studies (documented in 66 publications), associations between biological parameters and ASB were generally found to be stronger in the context of adverse social environments. In addition, associations between biosocial interaction and ASB were stronger for more severe and violent types of ASB. Further, in the context of social risk, under-arousal was associated with proactive aggression, whereas over-arousal was associated with reactive aggression. Empirical findings are discussed in terms of distinct biosocial theoretical perspectives that aim to explain ASB, and important unresolved empirical issues are outlined.

Key Words
Biosocial interaction, antisocial behavior, systematic review

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2.1 Introduction

Antisocial behavior (ASB) is costly for society and causes harm to individuals (M. A. Cohen & Piquero, 2009; Scott, Knapp, Henderson, & Maughan, 2001). ASB (i.e., chronic violations of social rules and norms; Hinshaw & Zupan, 1997) generates victims and high criminal justice system and treatment costs (M. A. Cohen, 1998). In addition, many antisocial individuals struggle with drug and/or alcohol addictions, experience psychiatric problems, and have numerous social problems, such as unemployment, homelessness, and financial difficulties (Dembo et al., 2008; Loeber & Farrington, 2000; Moffitt & Caspi, 2001).

In order to reduce the above-mentioned problems, it is important to develop and advance existing etiological theories on determinants of ASB. Knowledge of underlying factors associated with antisocial development can provide directions for effective prevention and intervention programs, as it allows for programs to target individuals' specific needs. Addressing such needs will reduce crime-related societal costs, registered crime, and individuals' adverse mental health outcomes (Chung et al., 2002; Raine et al., 2005).

For several decades, psychologists and sociologists have identified numerous social and environmental factors related to ASB. Theories in these fields highlight the role of personality traits, relationships with parents and peers, as well as environmental processes as being the cause of antisocial development. For example, low self-control (Gottfredson & Hirshi, 1990), parental criminal behavior (Farrington, 1979), and insufficient parental supervision (Gottfredson & Hirshi, 1990) are theorized to instigate ASB. Further, exposure to delinquent peers (Warr, 1993), and adverse community characteristics, such as residing in disadvantaged neighborhoods (Shaw & McKay, 1942), are hypothesized to increase antisocial development.

Independently, biological studies have more recently made enormous progress in identifying biological factors that are associated with ASB. Nowadays, there is a large body of evidence supporting the idea that biological factors are equally important in explaining antisocial development, emphasizing that these factors should be considered alongside social and environmental influences. Evidence has been gathered by an abundance of twin, family, and adoption studies as well as laboratory experiments.

There is now a long list of biological factors that have been empirically linked to ASB. For example, twin and adoption studies have shown that about 50% of individual differences in ASB can be explained by genetic variation (Polderman et al., 2015; Rhee & Waldman, 2002). Further, there is evidence that peri/prenatal factors, such as maternal smoking during pregnancy, predict ASB in offspring (for a review see Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002). Additionally, brain imaging research has linked damage to brain regions (for a meta-analysis see Yang & Raine, 2009), as well as gray matter abnormalities (for a meta-analysis see Rogers & De Brito, 2016) to ASB. Psychophysiological studies have specified the importance of direct relations

between resting heart rate and ASB (for a review see Portnoy & Farrington, 2015). Lastly, recent studies have also shown that neuropsychological functioning influences antisocial development, as high IQ was found to function as a protective factor against developing ASB (for a review see Ttofi et al., 2016).

Although research in several disciplines have independently provided adequate empirical support for the importance of their research field, they have failed to explain why individuals are differentially affected by biological, social and environmental influences. Although some individuals develop ASB in the most benign environments, others abstain from developing ASB in the most criminogenic environments. In between these two extremes are individuals whose criminal tendencies might come to surface when triggered by certain environmental influences (Walsh & Beaver, 2009).

With the intention of explaining why individuals differ in their tendency to develop ASB in similar environments, it is essential to combine biological and social/environmental factors into a multidisciplinary (i.e., biosocial) perspective on ASB. In response to advances in biological sciences and in order to explain the dynamic nature of ASB, scholars have come to understand that we have to incorporate biological and social/environmental factors into theoretical frameworks on ASB. We need to break through the fences that previously separated research areas and study the extent to which different people behave differently in comparable social environments, and vice versa (Walsh & Beaver, 2009). Such an interdisciplinary approach is crucial to further our understanding of ASB and provide new insights for potentially more effective prevention and intervention programs.

The current study therefore aims to provide an overview of the rapidly growing body of literature on interrelations between biological and social correlates of ASB. By focusing on biosocial research on ASB, we hope to evaluate some detailed, yet contradictory, expectations formulated in biosocial theories of ASB. In addition, we hope to increase our understanding of this research field, which has been hampered by studies testing markedly different research questions via different designs, in varying samples, using a range of assessment methods. We therefore aim to synthesize and evaluate their findings in order to offer new interpretations that transcend findings from individual studies as well as help steer future research questions by pointing out open empirical issues.

2.1.1 Theoretical framework

From a biosocial standpoint, different theoretical views on ASB can be distinguished. These views offer conflicting predictions on the way biological and social factors simultaneously influence antisocial development. As we aim to interpret study findings in light of these theories, we introduce them in the following paragraphs.

First, the *social push* hypothesis states that the biology-ASB relation is stronger for those from more benign home backgrounds (Mednick, 1977; Raine & Venables, 1981). For these individuals, the social push toward crime is relatively weak, allowing for the relation between biology and ASB to shine through (Mednick, 1977; Raine & Venables, 1981). When 'the social push' toward ASB is stronger, these social causes of crime are thought to overshadow biological contributions to ASB.

Alternatively, *diathesis-stress/dual risk* theory suggests that individuals with biological diatheses (i.e., vulnerabilities) are disproportionately at risk for developing ASB when they are exposed to adverse social and environmental contexts (Monroe & Simons, 1991; Zuckerman, 1999). Such vulnerabilities are considered stable, but not unchangeable over the life-course. When biologically vulnerable individuals are confronted with adverse life experiences, the combination of the biological predisposition and stress associated with these experiences may exceed a certain threshold and catalyze the development of ASB (Monroe & Simons, 1991; Zuckerman, 1999).

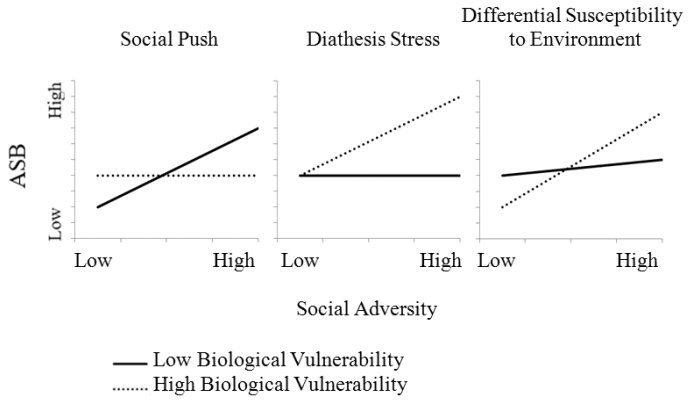
This last-mentioned theoretical perspective has been extended to encompass the idea that individuals with biological vulnerabilities have the lowest levels of ASB in privileged social environments (Belsky, 1997; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009; Boyce & Ellis, 2005; B. J. Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). This *differential susceptibility to environment* hypothesis suggests that biological vulnerabilities are better described as plasticity or malleability traits that sensitize individuals to negative as well as positive social contexts. Subjected to stressful life experiences, biological sensitivity would increase the likelihood of negative behavioral outcomes (dual risk). However, when exposed to positive environments, biologically sensitive individuals would have better outcomes than peers without biological sensitivity traits. The argument is that biological sensitivity allows individuals to acquire more social skills in prosocial environments and develop adaptive ways to deal with stress, lowering the threshold for developing ASB (Belsky, 1997; Belsky et al., 2007; Belsky & Pluess, 2009; Boyce & Ellis, 2005; B. J. Ellis et al., 2011).

2.1.2 Biosocial interaction

Much of the research on ways in which biological and social factors produce variation in behavioral outcomes has been guided by the logic of biosocial interaction. The question behind studies on biosocial interaction is whether or not biological risk factors are more strongly related to behavioral outcomes, for different levels of social risk. As the literature is supportive of the view that negative and positive social contexts can be found at both extremes of the same variables (see Stouthamer-Loeber et al., 1993), studies on biosocial interaction are capable of testing all three theoretical perspectives.

Different interaction effects are expected based on the above-mentioned theoretical models (see Figure 2.1). If the social push perspective is correct, the relation between biological parameters and ASB will be stronger when social adversity is weaker. If the diathesis-stress model is correct, the relation between biology and ASB will be stronger when social adversity is higher. The differential-susceptibility perspective adds that individuals higher on biological vulnerabilities, have the lowest levels of ASB in positive social environments.

Figure 2.1: Biosocial theories of biosocial interaction



Many biological parameters are studied as a biological vulnerability interacting with social adversity. In accordance with previous narrative reviews on the biosocial bases of ASB (F. R. Chen et al., 2015; Raine, 2002a; Rudo-Hutt, 2011; Yang et al., 2014), we distinguish between the following biological research areas: peri/prenatal complications, genetics, brain abnormalities, neuropsychology, psychophysiology, neurotransmitters, and hormones.

Some of the most significant evidence that interactions of biological and social risk factors increase risk for ASB has been provided by research on peri/prenatal risk and psychophysiological measures (for narrative reviews see Raine, 2002a; Rudo-Hutt, 2011; Yang et al., 2014). As research has produced a rich body of literature on biosocial interaction using these two biological parameters as compared with other biological factors, reviewing literature on biosocial interactions within the areas of peri/prenatal and psychophysiological factors is currently considered most fruitful. They are therefore the focus of the current systematic review. Accordingly, biosocial interactions using other biological measures are outside of the scope of this review. We refer the interested reader to other publications on biosocial interaction in the area of

genetics[1] (see Janssens et al., 2015; King et al., 2016; Marsman, Oldehinkel, Ormel, & Buitelaar, 2013; Tuvblad et al., 2016; Watts & McNulty, 2016), brain abnormalities (Raine et al., 2001), neuropsychology (see Jackson & Beaver, 2016; Levine, 2011; Yun & Lee, 2013), neurotransmitters (see Moffitt et al., 1997), and hormones (L. Ellis & Das, 2013; Pascual-Sagastizabal et al., 2014; Steeger, Cook, & Connell, 2017; Yu et al., 2016).

The first biological parameter, peri/prenatal complications, encompasses prenatal substance exposure, pregnancy and delivery complications (Griffith, Azuma, & Chasnoff, 1994; Steinhausen & Spohr, 1998; Wakschlag et al., 1997), and biomarkers for fetal neural maldevelopment such as low birth weight and minor physical anomalies (i.e., slight defects of head, hair, eyes, mouth, hand, and feet; Waldrop, Pedersen, & Bell, 1968). These complications are assumed to constitute a biological vulnerability for ASB, because they would cause fetal brain damage and neuropsychological deficits, which in turn may lead to ASB (Farrington, 1987; Moffitt, Lynam, & Silva, 1994; Raine, 2002b).

The second biological parameter, psychophysiological measures, covers cognition and emotions as revealed through autonomic nervous system (ANS) (re)activity (Hugdahl, 2001), and influences individuals' 'fight or flight' response to stressful situations. Different pathways from ANS (re)activity to ASB are proposed. One possibility is that psychophysiological under-arousal (i.e., representing insensitivity to stressful events) causes individuals to show ASB to increase their arousal to more comfortable levels (Zuckerman, 1999). In addition, lower psychophysiological responses to adverse circumstances are thought to reflect fearlessness. As a result, fear of negative consequences would not inhibit these individuals from showing ASB (Beauchaine, 2001; Fung et al., 2005). Another possibility is that psychophysiological over-arousal (i.e., representing sensitivity to stressful events) energizes antisocial responding (Scarpa & Raine, 1997), and lead to angry responses to perceived provocation (Berkowitz, 1962; Dollard, Miller, Doob, Mowrer, & Sears, 1939). Alternatively, higher levels of ANS responsiveness are thought to reflect emotion regulation and conscience development, and therefore lead to more positive behavioral outcomes in high-risk environments compared with individuals with lower levels of ANS responsiveness (Beauchaine, 2001; Katz & Gottman, 1997).

1 Although important advances have been made to study associations between candidate gene-environment interactions and ASB, findings have generally been inconclusive and are typically characterized by underpowered samples (Dick et al., 2015; Duncan & Keller, 2011; Okbay & Rietveld, 2015). Tielbeek et al. (2016) therefore suggested that future studies should focus on interactions between boarder polygenetic profiles and environmental factors to achieve better insight into biosocial interactions and ASB. As such, the study of biosocial interactions in the area of genetics requires different methodological approaches (i.e., twin or adoption studies or genome-wide data) than studies in the areas of peri/prenatal risk and psychophysiological functioning. Studies on biosocial interactions in the area of genetics are therefore not included in the current systematic review.

2.1.3 The current study

As empirical literature on biosocial interaction accumulates rapidly, it is important to continuously conduct reviews in this research area. The current systematic review aims to (1) systematically analyse empirical studies on associations between biosocial interactions in the areas of peri/prenatal complications and psychophysiological functioning and ASB, (2) examine the extent to which empirical evidence supports conflicting theoretical models on the association between biosocial interactions and ASB, and (3) make recommendations for future biosocial research.

In doing so, we aim to update and extend previous (mostly narrative) reviews. First, as previous reviews (P. A. Brennan & Raine, 1997; F. R. Chen et al., 2015; Raine, 2002b; Rudo-Hutt, 2011; Yang et al., 2014) are mostly based on studies published before 2000, we aim to answer some specific questions that remained unanswered in previous narrative reviews by reviewing research published after 2000. Specifically, we address the following questions: Do specific peri/prenatal and psychophysiological risk factors interact with specific social/environmental risk factors, or does any combination increase the likelihood of individuals showing ASB? Does the interaction between peri/prenatal and psychophysiological parameters with social risk contribute equally to the prediction of all subtypes of ASB, or is the relationship between biological risk and specific subtypes of ASB more influenced by social risk? Second, as methodological progress has been made in measuring biological parameters since 2000 (Bar-Oz, Klein, Karaskov, & Koren, 2003; D'Onofrio & Lahey, 2010; Gray et al., 2010; Konijnenberg, 2015; Lester, Andreozzi, & Appiah, 2004), the internal validity in empirical studies summarized in this review has increased compared with studies published before 2000. Third, by conducting a systematic review rather than a narrative review, we aim to provide a greater level of validity in our findings and minimalize bias by study selection.

Two important considerations need to be noted. First, this reading is organized using the conceptual framework in which biological parameters increase or decrease the likelihood of antisocial development in the context of varying levels of social risk. In order to examine whether this is true for all or for specific biological measures, studies on biosocial interaction within the research areas of peri/prenatal complications and psychophysiological measures are summarized separately. Second, throughout this study the term 'antisocial behavior' is used as a generic term for various behavioral problems, including aggressive, externalizing and delinquent behavior, as well as oppositional defiant disorder (ODD) and conduct disorder (CD). Although we recognize that this led to the inclusion of a variety of studies in this review, it allowed us to address the possibility that different types of ASB are associated with different underlying biosocial mechanisms.

2.2 Method

In accordance with standard methodology for conducting systematic reviews (see Kitchenham, 2004; Petticrew & Roberts, 2006), we identified and processed relevant studies via the multistage procedure described below.

2.2.1 Literature search

First, we used the following ten databases to identify eligible studies published from January 2000 to March 2018: Web of Science, PsychInfo, PubMed, EMBASE, PsychARTICLES, Psychological and Behavioral Sciences Collection, Criminal Justice Abstracts, ERIC, Academic Search Premier, and Social Services Abstracts. The electronic search strategy required articles to report on (1) an area of biological research, (2) a social risk factor, and (3) antisocial behavior. Multiple spellings were used, such as *antisocial*, *anti-social*, and *anti social*. Punctuation marks (*) made sure that search results would include articles using different word endings. For example, by using *delinquen**, we were able to find studies on *delinquent* (behavior) and *delinquency* (see Appendix A for the scripts we used for our search strategy for Web of Science[2]). Additionally, relevant studies were identified via examination of reference lists of included studies.

The online search led to a total of 5589 hits (after removing obvious duplicates). Titles and abstracts were read, and potentially relevant articles were flagged for further examination. All titles and abstracts were independently judged on eligibility by two researchers.

2.2.2 Inclusion and exclusion criteria

The following inclusion criteria were applied to determine eligibility: (1) the interaction between either peri/prenatal complications or psychophysiological functioning and a social risk factor was reported; (2) studies used antisocial behavior as the outcome variable, those focused on attention problems or substance use were excluded; (3) studies used humans as subjects, those focused on animals as subjects were excluded; (4) manuscripts had to report on primary studies including multiple subjects ($N > 1$), whereas reviews and case studies were excluded; and (5) studies were published in English, in international peer reviewed journals. When one publication reported on distinguishable samples or studies (i.e., different number of participants, age cohort or experiment), these samples were treated as independent. When

2 Scripts for the remaining databases are available upon request.

multiple articles were based on the same sample, study findings were clustered to prevent overrepresentation of findings on the same sample.

Studies based on both high-risk and community samples were included and the search was not restricted in terms of participants' age. In addition, no restrictions were placed on study methodology other than the use of interaction analyses. Research in the field of biosocial interaction is still relatively new, and is therefore mostly cross-sectional, and lacks unity in use of covariates and the way findings are reported. Available studies on prenatal testosterone exposure (n = 1), minor physical anomalies (n = 1), blood pressure (n = 2), electrodermal activity (n = 1), and salivary alpha-amylase (n = 3) were not sufficient in number to contribute meaningfully to the qualitative analysis. Therefore, these studies were excluded.

This process resulted in inclusion of 16 studies in the area of peri/prenatal complications and 34 studies in the area of psychophysiology. A flowchart of the literature selection process is presented in Appendix B.

2.2.3 Data extraction

Included studies were processed using a data extraction form designed for this review (see PRISMA Statement for the original checklist; Moher, Liberati, Tetzlaff, Altman, & The PRISMA Group, 2009). After studies were given an ID number, and general information was documented (such as information about the authors, title, and year of publication), information on samples and research instruments was subtracted. Samples were divided into community samples, and low- or high-risk samples. This distinction was based on sampling goals as specified in the original manuscripts. Samples were labelled as 'community samples' when authors had indicated that participants were drawn from the general population (El-Sheikh et al., 2009; Kochanska, Brock, Chen, Aksan, & Anderson, 2015; Murray-Close et al., 2014), or 'birth cohorts' (W. Chen, Lin, & Liu, 2010; Huijbregts, Seguin, Zoccolillo, Boivin, & Tremblay, 2008). In addition, samples were identified as being 'low-risk' when they consisted of (for example) 'college students' (Wagner & Abaied, 2015; Zhang & Gao, 2015). Lastly, the label 'high-risk' was given to samples from 'neighborhoods with lower socioeconomic status' (Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007), and 'urban areas with high prevalence of cocaine use' (Bennett, Marini, Berzenski, Carmody, & Lewis, 2013), as well as to samples consisting of individuals with 'at least one recorded offense' (Gibson & Tibbetts, 2000). Age groups were coded as follows: infancy (0-1) childhood (2-11) adolescence (12-18), and adulthood (>18).

Subsequently, we documented which biological parameter was measured. We distinguished between (1) peri/prenatal, and (2) psychophysiological parameters. Regarding peri/prenatal risk factors, studies targeted (a) prenatal substance exposure, (b) pregnancy and delivery complications, (c) birth weight,

and (d) a combined measure of these peri/prenatal risk factors. Regarding psychophysiological (re)activity, we further distinguished between (a) general ANS functioning, (b) sympathetic (SNS) functioning (i.e., fight or flight system responding to threatening situations), and (c) parasympathetic (PNS) functioning (i.e., the system regulating rest and recovery from stress). General ANS activity was measured with heart rate (HR).[3] Studies on SNS (re)activity reported on skin conductance (SCL),[4] and cardiac preejection period (PEP).[5] PNS (re)activity was operationalized as heart rate variability (HRV),[6] respiratory sinus arrhythmia (RSA),[7] and vagal tone (VT).[8] When measured at rest, these parameters reflect the assessment of autonomic activity in the absence of external stimuli, whereas reactivity is expressed as a change from rest to activity during a laboratory task (Lorber, 2004). Such laboratory tasks encompassed listening to an interadult argument on tape (see Erath, El-Sheikh, Hinnant, & Cummings, 2011), or playing an online game of Cyberball in which the other players only throw the ball at each other (see Sijtsema, Shoulberg, & Murray-Close, 2011).

Concerning social risk factors, we distinguished between (1) familial, (2) peer, and (3) environmental related risk factors. In the area of peri/prenatal risk, studies reported on interactions with familial and environmental factors, as well as with index scores based on a compilation of multiple social risk factors. In the area of psychophysiological (re)activity, studies were focused on interactions with social risk factors related to participant's family, peers, and larger social environments. Biosocial interactions were mostly studied by adding an interaction term to regression models (psychophysiological parameter × social risk). When significant, associations between social risk and ASB were typically tested at high versus low levels of psychophysiological (re)activity.

Behavioral outcomes were coded as one of the following five categories: antisocial behavior, aggressive behavior, externalizing behavior (including 'externalizing problems'), delinquent behavior (including 'arrest rate'), and conduct disorder. We further distinguished between proactive and reactive aggression, relational and physical aggression, as well as overt and covert conduct disorder. We also documented further specification of outcome variables, such as 'early onset', or 'persistent' antisocial behavioral outcomes.

Finally, study results of interaction analysis were collected.

As included studies varied notably in biological, social, and behavioral measures, analytic techniques, use of covariates, and methods of reporting results (for details see Table 2.1 and 2.2), they could not be considered as a

3 HR (SNS + PNS): heart beats per minute.
4 SCL (SNS): reflects fluctuations in sweat gland activity.
5 PEP (SNS): time between when the heart fills with blood and when blood is ejected from the heart.
6 HRV (PNS): variation of intervals between heart beats as a function of respiration.
7 RSA (PNS): reflects heart rate variability in synchrony with respiration.
8 VT (PNS): degree of activity of the vagus nerve resulting in changes in heart rate.

homogeneous group for the purposes of meta-analysis. However, by classifying and evaluating studies according to research question, we were able to clarify associations between biosocial interaction and ASB in a narrative synthesis. In doing so, we attempted to rank studies according to strength of evidence. In accordance with Petticrew and Roberts (2006), we systematically evaluated studies using the following criteria: (1) sample size; (2) sample characteristics (e.g., community vs. low- and high-risk; male vs. female); (3) type of biological parameters; (4) type of social risk; and (5) type of ASB.

2.3 RESULTS

2.3.1 Interactions between peri/prenatal complications and social risk factors

Study characteristics
Results of 16 studies, reported in 19 publications, included between 77 and 715.262 participants (*Mdn* = 513). Studies were conducted in the following countries: United States (*n* = 9), Canada (*n* = 2), England (*n* = 1), Sweden (*n* = 2), Taiwan (*n* = 1), and the Netherlands (*n* = 1). Most studies were longitudinal (*n* = 14), included males and females (*n* = 13), and were conducted among children up to age 12 (*n* = 9). Various studies used high-risk samples (*n* = 7).

Study findings
To examine whether interactions between specific peri/prenatal and social risk factors are associated with ASB, studies were categorized according to peri/prenatal measures into the following categories: (1) prenatal substance exposure (*n* = 10), (2) pregnancy and delivery complications (*n* = 4), (3) birth weight (*n* = 4), and (4) perinatal risk (*n* = 1). Several studies examined risk factors belonging to more than one category, and therefore appear in multiple sections of the review. A summary of study characteristics and significant interaction effects are presented in Table 2.1.

Prenatal substance exposure
Studies on interactions between prenatal substance exposure and social risk show mixed results. On the one hand, six out of eight studies on prenatal smoking and alcohol exposure showed that the relation with ASB is stronger in the context of higher social risk (Gibson & Tibbetts, 2000; Huijbregts et al., 2008; Monuteaux, Blacker, Biederman, Fitzmaurice, & Buka, 2006; Turner, Hartman, & Bishop, 2007; Wakschlag & Hans, 2002; Yumoto, Jacobson, & Jacobson, 2008). For example, children exposed to prenatal smoking or alcohol use were more likely to show ASB when they had an unresponsive mother (Wakschlag & Hans, 2002), absent father (Gibson & Tibbetts, 2000), antisocial parents (Huijbregts et al., 2008), or a low socioeconomic status (Monuteaux

et al., 2006). On the other hand, none of the studies on prenatal drug exposure found interaction effects with social risk (Bagner et al., 2009; Bennett et al., 2013; Veira, Finger, Eiden, & Colder, 2014).

Taking study characteristics into account, interactions between prenatal smoking and alcohol exposure and social risk were found in small (Wakschlag & Hans, 2002) as well as large samples (Huijbregts et al., 2008), and in studies using official report (Gibson & Tibbetts, 2000) as well as self (Monuteaux et al., 2006) and parent (Huijbregts et al., 2008) reports of biological, social, and behavioral measures. However, there is some evidence that the interaction between prenatal smoking and alcohol exposure and social risk is mostly related to ASB in high-risk samples. Although all studies among high-risk samples (n = 4) found support for the relation between biosocial interaction and ASB, inconsistent results were reported in studies among general population and low-risk samples (n = 4). Two studies among low-risk samples found no interaction effect (Buschgens et al., 2009; Wakschlag, Leventhal, Pine, Pickett, & Carter, 2006). In contrast, Huijbregts et al. (2008) found that children from a general population sample showed increased levels of aggressive behavior when they were exposed to prenatal smoking and had antisocial parents. One study (Turner et al., 2007) found a three-way interaction showing that prenatal exposure to nicotine and alcohol was associated with life-course persistent ASB in the context of familial adversity, but only for those individuals living in the most disadvantaged neighborhoods. Last-mentioned finding supports the idea that significant biosocial interactions are mostly found among high-risk samples.

Pregnancy and delivery complications

Two out of four studies on pregnancy and delivery complications found stronger associations with ASB in the context of higher familial adversity (Arseneault, Tremblay, Boulerice, & Saucier, 2002; Hodgins, Kratzer, & McNeil, 2001). For example, the relation between pregnancy and delivery complications and increased aggressive and violent delinquent behavior was stronger for those exposed to overall higher family adversity (Arseneault et al., 2002). In contrast, one study did not find significant interaction effects between pregnancy complications and inadequate parenting or socioeconomic status (Hodgins, Kratzer, & McNeil, 2002). Lastly, Buschgens et al. (2009) found that the relation between pregnancy and delivery complications and aggressive behavior was stronger when familial risk was lower. The authors suggested that strong environmental risk factors might have overshadowed the contribution of biological risk to ASB (Buschgens et al., 2009). However, it should be noted that this study is the only cross-sectional study in this category, and relations between interaction effects and outcome should perhaps be interpreted with a little more caution.

Birth weight

Two out of four studies on birth weight showed that the relation between low birth weight and ASB is stronger in the context of higher familial adversity (W. Chen et al., 2010; Piquero & Lawton, 2002). Specifically, children with low birth weight had longer delinquent careers when they were exposed to higher levels of familial adversity (Piquero & Lawton, 2002). Also, children with lower birth weight showed increased levels of delinquent behavior when their mother was either at the lower (below 18 years old) or higher end (between 40 and 49 years old) of maternal age at childbirth (Chen et al., 2010). In contrast, studies on interactions between birth weight and overall familial adversity (Buschgens et al., 2009) and social class (Kelly, Nazroo, McMunn, Borehamb, & Marmota, 2001) did not find significant interaction effects.

Studies that did and did not find support for biosocial interaction effects differed in two important ways. First, studies reporting significant biosocial interactions focused on delinquent behavior as outcome variable (W. Chen et al., 2010; Piquero & Lawton, 2002), whereas studies reporting insignificant results focused on conduct disorder (Kelly et al., 2001), and aggressive behavior (Buschgens et al., 2009). Thus, differences in behavioral outcomes may have influenced the significance of interaction effects. Second, both studies supporting biosocial interaction used stronger research designs, as they both used official reports to measure birth weight as opposed to parental report, and were based on longitudinal research as opposed to cross-sectional research.

Perinatal risk

Only one study used a combined measure of pregnancy and delivery complications and birth weight (i.e., perinatal risk; Beck & Shaw, 2005). In this study, the relation between perinatal risk and delinquent behavior was stronger for children exposed to higher levels of overall familial adversity. However, no biosocial interaction was found between perinatal risk and family adversity in relation to externalizing behavior. Furthermore, risk of showing delinquent behavior among participants exposed to perinatal risk was not elevated when parents had a rejecting parenting style (Beck & Shaw, 2005).

Summary

Overall, studies varied in the extent to which they provided support for associations between biosocial interaction and ASB. Studies that found significant interaction effects (n = 9) typically showed that associations between peri/prenatal risk and ASB were stronger in the context of higher social adversity (n = 8). Studies on prenatal smoking, pregnancy and delivery complications, and studies conducted among high-risk samples found the most consistent support for biosocial interaction. Further, studies distinguishing between subtypes of ASB suggested that interactions between peri/prenatal complications and social risk are particularly associated with more severe and violent types of ASB.

Table 2.1: Overview of studies on interactions between peri/prenatal and social risk factors

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	Peri/Prenatal[6,7] (% exposed)
Associations of the interaction between prenatal substance exposure and social risk and ASB						
1	Wakschlag and Hans (2002)	N = 77 B/G United States	CH	HR	L	PS^{P} (71%)
2	Gibson and Tibbetts (2000)	N = 215 B/G United States NCPP	CH-AL	HR	L	PS^{P} (51%)
3	Huijbregts et al. (2008)	N = 1745 B/G Canada	IN-CH	GP	L	PS^{P} (25.2%)
4	Wakschlag et al. (2006)	N = 93 B/G United States FHDP	IN	LR	L	PS^{O+P} (50%)
2	Monuteaux et al. (2006)	N = 682 B/G United States NCPP	IN-AL	HR	L	PS^{P}
5	Buschgens et al. (2009)	N = 2230 B/G Netherlands TRIALS	CH	LR	CS	PS^{P} (30.5%)
6	Turner et al. (2007)	N = 513 B(↑)/G United States National Longitudinal Survey of Youth	IN-AL	LR	L	$PS+A^{P}$
7	Yumoto et al. (2008)	N = 337 B/G, United States	CH	HR	L	PA^{P} (67,4%)
8	Bennett, Bendersky, and Lewis (2002)	N = 223 B/G United States (See Bennet et al., 2013)	IN-CH	HR	L	PCE^{P} (38; 41%)
8	Bennett et al. (2013)	N = 179 B/G United States (See Bennett et al., 2002)	IN-CH	HR	L	PCE^{O} (41%)
9	Veira et al. (2014)	N = 216 B/G United States	IN-CH	HR	L	PCE^{O+P} (54%)
10	Bagner et al. (2009)	N = 607 B/G United States MLS	IN-CH	HR	L	$PDE^{O\ or\ P}$ (36%)

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Maternal responsiveness[OB]	CD[S+P]	B	Yes	PS x maternal responsiveness → CD for *boys* ↑PS → ↑CD, for *boys* with unresponsive mothers For *girls*, PS was not associated with CD
Absence of father or husband[P]	Early onset DB[O]	B	NR	PS x absence father/husband → early onset DB ↑PS → ↑early onset DB, stronger for absent father or husband
Antisocial parents[P]	PHY-AGB[P]	B	NR	PS x parental history of ASB → PHY-AGB ↑PS → ↑PHY-AGB, for ↑antisocial parents
Family income[P]		B		PS x family income → PHY-AGB ↑PS → ↑AGB, only for ↓family income
Cumulative risk (index; mostly social status)[P]	EXB[P+OB]	-	NR	n.s.
Socioeconomic status[S]	(c)overt CD[S]	B	NR	PS x SES → overt CD ↑PS → ↑overt CD, only for ↓SES No interaction effect for covert CD
Familial risk (index; mostly parental characteristics)[P]	AGB[P] DB[P+T]	-	NR	n.s.
Family adversity (index; mostly social status) Neighborhood disadvantage[P]	Violence[S] LCP[S] ASB (25%)	B	NR	PS+A x family adversity x neighborhood disadvantage → LCP ASB ↑PS+A x ↑family adversity → ↑LCP, only for ↑neighborhood disadvantage
Number of social risk factors[P]	AGB[T] DB[T]	B	NR	PA x cumulative risk → DB[A] Cumulative risk → ↑DB, only in exposed group
Environmental risk (index; mostly social status)[P] Maternal depression[P] Maternal harsh discipline[P] Maternal verbal IQ[P]	EXB[P]	-	NR	n.s.
Environmental risk (index; mostly social status)[P]	EXB[P+T+OB] DB[S]	-	NR	n.s.
Maternal warmth/ sensitivity[OB], Maternal harshness[OB]	EXB[P]	-	NR	n.s.
Parenting stress[P]	EXB[P]	-	NR	n.s.

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	Peri/Prenatal[6,7] (% exposed)
Associations of the interaction between pregnancy and delivery complications and social risk and ASB						
5	Buschgens et al. (2009)	N = 2230 B/G Netherlands TRIALS	CH	LR	CS	PDC^{P} (10%)
11	Arseneault et al. (2002)	N = 849 B Canada	AL	LR	L	PDC^{O}
12	Hodgins et al. (2001)	N = 13852 B/G Sweden (Sample without mental disorder)	AH	GP	L	PC^{O}
13	Hodgins et al. (2002)	N = 161 B/G Sweden (Sample with mental disorder)	AH	HR	L	PC^{O}
Associations of the interaction between birth weight and social risk and ASB						
5	Buschgens et al. (2009)	N = 2230 B/G Netherlands TRIALS	CH	LR	CS	BW^{P} (3.6%)
2	Piquero and Lawton (2002)	N = 1758 B/G United States NCPP	IN-AL	HR	L	BW^{O}
14	W. Chen et al. (2010)	N = 715262 B Taiwan	CH-AH	GP	L	BW^{O}
15	Kelly et al. (2001)	N = 5181 B/G England	CH-AL	GP	CS	BW^{P} (8,9%)
Associations of the interaction between perinatal risk and social risk and ASB						
16	Beck and Shaw (2005)	N = 250 B United States Pitt Mother and Child Project	IN-CH	HR	L	PERIR^{O}

Note.
[1]ID = Study ID;
[2]Sample: B = Boys; G = Girls; NCPP = National Collaborative Perinatal Project; FHDP = Family Health and Development Project; MILS = Maternal Lifestye Study; TRIALS = Netherlands Tracking Adolescents' Individual Lives Survey;
[3]Age: IN = Infancy (0-1); CH = Childhood (2-12); AL = Adolescence (13-18); AH = Adulthood (>18);
[4]Risk: LR = Low-Risk sample; HR = High-Risk sample; GP = General Population sample;
[5]CS/L: L = Longitudinal; CS = Cross-sectional;
[6]Peri/Prenatal Risk: PS = Prenatal Smoking; PS+A = Prenatal Smoking and Alcohol use; PA = Prenatal Alcohol Exposure; PCE = Prenatal Cocaine Exposure; PDE = Prenatal Drug exposure; PDC = Pregnancy and Delivery Complications; PC = Pregnancy Complications; BW = Birth Weight; PERIR = Perinatal risk (i.e., birth weight, eclampsia, bleeding at beginning of delivery, premature birth);
[7]Source: O = Official Records; S = Self Report; P = Parent report; T = Teacher Report, OB = Observational Data;
[8]Behavior: EXB = Externalizing Behavior; CD = Conduct Disorder; DB = Delinquent Behavior; LCP = Life-Course Persistent ASB; (PHY) AGB = (Physical) Aggressive Behavior;
[9]Theory: A = social push hypotheses; B = diathesis stress; C = differential susceptibility; – = no support for biosocial theory; ? = support for theory unknown;
[10]Gender Diff = Gender Differences in interaction effects (i.e., whether the interaction effect was gender specific); n/a = not applicable (i.e., because of sample characteristics); NR = not reported;
[11]Interaction Effects: n.s. = non-significant.

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Familial risk^{P} (index; mostly parental characteristics)	AGB^{P}, DB^{P+T}	A	NR	PDC x familial risk → AGB ↑PDC →↑AGB, stronger for ↓familial risk
Family adversity^{P} (index; mostly social status)	AGB^{T} (non) violent DB^{S}	B	n/a	PDC x family adversity → AGB, violent DB ↑PDC →↑AGB, (non) violent DB, stronger for ↑family adversity
Inadequate parenting^{O}	(Violent + early onset) DB^{0}	B	Yes	PC x inadequate parenting → (violent) DB for *men* ↑PC → ↑(violent) DB among *men*, stronger for ↑inadequate parenting Relation between PC and DB not stronger for *women* exposed to PC
Inadequate parenting^{O} Socioeconomic status^{O}	DB^{O}	-	NR	n.s.
Familial risk^{P} (index; mostly parental characteristics)	AGB^{P} DB^{P+T}	-	NR	n.s.
Family adversity^{P} (index; mostly social status)	DB^{S} (LCP) DB^{S}	B	NR	BW x family adversity → LCP DB ↓BW →↑LCP DB, stronger for ↑family adversity
Parents (not) married Mother's education Maternal age at childbirth	(non) violent DB^{O}	B	n/a	BW x maternal age → violent DB ↓BW →↑violent DB, only for low (<18) and high (40-49) maternal age at childbirth
Social class	CD^{P}	-	NR	n.s.
Family adversity (index; mostly social status)^{P} Rejecting parenting^{OB}	EXB^{P} DB^{S}	B	n/a	PERIR x family adversity → DB ↑PERIR → ↑DB, stronger for ↑family adversity

2.3.2 Interactions between psychophysiological and social risk factors

Study characteristics. Results of 34 studies, reported in 47 articles, included between 23 and 2230 participants (*Mdn* = 150). Studies were conducted in the United States (*n* = 24), the Netherlands (*n* = 3), Italy (*n* = 1), and China (*n* = 1). Studies were mostly cross-sectional (*n* = 24), included males and females (*n* = 25), covered childhood (*n* = 19), and used general population or low-risk samples (*n* = 23).

Study findings
To synthesize study findings, studies were divided into the following categories: (1) general ANS (re)activity (*n* = 8), (2) SNS (re)activity (*n* = 19), and (3) PNS (re)activity (*n* = 25). When studies examined more than one research question, they appear in multiple sections of the review. A summary of main findings is presented in Table 2.2, showing interactions associated with ASB significant at the $p < 0.05$ level.

General ANS functioning

(a) Rest
Four out of five studies on general baseline ANS found support for an association between biosocial interactions and ASB. These studies showed that associations between low resting heart rate (RHR) and increased levels of ASB were stronger in the context of overall higher social adversity (Raine, Fung, Portnoy, Choy, & Spring, 2014), higher maternal psychiatric problems (Dierckx et al., 2011), and maintaining friendships with bullies (Sijtsema, Veenstra, et al., 2013). One study found that higher RHR protected subjects against developing proactive aggression in the context of community violence victimization (Scarpa, Tanaka, & Haden, 2008). In contrast, interactions between RHR and fathers' criminal history were not associated with delinquent behavior (van de Weijer, de Jong, Bijleveld, Blokland, & Raine, 2017).

Concerning different subtypes of ASB (see Raine et al., 2014; Scarpa et al., 2008), studies showed inconsistent results. Although Raine et al. (2014) found that biosocial interactions were associated with reactive and not proactive aggression, Scarpa et al. (2008) found associations with proactive and not reactive aggression. Although both studies are cross-sectional, based on children and adolescent, and high-risk samples, they differ in sample size. Raine et al. (2014) based their study on 334 participants, whereas Scarpa et al. (2008) only included 40 participants. As last-mentioned study is based on a relatively small sample, results reported by Raine et al. (2014) are considered to be of more value when drawing conclusion on interactions between RHR and social risk.

(b) Reactivity

Studies on interactions between heart rate reactivity (HRR) and social risk (n = 4) showed mixed results. Although two studies found interaction effects between HRR and social risk (Murray-Close & Rellini, 2012; Sijtsema, Nederhof, et al., 2013), two other studies did not (Murray-Close, 2011; Shoulberg, Sijtsema, & Murray-Close, 2011; Sijtsema et al., 2011). It is difficult to explain these mixed findings based on study characteristics, as differences in type of social risk and type of ASB are clustered within studies. When considering differences in social risk factors, interaction effects were found in studies on HRR and family and childhood related risk factors (Murray-Close & Rellini, 2012; Sijtsema, Nederhof, et al., 2013), and not in studies on peer-related risk factors (Murray-Close, 2011; Shoulberg et al., 2011; Sijtsema et al., 2011). For example, family cohesion was negatively associated with aggressive behavior for boys with low HRR (Sijtsema, Nederhof, et al., 2013). However, no interaction was found between HRR and peer rejection (Sijtsema et al., 2011). When considering differences in types of ASB, significant interaction effects were specifically found for proactive relational aggressive behavior. For example, Murray-Close and Rellini (2012) found that low HRR was associated with high proactive relational aggressive behavior when their female participants were sexually victimized during childhood. In contrast, studies on relational and physical aggressive behavior did not find support for interactions between HRR and social risk (Murray-Close, 2011; Shoulberg et al., 2011; Sijtsema et al., 2011).

SNS functioning

(a) Rest

Four out of six studies on interactions between baseline SNS and social risk did not find significant interaction effects. SNS activity at rest did not interact with marital conflict (El-Sheikh et al., 2009), parental antisocial personality disorders, maternal melancholia (Shannon et al., 2007), or maltreatment victimization (Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010). Two studies showed that lower baseline SNS was associated with increased levels of ASB in the context of higher social risk, such as higher maternal power assertion (Kochanska et al., 2015), and lower neighborhood cohesion (Bubier, Drabick, & Breiner, 2009). Higher SNS baseline combined with higher levels of harsh parenting was also associated with increased levels of externalizing behavior (Bubier et al., 2009). On the other hand, higher levels of social risk were also found to be associated with decreased levels of ASB for individuals with higher SNS baseline functioning (Bubier et al., 2009). Lastly, when children with lower SNS baseline functioning had positive relationships with their fathers, they showed lower levels of ASB than peers with higher SNS baseline functioning (Kochanska et al., 2015).

Table 2.2: Overview of studies on interactions between psychophysiology and social risk factors

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
Associations of the interaction between general ANS (re)activity and social risk and ASB						
1	Raine et al. (2014)	N = 334 B/G China	CH/AD	GP	CS	RHR
2	Dierckx et al. (2011)	N = 514 B/G Netherlands Generation R Study	IN	GP	CS	RHR
3	van de Weijer et al. (2017)	N = 794 B Transfive Netherlands	AH	HR	L	RHR
4	Scarpa et al. (2008)	N = 40 B/G United States	CH/AL	GP	CS	RHR
5	Sijtsema, Veenstra, et al. (2013)	N = 2230 B/G Netherlands TRAILS	CH	HR	L	RHR
5	Sijtsema, Nederhof, et al. (2013)	N = 679 B/G Netherlands TRAILS	AL	HR	CS	HRR
6	Murray-Close and Rellini (2012)	N = 83 G United States	AL/AH	GP	CS	HRR
7	Murray-Close (2011)	N = 131 B United States	AH	LR	CS	HRR
8	Sijtsema et al. (2011)	N = 119 G Netherlands Summer Camp Study	CH	LR	CS	HRR
8	Shoulberg et al. (2011)	N = 126 G Netherlands Summer Camp Study	CH	LR	CS	HRR
Associations of the interaction between SNS (re)activity and social risk and ASB						
9	Kochanska et al. (2015)	N = 74 B/G United States	IN-CH	GP	L	RSCL
10	Shannon et al. (2007)	N = 180 B/G United States	CH	HR	CS	RPEP

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Social adversity (index)[P]	AGB[P] PRO-AGB[P] RE-AGB[P]	B	NR	HR x social adversity → AGB ↓HR → ↑AGB at ↑social adversity HR x social adversity → RE-AGB ↓HR → ↑RE-AGB at ↑social adversity
Maternal psychiatric symptoms[P]	AGB[P]	B	NR	HR x maternal psychiatric symptoms → AGB ↓HR → ↑AGB at ↑maternal psychiatric problems
Fathers' criminal history[O]	DB[O]	-	n/a	n.s.
Heard about community violence (HCV)[S] Witnessed violence victimization (WCV)[S] Community violence victimization (CVIC)[S]	PRO-AGB[P] RE-AGB[P]	B C	NR	HR x CVIC → PRO-AGB ↑CVIC → ↑PRO-AGB at ↓HR ↑CVIC → ↓PRO-AGB at ↑HR
Affiliation with bullies[PEER]	ASB[S]	B	No	HR x affiliation with bullies → ASB ↓HR→ ↑ASB, only for ↑affiliation with bullies
Family cohesion[P]	ASB[P]	B	Yes	HRR x family cohesion → ASB for boys ↓Cohesion → ↑ASB, only for *boys* at ↓HRR ↓Cohesion → ↑ASB for *girls*, independent of HRR
Childhood victimization of sexual abuse[S]	RE-REL-AGB[S] PRO-REL-AGB[S]	B	n/a	HRR x sexual VIC → PRO-REL-AGB ↓HRR → ↑ PRO-REL-AGB at sexual VIC
Relational victimization[S]	REL-AGB[S]	-	n/a	n.s.
Peer rejection[PEER]	REL-AGB[T] PHY-AGB[T]	-	n/a	n.s.
Peer popularity[PEER]	REL-AGB[PEER]	-	n/a	n.s.
Security with parents[S] Power assertion[OB] Mutually responsive orientation[OB]	EXB[P]	B C	NR	SCL x maternal power assertion → EXB ↑Maternal power assertion → ↑EXB only at ↓SCL SCL x father-child MRO → EXB Positive father-child MRO → ↓EXB at ↓SCL Absent positive father-child MRO → ↑EXB at ↓SCL
Parental ASPD[P] Maternal melancholia[P]	CD[P]	-	NR	n.s.

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
11	El-Sheikh et al. (2009)	N = 176 B/G N = 150 B/G N = 251 B/G United States Bioregulatory Effects Project	CH	GP	CS	RSCL SCLR
11	El-Sheikh et al. (2011)	N = 251 B/G United States Bioregulatory Effects Project	CH	GP	L	RSCL SCLR
12	Diamond et al. (2012)	N = 110 B/G United States			CS	SCLR
13	Gordis et al. (2010)	N = 362 B/G United States	CH/AL	HR	CS	RSCL SCLR
14	Bubier et al. (2009)	N = 57 B/G United States	CH	HR	CS	RPEP PEPR
11	Erath et al. (2009)	N = 251 B/G United States Bioregulatory Effects Project	CH	GP	CS	SCLR
11	Erath et al. (2011)	N = 251 B/G United States Bioregulatory Effects Project	CH	GP	L	SCLR
15	El-Sheikh (2005b)	N = 180 B/G United States (see Cummings et al., 2007; El-Sheikh, 2007)	CH	GP	CS	SCLR
15	El-Sheikh et al. (2007)	N = 157 B/G United States (See Cummings et al., 2007; El-Sheikh, 2005)	CH-AL	GP	L	SCLR
16	Obradović et al. (2011)	N = 260 B/G United States	CH	LR	CS	PEPR
17	Wagner and Abaied (2016)	N = 180 mostly G United States (See Wagner & Abaied, 2015)	AH	LR	CS	SCLR
15	Cummings et al. (2007)	N = 157 B/G United States (See El-Sheikh, 2005a; El-Sheikh et al., 2007)	CH	GP	L	SCLR

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Marital conflict^{S+P}	EXB^{P+T}	?	NR	SCLR x marital conflict → EXB Direction not reported
Marital conflict^{P}	DB^{P}	-	NR	n.s.
Family structure: one or two parent household	EXB^{P}	B	Yes	SCLR x family structure → EXB Single mother → ↑EXB for *boys* at ↑SCLR Single mother → ↑EXB for *girls* at ↓SCLR
Victimization: maltreatment^{O}	AGB^{P}	-	No	n.s.
Harsh parenting^{P} Neighborhood cohesion^{S}	EXB^{P}	B C A B	NR	PEP x neighborhood cohesion → EXB ↓Neighborhood cohesion → ↑EXB at ↓PEP ↓Neighborhood cohesion → ↓EXB at ↑PEP ↑Neighborhood cohesion → ↑EXB at ↑PEP PEP x harsh parenting → EXB ↑Harsh parenting → ↑EXB at ↑PEP
Harsh parenting^{P+S}	EXB^{P}	B	No	SCLR x harsh parenting → EXB Harsh parenting → ↑EXB stronger for children with ↓SCLR
Harsh parenting^{P}	EXB^{P}	B	Yes	SCLR x harsh parenting → EXB Harsh parenting → ↑EXB at ↑+↓SCLR for *girls* Harsh parenting → ↑EXB at ↑+↓(stronger) SCLR for *boys*
Marital conflict^{P}	EXB^{P}	B	Yes	SCLR x marital conflict → EXB for *girls* ↑Marital conflict → ↑EXB for *girls* at ↑SCLR No interaction effect for *boys*
Marital conflict^{P}	EXB^{P}	B	Yes	SCLR x marital conflict → EXB Marital conflict → ↑EXB for *girls* at ↑(stronger)+↓SCLR ↑Marital conflict → ↑EXB for *boys* at ↓SCLR
Marital conflict^{P}	EXB^{S+P+T}	-	NR	n.s.
Parental psychological control ^{S}	PRO-REL-AGB^{S} RE-REL-AGB^{S}	B B	NR	SCLR x parental control → RE-REL-AGB ↑Parental control → ↑RE-REL-AGB, only at ↑SCLR SCLR x parental control → PRO-REL-AGB ↑Parental control → ↑PRO-REL-AGB, only at ↓SCLR
Parental depressive symptoms^{P}	EXB^{P}	B	No	SCLR x paternal depressive symptoms → EXB ↑Paternal depression → ↑EXB at ↑SCLR

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
18	Buodo et al. (2013)	N = 61 B Italy	CH	LR	CS	SCLR
19	McQuade and Breaux (2017)	N = 61 B/G United States	CH	HR	L	SCLR
20	Stanger, Abaied, Wagner, and Sanders (2018)	N = 64 B/G United States	CH	GP	L	SCLR
5	Sijtsema et al. (2015)	N = 2230 B/G Netherlands TRAILS	CH-AL	HR	L	PEPR
21	Waters et al. (2016)	N = 99 B/G United States	CH	HR	CS	PEPR
22	Hinnant et al. (2016)	N = 199-53 B/G United States	AL	GP	CS	SCLR PEPR
8	Shoulberg et al. (2011)	N = 126 G Netherlands Summer Camp Study	CH	LR	CS	SCLR
8	Sijtsema et al. (2011)	N = 119 G Netherlands Summer Camp Study	CH	LR	CS	SCLR
17	Wagner and Abaied (2015)	N = 168 mostly G United States (See Wagner & Abaied, 2016)	AH	LR	CS	SCLR
7	Murray-Close (2011)	N = 131 B United States	AH	LR	CS	SCLR
23	Murray-Close et al. (2014)	N = 196 B/G United States	CH	GP	CS	SCLR
24	Gregson et al. (2014)	N = 123 B/G United States	AL	GP	CS	SCLR
Associations of the interaction between PNS (re)activity and social risk and ASB						
2	Dierckx et al. (2011)	N = 514 B/G Netherlands Generation R Study	IN	GP	CS	RHRV

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Parenting stress^{P}	EXB^{S+P}		n/a	SCLR x parenting stress → EXB
		B		↑Parenting stress → ↑EXB only at ↓SCLR
Parental (non-)supportive emotion socialization^{P}	AGB^{P+T}		NR	SCLR x non-supportive emotional socialization → AGB
		B		↑Non-support → ↑AGB, only at ↓SCLR
Parent socialization of coping^{OB}: (Dis-)engagement control suggestions (CE/DIS)	EXB^{P}	C	NR	SCLR x DIS → EXB ↑DIS → ↓EXB, only for ↑SCLR
Familial adversity^{S+P} (index; mostly parental characteristics)	ASB^{S}	B	Yes	PEPR x family adversity → ASB for *boys* ↑Family adversity → ↑ASB, only for *boys* with ↓PEPR Family adversity → ASB for *girls*, independent of PEPR
Maternal depression^{P} Overcrowded housing	EXB^{P}	C	NR	PEPR x maternal depression → EXB ↑Maternal depression → ↓EXB, at ↑PEPR
Permissive parenting^{S} Affiliation deviant peers^{S}	EXB^{S}	B	NR	PEPR x deviant peers → EXB ↑Deviant peers → ↑EXB at ↑+↓(stronger)PEPR
Peer popularity^{PEER}	REL-AGB^{PEER}	-	n/a	n.s.
Peer rejection^{PEER}	REL-AGB^{T} PHY-AGB^{T}	-	n/a	n.s.
Relational victimization^{S}	PRO-REL-AGB^{S} RE-REL-AGB^{S}	-	NR	n.s.
Relational victimization^{S}	REL-AGB^{S}	?	n/a	SCLR x REL-VIC → REL-AGB Follow-up n.s.
Relational victimization^{T}	REL-AGB^{T}		No	SCLR x PHY-VIC → REL-AGB for both genders
Physical victimization^{T}	PHY-AGB^{T}	A		↓SCLR → ↑REL-AGB, at ↓PHY-VIC
		B		↑SCLR → ↑REL-AGB, at ↑PHY-VIC
			Yes	SCLR x PHY-VIC → PHY-AGB, only for *girls*
		A		↓SCLR → ↑PHY-AGB, at ↓PHY-VIC
		B		↑SCLR → ↑PHY-AGB, at ↑PHY-VIC
Peer victimization^{S}	EXB^{P+T} AGB^{T}	B	NR	SCLR x peer victimization → EXB ↑Peer victimization → ↑EXB, at ↓SCLR
Maternal psychiatric symptoms^{P}	AGB^{P}	B	NR	HRV x maternal psychiatric symptoms → AGB ↑HRV → ↑AGB at ↑maternal psychiatric problems ↑HRV → ↓AGB at ↓maternal psychiatric problems

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
4	Scarpa et al. (2008)	N = 40 B/G United States	CH/AL	GP	CS	RHRV
25	Hastings and De (2008)	N = 105 B/G	CH	GP	CS	RRSA
26	Davis et al. (2017)	N = 94 B/G United States	CH	GP	CS	RRSA
10	Shannon et al. (2007)	N = 180 B/G United States	CH	HR	CS	RPEP RRSA
27	El-Sheikh (2005a)	N = 216 B/G (See El-Sheikh, 2001)	CH	GP	L	RVT
11	El-Sheikh et al. (2009)	N = 176 B/G N = 150 B/G N = 251 B/G United States Bioregulatory Effects Project	CH	GP	CS	RRSA RSAR
11	El-Sheikh et al. (2011)	N = 251 B/G United States Bioregulatory Effects Project	CH	GP	L	RRSA RSAR
11	El-Sheikh and Hinnant (2011)	N = 222 B/G United States Bioregulatory Effects Project	CH	GP	L	RRSA RSAR
28	El-Sheikh, Harger, and Whitson (2001)	N = 75 B/G	CH	LR	CS	RVT VTR
29	Whitson and El-Sheikh (2003)	N = 64 B/G	CH	LR	CS	RVT RSAR VTR
11	Hinnant et al. (2015)	N = 251 B/G United States Bioregulatory Effects Project	CH-AL	GP	L	RRSA RSAR
14	Bubier et al. (2009)	N = 57 B/G United States	CH	HR	CS	RRSA RSAR

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Heard about community violence (HCV)[S] Witnessed violence victimization (WCV)[S] Community violence victimization (CVIC)[S]	PRO-AGB[P] RE-AGB[P]	 B C	NR	HRV x witnessed CV → RE-AGB ↑witnessed CV → ↑RE-AGB at ↑HRV ↑witnessed CV → ↓RE-AGB at ↓HRV
Response to children's emotions[P]	EXB[P]	 C C	NR	RSA x father override of anger → EXB Fathers' override → ↓EP at ↓RSA RSA x mothers neglect of fear/sadness → EXB Maternal neglect → ↓EXB at ↓RSA
Parenting Stress[P]	EXB[P]	-	NR	n.s.
Parental ASPD[P] Maternal melancholia[P]	CD[P]	 B	NR	RSA x paternal ASPD → CD ↑Paternal ASPD → ↑CD only at ↑RSA
Parental problem drinking[P]	EXB[P]	 B	NR	VT x parental problem drinking → EXB Parental problem drinking → ↑EXB at ↓VT
Marital conflict[S+P]	EXB[P +T]	?	NR	RSA x marital conflict → EXB RSAR x marital conflict → EXB Direction not reported
Marital conflict[S+P]	DB[P]	 B	Yes	RSA x martial conflict → DB for *boys* ↑Marital conflict → ↑DB, for *boys* with ↓RSA No interaction effect found for *girls* RSAR x martial conflict → DB for *boys* ↑Marital conflict → ↑DB, for *boys* with ↓RSAR No interaction effect found for *girls*
Marital conflict[S+P]	EXB[P]	-	NR	n.s.
Marital conflict[S+P]	EXB[P]	 B C	Yes	RVT x marital conflict → EXB ↑Marital conflict → ↑EXB only at ↓VT VTR x marital conflict → EXB for *boys* ↑Marital conflict → ↓EXB for *boys* at ↑VTR No interaction between VTR and marital conflict for *girls*
Marital conflict[S+P] Mother-child conflict[S+P]	EXB[P]	 B	Yes	RSAR x MC-conflict → EXB VTR x MC-conflict → EXB ↑Marital conflict → ↑EXB for *girls* at ↑ANS reactivity
Harsh parenting[S]	DB[P]	 B C C	Yes	RSA x harsh parenting → DB ↑Harsh parenting → ↑DB for *boys* with ↓RSA ↑Harsh parenting → ↓DB for *boys* with ↑RSA ↑Harsh parenting → ↓DB for *girls* at ↓RSA
Harsh parenting[S] Neighborhood cohesion[S]	EXB[P]	-	NR	n.s.

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
13	Gordis et al. (2010)	N = 362 B/G United States	CH/AL	HR	CS	RRSA RSAR
30	Zhang and Gao (2015)	N = 84 B/(↑)G United States	AH	LR	CS	RRSA RSAR
31	Zhang et al. (2017)	N = 253 B/G United States	CH	GP	L	RRSA RSAR
32	Eisenberg et al. (2012)	N = 213 B/G United Status	IN/CH	LR	CS	RRSA RSAR
33	Calkins, Blandon, Williford, and Keane (2007)	N = 441 B/G		GP	CS	RRSA RSAR
34	Dyer et al. (2016)	N = 262 B/G United States Flourishing Families Project	AL	LR	CS	RRSA RSAR
12	Diamond et al. (2012)	N = 110 B/G United States	CH		CS	RSAR
19	McQuade and Breaux (2017)	N = 23 B/G United States	CH	HR	L	RSAR
27	El-Sheikh (2001)	N = 216 B/G (See El-Sheikh, 2005b)	CH	GP	CS	VTR

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Victimization: maltreatment[O]	AGB[P]	B	Yes	RSA x maltreatment → ABG for *boys* Maltreatment → ↑AGB for *boys* with ↓RSA No interaction effect between RSA and maltreatment for *girls*
Social adversity[S] (index; mostly social status)	PRO-AGB[S] RE-AGB[S]	B A A	NR	RSA x social adversity → RE-AGB ↑RSA → ↑RE-AGB only at ↑social adversity RSAR x social adversity → RE-AGB ↑RSAR → ↑RE-AGB only at ↓social adversity RSAR x social adversity → PRO-AGB ↓RSAR → ↑PRO-AGB at ↓social adversity
Social adversity[P] (index: mostly parental characteristics)	EXB[P]	B	Yes	RSA x social adversity → EXB ↓RSA → ↑EXB, only for *boys* at ↑social adversity No interaction between RSA and social adversity for *girls*
Familial adversity[P] (index: mostly social status)	AGB[P]	B	Yes	RSA x familial adversity → AGB for *girls* ↓Environmental quality → ↑AGB for *girls* at ↑RSA No relation between environmental quality and AGB for *girls* with ↓RSA No interaction effect between RSA and familial adversity for *boys*
Familial adversity (index; mostly social status)	EXB[P]	-	NR	n.s.
Parenting style[S]	EXB[S]	B A C A+B	Yes	RSA x authoritative parenting → EXB for *boys* ↓Authoritative parenting → ↑EXB for *boys* at ↓RSA RSAR x authoritative parenting → EXB for *girls* ↑Authoritative parenting → ↑EXB for *girls* at ↑RSAR ↓Authoritative parenting → ↓EXB for *girls* at ↓RSAR RSAR x authoritarian parenting → EXB for *girls* ↑RSAR → ↑EXB for *girls* at ↑+↓authoritarian parenting
Family structure: one or two parent household	EXB[P]	B	Yes	RSAR x family structure → EXB for *girls* Single mother → ↑EXB only for *girls* at ↓RSAR No interaction between single mother households and RSAR for *boys*
Parental (non-)supportive emotion socialization[P]	AGB[P+T]	B	NR	RSAR x non-supportive emotional socialization → AGB ↑Non-support → ↑AGB, only at ↓RSAR
Parental problem drinking[P]	EXB[P]	B C	Yes	VTR x parental problem drinking → EXB ↑Parental problem drinking → ↑EXB, only at ↓VTR ↑Parental problem drinking → ↓EXB at ↑VTR, especially for *girls*

ID[1]	Publication	Sample[2]	Age[3]	Risk[4]	CS/L[5]	ANS[6]
6	Murray-Close and Rellini (2012)	N = 83 G United States	AL/AH	GP	CS	RSAR
8	Shoulberg et al. (2011)	N = 126 G Netherlands Summer Camp Study	CH	LR	CS	RSAR
8	Sijtsema et al. (2011)	N = 119 G Netherlands Summer Camp Study	CH	LR	CS	RSAR
7	Murray-Close (2011)	N = 131 B United States	AH	LR	CS	RSAR
17	Wagner and Abaied (2015)	N = 168 mostly G United States (See Wagner & Abaied, 2016)	AH	LR	CS	RSAR
5	Sijtsema et al. (2015)	N = 2230 B/G Netherlands TRAILS	CH-AL	HR	L	RSAR
16	Obradović et al. (2011)	N = 260 B/G United States (See Obradovic et al., 2010)	CH	LR	CS	RSAR
16	Obradović et al. (2010)	N = 338 B/G United States (See Obradovic et al., 2011)	CH	LR	L	RSAR
21	Waters et al. (2016)	N = 99 B/G United States	CH	HR	CS	RSAR

Note.
[1]ID = Study ID;
[2]Sample: B = Boys; G = Girls; Generation R Study = Focus Cohort of the Generation R Study; TRIALS = Tracking Adolescents' Individual Lives' Survey; Summer Camp Study = Private Residential Summer Camp for Girls; Bioregulatory Effects Project = Family Stress and Youth Development: Bioregulatory Effects Project;
[3]Age: IN = Infancy (0-1); CH = Childhood (2-12); AL = Adolescence (13-18); AH = Adulthood (>18);
[4]Risk: LR = Low-Risk sample; HR = High-Risk sample; GP = General Population sample;
[5]CS/L: L = Longitudinal; CS = Cross-sectional;
[6]ANS: RHR = Resting Heart Rate; HRR = Heart Rate Reactivity; RSCL = Resting Skin Conductance; RPEP = Resting Cardiac Preejection Period; SCLR = Skin Conductance Reactivity; PEPR = Cardiac Preejection Period Reactivity; RHRV = Resting Heart Rate Variability; RRSA = Resting Respiratory Sinus Arrhythmia; RVT = Resting Vagal Tone; RSAR = Respiratory Arrhythmia Reactivity; VTR = Vagal Tone Reactivity;
[7]Source: O = Official Records; S = Self Report; P = Parent report; T = Teacher Report, OB = Observational Data;
[8]Behavior: EXB = Externalizing Behavior; ASB = Antisocial Behavior; DB = Delinquent Behavior; AGB = Aggressive Behavior; PHY/REL-AGB = Physical/Relational Aggressive Behavior; PRO/RE-AGB = Proactive/Reactivity Aggressive Behavior; PRO/RE-REL-AGB = Proactive/Reactive Relational Aggressive Behavior; CD = Conduct Disorder
[9]Theory: A = social push hypotheses; B = diathesis stress; C = differential susceptibility; – = no support for biosocial theory; ? = support for theory unknown;
[10]Gender Diff = Gender Differences in interaction effects (i.e., whether the interaction effect was gender specific); n/a = not applicable (i.e., because of sample characteristics); NR = not reported;
[11]Interaction Effects: n.s. = non-significant.

Social Risk[7]	Behavior[7,8]	Theory[9]	Gender Diff[10]	Interaction Effects[11]
Childhood victimization of sexual abuse[S] (sexual VIC)	RE-REL-AGB[S] PRO-REL-AGB[S]	B	n/a	RSAR x sexual VIC → PRO-REL-AGB ↑RSAR → ↑PRO-REL-AGB at sexual VIC
Peer popularity[PEER]	REL-AGB[PEER+T]	-	n/a	n.s.
Peer rejection[PEER]	REL-AGB[T] PHY-AGB[T]	-	n/a	n.s.
Relational victimization[S]	REL-AGB[S]	?	n/a	RSAR x REL-VIC → REL-AGB Follow-up n.s.
Relational victimization[S]	PRO-REL-AGB[S] RE-REL-AGB[S]	-	NR	n.s.
Familial adversity[S+P] (index; mostly parental characteristics)	ASB[S]	B	Yes	RSAR x family adversity → ASB ↑Family adversity → ↑ASB for *boys* at ↑+↓RSAR ↑Family adversity → ↑ASB for *girls* at ↑RSAR
Marital conflict[P]	EXB[S+P+T]	B	NR	RSAR x marital conflict → EXB ↑Marital conflict → ↑EXB at ↑+↓RSAR
Familial adversity index[P]	EXB[S+P+T]	B	No	RSAR x familial adversity index → EXB ↑Familial adversity → ↑EXB at ↑(stronger)+↓RSAR
Maternal chronic depression[P] Overcrowded housing	EXB[P]	B C	NR	RSAR x maternal depression → EXB ↑Maternal depression → ↑EXB at ↓RSAR ↑Maternal depression→ ↓EXB at ↓PEPR

Considering study characteristics, the two studies reporting significant biosocial interactions did so among high-risk (Bubier et al., 2009) and general population samples (Kochanska et al., 2015), based on cross-sectional (Bubier et al., 2009) and longitudinal (Kochanska et al., 2015) studies, using multiple measures of SNS functioning (Bubier et al., 2009; Kochanska et al., 2015). However, Bubier et al. (2009) and Kochanska et al. (2015) both conducted studies based on small samples (of 57 and 74 individuals, respectively). Thus, results of the two last-mentioned studies have to be interpreted carefully and considered alongside results based on other – larger – samples.

(b) Reactivity

Overall, studies on SNS reactivity (n = 17) found that biosocial interactions are associated with ASB (n = 11). Studies showed that lower (Gregson, Tu, & Erath, 2014; Hinnant, Erath, Tu, & El-Sheikh, 2016; McQuade & Breaux, 2017; Waters, Boyce, Eskenazi, & Alkon, 2016) as well as higher (Cummings, El-Sheikh, Kouros, & Keller, 2007; Hinnant et al., 2016) SNS reactivity functions as a vulnerability factor for developing ASB in the context of higher social risk. Interaction effects were found between SNS reactivity and familial (El-Sheikh, 2005b; Erath, El-Sheikh, & Cummings, 2009; Wagner & Abaied, 2016), as well as peer (Gregson et al., 2014; Hinnant et al., 2016; Murray-Close, 2011) related social risk factors. For example, Hinnant et al. (2016) found that the association between affiliation with deviant peers and ASB is stronger among adolescents with higher as well as lower SNS reactivity. In contrast, one study found that lower SNS reactivity was associated with increased levels of ASB in the context of low peer-related risk (Murray-Close et al., 2014). SNS reactivity did not interact with environmental (i.e., overcrowded housing) risk factors (Waters et al., 2016).

The finding that individuals on both opposites of SNS reactivity are more likely to develop ASB when exposed to social risk factors might result from gender differences, and differential interaction mechanisms underlying different subtypes of ASB. Regarding gender differences, studies consistently showed that boys with lower SNS reactivity are more likely to develop ASB when exposed to harsh parenting (Erath et al., 2011), marital conflict ((El-Sheikh, Keller, & Erath, 2007), familial adversity (Sijtsema, van Roon, Groot, & Riese, 2015), and parenting stress (Buodo, Moscardino, Scrimin, Altoe, & Palomba, 2013). For girls, studies showed inconsistent results. On the one hand, social risk was associated with girls' ASB independent of levels of SNS reactivity (Sijtsema et al., 2015). The absence of biosocial interaction for girls is supported by the fact that studies based on (mostly) girls (Sijtsema et al., 2011; Wagner & Abaied, 2015) belong to the studies that did not find significant biosocial interaction effects. On the other hand, girls high on SNS reactivity were more likely to develop ASB in the context of marital conflict (El-Sheikh, 2005b; El-Sheikh et al., 2007). Inconsistencies among girls were evident across low- and high-risk samples, cross-sectional and longitudinal studies, among children

and adolescents, and across several measures of SNS reactivity (El-Sheikh, 2005b; El-Sheikh et al., 2007; Erath et al., 2011; Sijtsema et al., 2015; Wagner & Abaied, 2015, 2016).

PNS functioning

(a) Rest

Most studies (n = 12 out of 17) on interactions between baseline PNS and social risk showed that lower (El-Sheikh, Hinnant, & Erath, 2011; Hinnant, Erath, & El-Sheikh, 2015; Zhang, Fagan, & Gao, 2017), as well as higher (Dierckx et al., 2011; Scarpa et al., 2008; Shannon et al., 2007) PNS activity exacerbated the positive relation between social risk and ASB. Children with lower baseline PNS functioning were more likely to show ASB in the context of parental problem drinking (El-Sheikh, 2005a), material conflict (El-Sheikh et al., 2011), and harsh parenting (Hinnant et al., 2015). Children with higher PNS activity were more likely to show ASB when their mother had psychiatric problems (Dierckx et al., 2011), when their parents were diagnosed with an antisocial personality disorder (Shannon et al., 2007), and when they had witnessed increased levels of community violence (Scarpa et al., 2008). Furthermore, three studies have shown that higher PNS activity is associated with decreased levels of ASB in the context of social risk (Hastings & De, 2008; Hinnant et al., 2015; Scarpa et al., 2008). For example, children exposed to harsh parenting showed less delinquent behavior when their baseline PNS functioning was higher (Hinnant et al., 2015).

Although studies among boys consistently found interactions between PNS baseline activity and social risk (Dyer, Blocker, Day, & Bean, 2016; El-Sheikh et al., 2011; Gordis et al., 2010; Hinnant et al., 2015; Zhang et al., 2017), most studies did not find significant interaction effects among girls (Dyer et al., 2016; El-Sheikh et al., 2009; Gordis et al., 2010; Zhang et al., 2017). The two studies that did report significant biosocial interactions among girls, found either a negative relationship between social risk and ASB among girls with lower levels of PNS activity (Hinnant et al., 2015), or a stronger relation between familial risk and ASB for girls with higher PNS activity (Eisenberg et al., 2012).

(b) Reactivity

Although some studies (n = 14) showed that relations between social risk and ASB is effected by levels of PNS reactivity, other studies (n = 7) did not support this assumption. Studies that reported significant interaction effects, showed that interactions between higher as well as lower PNS reactivity and social risk factors were associated with ASB (Obradović, Bush, & Boyce, 2011; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Sijtsema et al., 2015). Studies that found significant interaction effects mostly focused on familial risk (Diamond, Fagundes, & Cribbet, 2012; El-Sheikh et al., 2009; McQuade & Breaux, 2017; Zhang & Gao, 2015), as opposed to peer-related risk factors (Shoulberg et al.,

2011; Wagner & Abaied, 2015). For example, children with higher and lower PNS reactivity showed increased levels of externalizing behavior when exposed to higher levels of marital conflict (Obradović et al., 2011). In contrast, PNS reactivity did not interact with peer popularity (Shoulberg et al., 2011), peer rejection (Sijtsema et al., 2011), or relational victimization (Wagner & Abaied, 2015).

When considering differences in types of ASB, studies showed inconsistent findings that might result from sex differences. For example, Zhang and Gao (2015) distinguished between proactive and reactive aggression in a sample of mostly boys. They found that in the context of higher social adversity, higher PNS reactivity was associated with reactive aggression, whereas lower PNS reactivity was associated with proactive aggression. The opposite was found among adolescent girls who were sexually victimized as children. In a study by Murray-Close and Rellini (2012), higher PNS reactivity was more strongly related to proactive aggression for victimized girls.

Summary

Studies typically demonstrated that interactions between general ANS (re)activity, SNS reactivity, and PNS (re)activity and social risk factors are associated with ASB. Findings on baseline SNS functioning were less supportive of a biosocial view on ASB. In general, findings indicated that individuals at both extremes of psychophysiological (re)activity are more likely to show ASB when exposed to higher levels of social adversity. In the context of higher social risk, blunted arousal was found to be associated with proactive and relational ASB, whereas heightened arousal was associated with reactive and physical ASB. In addition, interactions between psychophysiological (re)activity were found more often in studies focused on familial social risk as opposed to peer-related risk factors. Regarding gender, studies showed that lower psychophysiological reactivity exacerbated associations between social risk and ASB among boys. Among girls, studies showed that the negative relationship between social risk and ASB was either unaffected or stronger or weaker as a result of their psychophysiological functioning.

2.4 Discussion

A systematic review was conducted to examine the extent to which peri/prenatal complications and psychophysiological functioning interact with social risk in predicting ASB. In doing so, we examined whether *specific* peri/prenatal and psychophysiological measures interact with *specific* social risk factors in explaining *specific* subtypes of ASB. Overall, a total of 50 included studies (66 publications) provided support for a biosocial perspective on ASB. Yet, findings varied in direction, and across particular measures of biological parameters, types of ASB, and gender.

Overall, – and in accordance with previous narrative reviews (F. R. Chen et al., 2015; Raine, 2002b; Rudo-Hutt, 2011; Yang et al., 2014) – studies offer considerable evidence that exposure to peri/prenatal complications, as well as dysregulated physiological (re)activity increases the likelihood of ASB when combined with social risk (Raine, 2002b; Rudo-Hutt, 2011; Yang et al., 2014). Few studies report a stronger relationship between psychophysiological measures and ASB in those from benign social backgrounds that lack social risk factors for ASB (see also F. R. Chen et al., 2015; Yang et al., 2014). Lastly, studies documenting protective effects of psychophysiological parameters against antisocial development in the context of social risk have also been identified (see also Rudo-Hutt, 2011).

Furthermore, studies reveal that specific peri/prenatal, psychophysiological and social measures are important when considering associations between biosocial interactions and ASB. We add to previous narrative reviews by showing that in the area of peri-prenatal factors, biosocial interaction is mostly associated with ASB for children exposed to prenatal smoking as opposed to prenatal drug use. In the area of psychophysiology, studies showed that individuals with lower as well as higher ANS (re)activity are more likely to develop ASB when they are exposed to social adversity. Although previous narrative reviews only summarized interactions between social risk and general ANS (i.e., heart rate) and SNS (i.e., skin conductance) activity, we expanded this view by showing that PNS dysregulation also exacerbates the positive relation between social risk and ASB. Furthermore, we provided increased insight into biosocial interactions in the area of psychophysiology, by showing that psychophysiological dysregulation is especially related to ASB in the context of familial as opposed to peer-related adversity.

In addition, studies supported the idea that biosocial interactions in our two biological research areas are differentially associated with different types of ASB. In accordance with previous narrative reviews (see Raine, 2002b; Rudo-Hutt, 2011; Yang et al., 2014), studies showed that in the area of peri/prenatal complications, biosocial interaction is mostly associated with more severe, violent, and persistent subtypes of ASB. We add to previous research by showing that psychophysiological under- and over-arousal are differentially associated with different ASB outcomes. In the area of psychophysiology, interactions between blunted ANS reactivity and social risk were more often related to proactive aggression, whereas interactions between heightened ANS reactivity and social risk were more often associated with reactive aggression.

Lastly, studies seem to suggest that biosocial interaction plays a more significant role in antisocial development among males. For males, the combination of biological vulnerability and social risk factors seems to substantially heighten the risk of ASB. However, findings on associations between biosocial interactions and ASB among girls were less consistent. At this point, we know too little about the association between biosocial risk and girls' ASB to draw

firm conclusions. Future research should be aimed at explaining biosocial mechanisms underlying antisocial development among girls.

2.4.1 Theoretical implications

Overall, studies were most consistent with the diathesis-stress theory, and differential susceptibility to environment hypothesis. Findings provided support for the diathesis-stress hypothesis by showing that individuals with biological vulnerabilities show worse adaptive functioning in the context of higher social adversity. Consistent with the differential susceptibility to environment hypothesis, children with higher ANS reactivity to laboratory stressors, were also found to have better outcomes in positive environments than their low reactive peers. However, a few studies found opposite effects, showing that biological vulnerability was associated with ASB at lower levels of familial risk. These study findings seem to be best explained by the social push hypothesis, which states that the relation between biological factors and ASB is stronger when social risk factors are lacking (Mednick, 1977; Raine & Venables, 1981). Studies supporting this hypothesis were mostly performed among low-risk samples (see Buschgens et al., 2009; Zhang & Gao, 2015), suggesting that biological vulnerability might be an important explanation for ASB in children from benign social backgrounds.

Further, studies support under- as well as over-arousal models of ASB, showing that dysregulated ANS functioning interacts with social risk in explaining ASB. These findings point to the possibility of the existence of heterogeneous groups of antisocial individuals that might score on opposite extremes on physiological measures of arousal. Support for that assumption was found in studies distinguishing between subtypes of ASB. Findings on baseline under-arousal suggest that individuals try to raise their arousal levels (i.e., sensation seeking; Ortiz & Raine, 2004) by showing proactive as opposed to reactive aggression. Under-aroused physiological reactivity (i.e., theorized to reflect fearlessness) was associated with proactive aggression in the context of adverse social environments. Findings on psychophysiological over-arousal suggest that over-arousal energizes antisocial responses in adverse social contexts (Scarpa & Raine, 1997), resulting in reactive aggression. Thus, findings suggest that fearlessness (under-arousal) is more strongly associated with proactive aggression and fearfulness (over-arousal) with reactive/impulsive aggression.

2.4.2 Recommendations for future research

This systematic review draws attention to several methodological issues, which are relevant to future studies on biosocial interaction. First, many studies did not provide data that were needed to adequately compare effect sizes.

Consequently, conclusions about the strengths of differential interaction effects cannot be drawn. In order to compare interaction effects in the future, researchers could for example report a model without covariates, in which both (1) the biological, and (2) social risk factors, as well as (3) the interaction term are regressed on the outcome variable. Alternatively, researchers could specify means and standard deviations of ASB and correlations between biological parameters and social adversity for all combinations of low versus high biological vulnerability, and low versus high social adversity.

Second, most empirical studies on interactions between social risk and peri/prenatal, as well as psychophysiological measures were focused on childhood ASB. Future research could investigate if biosocial interaction can also explain variance in adult ASB, or if the relationship between biology and ASB becomes weaker as the effect of social contexts increases (supporting the social-push hypothesis).

Third, interactions between psychophysiological measures and social risk have mainly been analysed in cross-sectional studies, and among general population samples. Longitudinal study designs are required to investigate whether interactions remain significant over time, as social adversity is theorized to alter or disrupt psychophysiological functioning (Lovallo, 2013). Further, research among high-risk samples is necessary to examine whether interactions between psychophysiology and social risk are also associated with variance in ASB among high-risk youth, or whether social risk overshadows their biological vulnerability (testing the social-push hypothesis).

Lastly, as not all peri/prenatal and psychophysiological parameters were repeatedly studied, future studies could investigate interactions between social risk and prenatal testosterone exposure (n = 1), minor physical anomalies (n = 1), blood pressure (SBP, DBP) (n = 2), electrodermal activity (EDR) and salivary alpha-amylase (sAA) (n = 1) in explaining ASB.

2.4.3 Limitations

Although the current review shed a unique light on determinants of antisocial development, several limitations should be considered alongside the results. First, our search command was not specifically designed to collect studies on biosocial interaction in the two biological research areas discussed in the review. As a consequence, we might have missed relevant search terms regarding peri/prenatal complications and psychophysiological functioning. Although we scanned reference lists of included studies in order to find studies that were missed in the electronic search, we still might have overlooked some relevant studies. Second, in an attempt to address questions on the association of biosocial interaction and different types of ASB, the current review included studies on all possible related outcome measures. Although this led to an extensive overview of studies on biosocial interaction and ASB, included studies

were considered to be too much of a heterogeneous group to conduct a meta-analysis. Third, based on our search strategy, potentially unpublished findings could not be identified. Because positive results are more likely to be published than negative results (i.e., publication bias), findings summarized in this review might be biased. As non-significant findings were more often reported in studies that examined multiple biological risk factors, selective reporting and publishing may be a source of bias in this systematic review. Fourth, the overrepresentation of studies from the Unites States might have led to potential bias in study results, as for example contrasts in neighborhood SES are larger in the United States than in Europe (Weijters, Scheepers, & Gerris, 2007). Future research could study the generalizability of findings based on American samples to non-American samples. Finally, we only included studies focused on a biosocial model as opposed to a biopsychosocial model of ASB. As interactions might between biological and psychological factors might also explain variance in ASB, future reviews could summarize empirical evidence on the more encompassing biopsychosocial model.

2.4.4 Practical implications

We believe that studies in the field of biosocial criminology can improve public policy aimed at reducing ASB. Before discussing practical implications of biosocial criminology, it is important to recognize that biological factors can be viewed as risk factors for ASB, without implying that antisocial development is predetermined or unchangeable. In contrast, biological parameters and social risk factors influence and change each other throughout development, in addition to interacting in complicated ways (DiLalla & Bersted, 2015). As a result, biosocial criminology can inform crime prevention by detecting the most influential environmental factors after controlling for biological factors. In addition, biosocial criminology could help maximize overall treatment effectiveness by improving the ability to identify individuals with biological vulnerabilities growing up in high-risk environments (diathesis stress), as well as individuals who are more susceptible to environmental influences and would therefore be most at-risk for ASB, but would also gain the most benefit from social programs (i.e., differential susceptibility) (Glenn et al., 2018). Such information would allow practitioners to alter types or levels of interventions to the individuals' specific needs (Glenn, 2018). In this way, programming could be better matched to participants' needs (Gajos, Fagan, & Beaver, 2016). This is in line with the responsivity approach in corrections, in which individual characteristics (e.g., learning styles) are matched to particular prevention and rehabilitation approaches (see Andrews & Bonta, 2010; Andrews & Dowden, 2007).

Although more research on biosocial interaction is needed to reach these goals, we do want to attempt translating some of our findings into practical

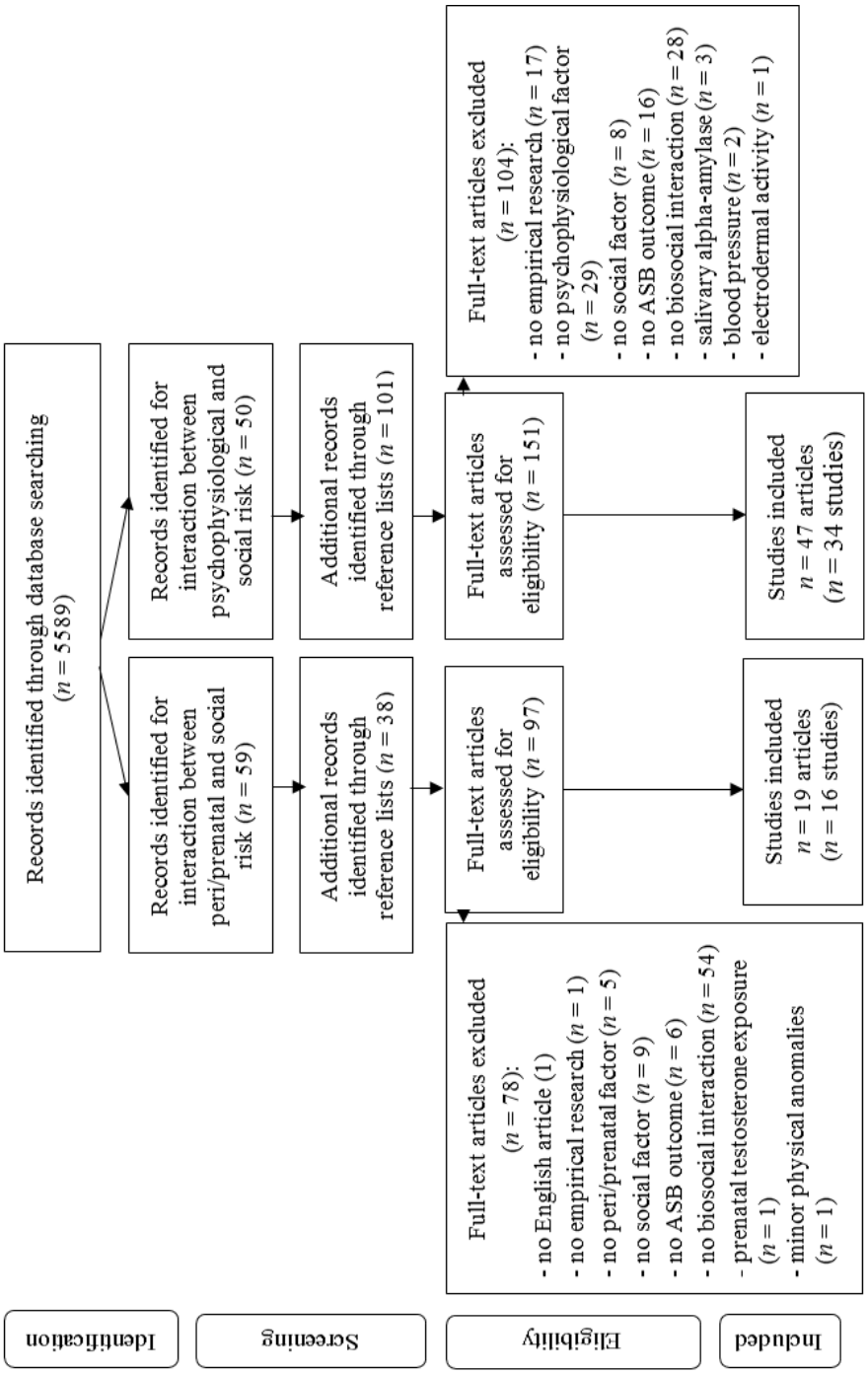
implications. Alongside these implications, it must be recognized that (1) research findings based on groups of individuals may not be directly applicable to treating antisocial individuals, (2) desirability of implementing interventions depends largely on individual's preferences and practitioners' considerations regarding individuals' unique circumstances, and (3) mentioned applications will mostly be relevant for interventions focused on young antisocial individuals as most studies were conducted among children. First, as studies have indicated that ASB is most common and severe among children exposed to prenatal smoking and adverse home environments, prevention programs could target mothers who report smoking during pregnancy. It is extra important for these mothers to be responsive toward their children. In addition, as under-aroused children show more (proactive) ASB in unsupportive environment, parents' attempts to punish these children through harsh discipline may be especially ineffective or even counterproductive. However, when biologically sensitive children are exposed to supportive environments, they tend to have better behavioral outcomes. Therefore, we suggest that prevention and intervention methods should especially focus on creating positive parent-child relationships among biologically vulnerable children.

Appendix A: Search strategy for Web of Science

(((TI=("biosocial" OR "bio-social" OR "bio social" OR "biopsychosocial" OR "bio-psycho-social" OR "bio psycho social" OR biosocial* OR bio-social* OR "bio social*" OR biopsychosocial* OR bio-psycho-social* OR "bio psycho social*" OR psychobiol* OR (biological NEAR/3 (social OR psychological))) OR (TI=(biolog* *OR "gene" OR "genes" OR genetic* OR genotyp* OR perinatal* OR prenatal* OR obstetric* OR hormon* OR neurotransmitt* OR brain OR psychophysiol* OR neuro* OR "MAOA"* OR "Monoamine Oxidase" OR "MAO" OR testosteron* OR cortex OR cortisol* OR HPA OR (("ANS" OR "CNS") AND "nervous") OR "central nervous system" OR "autonomic nervous system" OR "nervous system" OR serotonin* OR DRD2 OR "DRD-2"* OR striatum OR hemispher* OR "heart rate" OR "skin conductance" OR "IQ" OR "IQs" OR "intelligence" OR "executive functioning" OR reward* OR "sensation seeking")
AND TI=(psychosocial* OR environment* *OR family OR families OR peer* OR peers OR school OR school* OR friend OR friend* OR parent* OR father* OR mother OR neighbor* OR neighbour* OR socio-econom* OR socioecon* OR "social class*" OR abandon* OR abus* OR neglect* OR maltreat* OR empath* OR temperament* OR impulsiv* OR callous* OR unemotion* OR "emotion regulation")))
AND TS=(antisocial* OR anti-social* OR "anti social*" OR delinquen* OR aggression OR "aggressive behav*" OR offend* OR violen* OR "crime" OR "crimes" OR criminol* OR "conduct disorder*" OR "conduct problem*" OR "externalizing behav*" OR "externalising behav*" OR assault* OR criminal* OR murder*)) OR ((TS=("biosocial" OR "bio-social" OR "bio social" OR "biopsychosocial" OR "bio-psycho-social" OR "bio psycho social" OR biosocial* OR bio-social* OR "bio social*" OR biopsychosocial* OR bio-psycho-social* OR "bio psycho social*" OR psychobiol* OR (biological NEAR/3 (social OR psychological))) OR (TS=(biolog* *OR "gene" OR "genes" OR genetic* OR genotyp* OR perinatal* OR prenatal* OR obstetric* OR hormon* OR neurotransmitt* OR brain OR psychophysiol* OR neuro* OR "MAOA"* OR "Monoamine Oxidase" OR "MAO" OR testosteron* OR cortex OR cortisol* OR HPA OR (("ANS" OR "CNS") AND "nervous") OR "central nervous system" OR "autonomic nervous system" OR "nervous system" OR serotonin* OR DRD2 OR "DRD-2"* OR striatum OR hemispher* OR "heart rate" OR "skin conductance" OR "IQ" OR "IQs" OR "intelligence" OR "executive functioning" OR reward* OR "sensation seeking")
AND TI=(psychosocial* OR environment* *OR family OR families OR peer* OR peers OR school OR school* OR friend OR friend* OR parent* OR father* OR mother OR neighbor* OR neighbour* OR socio-econom* OR socioecon* OR "social class*" OR abandon* OR abus* OR neglect* OR maltreat* OR empath* OR temperament* OR impulsiv* OR callous* OR unemotion* OR "emotion regulation")) OR (TI=(biolog* *OR "gene" OR "genes" OR genetic* OR genotyp* OR perinatal* OR prenatal* OR obstetric* OR hormon* OR neurotransmitt* OR brain OR psychophysiol* OR neuro* OR "MAOA"* OR "Monoamine Oxidase" OR "MAO" OR testosteron* OR cortex OR cortisol* OR HPA OR (("ANS" OR "CNS") AND "nervous") OR "central nervous system" OR "autonomic nervous system" OR "nervous system" OR serotonin* OR DRD2 OR "DRD-2"* OR striatum OR hemispher* OR "heart rate" OR "skin conductance" OR "IQ" OR "IQs" OR "intelligence" OR "executive functioning" OR reward* OR "sensation seeking")
AND TS=(psychosocial* OR environment* *OR family OR families OR peer* OR peers OR school OR school* OR friend OR friend* OR parent* OR father* OR mother OR

neighbor* OR neighbour* OR socio-econom* OR socioecon* OR "social class*" OR abandon* OR abus* OR neglect* OR maltreat* OR empath* OR temperament* OR impulsiv* OR callous* OR unemotion* OR "emotion regulation")))
AND TI=(antisocial* OR anti-social* OR "anti social*" OR delinquen* OR aggression OR "aggressive behav*" OR offend* OR violen* OR "crime" OR "crimes" OR criminol* OR "conduct disorder*" OR "conduct problem*" OR "externalizing behav*" OR "externalising behav*" OR assault* OR criminal* OR murder* OR "psychiatric impairment")))
NOT ti=(veterinary OR rabbit OR rabbits OR animal OR animals OR mouse OR mice OR rodent OR rodents OR rat OR rats OR pig OR pigs OR porcine OR horse* OR equine OR cow OR cows OR bovine OR goat OR goats OR sheep OR ovine OR canine OR dog OR dogs OR feline OR cat OR cats) AND la=(english OR dutch)

APPENDIX B: PRISMA FLOWCHART OF PRIMARY STUDY SELECTION



3 Delinquent development among early onset offenders

Identifying and characterizing trajectories based on frequency across types of offending ▪

ABSTRACT

Early onset offending is generally recognized as a risk factor for persistent criminal behavior. However, *variation* in long-term delinquent development among early onset offenders remains rather underexplored and poorly understood. We therefore used multi-trajectory modeling to identify distinct subgroups of early onset offenders (N = 708) based on the frequency of offending across several types of offenses up to age 25. We used multinomial regression analysis to characterize subgroups on gender, ethnicity, and childhood neighborhood characteristics. Six offender subgroups could be distinguished in our data: non-recidivists (51%), sporadic recidivists (25%), and low-rate (8%), moderate-rate (10%), high-rate adult peaked (3%), and high-rate adolescence peaked recidivists (3%). Males, minorities, and children from disadvantaged neighborhoods were more likely to follow re-offending trajectories characterized by increased levels of property crime, vandalism, and violent and sexual offenses. Findings are discussed in relation to criminological theory, and recommendations are made for future life-course criminological research.

Key Words
Delinquent trajectories, early onset offenders, multi-trajectory modeling

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3.1 Introduction

Early onset offenders – those offenders who start before age twelve – are at high risk of developing persistent criminal behavior across the life-course (Loeber & Farrington, 2000; Loeber, Slot, van der Laan, & Hoeve, 2008; Snyder, 2001). Childhood onset offenders are two to three times more likely to become chronic offenders than youth who start offending in adolescence (Loeber, Farrington, & Petechuk, 2003; Moffitt et al., 2002). Early onset offenders also tend to engage in more serious types of offending, including violence (Loeber et al., 2003).

Despite their elevated risk of becoming persistent offenders, a substantial portion of early onset offenders desists from crime before reaching adulthood (Moffitt et al., 1996; Stouthamer-Loeber, Loeber, Stallings, & Lacourse, 2008). In fact, only half of all children displaying antisocial and offending behavior during childhood persists in offending in adulthood (Moffitt et al., 2002). Heterogeneity among early onset offenders poses a challenge to policy-makers. Although intervening at an early age may seem necessary to prevent further escalation into chronic offending, unnecessary intervention resulting from false-positive identification of high-risk youth should be avoided. Apart from being cost-ineffective, excessive interventions and potential stigmatization may even be harmful (L. G. Hill, Coie, Lochman, & Greenberg, 2004). At present however, identification of would-be chronic offenders is hampered by a lack of knowledge on differential re-offense patterns of early starters and factors associated with distinct delinquent pathways.

Extant studies aimed at identifying distinct offending patterns across the life-course (for reviews see Jennings & Reingle, 2012; Piquero, 2008) unfortunately offer very limited insight into differences in delinquent pathways among early onset offenders. Current trajectory-based studies largely use general population and general offender samples (Allard et al., 2017; Blokland et al., 2005; Broidy et al., 2015; Ferrante, 2013; Jennings & Reingle, 2012; Yessine & Bonta, 2009). Such samples however, might have precluded meaningful differentiation among childhood onset offenders, as the expected prevalence of early onset offenders in general and offender populations is low (Moffitt et al., 2002; Snyder, 2001; van der Laan, van Domburgh, Hoeve, Loeber, & Slot, 2008). As a result, variation in offending patterns among childhood onset offenders is overshadowed by the identification of large non- (e.g., D'Unger, Land, McCall, & Nagin, 1988; Land, McCall, & Nagin, 1996; Piquero, Farrington, Nagin, & Moffitt, 2010) and low-rate (e.g., Allard et al., 2017; Blokland et al., 2005; Broidy et al., 2015; Ferrante, 2013) offending subgroups.

The inability to differentiate between offending patterns of childhood onset offenders constitutes an important gap in our knowledge, as identifying and characterizing their distinct re-offense patterns has both theoretical and practical merit. First, it could help confirm or challenge theoretical assumptions (Moffitt, 1993, 2006) on the existence of distinct offending patterns of early

onset offenders and associated offender characteristics. Second, to the extent that individual and neighborhood characteristics differentiate between offending trajectories (see Moffitt, 1993; Moffitt et al., 1996), this could strengthen efforts to identify children at low- and high-risk of long-term criminal involvement.

To fill this knowledge-gap, two key methodological challenges need to be overcome. First, studies have to include solely known childhood onset offenders to be able to explore meaningful variation in delinquent pathways within this specific offender subgroup. Second, early starters have to be identified in childhood and followed beyond adolescence (Jennings & Reingle, 2012), during which delinquent behavior is theorized to peak for all individuals regardless of age of onset (Moffitt, 1993).

The purpose of the current study was to address the above-mentioned issues by asking: (1) whether subgroups of early onset offenders can be identified based on patterns in the frequency and nature of their offending from childhood into adulthood, and (2) whether gender, ethnicity, and childhood neighborhood characteristics can help characterize subgroups following distinct offense patterns. To address these questions, data were used on more than 700 individuals who were registered by the police for an offense before age 12. As a police contact/arrest at a young age has emerged as the most consistent indicator of persistence in offending (DeLisi et al., 2013), this sample enables us to draw a detailed picture of heterogeneity in delinquent development among childhood onset offenders.

3.1.1 Theoretical framework

Moffitt's (1993, 1997, 2006) developmental taxonomic theory is the most influential theoretical model arguing that offense patterns in the general population can be divided into distinctive offending trajectories. Moffitt (1993) originally stated that, although the majority of offenders is theorized to only temporarily engage in crime during adolescence, a small number of individuals will start offending early in the life-course and continue offending at a high-rate during adolescence. Based on emerging empirical findings, Moffitt later expanded her original taxonomic theory by including a third offending pathway. By then, several longitudinal studies had identified a small group of early starters who engaged only in low to moderate delinquency during adolescence (Moffitt et al., 1996; Nagin, Farrington, & Moffitt, 1995; Raine et al., 2005). From this, Moffitt (2006) concluded that some early onset offenders will show delinquent behavior up to age 18 that is too infrequent to be classified as classic life-course persistent offending (see Moffitt, 2006 for her altered theory and empirical underpinnings). Thus, although Moffitt (2006) still argues that the majority of early onset offenders will follow the delinquent pathway of the original life-course persistent offender, she now anticipates that a small group

of early starters will show low-rate offending behavior during adolescence and will desist before reaching early adulthood (i.e., low level chronics) (Moffitt, 2006).

In addition to duration and overall levels of offending, the offender subgroups are hypothesized to commit different types of crime. Although life-course persistent offending is generally thought to be more versatile than that of adolescence-limited delinquents, persistent offenders are believed to increasingly commit violent types of offenses with age (Moffitt, 2003, 2006). Offenders on the life-course persistent trajectory are thought to continuously suffer from the individual and familial risk factors that instigated their problems in childhood (i.e., contemporary continuity). As life-course persisters are exposed to new life domains with age (e.g., school, peers, neighborhood), accumulating negative experiences enhance a process of escalation of offending (i.e., cumulative continuity). Longer involvement in crime and new social circumstances are believed to change the manifestation of tendencies toward offending with age (i.e., heterotypic continuity). As a result, life-course persisters are thought to engage in all types of age-appropriate delinquent behaviors in all stages of life; they are thought to hit and kick at onset, exhibit theft and drug use throughout the teenage years and turn toward violent offending upon entering adulthood (Moffitt, 1993; Moffitt et al., 2002). In contrast, low level chronic offenders are thought to suffer from depression and anxiety, excluding them from deviant social peer groups. As a result, although low level chronics are subject to both contemporary and heterotypic continuity, they would be less affected by the process of cumulative continuity causing the escalation of offending among their high-level counterparts (Moffitt, 2006).

Moffitt's (1993) taxonomy also offers expectations on the associations between gender, ethnicity, and early environmental differences and offending subgroups. Life-course-persistent offending would mainly be limited to males, as they tend to suffer more from the neuropsychological deficits assumed to underlie this pathway than females. Minorities would also be at elevated risk for life-course persistent offending, because, as a group, they are likely to grow up in the most disadvantaged familial and economic environments (see Piquero, Moffitt, & Lawton, 2005). Childhood environmental characteristics are also thought to distinguish between low- and high-rate persistent offenders, with low-level offenders residing in more adaptive social environments than their high-level counterparts (Moffitt, 2006).

The Moffitt-taxonomy leads us to expect that most early starters frequently commit crimes across the entire life-course, whereas a small group of early onset offenders engages only in low to moderate delinquency during adolescence. In addition, we would expect persistent offenders to engage in all types of offending, and high-rate chronic offenders to disproportionally, and increasingly, engage in violent crime. Furthermore, males, minorities and children from low socioeconomic status (SES) and highly urban neighborhoods would be most likely to show persistent and violent delinquent behavior.

3.1.2 Prior research

The potentially meaningful differentiation of re-offense patterns among childhood onset offenders is vastly understudied. To exemplify: a systematic review on delinquent trajectories (Jennings & Reingle, 2012) found that out of the 105 studies included, only one study (i.e., van Domburgh, Vermeiren, et al., 2009) was based on a sample of childhood onset offenders. To provide empirical groundwork to the current study, we therefore draw from trajectory-based studies using juvenile offender samples.

Prior work among adolescent offenders has identified between three and seven offending trajectories (Baglivio et al., 2015; Day et al., 2012; Laub et al., 1998; Livingston, Stewart, Allard, & Ogilvie, 2008; Monahan, Steinberg, Cauffman, & Mulvey, 2009; Mulvey et al., 2010; van der Geest et al., 2009; Ward et al., 2010). Trajectory subgroups generally include low-, moderate, and high-rate offending groups. The low-rate group typically consists of the largest portion of the sample, whereas the high-rate group makes up the smallest trajectory subgroup (Day et al., 2012; Laub et al., 1998; Monahan et al., 2009; Mulvey et al., 2010; van der Geest et al., 2009; Ward et al., 2010).

Studies on juvenile offender samples confirm Moffitt's assumptions on (heterotypic) continuity among early onset offenders, by reporting that early onset offenders follow the most chronic delinquent pathways, and commit the highest amount and most diverse types of offenses (Broidy et al., 2015; Day et al., 2012). In a large sample of youth arrested before age 18, Baglivio et al. (2015) found distinct delinquent pathways among juveniles who started offending in childhood. Although some early starters desisted shortly after age 13 years (32%), others either had a steady but low number of arrests up to the age of 17 years (29%), or showed high-rate persistent offending throughout the adolescent period (7%). Data on 287 male childhood onset offenders also revealed heterogeneity in re-offending patterns by supporting a model with three distinct subgroups: a low-rate group (68.3%), an escalating group (24.7%), and a high-rate group (7.0%) (van Domburgh, Vermeiren, et al., 2009).

Unfortunately, it remains relatively unclear whether offending subgroups vary by demographic factors such as gender, as most trajectory-based studies are based on male subjects (Day et al., 2012; Laub et al., 1998; Monahan et al., 2009; Mulvey et al., 2010; van der Geest et al., 2009; Ward et al., 2010). Studies that were able to characterize trajectory subgroups based on demographic characteristics found that males were more likely to populate the chronic offending groups than females (e.g. Block, Blokland, van der Werff, van Os, & Nieuwbeerta, 2010; Livingston et al., 2008; Piquero, Brame, & Moffitt, 2005). Findings on ethnicity seem context dependent. In the United States, minorities (i.e., non-Whites) were more likely to belong to the trajectory subgroup demonstrating an early onset and chronic offending trajectory (e.g. Baglivio et al., 2015), whereas Australian Indigenous offenders were more likely than non-Indigenous offenders to populate the early onset/chronic trajectory

(e.g. Livingston et al., 2008). After gender and ethnicity were controlled for, neighborhood disadvantage was no longer associated with trajectory group membership (Livingston et al., 2008). In a childhood onset sample (van Domburgh, Vermeiren, et al., 2009), minorities were found to be more likely to follow the high-level than the low-level offending pathway, and children from disadvantaged neighborhoods were overrepresented in the escalating offender subgroup.

Although above-mentioned studies highlight heterogeneity in offending patterns, the literature is characterized by a number of limitations. First, the generalizability of findings among juvenile offenders to childhood onset offenders is limited. Childhood onset offenders without an arrest during adolescence are not included in juvenile offender samples, whereas variation in offending among children that do re-offend might be overshadowed by offending behavior of the more common adolescent-onset offender. Second, in previous studies offending subgroups were categorized based on frequency of offending alone (e.g. Day et al., 2012; Laub et al., 1998; Ward et al., 2010), or broad distinctions between non-serious and serious types of crime (van der Geest et al., 2009; van Domburgh, Vermeiren, et al., 2009). However, a relevant taxonomic theory (Moffitt, 1993) as well as critiques of life-course criminological research (Evans, Simons, & Simons, 2016; Hasking, Scheier, & Abdallah, 2011; Odgers et al., 2007) highlight that the complexity of delinquency is underestimated when it is defined solely as the frequency of offending. Scholars state that it is important to distinguish between several types of offending behaviors when identifying offender subgroups (Evans et al., 2016; Hasking et al., 2011; van Domburgh, Vermeiren, et al., 2009). Third, a limitation of past research featuring early onset offenders is the use of short follow-up periods (van Domburgh, Vermeiren, et al., 2009). This is unfortunate, because to test the popular notion that an early onset elevates the risk of becoming a chronic offender (see Moffitt, 1993), studies have to be focused on delinquent development beyond adolescence, during which delinquent behavior is theorized to peak for all offenders.

3.1.3 The current study

Given the paucity of studies focused on long-term re-offense patterns of childhood onset offenders, the current study explores the extent to which distinct delinquent trajectories can be identified within a sample of approximately 700 early onset offenders. These data provide us with a follow-up period of 3 to 14 years, which gives us insight into re-offending in the theoretically relevant period beyond adolescence. To take offense types into account, we use a multi-group trajectory model to identify clusters of individuals following similar trajectories *across multiple types of crime*. In doing so, we hope to uncover the most useful taxonomy of early onset offenders by accurately reflecting the

full range of their offending behavior. Finally, the current study incorporates key demographic and early neighborhood explanatory factors to examine the assumption that males, youths from non-Western backgrounds, and individuals from disadvantaged neighborhoods are overrepresented in the high-rate persistent delinquent trajectories.

3.2 Method

3.2.1 Participants and procedures

This study was based on participants of the *Dutch Childhood Arrestees Study*, a prospective longitudinal study on children registered by the police for committing a first offense prior to age 12. Although offenses committed before the age of criminal responsibility (i.e., 12 years in the Netherlands) are not registered in national crime statistics, they are documented in local police registration systems. These registration systems were used to select first-time registered children from three different Dutch police districts (Gelderland-Midden, Utrecht and Rotterdam-Rijnmond) in the Netherlands, ensuring that neighborhoods with a sufficient range in levels of SES and urbanization were included. Children were not eligible for inclusion when (1) they committed status offenses (i.e., behavior that is only prosecutable for certain (age) groups, such as truancy), (2) they were not legal residents of the Netherlands (because of foreseeable problems at follow-up), (3) they committed the crime on the command of their parents, or (4) inclusion would complicate police procedures.

Data from two cohorts were combined; (1) children registered in 2000-2001 (n = 351, 82.6% male, 46.2% non-Western) and (2) children registered between 2003 and 2005 (n = 357, 84.9% male, 44.8% non-Western). The total sample encompassed 708 childhood arrestees (83.8% male, 45.5% non-Western) who were registered by the police between the ages of 5 and 11 years (M = 9.66, SD = 1.50, Mdn = 10). Most participants (52.3%) were registered for committing vandalism prior to age 12, approximately one fourth (25.3%) was registered for property crime, and 14.7% was registered for a violent offense. Comparison with available data from the United States (Snyder, 2001) suggests that sample characteristics are similar in terms of gender and offense profile.

3.2.2 Measures

Measures used for multi-trajectory modeling. Two registration systems were used to reconstruct participants' criminal history. We used the Dutch police registration system HKS (Herkennings Dienst Systeem) to gather information on frequency and type of re-offending from age 12 years and above over a 3 to 14-year follow-up period, from January 2000 until February 2015. The

HKS has information on all the times a person was identified as a suspect in a criminal case from age 12 years and above. As such, HKS contains information on suspects and not convicted offenders. However, given that cases that are discarded in an early stage of investigation do not end up being registered in HKS, and given that approximately 90% of all HKS registered suspects are found guilty at a later stage (Besjes & van Gaalen, 2008), HKS data are closer to conviction than to arrest data. In order to correct for reduced time at risk, we gathered information on date of death and criminal sanctions from the Research and Policy Database Judicial Documentation ('Onderzoek- en Beleidsdatabase Justitiële Documentatie', OBJD) of the Research and Documentation Centre of the Ministry of Justice (WODC). As there was no participant mortality during the follow-up, we corrected for the possible reduction in police registrations due to time spent incarcerated (Piquero et al., 2001).[1] Relatively few participants (n = 75, 10.59%) had been incarcerated during the observation period. The average incarceration time among recidivists was 6.5 months (SD = 1 year and 2 months, Mdn = 1.4 months).

We determined frequency of re-offending per type of offense per age based on date of birth, date of police registrations, and crime descriptions from the police registration system (HKS). Crime descriptions in HKS were based on the following standard crime categorization employed by Statistics Netherlands (see Kalidien, de Heer-de Lange, & van Rosmalen, 2011; Statistics Netherlands, 2019); (1) property crime (including theft, embezzlement and fencing); (2) vandalism and crimes against the Public Order and Authority (such as discrimination and sedition); (3) violent and sexual offenses (including rape and (sexual) assault); (4) traffic offenses (including DUI, hit and run, joyriding and refusal of a breathalyzer test); (5) drug crimes (such as owning or selling illegal amounts of soft and hard drugs)[2]; and (6) weapons offenses (including carrying a weapon in public).

In order to correct for spells of incarceration, we calculated the number of offenses individuals might have committed had they not been imprisoned (see Bijleveld, van de Weijer, Ruiter, & van der Geest, 2015). We multiplied the number of times participants had been registered by the police during the time they were free within a year's time period, by the inverse of the proportion of that year. For example, if an individual was detained for six months at age 20 and committed two offenses during the remaining six months of that year, this person was expected to have committed four offenses were he or she not have been incapacitated at age 20 years. As this correction led to

1 To account for early release up to July 1st 2008 (when early release was standard in The Netherlands after two third of the sentence was completed), we multiplied the number of days spent in detention before this date by 2/3.

2 Note that in the Netherlands possession of small 'user' amounts of drugs or drug paraphernalia is not liable to prosecution. Drug offenses in the Netherlands thus pertain to commercial quantities of drugs produced, stored, sold, or smuggled.

disproportionately high offending rates per age for a few participants (e.g., because they had two police registrations in a year during which they were incarcerated for 11 months), we stabilized the correction effect (see also van der Geest et al., 2009) by capping their offending rates at the maximum of offenses committed by any individual in the sample without stabilization + 1. Thus, stabilizing the correction for incarceration put them within the sample's observed range, but still indicated that these individuals were more likely to have committed a disproportionately high number of offenses in the years they were incapacitated.

Risk factors

As *ethnicity* was not registered at participants' arrest below age 12, we determined ethnic background based on family name. This method is likely to be accurate in the vast majority of the cases, as intercultural marriages of foreign women to Dutch men (in which case the child is likely to have a Dutch family name) were rare at the time our participants were born (Statistics Netherlands, 2001). Most non-Western family names were easily recognizable. However, when a specific family name was common in both the Netherlands and in foreign countries, ethnicity was coded as 'unknown' (n = 57). This process led to high agreement among three coders (89%), and a high degree of inter-rater reliability was achieved ($\kappa > .86$, $p < .001$). As country of birth was registered at follow-up, we were able to check our initial coding among recidivists. We found that of the 66 recidivists born in non-Western countries, 89% was correctly classified as having a non-Western ethnicity based on their family name. For the 8% (n = 5) that were wrongfully categorized as Western, and the 3% (n = 2) originally categorized as 'unknown', we adjusted their ethnicity into non-Western based on their country of origin.

Postal codes were used as a proxy for *neighborhood levels of SES and urbanization* prior to age 12. The postal code classification of neighborhood SES was available in quintiles based on mean income, unemployment, and education level (Knol, 1998; Social and Cultural Planning Office of the Netherlands, 2002), with higher scores representing lower neighborhood SES. Information on urbanization levels was based on the number of households per square kilometers (Statistics Netherlands, 2006), and ranged from (1) 'no-urbanization: less than 500 households per km²' to (5) 'very high urbanization: 2,500 or more households per km²'. With the intent of using analytical techniques to compare groups, we increased the likelihood of having observations per trajectory group and levels of SES and urbanization by dichotomizing the neighborhood-related variables. We combined the lower three (low risk) and upper two (increased risk) categories for both variables. Hence, we separated 'very high to average neighborhood SES' (1-3) from 'low to very low neighborhood SES' (4-5) and 'very low to average urbanization' (1-3) from 'high to very high urbanization' (4-5). To deal with missing data, we added a category 'unknown' to all predictor variables.

3.2.3 Analyses

Our analyses proceeded in three steps. First, we studied the overall delinquent development in our sample by examining total recidivism rates and recidivism rates per type of offense. Second, we used a group-based multi-trajectory model (Nagin, Jones, Passos, & Tremblay, 2018) to identify distinctive clusters of individuals displaying similar offending patterns across several types of offending. Parameters defining these patterns were allowed to vary freely across groups, so that groups could differ in both the level and shape of their delinquent pathways. When estimating offending trajectories, we excluded participants without an additional police registration during the follow-up period (Broidy et al., 2015; Ferrante, 2013), as including non-recidivists would only add a flat trajectory to the model. Furthermore, it would increase the risk of low-level recidivists being pulled into the non-recidivist group, which would complicate distinguishing between non- and low-level recidivists. In the third step, we assigned individuals to subgroups based on their maximum posterior group probabilities, and used group membership – with non-recidivists denoted as a separate group – as a multiple nominal outcome in a series of multinomial logistic regression analysis. We studied whether gender, ethnicity and levels of childhood neighborhood SES and urbanization (all dummy-coded) differentiated between trajectory subgroups. Additional analysis showed that there was no evidence of multicollinearity among these predictors, as variance inflation factor (VIF) scores were between 1 and 10 (Bowerman & O'Connell, 1990; Mertler & Vannatta, 2005), and tolerance values above 0.2 (Menard, 1995).

Multi-trajectory model

The multi-trajectory models were run using the STATA Trajectory Procedure (Jones, Nagin, & Roeder, 2001) in STATA 13. We identified the best fitting model based on cubic shaped trajectories, and a count-specific zero-inflated Poisson regression model. In doing so, we were able to prevent disproportional change in delinquent trajectories caused by years with zero police registrations (Lambert, 1992).

Due to low rates of participation in drug and weapons offenses, we combined these two types of offending to display delinquent development across different types of crime. As a result, trajectory subgroups were identified based on similarity of their offending patterns across five outcome variables: (1) property crime, (2) vandalism and crimes against the public order and authority, (3) violent and sexual offenses, (4) drug and weapons offenses, and (5) traffic offenses.

In line with Nagin's recommendations (see Nagin, 2005, 2010), the best fitting model was selected based on three criteria: (1) Bayesian Information Criterion (BIC; Schwartz, 1978) values closest to zero, indicative of increased model fit compared with alternative models; (2) highest average posterior

probabilities, representing higher degrees of classification certainty of each individual to his or her most likely trajectory; and (3) highest odds of correct classification (OCC; Nagin, 2005), indicating improvement of assignment accuracy over random assignment, while accounting for differences in group sizes. Mean posterior probabilities above .70 and OCCs of 5 or larger for all trajectory groups were considered indicative of satisfactory model fit and assignment accuracy (Nagin, 2005, 2010). In addition, Wald tests were performed to test for group differences in terms of intercepts and cubic slopes across all outcome variables.

Attrition

As the year of inclusion and age at first offense differed between participants, the average age at the end of follow-up ranged from 15 to 27 years (M = 22.10, SD = 2.47, Mdn = 22; see bottom part of Table 3.1 for percentages of the original sample across age). To avoid problems associated with defining parts of offending trajectories based on a small number of individuals, we limited the trajectories to ages for which data were available on at least 100 individuals. We therefore ended our observation of offending trajectories at age 25. In addition, we corrected for differences in participants' age at the end of the observation period by coding non-observed years as missing (see also van der Geest et al., 2009). For example, when participants reached the age of 20 at the end of the follow-up period, we coded their police registrations from ages 21 to 25 years as missing. As a consequence, they did not contribute to the estimation of the trajectories from age 21 to 25 years.

Independent sample t-tests showed that there were no significant differences in participants' age at the end of the follow-up period across gender ($t(699) = 1.56, p = .12$), ethnicity ($t(648) = .82, p = .41$), or neighborhood urbanization levels ($t(668) = 1.80, p = .07$). However, there was a significant difference in the level of neighborhood SES. Children from higher SES neighborhoods were older (M = 22.20, SD = 2.32) at the end of the observation period, than children from lower SES neighborhoods (M = 21.80, SD = 2.36), $t(699) = 2.23, p = .03$.[3] Thus, although attrition led to decreasing power with age, substantial bias due to differential attrition is not likely to be an important threat to the validity of our conclusions.

3 This significant association between neighborhood SES and age is due to differences in years of inclusion, $\chi^2(4) = 61.42, p < .001$. Children from high and low SES neighborhoods did not differ in their mean age of onset ($t(699) = 0.33, p = .74$).

3.3 Results

3.3.1 Development of re-offending

Results showed that half (48.9%, $n = 346$) of early onset offenders had a second police registration between the ages of 12 and 25 years (see Table 3.1). Most participants were registered for property crime (35.3%), vandalism (29.7%), and violent or sexual offenses (25.4%), whereas drug (6.1%), weapons (6.6%), and traffic (10.7%) offenses were less common.[4] Regarding involvement in crime across age, Table 3.1 shows that early onset recidivists were typically criminally active during adolescence, as the largest share of participants was registered by the police at ages 16 and 17 years.

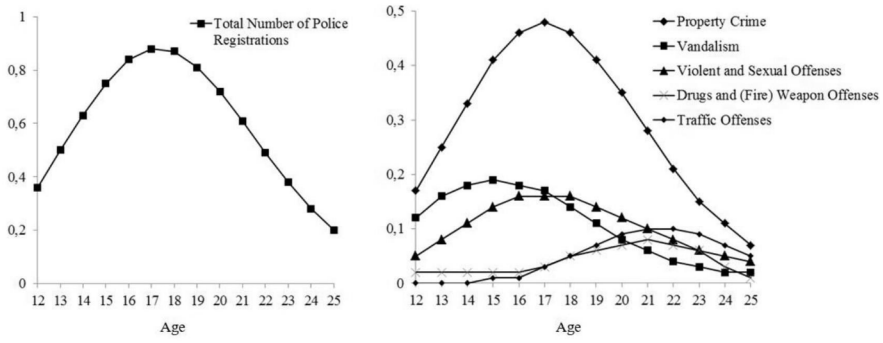
Table 3.1: Percent of sample with a police registration by age and offense types

	Total	12	13	14	15	16	17	18	19	20	21	22	23	24	25
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
Total	48.9	7.8	13.3	16.1	17.4	18.4	17.9	17.1	14.9	15.9	11.9	9.4	10.7	5.9	1.2
Property crime	35.3	3.7	8.6	10.6	11.3	11.2	11.0	9.2	7.7	7.6	6.5	5.3	4.4	2.1	0.6
Vandalism	29.7	4.2	5.8	5.9	6.2	6.8	5.6	5.7	4.1	2.2	2.5	1.8	1.7	1.3	0
Violent and sexual offenses	25.4	2.0	3.0	3.7	5.1	6.6	5.3	4.4	3.9	4.0	3.1	3.0	3.4	1.3	0
Drug offenses	6.1	0.1	0	0.3	0.3	0.4	0.7	0.9	0.8	1.5	1.7	1.5	1.3	0.8	0.6
Weapons offenses	6.6	0.3	0.4	0.6	1.0	0.7	0.9	0.7	1.1	1.2	1.0	1.3	1.0	0.4	0
Traffic offenses	10.7	0	0	0.3	0.3	0.4	1.0	2.0	2.4	3.8	1.9	3.3	3.4	1.3	1.2
Percentage of original sample size across age		100	100	100	99.9	98.9	96.8	93.1	85.2	73.4	55.6	42.1	33.6	23.4	8.3

Regarding frequency of re-offending, Figure 3.1 shows that re-offending rates across age displayed the age-crime curve, with police registrations increasing up to age 17 and then declining into adulthood. However, the typical age-crime curve did not apply to all types of crime. On the one hand, property crime, vandalism, and violent and sexual offenses followed the typical age-crime curve. Traffic offenses, and drug and weapons offenses on the other hand, were virtually absent up to late adolescence to peak only in early adulthood.

4 A possible explanation for the low rate of traffic offenses in this sample, is that the age limit for driving a car is 17 years under Dutch law.

Figure 3.1: Average number of police registrations per age, corrected for days of incapacitation



3.3.2 Offending trajectories

To select the optimal number of trajectory groups, we estimated multi-trajectory models with up to six groups and compared their fit. As illustrated in Table 3.2, the fit indices indicated that the five-group model described the data best. In this five-group solution, BIC values were lowest. In addition, average posterior group membership probabilities were high: all averaging above .90. The OCC ranged from 12 to 1091, indicating high assignment accuracy. Although this five-group model identified two relatively small subgroups (less than 5%), model fit was substantially better than that of the four-group model.

Table 3.2: Fit measures for one- to six multi-group models

Groups	BIC	2(?BIC)[1]	Average Posterior Probabilities	OCC	Group membership (n)
1	-7800.55		1		346
2	-7061.60	1477.9	.99; .96	28; 82	270; 76
3	-6947.57	228.06	.98; .93; .96	25; 45; 207	231; 77; 38
4	-6926.55	42.04	.93; .90; .94; .98	12; 42; 58; 325	181; 58; 70; 37
5	**-6853.07**	**146.97**	**.93; 90; .94; .97; .98**	**12; 42; 65; 549; 1091**	**180; 59; 68; 21; 18**
6	-6882.12	-29.05	.88; .89; .98; .98; .97; .76	9; 39; 94; 651; 610; 31	154; 61; 60; 21; 19; 31

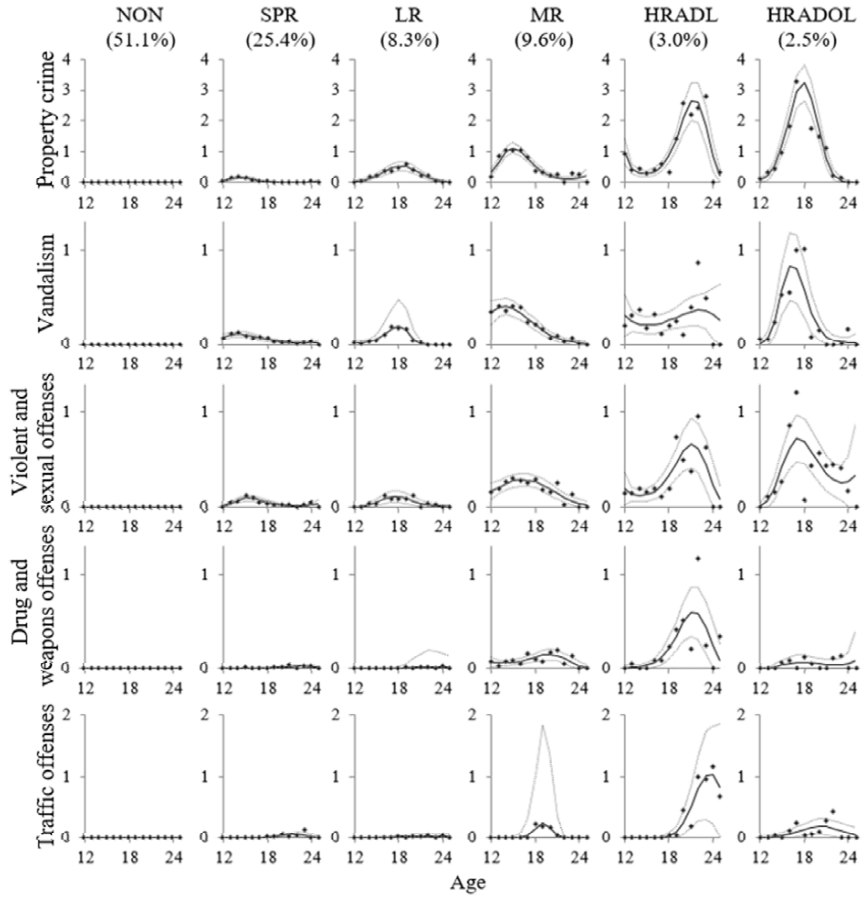
Note: N = 346; Starting values were required to converge the five- and six-group models. We therefore re-estimated these models, using intercepts and cubic slopes from the $k - 1$ group model as starting values. Bold text represents model fit indices for final group-model.

[1] $2(\Delta BIC) > 10$ indicates there is very strong evidence that the more complex model is favored above the simpler model (Jones et al., 2001).

Further examination of the five-group model indicated that distinguishing between the two smallest groups yielded unique information about their levels as well as developmental patterns of recidivism (see Figure 3.2 and Table 3.3);

information that was lost in the four-group model. We opted for the model with five re-offending subgroups. We note however, that additional analyses (not reported here, but available upon request) showed that current conclusions were not substantively affected by choosing the five- over the four-group model.

Figure 3.2: Multi-trajectory model of observed and predicted frequency of offense types per trajectory subgroup



Apart from the a-priori defined group of non-recidivists (NON) (51.1%, n = 362), the five recidivist groups were assigned the following labels based on overall level differences in re-offending: sporadic recidivists (SPR) (25.4%, n = 180), low-rate recidivists (LR) (8.3%, n = 59), moderate-rate recidivists (MR) (9.6%, n = 68), high-rate adult peaked recidivists (HRADL) (3.0%, n = 21), and high-rate adolescence peaked recidivists (HRADOL) (2.5%, n = 18). Re-offending patterns for each group across five types of crime are illustrated in Figure 3.2,

with columns representing offending trajectories per subgroup.[5] Figure 3.3 builds on the findings presented in Figure 3.2, by displaying the total frequency of offending, as well as the relative share of each type of offense per subgroup.

Most participants belonged to the subgroup that is characterized by non-involvement in any type of offending as registered by the police during the observation period. Mean offending rates per year were low in both the SPR (M = 0.20, SD = 0.16) and LR (M = 0.44, SD = 0.20) subgroups. The SPR and LR groups differed in their development of property crime and vandalism, which peaked at age 13 to 14 for the SPR group, whereas the LR group showed a (low) peak at age 18. The MR group had higher average offending rates per year (M = 1.25, SD = 0.57) than the first three trajectory subgroups. The MR group was mostly characterized by higher levels of violent and sexual offenses than the SPR group, and an earlier peak in property crime (age 16) than the LR group (age 18; see Figures 3.2 and 3.3).

Table 3.3: Wald tests on differences between intercepts and cubic slopes per type of offense across five trajectory subgroups

		LR vs. SPR	MR vs. SPR	HRADL vs. SPR	HRADOL vs. SPR	MR vs. LR	HRADL vs. LR	HRADOL vs. LR	HRADL vs. MR	HRADOL vs. MR	HRADOL vs. HRADL
Property	Intercept	4.88*	2.09	45.81***	5.45*	2.26	15.29***	0.00	99.47***	2.63	15.16***
Property	Slope	10.19**	2.68	43.78***	12.00***	6.69**	8.93**	0.18	94.25***	8.77**	5.05*
Vandalism	Intercept	2.20	0.55	4.44*	4.28*	1.71	0.77	5.82*	2.09	5.89*	9.48**
Vandalism	Slope	3.97*	0.64	3.67	1.77	3.27	2.33	6.04*	1.21	3.02	5.35*
Violence	Intercept	0.02	10.12**	22.68***	0.01	1.64	5.04*	0.01	4.91*	5.72*	14.64***
Violence	Slope	0.45	9.79**	22.55***	0.29	0.71	3.51	0.12	4.28*	4.40*	13.15***
Drug/ Weapons	Intercept	0.13	0.00	0.06	1.92	0.13	0.12	0.02	0.05	2.01	1.51
Drug/ Weapons	Slope	0.10	0.00	0.00	1.92	0.10	0.10	0.00	0.00	2.14	2.01
Traffic	Intercept	0.01	0.64	0.14	0.00	0.77	0.10	0.00	0.76	0.72	0.12
Traffic	Slope	0.17	1.47	0.10	0.08	1.91	0.00	0.02	1.57	1.72	0.02

Note: Lower group is reference category. SPR = sporadic recidivists; LR = low-rate recidivists; MR = moderate-rate recidivists; HRADL = high-rate adult peaked recidivists; HRADOL = high-rate adolescence peaked recidivists. *$p < .05$; **$p < .01$, ***$p < .001$

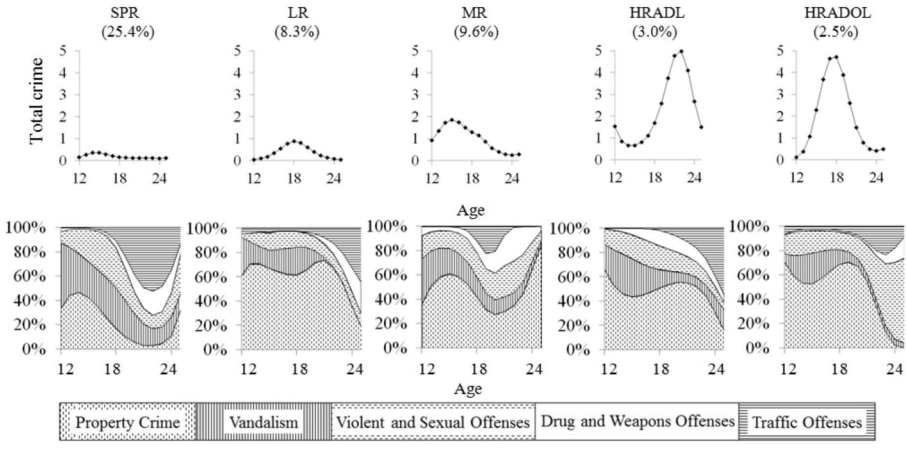
In addition, Figure 3.2 shows that mean offending rates per year were highest in the HRADL (M = 1.94, SD = 1.00) and HRADOL (M = 2.36, SD = 0.74) subgroups. These two high-rate groups differed in levels of offending at age 12 and developmental patterns of property crime, vandalism, and violent and sexual offenses (see Table 3.3 for Wald tests). Although the HRADL group had

5 We studied whether group assignment was determined by the combinations of different types of crimes. Results showed that participants who committed sexual offenses (n = 30), drug offenses (n = 43), or weapons offenses (n = 47) were represented in all trajectory groups, indicating that the fact that they committed these specific types of offenses, did not determine their group membership classification.

higher initial levels of property crime, vandalism, and violent and sexual offenses, their patterns of offending started to steadily rise around age 18, peaked around age 22, and decreased toward age 25. The HRADOL group however, showed lower levels of offending at age 12, but police registrations increased rapidly to a high frequency at age 18.

Regarding distributions of offense types, Figure 3.3 shows that SPR and MR offenders resembled each other in that for offenders in both trajectory groups property offending started to decline from the mid-teens onward to a low-rate in the early 20s, followed by a small increase up to age 25. Only MR offenders also showed an increase in the proportion of drug and weapons offenses during this period. For the other recidivist groups the proportion of property crimes did not decline until the early 20s, before giving way to traffic offenses in the LR and HRADL group, and violent and – to a lesser extent - drug and weapons offenses in the HRADOL group. Thus, violent and sexual offenses made up an increasing part of total crime among the HRADOL subgroup, indicating possible escalation of delinquent behavior toward the end of the follow-up period.

Figure 3.3: Total predicted amount of police registrations and distribution of types of offenses per trajectory subgroup across age



3.3.3 Characterizing trajectory subgroups

As the trajectory model identified two small subgroups with high rates of offending, the high-rate adult and adolescence peaked groups were combined to form the 'high-rate' (HR) recidivist group in follow-up analysis to create sufficient power.[6] The overall multinomial model with group membership as dependent variable was statistically significant, although predictors explained no more than 17% of group assignment (see Table 3.4).

In the model with non-recidivists as the comparison group, Table 3.4 shows that males were more likely to belong to the SPR (odds ratio [OR] = 2.39), and MR (OR = 6.06) groups than to the NON group. In addition, non-Western participants were more likely to be classified to the LR (OR = 3.08), MR (OR = 2.04), and HR (OR = 6.36) groups than to the NON group. Furthermore, residing in low SES neighborhoods as a child substantially increased the chances of being assigned to the MR (OR = 2.06) and HR (OR = 2.62) groups compared with the NON group.

When offender subgroups were compared, non-Western participants were more likely to belong to the LR (OR = 3.06), MR (OR = 2.03), and HR (OR = 6.31) groups than to the SPR group. Members of the LR, MR and HR groups did not differ in terms of gender, ethnicity or neighborhood levels of SES and urbanization.

In sum, multinomial regression analyses indicated that gender differentiated the SPR and LR groups from the NON subgroup. Minorities were more likely to be classified to the LR, MR, and HR groups than to the NON and SPR groups. Residing in a low SES neighborhood as a child only differentiated the MR and HR groups from the NON subgroup.

6 Results for multinomial regression analysis with high-rate adult and adolescence peaked offenders as two separate groups are available upon request.

Table 3.4 Odds ratio results from multinomial regression analysis: Effects of demographic and childhood

	Total Sample %	SPR vs. NON	LR vs. NON	MR vs. NON	HR vs. NON
Sex					
Female (*ref.*)					
Male	83.8	2.39**	1.84	6.06**	NI
		1.38-4.14	0.83-4.11	1.83-20.02	
Unknown	1.0	10.38*	NI	NI	NI
		1.74-61.78			
Ethnicity					
Western (*ref.*)					
Non-Western	45.5	1.01	3.08**	2.04*	6.36***
		0.67-1.53	1.57-6.05	1.10-3.81	2.25-18.00
Unknown	8.2	0.73	1.49	1.24	5.57*
		0.35-1.52	0.46-4.80	0.42-3.63	1.47-21.01
SES					
High (*ref.*)					
Low	48.0	1.15	0.94	2.06*	2.62*
		0.74-1.76	0.50-1.78	1.06-3.99	1.04-6.63
Unknown	1.0	0.63	NI	1.40	NI
		0.08-5.02		0.10-20.23	
Urbanization					
Low (*ref.*)					
High	69.9	1.13	1.55	1.32	0.66
		0.70-1.82	0.67-3.61	0.56-3.14	0.21-2.05
Unknown	5.4	2.49	3.75	3.61	2.39
		0.84-7.41	0.84-16.69	0.90-14.56	0.48-12.03

Note. $N = 708$. Percentages may not sum to 100 due to rounding. $R^2 = .15$ (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(32) = 117.27$***. Lower group is reference category. Odds ratios that could not be calculated – for example because trajectory subgroups consisted entirely of males – were labelled as 'not identified' (NI). NON = non-recidivists ($n = 362$) SPR = sporadic recidivists ($n = 180$); LR = low-rate recidivists ($n = 59$); MR = moderate-rate recidivists ($n = 68$); HR = high-rate recidivists ($n = 39$); SES = Socioeconomic status. Odds ratios greater than 1.00 indicate increased probability of group membership. *$p < .05$; **$p < .01$, ***$p < .001$.

3.4 Discussion

The aims of the current study were to identify delinquent trajectories and demographic and neighborhood characteristics related to trajectory group membership in a Dutch sample of early onset offenders. We employed multi-trajectory modeling to identify subgroups among approximately 700 early starters, following similar offense patterns across several types of offending. Results showed that delinquent development in early onset offenders is highly heterogeneous. Next to an a-priori defined non-recidivist trajectory, five re-offending trajectories were identified: a sporadic, low-, moderate-, and two high-rate re-offending subgroups, whose offending peaked either in adulthood

neighborhood factors on group membership

LR vs. SPR	MR vs. SPR	HR vs. SPR	MR vs. LR	HR vs. LR	HR vs. MR
0.77	2.54	NI	3.29	NI	NI
0.31-1.91	0.72-8.99		0.82-13.13		
NI	NI	NI	NI	NI	NI
3.06**	2.03*	6.31**	0.66	2.06	3.12
1.50-6.24	1.05-3.91	2.18-18.25	0.28-1.55	0.63-6.77	0.98-9.88
2.03	1.69	7.59**	0.83	3.73	4.49
0.57-7.20	0.53-5.43	1.87-30.86	0.19-3.68	0.70-19.97	0.91-22.16
0.82	1.80	2.29	2.19	2.78	1.27
0.42-1.63	0.89-3.63	0.88-5.95	0.95-5.05	0.97-7.98	0.44-3.70
NI	2.24	NI	NI	NI	NI
	0.14-35.94				
1.38	1.17	0.59	0.85	0.43	0.50
0.56-3.36	0.47-2.91	0.18-1.88	0.27-2.67	0.11-1.63	0.13-1.93
1.51	1.45	0.96	0.96	0.64	0.66
0.34-6.61	0.37-5.75	0.19-4.77	0.18-5.27	0.10-4.15	0.11-3.96

or adolescence. these findings extend prior trajectory-based studies, as knowledge of heterogeneity in long-term offending trajectories within the early onset offender population is at present virtually absent. Charting delinquent development in children experiencing their first police contact prior to age 12, this study represents an important contribution to our understanding of distinct delinquent pathways of early starters into young adulthood, and how these different childhood-onset trajectories are related to demographic and neighborhood characteristics. This is important, as assumptions on the delinquent development of early onset offenders constitute an important cornerstone of criminological theory and drives policies and treatment of delinquent children. Revealing that delinquent development in early starters is highly heterogeneous

and often discontinuous, the current findings advance theory and policies as well as fuel future empirical work on offenders with an onset in childhood. Directions for future studies are described below.

Contrary to expectations of taxonomic theories, we found that over half of the early onset offenders did not come into contact with the police again over the 10 to 14 year follow-up period. Even among those early onset offenders who did re-offend, over 25% did so only sporadically. To the extent that childhood onset offending is taken to signal personal or familial characteristics that continuously increase the likelihood of offending (i.e., contemporary continuity), the (near) absence of re-offending in a large part of the cohort is unexpected. This finding also diverges from prior empirical results. Among early onset offenders in the youngest cohort of the Pittsburgh Youth Study for instance, only 20% desisted offending – as reported by child, parent or teacher – between ages 14 and 19 years (van Domburgh, Loeber, Bezemer, Stallings, & Stouthamer-Loeber, 2009). However, despite high levels of desistance, prevalence of offending in the current sample was still three times higher than that of the general Dutch population, as only 14% of a Dutch birth cohort was registered by the police between 12 and 22 years of age (Blokland, Grimbergen, Bernasco, & Nieuwbeerta, 2010). Thus, compared with the general population, childhood onset offenders are at increased risk of having a police contact at the ages of criminal responsibility. Moreover, compared with adolescent onset offenders (onset at age 12 years or above), Dutch childhood onset offenders (onset below age 12 years) report higher rates of property crime, vandalism, and violent offenses (Hoeve et al., 2015). Thus, although childhood onset does not predestine offenders to a frequent and prolonged criminal career, in line with the Moffitt taxonomy, the present study still found that an early onset of offending elevates the risk of becoming a chronic offender (see Moffitt, 1993).

Early onset offenders who did re-offend at higher than sporadic rates showed re-offending patterns that differed both in shape and nature of offending, emphasizing the importance of distinguishing between types of offenses when categorizing subgroups of offenders. In line with a study by Ward et al. (2010), we found two high-rate groups; one with a peak in late adolescence and one with a peak in offending in early adulthood. The adolescence peaked subgroup was the smallest subgroup with the highest average offending rates up to early adulthood. Future research could examine whether this finding still holds when childhood or adolescent onset offenders are followed into late adulthood, as adult-peaked high-rate offenders might have longer criminal careers. Regarding distributions of offense types, the current study, as well as the study by Ward et al. (2010), indicated that high-rate offenders committed relatively more property crime than other trajectory subgroups. In the current study, the high-rate adolescence peaked offenders also displayed increasing amounts of violent and sexual offenses.

Relating trajectories to demographic and childhood neighborhood characteristics revealed that males, non-Western participants, and participants residing in low SES neighborhoods below age 12 were likely to populate the more frequent re-offending pathways. These results are in accordance with the Moffitt-taxonomy (Moffitt, 1993, 2006), and with findings from previous trajectory-based studies on associations between gender (Block et al., 2010; Livingston et al., 2008), ethnicity (van Domburgh, Vermeiren, et al., 2009) and trajectory group membership.

3.4.1 Theoretical implications

Although some trajectories identified in this study resemble those hypothesized by taxonomic theories, there are also clear differences. Besides the non-recidivists, the low- and moderate-rate recidivists do not necessarily fit within Moffitt's taxonomy. Although their total number of offenses was higher than that of the sporadic recidivists, their offending rates were lower than that of high-rate subgroups.

The high-rate adolescence peaked recidivists come closest to resembling the classic life-course persistent offender, as they displayed high-rate re-offending patterns into adulthood. Furthermore, the distribution of types of crime among high-rate adolescence peaked offenders showed an increase in overall violent and sexual offenses over time. Their tendency to increasingly commit violent offenses seems to be in accordance with Moffitt's (1993) assumption that violent crimes make up an increasing part of total crime among high-rate chronic offenders. In contrast, the high-rate adult peaked offenders did not seem to disproportionately commit violent crimes toward the end of the observation period. Rather, property crime, vandalism, and traffic offenses made up a large part of total crime rates among high-rate adult peaked offenders. The process of cumulative continuity thus seems to especially apply to adolescence peaked offenders, causing escalation of offending in this trajectory subgroup.

In addition, the sporadic recidivists seem to resemble Moffitt's low-rate chronic group, as they showed intermittent patterns of delinquent behavior up to age 18. As predicted by Moffitt (2006), these sporadic offenders seemed to desist from committing property crime, vandalism, as well as violent and sexual offenses upon entering adulthood. According to Moffitt (2006), the low-levels of offending in adolescence would result from specific individual characteristics (depression and anxiety) that exclude sporadic offenders from deviant social peer groups. As a result, offenders in the sporadic trajectory subgroup would not be exposed to the same process of cumulative continuity as their high-level counterparts.

Finally, there is evidence of heterotypic continuity among all recidivist subgroups. With increasing age, vandalism made up a smaller part of the total

criminal repertoire for all trajectory subgroups. In contrast, the proportion of traffic offenses and the proportion of offenses including drugs and weapons generally increased. The decrease in vandalism most likely reflects a shift in age appropriate behavior (see Blokland & Palmen, 2012). Increases in traffic and drug and weapons violations predominantly signal increased opportunities for such behaviors with increasing age.

3.4.2 Limitations and recommendations

Although this study offers unique insights into the delinquent pathways of early onset offenders, some limitations need to be considered. First, this study used a first police registration below age 12 as a proxy for early onset offending. Although we expected that by doing so, a group was selected that shows stable patterns of disruptive behavior in childhood, it may have also caused the inclusion of subjects whose registration was more or less coincidental. Although one might think that this would especially be the case for subjects registered for committing vandalism below age 12, it was found that type of first offense was unrelated to trajectory group membership in the current sample. An important task for future research is to examine whether current findings can be replicated among other samples of childhood onset offenders – for example defined as confessing to having committed more than one delinquent act before age 12 in a self-report survey. In this way, the generalizability of current findings to other samples of early onset offenders can be examined.

Second, shapes of delinquent trajectories might have been influenced by our reliance on police registrations as a measure for offending. As a result, we lack information on delinquent behavior unknown to the police. In addition, it remains unclear whether participants were only suspects in the criminal case registered by the police or were eventually found guilty by a judge. On the contrary, police records have the advantage of being more reliable than self-report data regarding the timing of offenses as well as the occurrence of more serious offenses. In addition, police records contain more information on less serious offenses than conviction data, as minor offenses are less likely to end up in court.

Third, rates of re-offending might have been affected by the method used to correct for imprisonment. By calculating the number of offenses individuals might have committed without being imprisoned, we may have overestimated offending rates at the ages individuals were incapacitated. However, as completely ignoring information on imprisonment has shown to affect group shape and membership (see Piquero et al., 2001), our attempt at correcting for exposure time probably led to a more accurate estimation of offending trajectories than if we had disregarded information on criminal sanctions. Future research

may strive to include information on the actual dates individuals entered and left detention.

Fourth, the current study lacked information on non-criminal justice interventions early onset offenders may have been subjected to during the observation period. To the extent their childhood police contacts signaled severe behavioral or familial problems, it is likely that parents, schools, child protection services, and other professionals were actively trying to curb these youths' delinquent development. The observed trajectories in our study could thus have evolved either because or despite such efforts. If not for such interventions, low-rate recidivists might have developed into high-rate recidivists, while efforts to make high-rate recidivists refrain from further offending may have been absent or in vain. Relatedly, the association between these delinquent trajectories and personal and background characteristics may reflect differential availability of these interventions for certain demographic groups as much as the direct influences of these individual characteristics.

Finally, the multinomial model explained a limited amount of variance in group membership. Childhood characteristics may be insufficient to differentiate between (especially low-, moderate- and high-rate) offending trajectories into adulthood in a group of children with histories of delinquency. Future research should therefore make an effort to incorporate important time varying risk factors for offending, including non-criminal justice intervention efforts, which might explain more variance in group assignment.

3.4.3 Practical implications

Trajectories identified in the current study illustrate heterogeneity among early onset offenders in terms of the frequency and type of re-offending. This heterogeneity poses a challenge to policy-makers that focus on young offenders. The following key considerations need to be addressed in discussions on criminal justice interventions for early onset offenders.

To the extent that discontinuity of offending among a large share of early onset offenders (i.e., non-recidivist subgroup) does not result entirely from prevailing intervention policy, this finding indicates that intervening at a young age might be unnecessary for a large group of justice involved children. Intervening would be particularly troubling in the light of findings on the effects of interventions directed toward individuals at low-risk of re-offending. Although focusing intervention efforts on children at high-risk of re-offending has been shown to substantially reduce offending rates (Andrews & Dowden, 2006), targeting low-risk offenders might actually increase re-offending behavior (Lowenkamp & Latessa, 2002). It has therefore been suggested that low-risk youth should be diverted away from the juvenile justice system to avoid labelling (Campbell et al., 2019), and deviancy training (Lowenkamp, Latessa, & Holsinger, 2006). An overemphasis on the timing of a first police contact

may lead to inaccurate decisions on the appropriateness of managing early onset offenders in the criminal justice system. As yet, the causal mechanisms that either inhibit or promote persistent offending among low-risk children remain subject to future study.

Regardless of a lack of continuity in offending, previous research based on the Dunedin (Moffitt et al., 2002) and Cambridge (Jennings, Rocque, Fox, Piquero, & Farrington, 2016) samples has shown that early onset offenders who were unlikely to be involved in crime during adolescence experienced numerous psychological and social problems. Findings revealed that although offending rates were low, 'recovering' early onset offenders suffered from mental illness (Jennings et al., 2016; Moffitt et al., 2002), social isolation (Moffitt et al., 2002), psychopathy, poor home conditions, and poor intimate relation status (Jennings et al., 2016). Thus, although a large share of the current sample did not re-offend during the observation period, they are likely to be at increased risk of experiencing long-term negative life consequences and might benefit from some kind of (preventive) treatment targeting problems associated with the onset of offending in childhood.

As a small proportion of early starters continues to inflict substantial harm on others, preventing the progression along persistent offending pathways is crucial. Current findings suggest that such prevention efforts would be most viable when aimed at children residing in lower SES neighborhoods. Although studies have begun to explore which risk factors differentiate between trajectory subgroups of adolescent offenders (for reviews see Assink et al., 2015; Jolliffe et al., 2017), further research into factors differentiating between early onset trajectory subgroups is essential in considering policy regarding this specific and important offender population.

4 Using risk profiles to differentiate between offending trajectories of early onset offenders

ABSTRACT

Taxonomic theories suggest that risk exposure across life domains influences offending behavior throughout the lifespan. However, empirical knowledge on whether functioning across life domains can help explain heterogeneity in offending trajectories is scarce, especially in childhood. By combining rich survey and official crime data on 348 childhood onset offenders from the *Dutch Childhood Arrestees Study*, we examine associations between risk profiles and offending trajectories from age 12 to 20. Next to an a-priori defined group of non-recidivists (55%), group-based trajectory modeling identified four offending trajectories: low-rate desisting (14%), low-rate persisting (18%), high-rate desisting (5%), and high-rate persisting (8%). Latent profile analysis further identified three risk profiles based on individual, familial, peer, school, and neighborhood characteristics: a low-problem/impulsive (31%), cognitive- and neighborhood-problem (48%), and multi-problem group (21%). Multinomial regression analysis showed that low-problem children were least likely to persist in offending during follow-up. Compared to low-problem children, multi-problem children were at increased risk of following the low-rate persistent trajectory, while children with both cognitive and neighborhood problems were at increased risk of following the high-rate persistent trajectory. Results offer implications for research on the development of offending, and for crime control policies and interventions for child delinquents.

Key words
Early onset offenders, offending trajectories, risk profiles

4.1 Introduction

A rich line of research has revealed heterogeneity in the development of offending by identifying distinct offending trajectories (for reviews see Jennings & Reingle, 2012; Piquero, 2008). With the aim of providing extensive insight into longitudinal patterns of offending, trajectory-based studies categorize individuals into trajectory subgroups displaying distinct offending patterns across age (see Nagin, 2005). Grouping individuals with homogeneous offending patterns allows researchers to explore the development of and persistence in offending in a given sample. Overall, this body of literature has shown that distinct offending trajectories can be identified, differing in offending rates, trajectory length, and peak age of offending.

The identification of distinct offending trajectories has triggered large research efforts aimed at characterizing offenders following distinct trajectories, mainly by studying differences in exposure to singular risk factors of offending from the individual, familial, peer, school, and neighborhood life domains (e.g. Chung et al., 2002; Ward et al., 2010; Wiesner & Capaldi, 2003). This work offers considerable insight into risk factors differentiating trajectory subgroups (mostly the high-rate chronic trajectory subgroup) from non-offenders. Unfortunately, scholars conclude that singular risk factors are less helpful in differentiating between offenders populating distinct offending trajectories (Day et al., 2012; Laub et al., 1998; Mulvey et al., 2010; Sampson & Laub, 2003), as offenders in all derived trajectory subgroups are exposed to some level of risk in several life domains (e.g. Assink et al., 2015; Day et al., 2012; Ferrante, 2013; Jolliffe et al., 2017; Ward et al., 2010). As a result, there is a lack of scientific knowledge on which offenders follow which offending trajectory. Prospectively explaining heterogeneity in offending trajectories is particularly challenging among early onset offenders, as singular risk factors identified in childhood differentiate even less between offending trajectories than risk factors identified in adolescence (Day et al., 2012; Ward et al., 2010). It is important to increase our insight into the etiology of distinct offending trajectories within the high-risk offender population of early starters (Moffitt et al., 1996), as this increases our understanding of why some trajectories progress while others discontinue. This is of great importance for policy and intervention efforts, as judicial interventions have been found to reduce crime among high-risk youth, while increasing re-offense rates among low-risk youth (see Lowenkamp & Latessa, 2002). With more detailed knowledge, the match between children's needs and criminal justice interventions can be improved, leading to less crime in society.

Instead of focusing on singular risk factors, recent research in a variety of disciplines has greatly progressed insight into heterogeneity in outcomes (e.g., youth depression, internet addiction, adolescent substance use, and adolescent internalizing symptoms) by focusing on differences in exposure to combinations of risk through the identification of risk profiles (J. R. Cohen

et al., 2015; Li et al., 2017; Simpson, Vannucci, & Ohannessian, 2018). Rather than estimating associations between singular risk factors and outcome measures, individuals are assigned to mutually exclusive subgroups exposed to similar combinations of risk. This approach allows researchers to adopt a holistic approach to risk exposure by simultaneously examining numerous types of risk, while accounting for possible confounding of singular risk factors. Together, this line of literature highlights the utility of risk profiles, as it reveals that distinct patterns of risk are differentially associated with a variety of outcome measures (e.g., J. R. Cohen et al., 2015; Li et al., 2017; Simpson et al., 2018).

The identification of risk profiles may also provide additional insight into the etiology of offending trajectories, as it is widely assumed that risk factors of offending do not operate in isolation, but rather co-occur and are often mutually reinforcing (e.g. Farrington & Welsh, 2008; Moffitt, 1993; van Hazebroek, Wermink, et al., 2019). Theory (Moffitt, 1993, 2006) and prior work (Assink et al., 2015; Jolliffe et al., 2017; Mulvey et al., 2010) suggest that we might increase our ability to differentiate between subgroups of offenders by adopting a holistic view on risk exposure, and study how distinct combinations of risk may differentially impact offending across age. However, research has not yet examined this possibility directly.

The current study therefore attempts to expand our understanding of heterogeneity in the development of offending by exploring whether and to what extent variation in exposure to risk across life domains can help explain heterogeneity in offending trajectories among early onset offenders. Specifically, we study the extent to which distinct offending trajectories and risk profiles can be identified in a group of early onset offenders. These trajectories and risk profiles are subsequently used to study whether children assigned to specific risk profiles in childhood are at increased risk of following specific offending trajectories. By addressing this issues, the current study intends to advance what is known about the etiology of patterns of offending across the lifespan.

4.1.1 Theoretical framework

Much research on offending trajectories has been guided by Moffitt's (1993, 1997) developmental taxonomy, arguing that distinct offending trajectories can be identified in the general population that differ in frequency and longitudinal pattern of offending. Building on a large body of literature, Moffitt (1993) hypothesized that the population of offenders can be divided into two subgroups, labeled adolescence-limited and life-course-persistent or *high-level chronic* offenders. Adolescence-limited offenders are theorized to engage in low-rate offending during adolescence. As such, adolescence-limited offenders are thought to abstain from offending in childhood, and desists from crime

before reaching adulthood. In contrast, life-course-persistent offenders are assumed to start offending in childhood and commit crimes at a high rate into adulthood. Based on additional empirical research testing for possible offender types (Moffitt et al., 1996; Nagin et al., 1995; Raine et al., 2005), Moffitt (2006) later added a third subgroup with an onset in childhood and persistent yet low offending rates during adolescence, labeled *low-level chronic* offenders.

Moffitt (1993, 2006) assumes that differences in offending trajectories result from varying etiological underpinnings of offending across offender subgroups (Moffitt, 1993, 2006). Adolescence-limited offenders are theorized to be affected by peer-related risk factors (Moffitt, 1993), as they mimic delinquent peers to demonstrate autonomy while experiencing a gap between biological and social maturity. They are expected to desist from crime when adult roles become available (Moffitt, 1993). In contrast, life-course-persistent or high-level chronic offenders are assumed to experience problems in multiple life domains. Biological deficits – caused by peri/prenatal problems such as maternal drug use or birth complications – are thought to manifest as cognitive deficits, impulsivity, and hyperactivity. Biological vulnerability is assumed to co-occur with familial disadvantage, and their combination is thought to place children at risk of offending. While individual and familial risk factors are predicted to remain relatively stable and influence behavior throughout the life-course (i.e., contemporary continuity), tendencies towards offending are assumed to escalate (i.e., cumulative disadvantage) as children are introduced to peer-, school-, and neighborhood-related risk (Moffitt, 1993, 1997). Low-level chronic offenders are theorized to share many individual (e.g., low intelligence), and familial (e.g., family adversity, and parental psychopathology) risk factors with the life-course-persistent group (Moffitt, 2006). However, Moffitt (2006) argues that low-level chronics are more likely to suffer from isolating individual characteristics (e.g., depression and anxiety) than their high-level counterparts. As a result, low-level chronic offenders are excluded from deviant social peer groups, and display low offending rates during adolescence.

In sum, Moffitt (1993, 2006) emphasizes the importance of exposure to distinct combinations of risk factors when studying heterogeneity in long-term offending behavior (Moffitt, 2006). As specific theoretical assumptions postulated by Moffitt (1993, 2006) can be extended based on prior empirical work (see Jennings & Reingle, 2012; van Domburgh, Vermeiren, et al., 2009), we first discuss prior studies focused on identifying offending trajectories and risk profiles in offender samples, before offering expectations on offending trajectories, risk profiles, and their association.

4.1.2 Prior research

By far, most trajectory-based studies have been conducted among general population and general offender samples, and identified between two and

seven trajectory subgroups, with three or four being the most common (for reviews see Jennings & Reingle, 2012; Piquero, 2008). In accordance with theory, the majority of trajectory-based studies identified (1) a very low/non-offending group (i.e., near-zero in offender samples); (2) an adolescence-peaked group, whose offending peaks at age 16 and then declines to zero in early adulthood; and (3) one or more persistent offending groups, that may differ in peak age yet have higher offending rates than all other groups at every age. A theoretically unanticipated group of late onset offenders – starting in adolescence and persisting into early adulthood – has also been identified.

The few prior studies that were able to explore which trajectories were populated by early onset offenders confirmed theoretical expectations, by showing that early onset offenders generally populate the most chronic trajectory subgroup (Allard et al., 2017; Broidy et al., 2015; Day et al., 2012). Despite the manifest theoretical and practical importance of identifying distinct re-offense patterns among child delinquents, studies on trajectories among early onset offenders are scarce (but see van Domburgh, Vermeiren, et al., 2009). Importantly, and in contrast to theory (Moffitt, 1993, 2006), prior work showed that even among child delinquents various offending trajectories can be identified, ranging from low- to high-level re-offending trajectory subgroups (van Domburgh, Vermeiren, et al., 2009).

In various samples, several singular risk factors have been associated with trajectory subgroup membership, but these factors mostly differentiated between offenders in the high-rate chronic trajectory and non- or sporadic offenders. Studies conducted among juveniles from offender and at-risk samples, as well as the limited work on early onset offender samples, revealed that offenders in high-rate/chronic offending trajectories can be differentiated from non-offenders based on heightened exposure to risk in several life domains. Risk factors characterizing high-level trajectories include increased levels of impulsivity (Baglivio et al., 2015), attention problems (Wiesner & Capaldi, 2003), substance use (versatility) (Corrado, McCuish, Hart, & DeLisi, 2015), and sensation seeking/'being adventurous' (Jennings et al., 2019; Laub et al., 1998). In addition, offenders in high-rate trajectory subgroups have been found to suffer from inconsistent parenting (Wiesner & Capaldi, 2003), low parental supervision/neglectful parenting (Hoeve et al., 2008; Monahan & Piquero, 2009; Wiesner & Capaldi, 2003), parental delinquency (van der Geest et al., 2009), deviant peers (Baglivio et al., 2015; Chung et al., 2002; Monahan & Piquero, 2009; van der Geest et al., 2009), and neighborhood disadvantage (van Hazebroek, Blokland, et al., 2019).

While offenders in high-rate trajectory subgroups are generally exposed to highest levels of risk, many other trajectory subgroups are – to some extent – characterized by exposure to similar types of risk. This is evidenced by studies reporting that many risk factors (i.e., impulsivity/hyperactivity, low intelligence/school success, antisocial family members, poor parental supervision, and neighborhood disadvantage) characterized offenders assigned to various

trajectory subgroups (e.g. Assink et al., 2015; Baglivio et al., 2015; Day et al., 2012; Jennings et al., 2019; Jolliffe et al., 2017; Wiesner & Capaldi, 2003). As a result, many other studies failed to differentiate between offending trajectories based on theoretically relevant risk factors (e.g., low IQ, substance use, depressive symptoms, neighborhood conditions, parental criminality, parental supervision, childrearing practices, proportion of arrested friends) (e.g., Laub et al., 1998; Sampson & Laub, 2003).

One limitation of this line of research however, is that the influence of risk factors on offending is studied in isolation, making it impossible to include information on functioning across life domains. While studying singular risk factors allows researchers to assess their relative impact on outcome measures, it fails to account for possible additive and interactive effects among multiple risk factors. As a result, it remains unclear whether exposure to specific combinations of risk might especially increase the odds of prolonged delinquent involvement.

In order to integrate influences from multiple life domains, a growing body of research focused on a variety of adverse psychosocial outcomes has identified naturally occurring subgroups of individuals based on similar experiences across life domains (e.g., Dunn et al., 2011; Li et al., 2017; Simpson et al., 2018). This integrated approach allows researchers to study the collective impact of (the absence of) risk in several life domains. As such, it represents an important departure from studies that simultaneously examine relationships between singular factors and outcomes that inevitably weigh relationships against each other. The body of evidence surrounding the identification of risk profiles supports the assumptions that there are subgroups of individuals exposed to distinct patterns of risk. While the number of identified subgroups differs across studies, most studies identified a group characterized by limited exposure to all risk factors, and a group with relatively high scores on all risk factors. In general, studies showed that individuals in the low-risk group have better adjustment outcomes than individuals in the high-risk group. Importantly, prior work highlights the utility of risk profile identification as they provided complementary information to more traditional models by revealing associations between specific patterns of risk and variation in outcome measures (e.g., J. R. Cohen et al., 2015; Dunn et al., 2011; Li et al., 2017).

To date, few studies have aimed to identify risk profiles within offender populations. These studies revealed heterogeneity in patterns of risk across populations of early onset (Geluk et al., 2014), adolescent (e.g., T. Brennan, Breitenbach, & Dieterich, 2008; Dembo et al., 2008; Hilterman, Vermunt, Nicholls, Bongers, & van Nieuwenhuizen, 2019; Lopez-Romero et al., 2019; Schwalbe et al., 2008), and adult offenders (Taxman & Caudy, 2015). Risk profiles of offenders could be distinguished based on differences in levels of risk (i.e., quantitative differences in risk exposure), differentiating between low-, moderate-, and high-risk subgroups (Dembo et al., 2008; Hilterman et al., 2019), as well as differences in exposure to specific combinations of risk

factors (i.e., qualitative differences in risk exposure), differentiating between subgroups exposed to similar levels yet distinctive combinations of risk (e.g. T. Brennan et al., 2008; Lopez-Romero et al., 2019; Mulder, Brand, Bullens, & van Marle, 2010; Onifade et al., 2008; Schwalbe et al., 2008; Taxman & Caudy, 2015).

Importantly, studies showed that quantitative as well as qualitative differences in patterns of risk help explain heterogeneity in offending. For instance, studies showed that high risk groups are most likely to re-offend (Campbell et al., 2019; Hilterman et al., 2019; Taxman & Caudy, 2015), and that they tend to commit more future crimes (Lopez-Romero et al., 2019). Additionally, studies showed that youth that share cumulative risk levels yet differ in their patterns of risk exposure can differ in their re-offending rates (Onifade et al., 2008). As such, the identification of risk profiles has proved superior to original coding schemes focused only on overall level differences in risk exposure, and improved our ability to predict future offending (Campbell et al., 2019; Onifade et al., 2008).

While studies that identified risk profiles of offenders have progressed our understanding of variability in offending, the majority of these studies covered short (i.e., at the most two years) follow-up periods (Campbell et al., 2019; Dembo et al., 2008; Lopez-Romero et al., 2019; Onifade et al., 2008; Schwalbe et al., 2008; Taxman & Caudy, 2015; van Domburgh, Geluk, Jansen, Vermeiren, & Doreleijers, 2016). Moreover, by estimating offending as a dichotomous or continuous measure, none of the previous studies captured differences in persistence and desistance in offending over time. Lastly, prior work did not account for incarceration time, and might have therefore underestimated offending rates, especially among frequent offenders (Piquero et al., 2001). Up to date, it therefore remains unclear if, and to what extent, risk profiles can be used to explain heterogeneity in the development in offending across the lifespan.

4.1.3 The current study

The goal of the current study was to synthesize the interrelated lines of research on the identification of offending trajectories on the one hand and risk profiles on the other hand. In doing so, the current study aims to overcome some of the limitations of prior work. First, by studying associations between assignment to risk profiles and offending trajectory subgroups, we characterize offenders populating distinct trajectories based on exposure to combinations of risk rather than singular factors. Theory and converging lines of research suggest that such a holistic view of risk exposure is important in trying to characterize offenders following distinct offending trajectories. We explore this line of reasoning by combining the identification of risk profiles and offending trajectories, allowing us to account for functioning across life

domains, while avoiding overestimations of associations between singular risk factors and offending-related outcomes. Second, we identify risk profiles based on a large array of risk factors associated with offending behavior from all life domains, by utilizing unique data from the *Dutch Childhood Arrestees Study* on children registered by the police for committing an offense before the age of twelve. These data include information on theoretically important risk factors from individual (e.g., prenatal problems, intelligence, hyperactivity), familial (e.g., poor parental supervision, parental mental health problems, familial delinquency), peer (e.g., peer rejection, affiliation with deviant peers), school (e.g., poor school achievement) and neighborhood (e.g., low socio-economic status) domains. Third, as data on offending behavior covers a lengthier follow-up period, we are able to estimate the development of offending behavior across a longer period of the lifespan than all of the previous studies. As a result, we are better able to study desistence and persistence in offending. In estimating offending trajectories, we control for decreased exposure time caused by spells of incarceration, thereby avoiding underestimations of offending frequency.

Based on the Moffitt-taxonomy (Moffitt, 1993, 2006), and studies on offending trajectories and risk profiles of offenders, expectations can be formulated regarding the shape of trajectories, the content of risk profiles, and their association. First, Moffitt (1993, 2006) expects early onset offenders to continuously engage in offending behavior, displaying either low (low-level chronic trajectory) or high (high-level chronic trajectory) offending rates during adolescence. Regarding risk exposure, Moffitt (2006) expects that early onset offenders suffer from increased risk in multiple life domains due to adverse individual, familial, peer, school, and neighborhood characteristics. While Moffitt (2006) would not expect differences in levels of risk in early onset offenders, heterogeneity in combinations of risk factors is expected. Specifically, Moffitt (2006) distinguishes between a group of early starters experiencing heightened depression and anxiety and consequently social isolation, while a second group of early starters would be less characterized by these isolating features. The group experiencing isolating individual characteristics is expected to display lower offending rates, resulting in a low-level chronic trajectory. Children suffering from fewer isolating features are expected to demonstrate a high-level chronic offending trajectory. Based on prior work however – revealing quantitative in addition to qualitative differences in risk exposure (Lopez-Romero et al., 2019; Onifade et al., 2008; Schwalbe et al., 2008), as well as a group of early onset offenders who did not or only sporadically re-offended during follow-up (see van Domburgh, Vermeiren, et al., 2009) – we additionally expected to identify a group of early onset offenders exposed to relatively low levels of risk, re-offending at a decreasing rate with age.

4.2 Method

4.2.1 Participants and procedures

The data used in the current study originate from the *Dutch Childhood Arrestees Study*, a larger study focused on children registered by the police for committing a first offense prior to the age of 12. While offenses committed prior to the age of 12 (i.e., the age of criminal responsibility in the Netherlands) are not registered in national crime statistics, they are documented in local police registration systems. These registration systems were used to select children registered for displaying behavior that could have been prosecuted or fined if displayed by someone older than 12 years. Detailed descriptions of this study have been published previously (see Geluk et al., 2014; van Domburgh, Vermeiren, et al., 2009). The current study was based on children who were followed up in order to gather information on risk exposure (N = 348). The sample is largely male (n = 302), and about half of the participants (n = 184) is of non-Dutch origin. Participants were registered by the police between 2003 and 2005 (M_{age} = 9.78, SD = 1.44), for committing vandalism (58.7%, n = 178), property crime (27.4%, n = 83), and violent offenses (13.9%, n = 42).

The current analysis used data collected during the first measurement wave of the study, when children were between 5 and 13 years old (M = 10.10, SD = 1.51, *Median* = 11). In order to gather information on a range of risk factors from multiple life domains, questionnaires and interviews were administered to the children and their primary caretakers (hereafter referred to as 'parents') at participants' homes. For the aim of the current study, we linked data from several sources to the Childhood Arrestees data. Data from the Dutch police registration system Herkennings Dienst Systeem (HKS) was used to measure offending over a 3 to 11-year follow-up period, from February 2004 (when the oldest participants turned 12) until February 2015. Additionally, information on mortality and criminal sanctions were collected from the Research and Policy Database Judicial Documentation ('Onderzoek- en Beleidsdatabase Justitiële Documentatie', OBJD) of the Research and Documentation Centre of the Ministry of Justice (WODC) to obtain information on exposure time or 'street time' (i.e., the amount of time participants were free to engage in offending) (see Piquero et al., 2001).

4.2.2 Measures

Measures used for trajectory modeling

Offending was defined as every entry in the Dutch police registration system HKS during follow-up. Frequency of offending across age was calculated using offender's date of birth and registration dates of offenses. The age at the end of follow-up ranged from 15 to 23 (M = 20.28, SD = 1.57), due to differences

in age at first arrest and year of inclusion. We limited the trajectories to ages for which data was available on at least 100 individuals, and therefore estimated offending trajectories up to age 20. When participants had not reached age 20 during follow-up, non-observed years were coded as missing to prevent contributions to trajectory estimations (see also van der Geest et al., 2009).[1] Between the age of 12 and 20, less than half (44.5%) of early onset offenders in the current sample was registered by the police for displaying offending behavior.

As there was no mortality during follow-up, we controlled for incarceration time by estimating the number of months individuals were not incarcerated within a year's time period. For example, individuals who had been incarcerated for 1 month at age 19, were coded 'free' for 11 months at that age (see also Piquero et al., 2001). We corrected for spells of incarceration by including exposure time (i.e., not incarcerated) as a time-varying covariate in the analysis (see also Mulvey et al., 2010; Piquero et al., 2001). About thirty percent of the recidivists had been incarcerated in the follow-up period (n = 46). The average incarceration time was 6.6 months, and none of the participants was incarcerated for the entire follow-up period.

Measures used as profile indicators

Information on risk factors from individual, familial, peer, school, and neighborhood domains was collected when children were between 5 and 13 years old (M = 10.10, SD = 1.51, $Median$ = 11).[2] In order to facilitate the interpretation of risk profiles for different groups, levels of risk were determined based on norm scores whenever information on norm scores was available for a given questionnaire (see also Decuyper et al., 2013). When norm scores were unavailable, we calculated average scores for continuous profile indicators (see also T. Brennan et al., 2008; Geiser, Okun, & Grano, 2014). Higher scores on profile indicators are indicative of more problems in that specific area. The 21 profile indicators are described in Appendix A, including instruments, informants, sample items, response scales, internal consistency estimates, and final measurement levels of profile indicators. Information on measures per life domain and overall sample characteristics are provided below (see Appendix B for descriptive statistics).

1 Of the 95 (27%) participants who did not reach the age of 20 during the observation period, 48 (13.8% of the total sample) participants reached the age of 19, and 73 (21.0% of the total sample) reached the age of 18 during follow-up. Assignment to trajectory subgroups did not differ between participants who did and did not turn 20 during follow-up, $\chi^2(4) = 7.57$, p = .11.

2 Because of potential problems with comprehensibility of the questionnaires, due to children being younger than eight years old or having below average verbal IQs (measured using the Wechsler Intelligence Scale for Children-Revised; Wechsler, 1974), self-report questionnaires from a total of 46 participants were coded as missing.

Individual

In the individual domain, we measured biological (indicators 1 and 2), cognitive (indicator 3), emotional (indicators 4 and 5), and behavioral (indicators 6 through 9) problems. Biological risk factors included whether children had suffered from *prenatal substance exposure*, and whether children's mothers had experienced *prenatal complications*. The cognitive indicator *intelligence* was categorized into seven categories using norm scores ranging from very high (IQ ≥ 130) to very low (IQ ≤ 69). Emotional risk factors encompassed a four-fold classification of *emotional problems* (i.e., close to average; slightly raised; high; very high), and a three-fold classification of *depression* (i.e., low; at risk; clinical range). Behavioral risk factors included a classification of levels of *hyperactivity/inattention* (i.e., close to average; slightly raised; high; very high), the number of *substance types* the child had ever used, and mean scores signifying levels of *sensation seeking behavior*. Levels of *social understanding difficulties* were divided into seven categories using norm scores ranging from very low to very high. Emotional problems, depression, and social understanding difficulties can be argued to represent isolating individual characteristics. While descriptive statistics showed that overall levels of risk in the individual domain were slightly elevated, still a sizeable share of the current sample suffered from substantial problems. For instance, about a third of the sample had a very low (IQ = 79) to extremely low (IQ = 69) estimated IQ, a fourth of the sample experienced high to very high emotional problems, and 10.8% scored in the clinical range of depression.

Familial

We used *parenting characteristics* (indicators 10 through 13), *familial delinquency* (indicator 14), *parental mental health problems* (indicator 15), and *parenting stress* (indicator 16) as measures of risk in the family domain. Children's perception of parenting characteristics was determined by calculating mean scores on the following subscales: *parental neglect* (opposite of parental supervision), *inconsistent parenting*, *parental indifference* (opposite of parental warmth), and *uninvolved parenting* (opposite of parental involvement). We calculated norm scores on a scale from 0 (very low) to 6 (very high) to specify levels of parental mental health problems and parenting stress. Appendix B shows that children barely experienced adverse parenting, and that parents experienced below average to average levels of mental health problems and parenting stress. Still, a fourth of children's parents suffered from high to very high levels of mental health problems and parenting stress.

Peers

Peer relationship problems were measured as mean scores indicating levels of *bullying victimization* (indicator 17), and *affiliation with antisocial peers* (indicator 18). Descriptive statistics showed that children were sometimes bullied, and virtually had no antisocial friends.

School
Poor school achievement (indicator 19) was measured as failing a reading test. A child was assumed to have failed the reading test, when the number of words the child could read within one minute was one year behind their appropriate level (44% in the current sample).

Neighborhood
Postal codes were used as a proxy for *neighborhood levels of socio-economic status* (indicator 20) and *urbanization* (indicator 21). Information on both was available in quintiles, with highest scores indicating very low levels of socio-economic status (SES) and very high levels of urbanization respectively. As shown in Appendix B, about half of the sample grew up in neighborhoods with low to very low SES levels, and about 75% resided in highly urban areas.

4.2.3 Analytical approach

The first step in our analysis was to estimate group-based trajectory models using STATA Trajectory Procedure in STATA 13 (Jones & Nagin, 2013; Nagin, 2005). We fitted cubic shaped trajectories using a zero-inflated Poisson model, allowing us to account for the large number of zero offenses in the data (Lambert, 1992). In estimating offending trajectories, parameters defining the level and shape of offending trajectories were allowed to vary freely across groups. The trajectory model was solely based on participants with at least one police registration during the observation period (see also Broidy et al., 2015), as the risk of low-level recividists being pulled into the non-recidivists group would have complicated comparisons between non- and low-rate recidivists. Wald (χ2-based) tests were conducted to explore differences in intercepts and cubic slopes across trajectory subgroups.

Second, we conducted latent profile analyses (Collins & Lanza, 2013) in MPlus 8.1 (Muthén & Muthén, 1998-2010) to determine the optimal number of groups exposed to distinct combinations of profile indicators. We used the full maximum likelihood procedure, allowing for model estimates to be calculated based on information provided by participants with complete and partially complete data.[3] Differences in scores on profile indicators across risk profiles were examined through analysis of variance (ANOVA) and chi-square tests for continuous and dichotomous indicators respectively. Significant overall

3 Of the 348 participants, 220 participants (63.2%) had missing data on at least one of the profile indicators, with 28% of the participants having missing data on one profile indicator and 80% of the participants having missing data on no more than four profile indicators. Whether or not participants had missing data, was not associated with eventual assignment to risk profiles, $\chi^2(2) = 3.41$, $p = .18$.

effects were followed by pairwise Bonferroni (equal variances assumed) or Tamhane (equal variances violated) post-hoc comparisons.

Third, participants were assigned to their most likely trajectory and risk profile – which is acceptable when assignment accuracy is above 80% (Clark & Muthén, 2009) – and both were used as observed variables in follow-up analyses. Assignment to trajectory subgroups across risk profiles (dummy-coded) was examined by using trajectory subgroup membership as a multiple nominal outcome in a series of multinomial logistic regression analyses.

4.2.4 Group assignment and diagnostics

In order to identify the optimal trajectory model, we estimated models with up to six groups and compared their fit (see upper half of Table 4.1). Model fit indices provided support for a four-group trajectory model, as the relative change in the Bayesian Information Criterion (BIC; Schwartz, 1978) decreased after the identification of the four-group model.[4] Additionally, average posterior probabilities (exceeding .80) and OCC values (exceeding 5.0) of the four-group model indicated adequate assignment accuracy (Nagin, 2005, 2010). A five-group solution was not preferred, as the fifth trajectory subgroup consisted of a very small share of participants (2.6%), who were conceptually embodied by a larger trajectory in the four-group model.

Next, fit indices for latent profile models with up to six groups were evaluated to identify the optimal risk profile solution (see bottom half of Table 4.1). The Lo-Mendell-Rubin likelihood-ratio test of model fit (LMR; Lo, Mendell, & Rubin, 2001) indicated that the model with three risk profiles was preferred, as adding a fourth group did not statistically improve model fit.[5] The three-group model also performed well on classification accuracy. The three-group model was therefore chosen for further analyses.

We will describe the four trajectory subgroups and three risk profiles among early onset offenders in the next section.

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4 The Bayesian Information Criterion (BIC; Schwartz, 1978), and the Akaike Information Criterion (AIC; Akaike, 1998) did not reach a minimum in the current study (see also Blokland et al., 2005), and therefore failed to clearly identify the best solution.

5 Nylund, Asparouhov, and Muthén (2007) suggest that a first non-significant LMR-test is a good indictor to stop increasing the number of groups. BIC values and the Bootstrap Likelihood Ratio Test (BLRT; McLachlan & Peel, 2000) failed to distinguish between the models identified in the current study, as they continued to prefer each model with an additional group (see also Geiser et al., 2014).

Table 4.1: Fit statistics of risk profiles and offending trajectories

#TRAJ[1]	BIC	2(ΔBIC)[2]	AIC				Lowest profile probability[3]	OCC[4]	Trajectory subgroup membership (*n*)
1	-1741.20		-1733.59				1	-	155
2	-1547.66	387.08	-1530.92				.96	22; 54	111; 44
3	-1534.44	26.44	-1508.57				.83	8; 13; 54	70; 43; 42
4	**-1519.82**	**29.24**	**-1484.83**				**.83**	**9; 11; 40; 73**	**62; 47; 27; 19**
5	-1513.52	12.6	-1469.39				.79	6; 60; 21; 86; 565	82; 23; 23; 18; 9
#LP[5]	BIC	2(ΔBIC)	AIC	LMR (*p* value)[6]	BLRT (*p* value)[6]	Ent	Lowest profile probability[3]		Risk profile membership (*n*)
1	14683.76		14537.37	-	-	-	1		348
2	14296.83	773.86	14065.70	.01	<.001	.89	.91		268; 80
3	**14231.89**	**129,88**	**13916.01**	**.01**	**<.001**	**.83**	**.92**		**167; 107; 74**
4	14180.46	102,86	13779.83	.45	<.001	.88	.76		206; 60; 42; 40
5	13632.18	1096.56	13146.80	.24	<.001	.90	.91		134; 88; 61; 49; 16

Note: Bold text represents model fit indices for final group-model.
#TRAJ = number of offending trajectories in estimated model; BIC = Bayesian Information Criterion; AIC = Akaike Information Criterion; #LP = number of risk profiles in estimated model; LMR = Lo-Mendell-Rubin Test; BLRT = bootstrap likelihood ratio test; Ent = Entropy.
[1] *N* = 155: participants with a police registration during follow-up. Fit statistics for the one- through five-group models are reported, as proper solutions for models with (more than) six groups could not be obtained.
[2] ΔBIC indicates the relative change in BIC values
[3] Average posterior probabilities above .70 indicate satisfactory assignment accuracy (Nagin, 2010).
[4] OCC > 5.0 indicates high assignment accuracy (Nagin, 2005).
[5] *N* = 348. Fit indices for models with up to five groups are displayed, as the six-group model failed to converge properly. We used a set of 600 random starting values to estimate each of the risk profile models (see also Mokros et al., 2015), and mean values were estimated independently within each profile.
[6] Significant LMR and BLRT tests indicate that the model with an additional subgroup (the *k*-group model) constitutes an important improvement over the *k* – 1 group model (Lo et al., 2001; Nylund et al., 2007; Vuong, 1989).

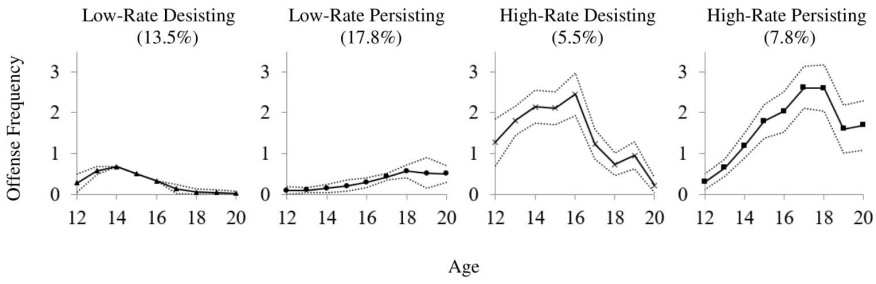
4.3 Results

4.3.1 Offending trajectories

Figure 4.1 displays the offending trajectories of the trajectory subgroups that were identified in addition to the a-priori defined group of non-recidivists (NON) (55.5%, *n* = 193): low-rate desisting (LR-D) (13.5%, *n* = 47), low-rate persisting (LR-P) (17.8%, *n* = 62), high-rate desisting (HR-D) (5.5%, *n* = 19), and high-rate persisting (HR-P) (7.8%, *n* = 27). Wald tests were non-significant (see Table 4.2), but the four offending trajectories clearly differed in offending rates, trajectory lengths, and peak age of offending (see Figure 4.1).[6]

6 Furthermore, trajectory subgroups differed in exposure to combinations of childhood risk factors, as reported below, suggesting that these trajectories should not be combined (see also Hoeve et al., 2008).

Figure 4.1: Longitudinal offending trajectories for the four-group model from age 12 to 20 corrected for time spent incarcerated



Mean offending rates were low in both the LR-D (M = 0.29, SD = 0.18) and LR-P (M = 0.32, SD = 0.20) subgroups, indicating that participants assigned to these trajectories committed one offense every three years. In contrast, average offending rates were high in both the HR-D (M = 1.61, SD = 0.74) and HR-P (M = 1.58, SD = 0.62) groups, demonstrating a level difference of 1.30 offenses per year between the low- and high-rate groups. Additionally, the desisting trajectories differed from their persisting equivalents in terms of development of offending. The LR-D group showed a rise and decline in offending rates with age, resembling the standard aggregated age-crime curve. As such, the LR-D group had the shortest offending trajectory, with a peak age of offending in early adolescence and declining offending rates towards abstinence around age 18. In contrast, the LR-P trajectory subgroup committed very few offenses until around age 15 and showed a (low) peak at age 18. Among the high-rate offenders, the HR-D group displayed high initial levels of offending that increased somewhat up to age 16, before steadily decreasing towards age 20. The HR-P group however, showed lower levels of offending at age 12, while offense rates rapidly increased, peaked around age 17-18, and persisted at a high rate into young adulthood.[7]

7 Regarding crime mix, follow-up analysis showed that property crime was the most common offense type in all trajectory-subgroups (over 40% of all criminal law offenses). Vandalism made up a relatively large part of total crime in both desisting groups (about 30% as opposed to 20% in the persisting groups). The LR-D group committed a relatively large amount of violent and sexual offenses (25%) compared to the other trajectory-subgroups (14%), possibly because the LR-D group did not continue to commit drug, weapon, or traffic offenses. Traffic offenses are relatively common in the LR-P group (10%), compared to other trajectory-subgroups (less than 4%).

Table 4.2: Wald tests testing differences between intercepts and cubic slopes across trajectory subgroups

	LR-P vs. LR-D	*HR-D vs. LR-D*	*HR-P vs. LR-D*	*HR-D vs. LR-P*	*HR-P vs. LR-P*	*HR-P vs. HR-D*
Intercepts	0.30	2.22	0.32	0.33	0.29	1.86
Lineair slopes	0.63	3.66	2.15	0.49	1.16	2.04
Cubic slopes	2.69	1.75	2.74†	0.46	0.56	0.45

Note: Last group is reference category.
LR-D = low-rate desister (n = 47); LR-P = low-rate persister (n = 62); HR-D = high-rate desister (n = 19); HR-P = high-rate persister (n = 27).
†p < .10, *p < .05 **p < .01, ***p < .001.

4.3.2 Risk profiles

As previously described, three risk profiles were identified in our data. Based on post-hoc comparisons between profiles (see Table 4.3 and Figure 4.2), the risk profiles were labelled as follows: (1) low-problem/impulsive group (30.7%, n = 107), (2) cognitive- and neighborhood-problem group (48.0%, n = 167), and (3) multi-problem group (21.3%, n = 74).

Profile 1 consisted of children displaying overall lowest scores on profile indicators yet elevated levels of hyperactivity/inattention and sensation seeking, and was therefore labelled as *low-problem/impulsive group*. Specifically, low-problem children had average estimated IQs (IQ = 90-109), and close to average emotional problems. Additionally, children assigned to Profile 1 were not depressed, had not used substances before the age of 12, and experienced very low to low levels of social understanding difficulties. Regarding familial risk, results revealed that members of Profile 1 hardly ever experienced adverse parenting characteristics, and that parents experienced low to average mental health problems and parenting stress. Furthermore, low-problem children had no antisocial friends, and resided in neighborhoods with average to high SES and average urbanization levels. However, besides elevated levels of hyperactivity and sensation seeking, low-problem children scored close to overall sample estimates on prenatal substance exposure, prenatal complications, and school achievement. Lastly, levels of familial delinquency were low compared to Profile 2 and Profile 3, yet still 17% of low-problem children had a delinquent family member.

Profile 2 encompassed children with the lowest IQs, residing in the most disadvantaged neighborhoods, and was therefore labeled as *cognitive- and neighborhood-problem group*. Specifically, children assigned to Profile 2 had well below (IQ = 70-79) to below (IQ = 80-89) average estimated IQs, and resided in low to very low SES and highly urban neighborhoods. Besides intelligence and neighborhood characteristics, Profile 2 differed from Profile 1 in terms of lower scores on hyperactivity/inattention and sensation seeking, yet higher

Table 4.3: Parameter estimates and prevalence rates for the total sample and across risk profiles

Risk Profile Indicator	Range	Sample	Risk Profiles				
			Profile 1 n (%) 107 (30.7)	Profile 2 n (%) 167 (48.0)	Profile 3 n (%) 74 (21.3)	Profile Comparison	Post Hoc Comparison
1 Prenatal Substance Exposure (%)	0, 1	36.1	29.9	36.5	40.5	$\chi^2(2) = 2.86$	
2 Prenatal Complications (%)	0, 1	42.1	43.9	37.7	43.2	$\chi^2(2) = 1.58$	
3 Low Intelligence (N)	0-6	3.96	3.21	**4.45**	*3.91*	$F = 41.19^{***}$	2 > 3 > 1
4 Emotional Problems (N)	0-3	0.74	0.30	*0.59*	**1.69**	$F = 52.68^{***}$	3 > 2 > 1
5 Depression (N)	0-2	0.34	0.07	*0.28*	**0.92**	$F = 24.24^{***}$	3 > 2 > 1
6 Hyperactivity/Inattention (N)	0-3	0.91	*0.77*	0.44	**2.21**	$F = 80.09^{***}$	3 > 1 > 2
7 Substance Use (M)	0-3	0.27	0.28	0.20	*0.43*	$F = 4.54^{*}$	3 > 2; 1 = 2, 3
8 Sensation Seeking (M)	0-4	1.59	*1.75*	1.31	*1.93*	$F = 16.65^{***}$	1, 3 > 2
9 Social Understanding Difficulties (N)	0-6	1.40	0.64	0.75	**3.81**	$F = 289.40^{***}$	3 > 1, 2
10 Parental Neglect (M)	0-3	0.58	0.57	0.56	0.63	$F = 0.55$	
11 Inconsistent Parenting (M)	0-3	1.26	1.22	1.18	*1.50*	$F = 7.19^{**}$	3 > 1, 2
12 Parental Indifference (M)	0-3	0.32	0.30	0.26	*0.47*	$F = 8.58^{***}$	3 > 1, 2
13 Uninvolved Parenting (M)	0-3	0.95	0.93	0.91	1.07	$F = 2.06$	
14 Familial Delinquency (%)	0, 1	36.8	16.8	**46.1**	**40.5**	$\chi^2(2) = 25.00^{***}$	2, 3 > 1
15 Parental Mental Health Problems (N)	0-6	2.50	1.53	*2.55*	**3.81**	$F = 28.07^{***}$	3 > 2 > 1
16 Parenting Stress (N)	0-6	3.05	2.50	2.34	**5.25**	$F = 97.31^{***}$	3 > 1, 2
17 Bullying victimization (M)	0-3	0.77	0.71	0.70	*1.00*	$F = 6.31^{**}$	3 > 1, 2
18 Antisocial Friends (M)	0-3	0.38	0.34	0.35	*0.48*	$F = 4.16^{*}$	3 > 1; 2 = 1, 3
19 Poor School Achievement (N)	0, 1	43.9	41.1	40.1	37.8	$\chi^2(2) = 0.10$	
20 Socio-economic status (M)	0-4	2.64	1.47	**3.37**	*2.69*	$F = 125.52^{***}$	2 > 3 > 1
21 Urbanization (M)	0-4	3.07	1.99	**3.77**	*3.13*	$F = 125.65^{***}$	2 > 3 > 1

Note: Profile defining parameters are outlined, with elevated scores presented in italics and high scores presented in bold. Profile 1 = low-problem/impulsive group; Profile 2 = cognitive- and neighborhood-problem group; Profile 3 = multi-problem group. M = Mean; N = norm score.

scores on emotional problems, depression, and parental mental health problems. Results revealed that children assigned to Profile 2 suffered from elevated emotional problems, while growing up around delinquent family members (46%), and parents experiencing (below) average parental mental health problems. Profile 2 resembled Profile 1 in terms of prenatal problems, substance use, parenting and peer-related problems, and school achievement.

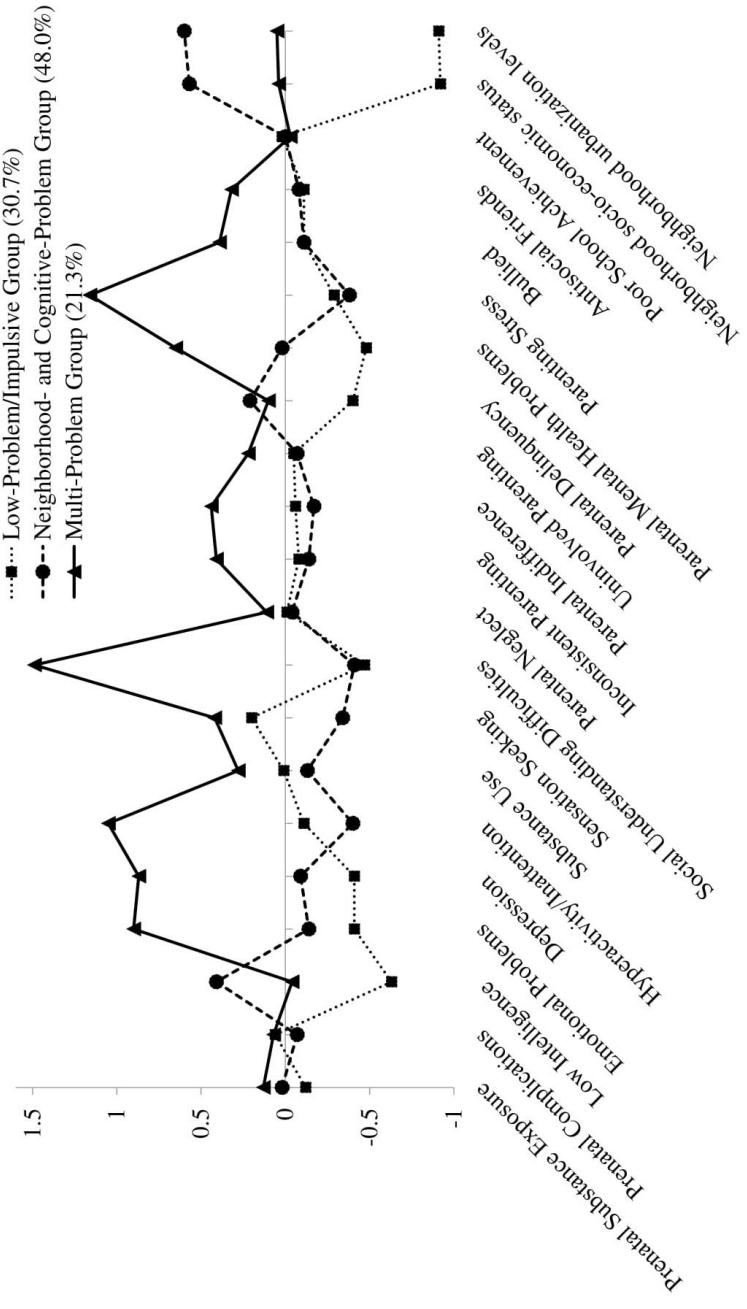
Profile 3 consisted of children experiencing overall highest levels of risk in individual, familial, and peer domains, and therefore received the label *multi-problem group*. Specifically, multi-problem children had slightly raised to high levels of emotional problems (versus close to average/slightly raised levels in Profiles 1 and 2), were at risk of developing clinical depression, and were highly hyperactive (versus close to average/slightly raised in the other two profiles). Also, children in Profile 3 had used none to 1 substance type prior to age 12, and experienced above average social understanding difficulties (as opposed to very low to low difficulties in Profiles 1 and 2).[8] Regarding familial problems, multi-problem children were exposed to slightly higher levels of inconsistent parenting and parental indifference than children populating Profile 1 and Profile 2. Additionally, 40.5% of children in Profile 3 had a delinquent family member. Furthermore, children's parents suffered from above average mental health problems (compared to low/below average problems in Profiles 1 and 2), and high parenting stress (compared to below average stress levels in Profiles 1 and 2). Indicators on peer-related risk revealed that children in Profile 3 were occasionally bullied, and had the most (i.e., none to a few) antisocial friends. Lastly, multi-problem children resided in neighborhoods with low to average SES and average urbanization levels, indicating that neighborhood-problems were less pronounced than in Profile 2, yet more prominent than in Profile 1.

4.3.3 Risk profiles and offending trajectories

Table 4.4 and Figure 4.3 illustrate that trajectory subgroup membership varied across risk profiles. Children in the low-problem/impulsive group were least likely to populate persisting offending trajectories. In comparison, children with cognitive- and neighborhood-problems (Profile 2) and children with multi-problems (Profile 3) were more likely to follow persistent trajectories. For instance, children assigned to the cognitive- and neighborhood-problem group

8 Additional analysis revealed that the multi-problem group scored higher than Profile 1 and Profile 2 on all six subscales of the questionnaire on social understanding difficulties (Hartman, Luteijn, Serra, & Minderaa, 2006). As such, multi-problem children experienced (1) difficulty in tuning their behavior/emotions to the social situation, (2) reduced contact and social interests, (3) orientation problems in time, place, or activity, (4) difficulties in understanding social information, (5) fear of and resistance to change, and (6) displayed stereotyped behavior.

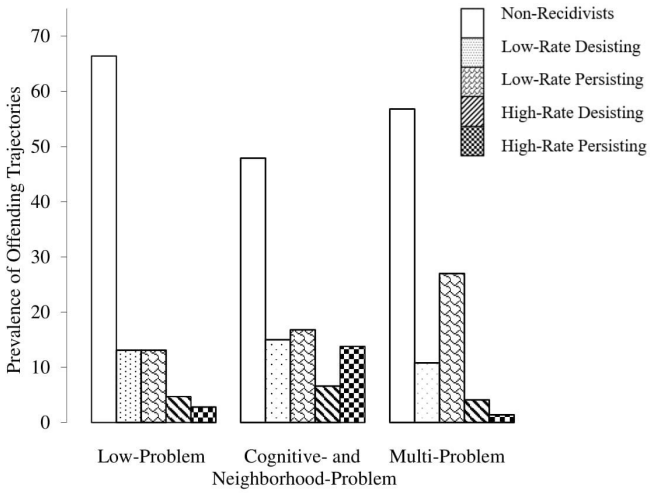
Figure 4.2: Mean z scores on profile indicators for the three-group model



Note. As continuous profile indicators differ in range, we present standardized mean scores across risk profiles (see also Hall, Howerd, & McCabe, 2010).

were more likely to populate the HR-P trajectory subgroup than to abstain from crime (OR = 6.80), or follow the LR-D (OR = 4.29) or LR-P (OR = 3.83, p = .053) trajectory. Compared to the low-problem/impulsive group, multi-problem children were more likely to be assigned to the LR-P trajectory than to abstain from crime (OR = 2.42). Low- and multi-problem children were equally likely to follow one of the high-rate offending trajectories. Children populating the multi-problem group also differed in important ways from children assigned to the cognitive- and neighborhood-problem group. Multi-problem children were less likely to follow the HR-P trajectory over abstaining from crime (OR = 0.08), and they were less likely to populate the HR-P than the LR-P (OR = 0.06) or LR-D (OR = 0.14, p = .069) trajectory subgroups.

Figure 4.3: Distribution of offending trajectories within risk profiles



In sum, results revealed that the low-problem/impulsive group was least likely to display persistent offending behavior. In contrast, children assigned to both the cognitive- and neighborhood-problem group and the multi-problem group were at increased odds of displaying offending behavior into young adulthood. While the cognitive- and neighborhood-problem group was over-represented among high-rate persistent offenders, the multi-problem group was most likely to cluster in the low-rate persistent offending trajectory.[9]

9 In accordance with our primary results, an additional chi square test with Bonferroni correction for multiple testing revealed that compared to low-problem children, children assigned to the cognitive- and neighborhood profile were at increased risk of following the high-rate persistent trajectory, while multi-problem children were at increased risk of following the low-rate persistent trajectory, $\chi^2(8) = 25.55$, p = .001.

Table 4.4: Odds ratios and confidence intervals from multinomial regression analysis: Risk profile membership on offending trajectory membership

	Risk Profiles	LR-D vs. NON	LR-P vs. NON	HR-D vs. NON	HR-P vs. NON	LR-P vs. LR-D	HR-D vs. LR-D	HR-P vs. LR-D	HR-D vs. LR-P	HR-P vs. LR-P	HR-P vs. HR-D
Mod.1	P1 (*ref.*)										
	P2	1.16 [0.77-3.28]	1.78 [0.87-3.64]	1.95 [0.65-5.89]	6.80** [1.96-23.63]	1.12 [0.45-2.80]	1.23 [0.36-4.27]	4.29* [1.09-16.89]	1.10 [0.32-3.79]	3.83† [0.98-14.99]	3.49 [0.70-17.29]
	P3	0.94 [0.37-2.50]	2.42* [1.10-5.28]	1.01 [0.23-4.46]	0.56 [0.06-5.59]	2.50 [0.83-7.55]	1.05 [0.20-5.60]	0.58 [0.05-6.59]	0.42 [0.09-2.05]	0.23 [0.02-2.48]	0.56 [0.04-8.09]
Mod.2	P1	0.63 [0.31-1.31]	0.56 [0.28-1.15]	0.51 [0.17-1.55]	0.15** [0.04-0.51]	0.89 [0.36-2.23]	0.81 [0.23-2.81]	0.23* [0.06-0.92]	0.91 [0.26-3.13]	0.26† [0.07-1.02]	0.29 [0.06-1.42]
	P2 (*ref.*)										
	P3	0.61 [0.25-1.47]	1.36 [0.69-2.70]	0.52 [0.14-1.96]	0.08* [0.01-0.64]	2.23 [0.84-5.96]	0.85 [0.19-3.84]	0.14† [0.02-1.17]	0.38 [0.09-1.55]	0.06** [0.01-0.49]	0.16 [0.02-1.71]

Note: $N = 348$. $R^2 = .07$ (Cox & Snell), .08 (Nagelkerke). Model $\chi^2(8) = 26.88$**. Lower group is reference category. Odds ratios greater than 1.00 indicate increased probability of group membership. Profile 1 = low-problem/impulsive group ($n = 107$); Profile 2 = cognitive- and neighborhood-problem group ($n = 167$); Profile 3 = multi-problem group ($n = 74$).
NON = non-recidivists ($n = 193$); LR-D = low-rate desisting ($n = 47$); LR-P = low-rate persisting ($n = 62$); HR-D = high-rate desisting ($n = 19$); HR-P = high-rate persisting ($n = 27$).
†p < .10, *p < .05 **p < .01, ***p < .001.

4.3.4 Sensitivity analyses

Two types of sensitivity analyses were conducted to determine whether similar results could be obtained for the multinomial logistic regression analyses when accounting for uncertainties in assignments to risk profiles and offending trajectories. First, we repeated the analyses among participants who were assigned to both risk profiles and offending trajectories with a probability of at least 70% (see also Diestelkamp et al., 2015). These analyses were based on 84% ($n = 293$) of the total sample, pointing out the high assignment accuracy in the current study. Second, the analyses were repeated among the entire sample while weighing for participant's posterior probabilities of belonging to each risk profile. Both sensitivity analyses confirmed our primary findings, producing similar directions, significance levels, and largely comparable odds ratios. Hence, the low-problem group was least likely to persist in offending. Also, associations between the cognitive- and neighborhood-problem profile (Profile 2) and the HR-P trajectory, and the multi-problem profile (Profile 3) and the LR-P trajectory remained significant. Thus, these additional analyses seem to strengthen the reliability of our main findings because uncertainty in group assignment did not appear to influence associations between risk profiles and offending trajectories.

In addition to testing the robustness of our findings, we performed two additional analyses to reflect on our methods of data-reduction, by alternatively defining risk and offender groups based on a count score of risk and overall frequency of offending, and revealing their association. First, associations between risk profiles and subgroups based on offense frequency were studied.[10] Multinomial regression analyses showed that children assigned to the cognitive- and neighborhood-problem groups were more likely to be classified as high-level recidivists than non-recidivists than the low-problem group (OR = 4.14, $p < .01$) and the multi-problem group (OR = 2.45, $p = .03$). Unfortunately, the increased risk of following low-rate persistent trajectories amongst children classified to the multi-problem profile could not be revealed when defining offender groups based on overall offense frequency, as the extent to which individuals display offending behavior over an extended period of time is lost when offending behavior is defined as merely the overall frequency of offending. Second, in order to study differences in mean counts of risk across offending trajectories, we created a sum score of risk exposure based on the presence of risk per life domain (i.e., individual, familial, peer, school, and neighborhood).[11] As a result, the count score of risk exposure ranged from 0 (no risk exposure in any of the life domains) to 5 (risk exposure in all five life domains). Descriptive statistics indicated that trajectory subgroups were exposed to risk in an equal number of life domains, as evidenced by the limited differences in mean scores of risk across trajectory-subgroups, ranging from 3.23 in the non-recidivist group to 3.65 in the high-rate desisting group.[12] These additional findings thus highlight the importance of taking specific patterns of risk into account when studying variation in long-term offending behavior.

10 Offender subgroups were defined as follows: (1) non-recidivists (i.e., participants without an additional police registration during follow-up), (2) sporadic recidivists (i.e., participants with one of two additional police registrations), (3) low-level recidivists (i.e., participants with three of four additional police registrations), and (4) high-level recidivists (i.e., participants with more than 4 additional police registrations).

11 As calculating a sum score of all 21 profile indicators would require participants to have a valid score on all profile indicators – which was the case for 128 participants – the count score of risk exposure was calculated based on the absence (0) or presence (1) of risk exposure per life domain – requiring a valid score on at least half of the indicators per life domain – before being summed into a measure of risk exposure across life domains.

12 When the sum score of risk exposure was measured as the sum of risk exposure on all 21 profile indicators – ranging from 0 (i.e., no risk exposure on any of the 21 profile indicators) to 21 (i.e., risk exposure on all of the 21 profile indicators) – mean scores of risk exposure across trajectory subgroups were still fairly similar; ranging from 6.01 in the non-recidivist group to 8.60 in the high-rate desisting group.

4.4 Discussion

The current study examined the extent to which exposure to distinct combinations of risk factors in childhood are associated with long-term re-offense patterns among early onset offenders. The reliance on registered data with a lengthy follow-up period, information on problems in various life domains, and the novel combination of risk profiles and offending trajectories enabled us to improve our knowledge on associations between childhood risks and long-term offending behavior.

Results showed that delinquent development in the current sample of early onset offenders was highly heterogeneous. In addition to an a-priori defined group of non-recidivists, the trajectory analysis yielded a model with four trajectories: two low-rate and two high-rate offending trajectories, with offending rates peaking either in adolescence or early adulthood. The finding that 55% of participants was assigned to the non-recidivist group diverges from theoretical expectations (Moffitt, 1993, 2006), as well as prior work on early onset offenders included in the Pittsburgh Youth Study, showing that only 20% desisted offending between ages 14 and 19 (van Domburgh, Loeber, et al., 2009). While not all early onset offenders in the current study continued to display offending behavior, the current sample can still be considered at increased risk of displaying offending behavior at the ages of criminal responsibility compared to national and cross-national general population samples (Blokland et al., 2010; Broidy et al., 2015). For instance, prevalence of offending in the current sample was three times higher than that of the general Dutch population, as only 14% of a Dutch birth cohort was registered by the police between 12 and 22 years old (Blokland et al., 2010).

The finding that both low- and high-rate recidivists were distributed across two offending trajectories resonates with findings from previous trajectory-based studies among adolescent and adult offender populations (Baglivio et al., 2015; Broidy et al., 2015; Day et al., 2012; Hoeve et al., 2008; Ward et al., 2010; Wiesner & Capaldi, 2003). As such, current empirical findings provide support for the robustness of trajectories repeatedly found in prior work, in that we found that both low- and high-rate offenders display either declining rates before reaching the mid-teens, or show rising offense rates into late adolescence that persist into adulthood. The fact that these trajectory shapes have been found repeatedly, despite studies varying in follow-up, provides support for the suggestion that trajectory modeling can be used to reveal variation in delinquent development that is not overly dependent upon specific study designs. We do however believe that it is important to consider that trajectory modeling is essentially exploratory in nature, and will extract a number of distinct trajectories in most datasets (Morizot, 2019; Sher, Jackson, & Steinley, 2011). Alternatively, trajectory modeling can be used to supplement or validate theoretically derived trajectories (Nagin & Tremblay, 2005; Sher, Gotham, & Watson, 2004) and provide insight into the extent to which theoret-

ical assumptions on delinquent development deviate from patterns of delinquency found in longitudinal data.

Results further revealed that three distinct profiles of childhood risk factors could be distinguished in our data. The latent profile analysis identified a low-problem/impulsive group (i.e., overall low levels of problems yet elevated hyperactivity/inattention and sensation seeking), a cognitive- and neighborhood-problem group (i.e., low intelligence levels and high neighborhood disadvantage), and a multi-problem group (i.e., high levels of individual, familial, and peer-related problems). As such, current profiles were characterized by overall level differences in risk exposure (quantitative differences) as well as exposure to specific combinations of risk (qualitative differences). While the low-problem group experienced overall low levels of risk exposure, the multi-problem group suffered from substantial problems in most life domains. Characteristics of the low- and multi-problem groups therefore support the assumption that problems in distinct life-domains – as well as internalizing and externalizing problems – are likely to co-occur (Caspi et al., 2014; Moffitt, 1993; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002). However, problems in one life domain did not necessarily co-occur with problems in other life domains, as is evidenced by the cognitive- and neighborhood-problem group, in which familial problems were largely lacking. It would be interesting to explore whether this group might be characterized by impaired parental cognitive abilities, which could explain the cognitive problems in children (see Plomin & Spinath, 2004).

While the finding that quantitative and qualitative differences characterized current risk profiles contradicts theoretical assumptions on delinquent development in early onset offenders (Moffitt, 1993), it corroborates findings from prior work on risk profiles in offender samples (e.g., T. Brennan et al., 2008; Lopez-Romero et al., 2019; Mulder et al., 2010; Schwalbe et al., 2008). It is challenging to compare our risk profiles to those found in previous studies in more detail, because of dissimilarities in profile indicators. However, some comparisons are worth highlighting. For example, the co-occurrence of impulsivity and sensation seeking is in accordance with findings from a study performed by Lopez-Romero et al. (2019) among adolescent and young adult offenders. However, these features characterized one of the two high-risk groups in their study, instead of the currently identified low-problem group. When comparing current findings to results reported by Schwalbe et al. (2008), it stands out that school problems as well as familial involvement in the justice system were distinguishing factors in their sample of juvenile court-involved youth. However, school problems characterized all three risk profiles identified in the current study, and familial delinquency was high in two of the three currently identified profiles. Overall, these comparisons seem to indicate that singular distinguishing risk factors are less common in the current sample of early onset offenders than in prior work identifying risk profiles among adolescent and young adult offenders.

Lastly, findings indicated that children assigned to risk profiles characterized by problems in multiple life domains were at increased risk of following persistent offending trajectories into young adulthood. On the one hand, findings showed that low-problem children were least likely to persist in offending during follow-up compared to the other two profiles. On the other hand, the specific combination of cognitive and neighborhood problems placed children at risk of displaying high-rate persistent offending, while multi-problem children were at increased risk of showing low-rate persistent offending. Besides supporting prior findings on IQ and offending (Farrington & Hawkins, 1991; Fergusson & Horwood, 2002), and neighborhood disadvantage and offending trajectories (Allard et al., 2017), current findings showed that – when considering influences from all life domains – the specific combination of both elevated the risk of following the most troublesome offending trajectory. Findings on associations between risk profiles and offending trajectories suggest that low estimated intelligence might increase children's susceptibility to criminogenic characteristics of disadvantaged environments (such as peer pressure), while residence in deprived neighborhoods might also result in under stimulation which may further worsen children's cognitive impairment.

Hence, results showed that accounting for functioning across life domains can help explain heterogeneity in longitudinal offending patterns among early onset offenders. We found significant associations between risk profiles and offending trajectories, even despite our relatively small and homogeneous sample. These findings highlight the potential of advancing the larger field of trajectory-based literature, by adopting a holistic view on risk exposure through the identification of risk profiles. By linking risk profiles to offending trajectories, we might drastically improve our insight into heterogeneity in longitudinal offense patterns.

A meaningful way to build on the current study would be to explore whether risk profiles can help explain heterogeneity in offending trajectories in general population and offender samples. Research on such samples will likely capture larger differences in levels of risk and trajectories of offending, enhancing the likelihood of detecting associations between risk profiles and offending trajectories and improving our understanding of the underlying causes of distinct offending trajectories.

Additionally, future research could strive to enlighten associations between risk profiles and other adverse adolescent and adult outcomes. According to theory (Moffitt, 1993) and prior research (Dembo et al., 2008; Espiritu, Huizinga, Crawford, & Loeber, 2001; Loeber & Farrington, 2000; Moffitt et al., 2002), an early onset of offending is associated with several adverse adult outcomes, such as drug and/or alcohol abuse, young parenthood, and unemployment. Even early onset offenders who do not engage in offending during adolescence are at increased risk of developing non-crime problems (Jennings et al., 2016; Moffitt et al., 2002). Knowledge on associations between risk profiles in childhood and poor adolescent and adult outcomes may (1) inform and further

justify paths of early intervention programs aimed at reducing offending, even for those children who desist from crime before reaching adolescence, while also (2) unveil characteristics of true recoveries.

Lastly, heterogeneity in trajectory subgroup membership within risk profiles raises important questions for future research. Findings showed that not all low-problem children desist from crime, nor did all children exposed to risk in multiple life domains follow persistent trajectories. It would be interesting to shed a light on the developmental process causing heterogeneity in long-term offending patterns within groups exposed to similar combinations of risk in childhood. According to Moffitt (1993), influences from different life domains are more or less important in different stages of the life-course, with familial influences decreasing during adolescence, while peer-influences increase. It would therefore be of theoretical importance to study the change in risk factors within individuals to be able to examine if, and how, this influences the development of offending behavior over the life-course.

4.4.1 Theoretical implications

The first finding of theoretical importance is that heterogeneity in offense patterns among child delinquents, while not completely unanticipated (Baglivio et al., 2015; van Domburgh, Vermeiren, et al., 2009; van Hazebroek, Blokland, et al., 2019), exceeds theoretical expectations. While Moffitt (1993) would expect all early onset offenders to continuously engage in crime during – at least – adolescence, current findings clearly indicate that this is not the case for a large part of the current sample. In fact, half of the sample desisted from crime before reaching adolescence (non-recidivist group), and therefore represents a substantial share of the *Childhood Arrestees Sample*. When comparing offense levels and trajectory shapes with Moffitt's early onset groups, the LR-P group – characterized by low yet persistent levels of offending during adolescence – mostly resembles the theoretically expected group of low-level chronic offenders. Our HR-P group – displaying continuously high offending rates – might be argued to represent Moffitt's high-level chronic offenders. On the other hand, we identified two additional offending trajectories that are not described in Moffitt's taxonomy. Both the HR-D and LR-D groups do not seem to resemble expected high- or low level chronic trajectories, as offending rates declined in both trajectory subgroups. Our findings therefore suggest that extant theory on the development of offending would have to allow for offending rates to decline with age (see for example Sampson & Laub, 1993), by for instance permitting developmental processes in the social environment to curb delinquent development, even in offenders with an onset in childhood.

Second, findings highlighted the high-risk nature of the current sample, as almost three in four participants (Profile 2 and Profile 3 combined) were characterized by problems in multiple life domains. Furthermore, all three

risk profiles were characterized by prenatal, cognitive, and school problems, supporting the assumption that biological risk and poor school achievement are associated with an early onset of offending (Moffitt, 1993). When considering profile-specific characteristics, the multi-problem profile exposed to a combination of individual (i.e., internalizing as well as externalizing), familial, and peer problems might be argued to resemble Moffitt's low-level chronic group in terms of risk exposure. The increased levels of emotional problems, depression, social understanding difficulties, and bullying victimization characterizing the multi-problem group could be interpreted as isolating individual characteristics, which further validates the argument that multi-problem children can be classified as Moffitt's (2006) low-level chronic group. In contrast, children populating the cognitive- and neighborhood-problem group experienced problems in multiple life domains yet fewer isolating features, and might therefore be argued to represent Moffitt's (1993) classic life-course-persistent group. However, levels of familial problems might be lower than what would be expected among the high-level chronic group. Lastly, the finding that low-problem children developed relatively well in most life domains is in contrast with Moffitt's (1993) assumption on heterotypic continuity in risk exposure, as biological vulnerability in these children did not elicit the process of cumulative disadvantage.

Third, associations between risk profiles and offending trajectories further justify the suggestion that the cognitive- and neighborhood-problem group resembles the theoretically expected group of high-level chronics, while the multi-problem group bears a resemblance to the low-level chronic group. In accordance with theory, the cognitive- and neighborhood-problem group experienced problems in multiple life domains yet low levels of internalizing problems, and displayed a high-rate chronic trajectory. In contrast, multi-problem children suffered from a combination of externalizing, internalizing, familial, and peer-related problems, and were at increased risk of following a low-rate chronic trajectory. Future studies including levels of anxiety are needed to further support or contradict hypotheses on differences in individual characteristics between low- and high-level chronic offenders.

4.4.2 Limitations and recommendations

Some limitations need to be considered alongside the interpretation of current findings. First, as the current study used data on a specific offender population (i.e., children with a police contact prior to age 12), findings may be due to specific characteristics of this sample. While we expected to include a group displaying stable patterns of disruptive behavior, the selection criteria of the current study may have also led to the inclusion of children whose registration was more or less coincidental. As Moffitt et al. (1996) defined an early onset as the manifestation of prolonged antisocial behavior at home and at school,

future research employing diverse definitions of an early onset could reveal the generalizability of our results. As non-criminal justice interventions may have influenced the shapes of the observed offending trajectories, generalizability of current findings could also be revealed by including information on parental, school, and professional intervention efforts in future studies. Second, current offending trajectories were based solely on overall frequency of police registrations. Prior work revealed a lack of overlap between officially registered and self-reported delinquent behavior (Feld & Bishop, 2012), and highlighted the importance of distinguishing between several types of crime when identifying trajectories (van Hazebroek, Blokland, et al., 2019). It would be interesting to see if current findings can be replicated when studies focus on self-reported delinquent acts, and differentiate between types of crime. Third, identified risk profiles and offending trajectories were used as observed variables in follow-up analyses. However, ways of incorporating (dichotomous and continuous) distal outcomes into mixture models are continuously being developed (Nylund-Gibson, Grimm, & Masyn, 2019). Future research focused on the likelihood or rates of offending could therefore account for uncertainty in group assignment in follow-up analyses.

4.4.3 Practical implications

Current findings have two important implications for prevention and intervention efforts. First, findings revealed that prevention and intervention efforts aimed at early onset offenders should be focused on a range of problems, as almost three in four children (70%) suffered from difficulties in multiple life domains. As such, findings suggest that the implementation of general intervention programs may be a promising avenue for risk reduction in childhood onset offenders. Second, intervening relatively early in the life course seems particularly relevant for the cognitive- and neighborhood problem group, as they are at increased risk of continuously engaging in crime at a high-rate. In contrast, the low-problem group might benefit most from being excluded from intervention programs and/or judicial interventions, as research has shown that interventions can be counterproductive and increase offending rates when implemented among low-risk youth (see Lowenkamp & Latessa, 2002). Further clinical implications for low-problem children await research on the developmental processes that cause some of these children to follow persistent offending trajectories.

APPENDIX A: DESCRIPTION OF PROFILE INDICATORS

Risk Profile Indicator	*Instrument*[1]	*C/P*[2]	*Items*	*Alpha*[3]	*Description or Sample Item (Response options)*	*Final*	*Scale Risk Profile Indicator*[4]
Individual							
1 Prenatal Substance Exposure	-	P	3	-	Whether mother had used substances (cigarettes, alcohol, drugs) during pregnancy (0 = *no*, 1 = *yes*)	D	0 = *no substance use*, 1 = *substance use*
2 Prenatal Complications	-	P	3	-	Whether mother had experienced complications during pregnancy (e.g. blood loss or sickness) and/or childbirth (e.g. navel cord entanglement or induction of labor) (0 = *no*, 1 = *yes*)	D	0 = *no complications*, 1 = *complications*
3 Intelligence	WISC-III	C	-	-	Test score on vocabulary (i.e. verbal intelligence) and block design (i.e. performal intelligence)	N	0 = *upper extreme* (IQ = 130), 6 = *lower extreme* (IQ = 69)
4 Emotional Problems	SDQ	C+P	5	.64	Often unhappy, down-hearted or tearful (0 = *not true*, 2 = *certainly true*)	N	0 = *close to average*, 3 = *very high*
5 Depression	KdvK	C	9	.79	I've been feeling down lately (0 = *not true*, 2 = *certainly true*)	N	0 = *not depressed*, 2 = *clinical depression*
6 Hyperactivity / Inattention	SDQ	C+P	5	.65	Restless, overactive, cannot stay still for long (0 = *not true*, 2 = *certainly true*)	N	0 = *close to average*, 3 = *very high*
7 Substance Use	OAB	C+P	5	-	Whether the child had ever used substances (i.e. alcohol, tobacco, and drugs) (0 = *no*, 1 = *yes*)	M	0 = *0 types of substances*, 3 = *3 types of substances*
8 Sensation Seeking	SAHA	C	7	.71	I like trying new things, even when they are not allowed (0 = *strongly disagree*, 4 = *strongly agree*)	M	0 = *strongly disagree*, 4 = *strongly agree*
9 Social Understanding Difficulties[5]	CSBQ	P	49	.94	Over-reacts to everything and everyone; Takes in information with difficulty (0 = *does not apply at all*, 3 = *applies very well*)	N	0 = *very low*, 6 = *very high*
Familial							
10 Parental Neglect	SAHA	C	8	.50	My parents [do not] want to know who I am meeting up with (0 = *never*, 3 = *often*)	M	0 = *never*, 3 = *often*
11 Inconsistent Parenting	SAHA	C	5	.43	My parents forget a rule that they've made themselves (0 = *never*, 3 = *often*)	M	0 = *never*, 3 = *often*
12 Parental Indifference	SAHA	C	6	.65	My parents [do not] hug me (0 = *never*, 3 = *often*)	M	0 = *never*, 3 = *often*
13 Uninvolved Parenting	SAHA	C	6	.60	My parents [do not] spend time with me (0 = *never*, 3 = *often*)	M	0 = *never*, 3 = *often*
14 Familial Delinquency	SAHA	P	1	-	Whether a family member had ever been in contact with the criminal justice system (0 = *no*, 1 = *yes*)	D	0 = *no*, 1 = *yes*
15 Parental Mental Health Problems	SCL-90	P*	90	.97	Headaches; Feelings of guilt; Being scared (0 = *not at all*, 4 = *very much*)	N	0 = *very low*, 6 = *very high*
16 Parenting Stress	NOSIK	P*	17	.95	My child demands more attention from me than I can give (0 = *strongly disagree*, 3 = *strongly agree*)	N	0 = *very low*, 6 = *very high*
Peers							
17 Bullying victimization	SAHA	C	9	.82	How often have children from school called you names (0 = *never*, 3 = *often*)	M	0 = *never*, 3 = *often*
18 Antisocial Friends	SAHA	C	6	.52	How many of your friends have been arrested by the police (0 = *none*, 3 = *most or all*)	M	0 = *none*, 3 = *most or all*

Risk Profile Indicator	*Instrument*[1]	*C/P*[2]	*Items*	*Alpha*[3]	*Description or Sample Item (Response options)*	*Final*	*Scale Risk Profile Indicator*[4]
School							
19 Poor school achievement	OMRT	C	-	-	Whether test scores on one-minute reading test indicated insufficient reading abilities	N	0 = *no*, 1 = *yes*
Neighborhood							
20 Socio-economic status	-	SCP	-	-	Neighborhood mean income, unemployment, and education levels (0 = *very high*, 4 = *very low*)	M	0 = *very high* , 4 = *very low*
21 Urbanization	-	ST	-	-	Number of households per km² (0 = *very low: less than 500 households per km²*, 4 = *very high: 2.500 or home households per km²*)	M	0 = *very low*, 4 = *very high*

Note: [1] Instrument: WISC-III = Wechsler Intelligence Scale for Children-Revised – version III (Wechsler, 1974); SDQ = Strengths and Difficulties Questionnaire (Goodman, 1997; Muris, Meesters, & van den Berg, 2003; van Widenfelt, Goedhart, Treffers, & Goodman, 2003; for information on norm scores see Youth-in-Mind, 2012); KdvK = Short Form Depression Questionnaire for Children (Korte Depressievragenlijst voor Kinderen; de Wit, 1987); OAB = Observed Antisocial Behavior Questionnaire (Loeber, Stouthamer-Loeber, van Kammen, & Farrington, 1989); SAHA = Social and Health Assessment (Weissberg, Voyce, Kasprow, Arthur, & Shriver, 1991); CSBQ = Children's Behavior Questionnaire (Hartman, Luteijn, Serra, & Minderaa, 2006); SCL-90 = Symptom Checklist (Arrindel & Ettema, 1986; Derogatis, Lipman, & Covi, 1973); NOSIK = Nijmeegse Ouderlijke Stress Index (Abidin, 1983; de Brock, Vermulst, Gerris, & Abidin, 1992); OMRT = One-Minute Reading Test (Brus & Voeten, 1995; Evers, van Vliet-Mulder, & Groot, 2000).

[2] Informant: C = Child; P = Parent; SCP = Social and Cultural Planning Office of the Netherlands 2002; ST = Statistics Netherlands, 2006. C+P indicates that the final score was determined by the informant reporting the most problems. P* indicates that the final score was determined by the parent reporting the most problems.

[3] Some of the profile indicators display low internal reliability (e.g. emotional problems and hyperactivity/inattention) as they are aimed at screening the entire concept with a limited number of items.

[4] Scale: D = dichotomous; N = norm scores; M = mean scores.

[5] The CSBQ consists of six subscales. Sample items are subtracted from subscales with the highest correlation with the total score in the current sample.

APPENDIX B: DESCRIPTIVE STATISTICS OF PROFILE INDICATORS

Risk Profile Indicator[1]	*N*	*Range*	*Mean / Proportion*	*SD*[2]	*Norms (valid %)*[3]						
					0	1	2	3	4	5	6
1 Prenatal Substance Exposure (D)	341	0, 1	36.1								
2 Prenatal Complications (D)	337	0, 1	42.1								
3 Low Intelligence (N)	319	0-6	3.96	1.19	0	1.3	5.3	34.8	27.6	16.9	14.1
4 Emotional Problems (N)	342	0-3	0.74	1.06	61.1	14.9	12.9	11.1			
5 Depression (N)	203	0-2	0.34	0.67	76.8	12.3	10.8				
6 Hyperactivity/Inattention (N)	342	0-3	0.91	1.16	54.1	19.0	9.1	17.8			
7 Substance Use (M)	347	0-3	0.27	0.56	77.8	17.6	4.0	0.6			
8 Sensation Seeking (M)	285	0-4	1.59	0.82							
9 Social Understanding Difficulties (N)	309	0-6	1.40	1.62	38.2	27.5	13.3	8.4	3.6	7.4	1.6
10 Parental Neglect (M)	285	0-3	0.58	0.41							
11 Inconsistent Parenting (M)	286	0-3	1.26	0.57							
12 Parental Indifference (M)	286	0-3	0.32	0.34							
13 Uninvolved Parenting (M)	286	0-3	0.95	0.54							
14 Familial Delinquency (D)	340	0, 1	36.8								
15 Parental Mental Health Problems (N)	267	0-6	2.50	2.02	25.1	12.7	14.2	12.4	13.9	13.9	7.9
16 Parenting Stress (N)	301	0-6	3.05	1.90	12.6	12.0	12.6	22.9	13.6	13.0	13.3
17 Bullying victimization (M)	286	0-3	0.77	0.60							
18 Antisocial Friends (M)	283	0-3	0.38	0.33							
19 Poor School Achievement (N)	317	0, 1	43.8								
20 Socio-economic status (M)	348	0-4	2.54	1.27	5.7	12.9	31.6	11.2	38.5		
21 Urbanization (M)	331	0-4	3.07	1.18	4.5	9.1	11.5	25.1	49.8		

Note: Valid percentage exclude missing data, and represent the share of the sample that was exposed to that specific risk factor. SD = Standard Deviations.

[1] Risk Profile Indicator: D = dichotomous; N = norm scores; M = mean scores.

[2] SD is not reported for dichotomous variables.

[3] Norms: *Intelligence*, *Social understanding difficulties*, *Parental mental health problems*, *Parenting stress*: 0 = very low, 1 = low, 2 = below average, 3 = average, 4 = above average, 5 = high, 6 = very high; *Emotional problems*, *Hyperactivity/Inattention*: 0 = close to average, 1 = slightly raised, 2 = high, 3 = very high; *Depression*: 0 = not depressed, 1 = at risk of depression, 2 = clinical depression; *Substance use*: 0 = 0 substance types, 1 = 1 substance type, 2 = 2 substance types, 3 = 3 substance types; *Socio-economic status*: 0 = very high, 1 = high, 2 = average, 3 = low, 4 = very low; *Urbanization*: 0 = very low, 1 = high, 2 = average, 3 = high, 4 = very high.

5 Studying the effects of social bonds on offending behavior varied by biological vulnerability in early onset offenders

ABSTRACT

Applying sociological and developmental perspectives on offending, the current study assesses the within-individual associations between changes in social bonds and offending over time in early onset offenders, as well as the moderating influence of biological vulnerability. Longitudinal data from the *Dutch Childhood Arrestees Study* was analyzed using random effects models in order to examine the effects of changes in bonds with parents, peers, and school, as well as the interactions between biological vulnerability and social bonds, on offense rates from childhood into adolescence. While we found no evidence for main effects of changes in bonds with parents and school on offense rates, results revealed that an increase in affiliation with delinquent peers acted in the expected offending-inducing direction. Furthermore, the effect of bonds with school on offense frequency depended on participants' biological vulnerability, as only biologically vulnerable children were found to show higher offense rates in the years they skipped class. Current findings reveal that dynamic processes are important in understanding delinquent development in early onset offenders. Furthermore, the current study highlights the importance of including interactions between biological vulnerability for delinquent development and time-varying social factors when studying variability in offending over time.

Key Words
Early onset offenders, social bonds, biological vulnerability, within-individual methodology

5.1 Introduction

Both criminological theory and empirical research suggests that within delinquent populations individual offense frequency varies considerably across the life-course (Jennings & Reingle, 2012; Moffitt, 1993). While some delinquents display either a stable-low or stable-high pattern of offending, others show either increasing or decreasing rates of offending with age (Lynne-Landsman et al., 2011; Odgers et al., 2008). Moreover, a substantial portion of former delinquents desists from committing delinquent acts during the early adult years (Sampson & Laub, 2003).

The developmental literature suggests that understanding variability in offending across the life-course requires a dynamic approach to the influence of key social risk factors of offending (Childs, Sullivan, & Gulledge, 2010; Paternoster & Brame, 1997), by allowing changes in life circumstances to affect individual criminal activity (Piquero, Brame, Mazerolle, & Haapanen, 2002). For instance, criminological theory states that social control originating from conventional social bonds is a key factor influencing delinquency and crime. Importantly, social bonds are hypothesized to change in response to changing life circumstances, and these changes in social bonds are assumed to be of primary importance to understand changes in offending over time (Moffitt, 1993; Sampson & Laub, 1993).

While research adopting a dynamic approach to key social risk factors has mainly focused on changes in offending during the transition from adolescence into early adulthood (see Averdijk, Elffers, & Ruiter, 2012; Kazemian & Farrington, 2015), studies addressing the question whether changes in social bonds may help explain variability in delinquency during the transition from childhood into early adolescence are scarce. Research on the transition into adulthood has for instance shown that marriage, being in a relationship, employment, and spending time in adult-like roles function as a positive source of change for offenders (Blokland & Nieuwbeerta, 2005; J. M. Hill, Blokland, & van der Geest, 2016; Horney, Osgood, & Marshall, 1995; Verbruggen, Blokland, & van der Geest, 2012), while divorce has been found to contribute to an increase in offending behavior (Bersani & Doherty, 2013). Transitioning into adolescence however is also accompanied by important changes in the social environment, involving changes in the importance of relationships with parents, peer networks, and school (Berndt, 1982; Larson & Richards, 1991). Whether or not individuals successfully navigate through changes in their social environment may result in a decrease or increase of offending behavior. It is especially relevant to study the effect of changes in social bonds on offending in an early onset offender population, as they are particularly crime-prone, yet also show substantial variability in offending during the transition from childhood into early adolescence (van Domburgh, Loeber, et al., 2009; van Hazebroek, Blokland, et al., 2019).

Importantly, both developmental (Moffitt, 1993), and biosocial (Monroe & Simons, 1991; Zuckerman, 1999) criminological theories argue that specific, relatively stable, individual characteristics developed early in the life-course (i.e., antisocial dispositions) may render individuals either less susceptible to changes in their social environment (Moffitt, 1997), or less equipped to benefit and more likely to suffer damage from these changes (Monroe & Simons, 1991; Zuckerman, 1999). For instance, antisocial dispositions resulting from peri/prenatal complications, neuropsychological problems, and impaired intelligence have been argued to affect both offending behavior as well as the extent to which individuals successfully navigate changes in social bonds (Moffitt, 1993). If we want to improve our efforts to support children experiencing difficulty in turning away from delinquent activities upon entering adolescence, it is therefore important to include antisocial dispositions and their interaction with the social environment, and social bonds in particular, in studies focused on variability in offending (Boman & Mowen, 2018; Moffitt, 1993). In the current study, we focus on biological vulnerability resulting from peri/prenatal complications, because theory (Moffitt, 1993) and prior research (for a review see van Hazebroek, Wermink, et al., 2019) have identified peri/prenatal problems as an important indicator of biological vulnerability interacting with social risk.

The current study examines the extent to which changes in social bonds with parents, peers, and school are related to variability in offending in early onset offenders during the transition from childhood into adolescence, and whether these effects vary across children differing in biological vulnerability resulting from peri/prenatal complications. To address its aims, the current study uses three waves of panel data on a sample of Dutch delinquents with an onset below age 12 who were followed from childhood into adolescence (Geluk et al., 2014; van Domburgh, Vermeiren, et al., 2009).

5.1.1 Theoretical framework

A variety of sociological theories explains delinquency based on individuals' social environment, such as social control theory (Hirschi, 1969), differential association (Sutherland, 1947), and social learning theory (Akers, 1973). Control theory argues that delinquency emerges when bonds to society are weak or broken. During the transition from childhood into adolescence, changes in social bonds with parents (i.e., the amount of parental supervision, and the amount of time parents spend with their children), peers (i.e., time spent with conventional peers), and school (i.e., perceived importance of education, and attachment to teachers) are thought to alter the likelihood of criminal involvement (Hirschi, 1969). In addition, from learning (Akers, 1973) and socialization (Sutherland, 1947) theories it can be derived that the effects of social bonds with parents and peers on delinquency may depend on whether

or not parents and peer themselves display law-abiding or rather offending behavior. Specifically, learning and socialization theories state that delinquent behavior is learned through social interactions, and social bonds with delinquent others will therefore increase the likelihood of delinquent involvement (Akers, 1973; Akers & Jennings, 2016; Hoeben et al., 2016; Sutherland, 1947).

The developmental criminological literature has highlighted the importance of changes in key social influences upon entering adolescence, arguing that changes in the social environment from childhood into adolescence may result in changes in delinquent activity within individuals over time (Moffitt, 1993). While family is the most prominent factor in the development of conventional norms in childhood, it is normal for children to break away from their parents during adolescence, and in turn, spend more time with peers (Berndt, 1982; Larson & Richards, 1991; Moretti & Peled, 2004). Consequently, as the role of parents decreases, peers become increasingly important in influencing behavioral development during the transition from childhood into adolescence (Cooper & Ayers-Lopez, 1985; Haynie & Payne, 2006), including delinquent development (Moffitt, 1993; Simons, Whitbeck, Conger, & Conger, 1991).

Developmental taxonomic theory (Moffitt, 1993) and biosocial theory (Monroe & Simons, 1991; Zuckerman, 1999) combine ideas from sociological, psychological and developmental criminology, by acknowledging the importance of both (changes in) social bonds and antisocial dispositions. Traditionally, psychological cirminology has explained offending behavior in terms of antisocial dispositions, that develop in childhood from both social and biological origins – such as low self-control (Wilson & Herrnstein, 1985) – and keep motivating delinquent behavior throughout the life-course.

Moffitt (1993) argues that children's antisocial disposition decreases the likelihood of experiencing prosocial interactions, as well as the ability to profit from these interactions. At the same time however, antisocial disposition increases the likelihood of experiencing antisocial interactions as well as the child's vulnerability to the negative effects of such interactions. Children displaying antisocial tendencies will therefore be more likely to bond with similarly antisocially inclined peers, as well as be more susceptible to their negative influences than are children without antisocial dispositions (Moffitt, 1993; Wright, Caspi, Moffitt, & Silva, 2001). However, when children with antisocial dispositions, despite them being unlikely to do so, do develop prosocial ties such as conventional bonds with parent or school, the effects of these bonds may appear larger than those in prosocial children (Wright et al., 2001). This is not because of antisocially inclined children are more receptive to the benefits of prosocial bonds – rather on the contrary –, but because prosocial children tend not to engage in delinquent behavior in the first place, leaving less room for behavioral improvement – a floor effect. Furthering this line of reasoning, one could expect the detrimental effects of antisocial bonds to be most outspoken for children without antisocial

dispositions, not because they are more vulnerable to these effect than children that do have antisocial dispositions – rather on the contrary – but because of ceiling effects.

Drawing from sociological and developmental criminology, we can sum up the following theoretical assumptions on associations between within-individual changes in social bonds and offending in early onset offenders. First, we expect that an increase in social bonds with conventional family members, peers, and school will result in a decrease in offending behavior. Second, we expect to observe an increase in offending behavior as a result of an increase in social bonds with criminal parents or delinquent peers. Third, the effects of within-individual changes in social bonds on delinquency are theorized to depend on biological vulnerability, with bonds with conventional others offering stronger protective effects against delinquent behavior in biologically vulnerable children, while bonds with delinquent others will have stronger offending-inducing effects in biologically nonvulnerable youth than among their biologically vulnerable counterparts.

5.1.2 Prior research

Two generations of longitudinal studies focusing on associations between social bonds with parents, peers, and school and delinquent behavior can be distinguished. The first generation of studies compared offenders and non-offenders on familial, peer, and school characteristics or studied the correlation of these characteristics with levels of delinquency (for a recent review, see Farrington, 2015). As such, these studies emphasized between-individual differences in social bonds and offending (Farrington et al., 2002; Flanagan, Auty, & Farrington, 2019). Such studies found that in the family domain, weak social bonds (e.g., poor parental supervision, low parental involvement) were associated with a higher probability of later offending (Derzon, 2010; Flanagan et al., 2019). In contrast, strong positive familial bonds were found to be associated with lower levels of offending behavior. In the peer domain, affiliation with delinquent peers was found to result in an increased risk of offending (Hemphill et al., 2009). Regarding bonds to school, prior work has shown low school commitment to be associated with a higher likelihood of offending behavior (Chung et al., 2002).

Prior work has also aimed to explain between-individual differences in offending by focusing on differences in antisocial dispositions, as well as by addressing the question whether the association between social bonds and offending varies by antisocial disposition (for a review, see Craig, Baglivio, Wolff, Piquero, & Epps, 2016; van Hazebroek, Wermink, et al., 2019). For example, studies have revealed associations between genetic (Rhee & Waldman, 2002), peri/prenatal (for a review see Wakschlag et al., 2002), and neuropsychological functioning (for a review see Ttofi et al., 2016) and antisocial develop-

ment. Furthermore, and in line with theoretical expectations, prior work has shown that the associations between social bonds and offending depend on individual differences in antisocial disposition (van Hazebroek, Wermink, et al., 2019; Wright et al., 2001). For instance, children exposed to peri/prenatal complications and adverse social circumstances have been found to display the highest levels of delinquent behaviors (van Hazebroek, Wermink, et al., 2019). On the other hand, strong social bonds have also been found to lower the likelihood of offending in children with low-self-control (Wright et al., 2001), as well as in children exposed to adverse childhood experiences (Craig et al., 2016).

While the first generation of studies provided consensus on between-individual differences in social bonds and delinquency, a second generation of studies was designed to increase our understanding of delinquent development by utilizing dynamic models that focus on developmental changes within individuals (Thornberry, 1996). This second generation of studies contributes to the literature on associations between social bonds and delinquency in two important ways. First, second generation-studies are better able to test developmental theories of offending, as they are focused on explaining changes in individual delinquent activity over time. Second, by focusing on within-individual changes in social bonds and offending, pre-existing differences between individuals are held constant and are therefore accounted for in second generation-studies (Allison, 2009). This is important, as there will always be pre-existing differences of interest – whether measured or not – that may affect changes in individual offending behavior (Farrington et al., 2002; Paternoster, Bushway, Apel, & Brame, 2003).

The few studies that have applied within-individual methodology to examine the effects of changes in social bonds with parents on offending behavior, have generated mixed findings. Some of the research on the impact of bonds with parents on offending showed that youths experiencing an increase in parental bonds over time – measured as parental attachment and parental supervision – displayed a decrease in their offending behavior (Childs et al., 2010; Craig, 2016; Peterson, Lee, Henninger, & Cubellis, 2016). Likewise, a decrease in parental bonds – operationalized and measured as low attachment, low parental involvement and poor parental supervision – was shown to be associated with an increase in subsequent offending behavior (Farrington et al., 2002; Hemphill et al., 2015). In contrast, other work (Beardslee et al., 2018; Childs et al., 2010) has shown that changes in parental supervision does not affect individuals' delinquent development.

A slightly larger body of literature examined the effects of social bonds with peers on offending, although in absolute numbers this type of study is also still rare. Most of this work suggests that peer delinquency is positively related to individuals' own engagement in offending behavior (i.e., Beardslee et al., 2018; Childs et al., 2010; Craig, 2016; Hemphill et al., 2015; Peterson et al., 2016; Unnever & Chouhy, 2019). In contrast, however, Farrington et al.

(2002) failed to find an association between changes in delinquent peers and individual's own future delinquent behavior.

Only a few studies investigated how within-individual changes in bonds with school affect offending, and these findings too vary. Peterson et al. (2016) found that an increase in bonds to school over time – measured as both school commitment and school achievement – significantly reduced offending behavior. Additionally, Na (2017) showed that youth who dropped out of school were significantly more likely to experience an increase in subsequent arrests compared to youth who did not drop out. However, other studies have found no significant effects of changes in school bonds on delinquent behavior (Farrington et al., 2002; Unnever & Chouhy, 2019).

Although the above-mentioned research adopting within-individual designs has added greatly to our understanding of the effects of changes in social bonds with parents, peers, and school on changes in offending behavior over time, they have been limited in three important ways. First, except for the study conducted by Na (2017), studies on within-individual changes in offending were based on the general adolescent population and inner-city samples. It is therefore likely that not many early onset offenders were included in prior samples. Consequently, prior results might not apply to Moffitt's (1993) early onset offenders, who are deemed most at risk of displaying persistent offending behavior. Various scholars have therefore recommended that future studies apply within-individual models in longitudinal surveys of especially at risk populations (Farrington, Ttofi, & Piquero, 2016; Hemphill et al., 2015). Second, prior work did not examine whether the effects of social bonds depend on pre-existing individual differences in biological vulnerability to delinquent development. This is important, as assumptions on interaction effects between biological vulnerability and social influences are key in developmental criminological theory on early onset offenders (Moffitt, 1993). Third, unlike studies examining the effects of associations with delinquent peers, none of the prior studies on the effects of parental social bonds on offending across adolescence captured differences in parental law-abiding or criminal behavior. Up to date, it therefore remains unclear if, and to what extent, bonds with conventional versus criminal parents differentially affect within-individual changes in youth's delinquent involvement.

5.1.3 The current study

The current study aims to increase our understanding of variability in offending, as well as overcome some of the shortcomings hampering earlier research, (1) by studying the effects of within-individual changes in social bonds with family, peers, and school on offense frequency, (2) by doing this in a high-risk sample of early onset delinquents, and (3) by paying specific

attention to pre-existing individual differences in biological vulnerability. In addition, as the data provide us with information on social bonds and offense frequency during the transition from childhood into early adolescence, we are able to expand our knowledge on associations between within-individual changes in social bonds and variability in offending to this crucial transition in the life-course. Furthermore, by specifically distinguishing between law-abiding and criminal parents, we are able to study the potential differential effects of bonds with conventional versus criminal parents on changes in offending behavior over time.

5.2 Method

5.2.1 Participants and procedures

This study is based on data from the *Dutch Childhood Arrestees Project*, a prospective longitudinal study on early onset delinquents, conducted by the Department of Child and Adolescent Psychiatry of the Amsterdam University Medical Centers (VUmc) (Geluk et al., 2014; van Domburgh, Vermeiren, et al., 2009).[1] Children with a registered police contact prior to age 12 (i.e., the minimum age of criminal responsibility in the Netherlands) were selected from three municipal police registries in the Netherlands (i.e., Gelderland-Midden, Utrecht, and Rotterdam-Rijnmond). A total of 348 children (302 males; 184 of non-Dutch origin) participated in the first measurement occasion (M_{age} = 10.63, SD = 1.48), shortly after they were registered by the police between 2003 and 2005 (M_{age} = 10.26, SD = 1.45). These participants formed the base sample that has since been followed-up on three more occasions, after 1-year (n = 295, 85%, M_{age} = 11.79, SD = 1.53), 2-years (n = 266, 76%, M_{age} = 12.85, SD = 1.54), and 6.5-years (n = 134, 39%, M_{age} = 17.61, SD = 1.50).

The current analysis used data from the first (T1), second (T2) and third (T3) measurement waves of the study. At each assessment, questionnaires and interviews were administered to the children and their primary caretakers (hereafter referred to as 'parents'), covering offending behavior and a range of risk factors from multiple life domains. Comparing police records of the baseline sample to those of a Dutch birth cohort suggests that the study achieved its goal of including a high-risk sample of early onset delinquents, as 45 percent of the baseline sample had a police record from age 12 into early adulthood compared to 14 percent of the Dutch birth cohort (Blokland et al., 2010).

We examined whether study members who completed all three waves differed from the baseline sample in terms of several background characteristics

1 This study was approved by the Dutch Ministry of Justice.

(see top half of Table 5.1 for demographic details of the sample across waves). Results revealed no differences in gender ($\chi^2(1) = .10$, $p = .75$), ethnicity ($\chi^2(1) = 2.72$, $p = .10$), or frequency of offending at wave 1 ($t(324) = -.68$, $p = .50$). In addition to missing data due to non-participation, we excluded self-report questionnaires of children younger than eight years old and children with below average verbal IQs at wave 1 (measured using the Wechsler Intelligence Scale for Children-Revised; Wechsler, 1974), because of potential problems with comprehensibility of the questionnaires. As a result, self-report questionnaires were excluded for 64 children at T1, 50 children at T2, and 48 children at T3. Children whose self-report questionnaires were and were not excluded did not differ in terms of gender or ethnicity. Besides the lack of bias in attrition resulting from differences in background characteristics, our choice of analyses – looking at within-individual change – further minimized bias resulting from loss of data, as each person serves as their own control by focusing on associations between change in each person's risk exposure and their offending behavior (Allison, 2009).

5.2.2 Measures

Offense frequency. Frequency of offending behavior was measured at all three waves using the child version of the Observed Antisocial Behavior Questionnaire (OAB: Vragenlijst Waargenomen AntiSociaal gedrag; Loeber, Stouthamer-Loeber, van Kammen, & Farrington, 1989; Slot, Orobio de Castro, & Duivenvoorden, 1998). Participants were asked whether they had committed any of 20 delinquent acts – over the past six months at baseline, and over subsequent intervals between waves (i.e., approximately 12 months in waves 2 and 3) – and if so, how many times they had committed these acts. The 20 items measuring offending behavior included: stealing outside the home (6 items), hitting or fighting outside the home (5 items), property damage and arson (5 items), rule breaking and fare dodging (3 items), and possession of a weapon (1 item).[2] Items on non-delinquent behaviors under Dutch law were not included in the scale. Across all waves, for only 4 out of 20 delinquent acts, over 5 percent of the sample reported to have committed the particular act 4 or more times. To prevent outliers in the frequency distribution, we therefore

2 The 20 items on delinquent behaviors under Dutch law included: (1) stealing a bicycle, (2) shoplifting, (3) stealing from school, (4) stealing from a car, (5) stealing from someone's pocket, coat, or bag, (6) burglary, (7) hitting a teacher, (8) hitting or kicking other children, (9) throwing stones or objects towards others, (10) taking part in a group fight, (11) threatening a child, (12) purposely damaging property at home, (13) purposely damaging school property, (14) purposely damaging property in other places, (15) making graffiti at public places, (16) arson, (17) fencing, (18) trespassing, (19) fare dodging, and (20) weapon possession.

capped reported frequency at 4 for all delinquent acts, and then summed the frequency of all items to create a count scale.,[3,4]

Social Bonds. Social bonds with family (i.e., parental supervision, and parental involvement), peers (i.e., affiliation with delinquent peers), and school (i.e., changes in schools, and skipping class) were measured using the Social and Health Assessment (Weissberg, Voyce, Kasprow, Arthur, & Shriver, 1991). Social bonds were measured as follows:

1) Parental supervision: average score on 8 items on children's perception of the degree of parental control over different aspects of their lives (e.g., 'My parents want to know who I am meeting up with', rated on a scale from 0 (*never*) to 3 (*often*) with higher scores indicating stronger bonds with family, α = .51 at T1);[5]
2) Parental involvement: average score on 6 items on the extent to which children feel that their parents are involved in several areas of their lives (e.g., 'My parents spend time with me', rated on a scale from 0 (*never*) to 3 (*often*) with higher scores representing stronger bonds with family, α = .61 at T1);
3) Affiliation with delinquent peers: 1 item asking how many of the child's friends have been arrested by the police, rated on a scale from 0 (*none*) to 3 (*most or all*) with higher scores indicating that a larger portion of the participant's friends consisted of delinquent peers;
4) Changing schools: 1 item asking how many times children had changed schools, rated on a scale from 0 (*0 times*) to 3 (*three or more times*) with higher scores representing weaker bonds with school;
5) Skipping class: 1 item asking whether or not the child had skipped class, rated on a scale from 0 (*no*) to 1 (*yes*) with higher scores representing weaker bonds with school.

Criminal Parents

In order to study whether parental bonds may have differential effects on offending when parents themselves are criminal or law-abiding, we constructed a dummy variable indicating whether the child's parents (i.e., biological parents or their current partners) had been in contact with the police across the three

3 The four delinquent acts that were committed four or more times by over 5 percent over the sample across waves included 'kicking or hitting other children', 'taking part in a group fight', 'trespassing' and 'fare dodging'.

4 Participants displaying the highest frequencies of offending according to the uncapped measure were also identified as frequent offenders in the capped measure of offense frequency. In order to suppress outliers, we continued our analyses with the capped measure of offense frequency.

5 The limited number of items in the parental supervision and parental involvement subscales might have suppressed the alpha values (Streiner & Norman, 1989). As the internal reliability could not be improved by deleting a specific item from the scale, we continued our study with these measures.

waves (0 = *non-criminal*, 1 = *criminal*). Subsequently, we constructed two interaction terms by multiplying scores on the dummy variable with variables measuring social bonds with parents.

Biological Vulnerability
The dummy variable on biological vulnerability (0 = *nonvulnerable*; 1 = *vulnerable*) was defined as the presence or absence of either prenatal exposure to substances (i.e., cigarettes, alcohol, or drugs), or pregnancy or birth complications (e.g., blood loss, or navel cord entanglement). Prior research shows both to be related to an elevated risk of delinquency (see for example Oddone-Paolucci, Violato, & Wilkes, 2000; Wakschlag et al., 2002). In order to assess whether within-individual effects of changes in social bonds on offense frequency varied across biological vulnerable and nonvulnerable children, we subsequently constructed six interaction terms by multiplying biological vulnerability by each of the social bond variables.

Control variables
We included two time-variant control variables in the analyses.[6] These time-variant control variables consisted of participant's age at each wave, and the monthly interval between waves, as these varied between participants and waves. By including interval between waves, we were able to control for the fact that the length of time between waves – and therefore the length of time participants reported on – varied to some extent.

5.2.3 Analyses

Stata version 15.0 was used to perform hybrid random effect negative binomial models, in order to examine the extent to which individual fluctuations in offense frequency systematically changed as a function of changes in social bonds with family, peers, and school. We used hybrid random effect models, as fixed effect negative binomial models fail to control for stable covariates when the outcome is an overdispersed count variable (Allison & Waterman, 2002), like offense frequency in the current study (see descriptive information in Table 5.1).[7] Allison (2005) suggests that hybrid models offer a solution in the case of overdispersed count variables, as these express time-varying independent variables at each measurement occasion as deviations from overall

6 As we are conducting within-individual analyses, there is no need to include time-invariant controls.

7 Estimating fixed effects models using a dichotomous measure of offending behavior was not preferred in the current study, as over a third of participants committed at least one delinquent act in all three waves (40.2% of the 209 children completing all three waves), and would therefore have been removed from the analyses due to a lack of within-individual variation on the outcome variable.

person means across measurement occasions. For example, if a participant's score on parental supervision was 1.0 at T1, 2.0 at T2, and 3.0 at T3, his or her person mean would be 2.0. Accordingly, his or her deviation scores would be -1.0 at T1, 0.0 at T2, and 1.0 at T3. By focusing on individual deviation scores, hybrid random effects models specifically estimate whether within-individual change in independent variables are related to change in the outcome variable, while simultaneously controlling for effects of other, time-stable risk factors (Allison, 2009).

The primary analyses in the current study consisted of both non-lagged (i.e., associations between concurrent changes in social bonds and offense frequency) and lagged (i.e., associations between changes in social bonds at one time-point and offense frequency at the next time-point) random effects models. While the lagged models allowed us to address questions on causal direction (Vaisey & Miles, 2017), scholars have shown that lagged models may to lead to biased estimates when the lag structure of the data does not accurately capture real-world causal lags between continuously varying states of independent variables, such as parental supervision, and the dependent variable (Unnever & Chouhy, 2019; Vaisey & Miles, 2017). In the current study, findings from both the non-lagged and lagged models are therefore presented.

In both non-lagged and lagged models, we first examined associations between changes in social bonds and changes in offense frequency. Second, we added a main effect of the criminal parent-dummy as well as the interaction terms between the dummy and social bonds with parents. Third, we added a main effect of biological vulnerability to the original model, as well as interaction terms between biological vulnerability and the social bond variables.

To complement our primary analysis, we examined potential reverse causation by estimating the effects of offense frequency on each of the social bond variables. If the initial and reserved models both reveal significant results, the process might be cyclical, indicating that social bonds affect offense frequency and offense frequency affects social bonds.

Sensitivity analyses were conducted to determine whether similar results could be obtained when offending was measured as a diversity score, or as the frequency of serious types of offending. The diversity score was defined by summing the presence or absence of each of the 20 different delinquent acts, based on self- and parent-reports, with the act considered present if either of the informants had reported it as present. In this case, a child that for example had stolen something twice and hit another child once, would have a diversity score of 2, resulting in a less skewed outcome variable. Alternatively, we defined the frequency of serious offenses as the sum of the offense frequency on items regarding stealing outside the home (6 items), and hitting or fighting outside the home (5 items).[8]

8 Property damage and arson, rule breaking and fare dodging, and weapon possession were excluded from the serious offense frequency scale.

5.3 Results

5.3.1 Descriptive statistics

Table 5.1 presents the descriptive statistics and mean scores for independent and dependent variables across waves of the final sample used in the analyses. Table 5.1 shows that, on average, participants were fairly well bonded to their parents ($M = 2.42$, $SD = 0.42$; $M = 2.04$, $SD = 0.53$ at T1 for parental supervision and parental involvement respectively), had 'a few' friends that had been arrested by the police ($M = 0.99$, $SD = 0.83$ at T1), and rarely changed school over a year-time period ($M = 0.12$, $SD = 0.41$ at T1). Less than 15 percent of participants reported skipping class across waves. The mean number of reported delinquent acts varied from 3.86 ($SD = 5.01$) at T1 to 4.00 ($SD = 6.34$) at T2. Independent samples t-tests revealed that biologically vulnerable and nonvulnerable children differed in levels of parental supervision at T3, $t(206) = 2.37$, $p = .02$, with biologically nonvulnerable children being more closely supervised ($M = 2.46$, $SD = .51$) than their biologically vulnerable peers ($M = 2.28$, $SD = .49$).

Examining absolute within-individual change in social bonds revealed that participants experienced change in all three domains (family, peers, school), with average absolute within-individual change ranging from 0.25 to 0.65 within the one-year time intervals between waves for variables measures on a scale from 0 to 3 (i.e., parental bonds, affiliation with delinquent peers, and changing schools). Among participants experiencing change in social bonds, about half experienced a decrease, while the other half experienced an increase in social bonds. Regarding offense frequency, participants displayed an absolute change of approximately 4 offenses between waves, with about 40% of participants displaying a decrease and about 35% displaying an increase in offense frequency.

5.3.2 Effects of social bonds on offense frequency

The non-lagged hybrid random effects models are presented in Models 1 through 3 in Table 5.2. Results showed that changes in social bonds with parents were not significantly associated with changes in offense frequency (Model 1). Furthermore, none of the interaction effects between parental bonds and parental criminal behavior (Model 2), or parental bonds and biological vulnerability (Model 3) were significant. In contrast, changes in bonds with delinquent peers had a significant positive effect on offense frequency, such that with every one-unit increase in our measure of affiliation with delinquent peers – where one-point indicates an increase from 'none' to 'a few' delinquent peers for example – the number of delinquents acts is expected to increase with 43 percent (IRR = 1.43, $p < .01$). Adding the interaction term with biological

Table 5.1: Descriptive statistics for demographic, dependent and independent variables by wave

	Total sample			Absolute within-individual change		Biologically vulnerable participants			Biologically nonvulnerable participants		
	T1 *N* = 284	T2 *N* = 254	T3 *N* = 232	T1-T2	T2-T3	T1 *N* = 171	T2 *N* = 161	T3 *N* = 149	T1 *N* = 107	T2 *N* = 89	T3 *N* = 80
Variables	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
Demographics											
Males[a]	0.87	0.87	0.87			0.90	0.90	0.90	0.83	0.81	0.81
Non-Dutch [a]	0.51	0.49	0.49			0.41	0.41	0.40	0.65	0.62	0.63
Age	10.84 (1.20)	11.86 (1.42)	12.87 (1.46)			10.82 (1.21)	11.78 (1.46)	12.76 (1.50)	10.90 (1.18)	12.00 (1.35)	13.06 (1.38)
Time interval (months)		14.17 (3.79)	13.16 (3.77)				14.03 (3.58)	13.36 (3.86)		14.51 (4.20)	12.82 (3.67)
Social bonds											
Parental supervision	2.42 (0.42)	2.42 (0.43)	2.34 (0.51)	0.34 (0.32)	0.35 (0.33)	2.42 (0.42)	2.38 (0.45)	2.28 (0.49)	2.43 (0.42)	2.47 (0.37)	2.46 (0.51)
Parental involvement	2.04 (0.53)	2.03 (0.49)	2.02 (0.54)	0.41 (0.36)	0.41 (0.36)	2.04 (0.53)	2.00 (0.49)	1.98 (0.53)	2.05 (0.56)	2.08 (0.48)	2.12 (0.54)
Delinquent peers	0.99 (0.83)	0.95 (0.84)	1.01 (0.92)	0.65 (0.70)	0.61 (0.72)	0.97 (0.81)	0.91 (0.84)	1.02 (0.94)	0.97 (0.85)	1.01 (0.81)	0.97 (0.86)
Changing schools	0.12 (0.41)	0.16 (0.44)	0.13 (0.41)	0.25 (0.53)	0.26 (0.55)	0.11 (0.38)	0.18 (0.48)	0.14 (0.44)	0.13 (0.44)	0.13 (0.37)	0.08 (0.27)
Skipping class [a]	0.13	0.07	0.15	0.16 (0.37)	0.16 (0.37)	0.15	0.05	0.13	0.08	0.10	0.22
Outcome											
Offense frequency	3.86 (5.01)	4.00 (6.34)	3.81 (5.82)	3.92 (5.35)	3.80 (4.84)	3.90 (4.92)	3.98 (5.92)	3.99 (6.17)	3.36 (4.51)	3.61 (5.68)	3.18 (4.42)

Note: [a] These variables are binary and the mean represents the proportion.

vulnerability, results in the effect of peer delinquency on offense frequency only approaching significance in the biologically nonvulnerable group (IRR = 1.33, $p < .10$) while remaining significant in the vulnerable group. The difference in the effect of delinquent peers between biologically nonvulnerable and vulnerable children however was non-significant (IRR = 1.02, $p = .91$).[9] Regarding bonds with school, main effects of changes in offense frequency were found to be non-significant (Model 1). The interaction effect between skipping class and biological vulnerability approached significance (IRR = 2.11, $p < .10$), suggesting that the effect of skipping class on offense frequency is over twice as strong in biologically vulnerable children than in biologically nonvulnerable children. In terms of the magnitude of the relationship, these findings suggest that at times biologically vulnerable children skipped class, they reported committing almost twice as many delinquent acts ($0.82 \times 2.11 = 1.73$, i.e., an increase of 73% in offense frequency).[10]

The lagged hybrid random effects models are presented in Models 4 through 6 in Table 5.2. In line with the non-lagged models, main effects of changes in social bonds with parents and school were unassociated with changes in next-year offense frequency (Model 1). Interaction effects between the criminal parent-dummy and parental social bonds were also shown to be non-significant (Model 5). In contrast with the non-lagged models, the main effect of affiliation with delinquent peers on offense frequency was non-significant (Model 4), indicating that change in the proportion of participants' delinquent peers did not affect offense frequency in the following time-period.

With respect to the interaction effects between biological vulnerability and social bonds, Model 6 overall showed that an increase in levels of parental supervision, affiliation with delinquent peers, and skipping class had offending-inducing effects in biologically nonvulnerable children, while they did not significantly affect biologically vulnerable children. Regarding parental supervision, the expected number of offenses in biologically nonvulnerable children was surprisingly found to increase when a child experienced a one-unit increase in the level of parental supervision (IRR = 2.89, $p < .05$). The effect

9 The absence of a significant effect of peer delinquency on offense frequency for the non-vulnerable group in Model 3 might be due to the fact that parameters were estimated for biologically nonvulnerable and vulnerable youth separately, resulting in smaller groups and therefore larger standard errors.

10 As prior studies found strongest biosocial interaction effects for prenatal smoking and prenatal complications (van Hazebroek, Wermink, et al., 2019), we also defined biological vulnerability as (1) as the presence ($n = 110$ at T1) or absence of prenatal exposure to nicotine or (2) as the presence ($n = 142$ at T1) or absence of pregnancy or birth complications. In addition to confirming findings from our primary analyses, results of hybrid random effects models with the alternative definitions of biological vulnerability showed that in both models the effect of affiliation with delinquent peers on offense frequency in biologically nonvulnerable children remained significant (IRR = 1.38, $p < .01$ for children who were not exposed to prenatal nicotine use, IRR = 1.43, $p < .01$ for children who were not exposed to prenatal complications).

Table 5.2 Random effects models for the effect of social bonds on offense frequency

	Non-lagged models					
	Model 1			Model 2		
	B	(SE)	IRR	*B*	(SE)	IRR
Individual characteristics						
Criminal parents				-.02	(.15)	0.98
Biological vulnerability						
Control variables						
Age	.04	(.04)	1.05	.05	(.04)	1.05
Wave interval	-.003	(.003)	1.00	-.004	(.003)	1.00
Social bonds						
Parental supervision	.12	(.19)	1.13	.17	(.24)	1.19
Parental involvement	-.09	(.16)	0.91	-.10	(.19)	0.91
Delinquent peers	.36**	(.09)	1.43	.35**	(.09)	1.41
Changing schools	-.15	(.17)	0.86	-.10	(.17)	0.90
Skipping class	.30	(.20)	1.35	.29	(.20)	1.34
Criminal parents × parental supervision				-.15	(.38)	0.86
Criminal parents × parental involvement				.14	(.38)	1.15
Biosocial interactions						
Bio × supervision						
Bio × involvement						
Bio × delinquent peers						
Bio × changing schools						
Bio × skipping class						
No. of observations	515			502		
No. of groups	239			232		
Wald χ^2	20.08**			19.33*		

Note. IRR = incidence rate ratio, indicating the percentage increase (IRR greater than 1) or decrease (IRR less than 1) in delinquency rates for every one-unit increase in the independent variable.
$^{\dagger}p < .10$, $^{*}p < .05$, $^{**}p < .01$.

of parental supervision on offense frequency was significantly smaller in biologically vulnerable children (IRR = 0.34, $p < .05$), rendering the effect of an increase parental supervision near zero in this group (i.e., 2.89 × 0.34 = [an IRR of] 0.98). Regarding change in bonds with delinquent peers, we found a positive effect on subsequent offending in biologically nonvulnerable children, such that with each one-unit increase in our measure of delinquent peers, the number of delinquent events participants reported in the following time-period increased by 42 percent (IRR = 1.42, $p < .05$). This effect was significantly smaller in biologically vulnerable children (IRR = 0.60, $p < .05$), resulting in a non-significant effect of bonds with delinquent peers on offense

Lagged models												
Model 3			Model 4			Model 5			Model 6			
B	(SE)	IRR	*B*	(SE)	IRR	*B*	(SE)	IRR	*B*	(SE)	IRR	
						.12	(.20)	1.13				
.15	(.13)	1.17							-.001	(.17)	1.00	
.07	(.04)	1.07	.08	(.05)	1.08	.08	(.06)	1.08	.04	(.06)	1.05	
-.003	(.003)	1.00	.03	(.02)	1.03	.03*	(.02)	1.03	.04*	(.02)	1.04	
-.11	(.36)	0.90	.35	(.26)	1.41	.54	(.34)	1.71	1.06*	(.45)	2.89	
-.24	(.32)	0.78	-.02	(.20)	0.98	-.05	(.23)	0.95	.05	(.35)	1.05	
.29†	(.16)	1.33	-.01	(.11)	0.99	-.001	(.11)	0.99	.35*	(.17)	1.42	
.08	(.32)	1.08	.30	(.23)	1.35	.14	(.23)	1.15	.12	(.39)	1.13	
-.19	(.33)	0.82	-.02	(.27)	0.98	.01	(.26)	1.01	.70†	(.45)	2.00	
						-.69	(.51)	0.50				
						-.01	(.45)	1.01				
.30	(.42)	1.35							-1.09*	(.55)	0.34	
.22	(.37)	1.24							-.07	(.43)	0.93	
.02	(.20)	1.02							-.51*	(.24)	0.60	
-.32	(.37)	0.73							.29	(.48)	1.33	
.75†	(.41)	2.11							-1.11†	(.57)	0.33	
	509			393			381			388		
	235			214			207			211		
	25.05*			8.45			8.27			19.52		

frequency (1.42 × 0.60 = [an IRR of] 0.85, p = 0.31). Lastly, findings from Model 6 suggest that when biologically nonvulnerable children skipped class during one time-period, they reported an increase in delinquent acts in the following time-period (IRR = 2.00, $p < .10$). However, and in contrast to the non-lagged models, Model 6 suggests that the effect of skipping class on offense frequency is 67% (IRR = 0.34, $p < .10$) smaller, and non-significant (2.00 × 0.33 = [an IRR of] 0.66, p = 0.22), in biologically vulnerable children.[11]

11 When biological vulnerability was defined as either the presence or absence of prenatal exposure to nicotine or prenatal complications, hybrid random effects models showed that none of the interaction effects were significant.

5.3.3 Potential reverse causation

As shown in Table 5.3, the effect of individual change in offense frequency was not systematically associated with changes in bonds with parents, the number of times participants changed schools, or whether or not they skipped class. However, Table 5.3 shows that within-individual changes in offense frequency affected bonds with delinquent peers. Findings revealed that in time-periods participants were exposed to a relative large proportion of delinquent peers, they were also more likely to offend more frequently ($B = .04$, $p < .001$).

5.3.4 Sensitivity analyses

Models with offending diversity and frequency of serious offending as outcome measures overall strengthen the reliability of our main findings, as they yielded substantially similar results, producing similar directions, significance levels, and largely comparable estimates.[12] Hence, results of the sensitivity analyses revealed non-significant effects of parental bonds on offending, while revealing a significant positive effect of increases in bonds with delinquent peers on offending. Differences between the primary and the sensitivity analyses were limited to the main effect of skipping class on offending, and the interaction effect between biological vulnerability and affiliation with delinquent peers. First, the non-lagged models showed that in time periods children were more likely to skip class, they were also more likely to display a higher diversity of

Table 5.3 Random effects models for the effect of offense frequency on social bonds

Independent variables	*Dependent variable*									
	Parental supervision		*Parental involvement*		*Delinquent peers*		*Changed schools*		*Skipped class*	
	B	(SE)	*B*	(SE)	*B*	(SE)	*B*	(SE)	*B*	(SE)
Control variables										
Age	-.04**	(.01)	-.01	(.02)	.10**	(.02)	.002	(.01)	.24*	(.13)
Wave interval	-.001	(.001)	-.001	(.001)	-.001	(.002)	-.001	(.001)	.001	(.01)
Behavioral variable										
Offense frequency	-.01†	(.004)	-.001	(.004)	.04**	(.01)	.002	(.004)	.06	(.04)
Observations	530		532		522		532		529	
Individuals	242		242		242		22		242	
Wald χ^2	16.82*		3.22		41.60*		0.53		8.43*	

Note: We used fixed effects linear regression models for continuous measures of social bonds (i.e., parental supervision, parental involvement, affiliation with delinquent peers, and changes in schools), and logistic fixed effects models for dichotomous measures of social bonds (i.e., skipping school).
†$p < .10$, *$p < .05$, **$p < .01$.

12 Results from sensitivity analyses are available upon request.

offending (IRR = 1.56, $p < .01$), as well as a higher frequency of serious offending (IRR = 1.65, $p < .05$). Furthermore, when offending diversity was used as the independent variable in the reversed models, it was shown that the diversity of offending also had a positive effect on skipping class ($B = .44$, $p < .001$). Second, the positive effect of affiliation with delinquent peers on offending remained significant for biologically nonvulnerable children in the non-lagged models with offending diversity as outcome variable (IRR = 1.23, $p < .05$). Lastly, none of the interactions between biological vulnerability and social bonds were significant in models with serious offending as the outcome variable.

5.4 Discussion

The present study examined the effects of social bonds on delinquent behavior in early onset offenders during the transition from childhood into early adulthood. Specifically, this study examined (1) the association between within-individual changes in bonds with parents, peers, and school and concurrent as well as next-year changes in delinquency rates, and (2) whether such associations varied by participants' biological vulnerability to delinquent development.

Consistent with peer-influence models, findings indicated that an increase in affiliation with delinquent peers acted in the expected offending-inducing direction. Specifically, the current study revealed that when a greater proportion of participants' peers had a police contact, participants' offending behavior increased in the same year. These findings corroborate prior work on changes in bonds with peers and delinquent behavior in adolescent general population and inner-city samples (e.g., Beardslee et al., 2018; Peterson et al., 2016; Unnever & Chouhy, 2019). Our findings add to this literature by revealing the importance of changes in friendships with delinquent peers during the transition from childhood into adolescence in early onset offenders.

Unlike the effect of affiliation with delinquent peers, there was no evidence of an association between change in parental bonds and offending behavior during the transition into early adolescence. The absence of an effect of bonds with parents on offending contradicts some previous findings on associations between changes in parental bonds and offending behavior (Farrington et al., 2002; Hemphill et al., 2015), yet is in accordance with other prior studies focused on within-individual changes in offending (Beardslee et al., 2018; Childs et al., 2010). The non-significant effect of parental bonds, combined with the non-significant interaction effect between criminal parents and parental bonds, may point to a general decline in the influence of parental bonds on behavioral outcomes in adolescents, be it good or bad (Berndt, 1982; Larson & Richards, 1991; Moretti & Peled, 2004). Another possible explanation for this finding might be related to the fact that we did not distinguish between

paternal and maternal bonds, as prior research found an association between growth in maternal bonds and a decrease in youth's delinquent behavior, while changes in paternal bonds did not affect youth's delinquent involvement (Craig, 2016). Further assessment of the differential effects of change in maternal and paternal bonds on delinquency is therefore needed. Lastly, the lack of an interaction effect between criminal parents and parental bonds might also be due to the way parental criminal behavior was measured in the current study, as parents were asked at T1 whether or not they had ever been in contact with the police. Thus, parents identified as criminal in the current study may have been in contact with the police only during their adolescent years. Future studies focused on associations between parental bonds and change in children's delinquent behavior over time could therefore strive to examine a more direct effect of parental criminal behavior by asking whether parents had been in contact with the police during or directly prior to the observation period.

With respect to our secondary aim, our work extends prior research in that findings showed that the association between concurrent social bonds with school and offending are conditional upon early onset offenders' biological vulnerability. Although offense rates in biological nonvulnerable children seemed to be unaffected by concurrent bonds with school, biologically vulnerable children were found to commit more offenses in years they skipped class. These results may explain varying findings found in earlier work. While studies that failed to find an effect of bonds with school were conducted in a birth cohort (Unnever & Chouhy, 2019) and an inner-city sample (Farrington et al., 2002), the study reporting an effect of school dropout and subsequent arrest frequency was conducted among a sample of serious adolescent offenders (Na, 2017). As Moffitt (1993) suggests that biologically vulnerable individuals are more likely to display offending behavior, the study by Na (2017) may have been based on a relatively large share of biologically vulnerable participants, and consequently revealed an effect of bonds with school and offending behavior. Clearly, future research would further increase our understanding of the nature of the effects of changes in social bonds on delinquency by replicating the current effort in considering possible interactions between antisocial dispositions, like biological vulnerability, and time-varying social factors, like social bonds, especially when considering high-risk groups.

Importantly, we also examined whether changes in offense frequency had an effect on social bonds. In doing so, the current study found that when youths display an increase in their offending behavior, they are likely to experience an increase in the number of delinquent peers they affiliate with. As offending behavior was found to affect bonds with delinquent peers – in this and other studies (see for instance Weerman, 2011) –, and bonds with delinquent peers affect offending behavior, this process might be cyclical.

Lastly, we want to reflect on the somewhat surprising estimates produced by the lagged models in our study. In contrast to our expectations, lagged

models showed that biologically nonvulnerable children were especially susceptible to changes in social bonds compared to biologically vulnerable children. While some scholars have suggested that changes in the social environment will mostly affect individual with (biological) antisocial dispositions (Monroe & Simons, 1991; Wright et al., 2001; Zuckerman, 1999), it has also been proposed that stronger associations between social risk and offending behavior will be found in children who lack biological risk factors of offending (Raine, 2005). The reasoning behind this second argument is that the association between the social environment and offending behavior might be stronger when the biological 'push' towards crime is weaker, allowing for the link between social bonds and offending to shine through. Another possible explanation for the results produced by the lagged models is that the yearly time intervals between waves in the current study may be too large to paint a detailed picture of the relationship between changes in social bonds and offending, as prior work indicated that lagged models may lead to biased estimates when the lag in the model does not match with the time lapse in the real world (Unnever & Chouhy, 2019; Vaisey & Miles, 2017). Questions surrounding the developmental processes underlying the differential effects of social bonds on future offending in biologically vulnerable and nonvulnerable children entering early adolescence therefore warrant future research.

5.4.1 Theoretical implications

Overall, current findings offer mixed support for sociological theories of offending (Akers, 1973; Hirschi, 1969; Sutherland, 1947). In contrast to assumptions from social control theory (Hirschi, 1969), current findings showed that changes in bonds with parents were not associated with concurrent changes in delinquency rates, regardless of whether parents themselves had displayed criminal or law-abiding behavior. However, the finding that an increase in social bonds with delinquent peers exacerbates children's' own engagement in delinquent activities provides support for differential association and social learning theories, which generally suggest that delinquent behavior is learned by interacting with delinquent others (Akers, 1973; Sutherland, 1947).

In line with developmental theories of offending (Moffitt, 1993), the results reported here confirm the importance of considering the dynamic processes that occur upon entering adolescence in order to understand variability in offending during this phase in the life-course. The transition from childhood into early adolescence was found to be a time where changes in the social environment affect delinquent behavior. Thus, failing to consider the influence of change in important social risk factors over time can lead to insufficient or partial explanations of offending behavior.

Lastly, findings furnished support for the theoretical assumption that the effect of changes in the social environment on delinquent behavior depend

on early biological differences (Moffitt, 1993; Monroe & Simons, 1991; Zuckerman, 1999). As such, sociological theories that dismiss these interaction effects appear to do so in error. While findings from the non-lagged models provide support for the hypothesis that biologically vulnerable children are more susceptible to their social bonds with others than their biologically nonvulnerable peers (Moffitt, 1993; Monroe & Simons, 1991; Zuckerman, 1999), the lagged models showed that social experiences in distinct life domains (at home, with peers, and at school) mostly affect biologically nonvulnerable children. Overall, the results of this study therefore show that social learning, biosocial, and developmental theories are complementary, as within-individual changes in offense frequency over time did not only vary due to changes in social bonds, but also due to variation in susceptibility to social influences based on biological makeup.

5.4.2 Limitations and recommendations

Some limitations need to be considered when interpreting the results. First, while this study is based on data collected across three waves in an important high-risk offender population, the use of three measurement occasions to study the effects of time-varying factors on offense rates may be somewhat limited. For example, prior studies on the effects of change in social bonds on offending across adolescence used about five to six waves (Childs et al., 2010; Peterson et al., 2016). Future research could further our analyses by including more measurement occasions over an extended period of the life-course, in order to reveal how stability and change in social bonds affect delinquent behavior across different periods of adolescence. Second, the current study is based on self-reported delinquency rather than registered offenses. A useful area for future research would be to test our findings using conviction data, as a judicial contact, in the form of either an official arrest or conviction, may have a more profound impact on social bonds. Third, no protective factors of offending in the peer and school domains were used in the current study. Future research including factors such as friendships with conventional peers, or connectedness between student and teachers, may offer a more detailed interpretation of the effects of changes in social bonds on offending. Lastly, the current study used biological vulnerability resulting from exposure to prenatal problems as a proxy for antisocial dispositions. It would be interesting to see if current findings on biosocial interactions are replicated when different definitions of biological vulnerability are used. For instance, future research could consider whether the effects of social bonds on offending vary across children differing in verbal and executive functioning (Moffitt, 1993), or psychophysiological functioning (i.e., individuals' 'fight or flight' response to stressful situations; for a study on the interaction between psychophysiological measures and social/environmental risk factors, see Raine et al., 2014).

5.4.3 Practical implications

Findings from the current study on the extent to which changes in social bonds affect variability in offending behavior in early onset offenders offer three main practical implications. First, that it is important for practitioners and clinicians to consider the type of peers early onset offenders surround themselves with, as an increase in affiliation with delinquent peers plays an important role in the increase of offense rates. Second, current findings suggest that it is beneficial to ensure that early onset offenders attend class. As skipping class has the potential to facilitate an increase in offense rates, it is important to address early signs of school disengagement in children who are at greatest risk of continuing their offending behavior. Lastly, criminal justice interventions may therefore be most effective when they are organized in ways to avoid hindering early onset offenders from following conventional developmental pathways, in order to prevent an increase in exposure to delinquent peers and school disengagement. On a final note, while no significant effect of changes in parental bonds on offending behavior was revealed, findings from the current study should not be taken to suggest that practitioners and clinicians should not focus on the parent-child relationship. In contrast, prior work has shown that interventions focused on the parent-child relationship can be effective (Baglivio, Jackowski, Greenwald, & Wolff, 2014). Questions on which changes in what particular aspects of the social bonds with parents may have protective effects in the early onset offender population as yet await future research.

6 General discussion

6.1 Aims

The current thesis intended to contribute to literature on offending over an extended period of the life-course, by focusing specifically on the delinquent development and its correlates in the high-risk population of early onset offenders, defined in the current thesis as children with a police contact prior to the age of 12 (i.e., childhood arrestees). Understanding long-term delinquent development in childhood arrestees is highly relevant from a policy perspective, as a police contact/arrest below age 12 has emerged as an important indicator for persistence in offending (DeLisi et al., 2013). Increased knowledge on this specific offender population may provide vital information for prevention and intervention programs aimed at limiting the continuation of delinquent behavior in early onset offenders and improving their mental health outcomes. Furthermore, studying offending behavior and associated offenders characteristics from onset into early adulthood allows for addressing key theoretical predictions from developmental criminological theory (Moffitt et al., 1996). Unfortunately, knowledge on the development of offending behavior in early onset offenders known to the police is scarce, as their age of onset is below the age of criminal responsibility in many Western countries (e.g., 12 years in the Netherlands), and offenses committed below age 12 do therefore not appear in national crime statistics.

Specifically, the current thesis addressed two general aims. First, it aimed to provide empirical insight into (variation in) the development of offending behavior from childhood into early adulthood and associated singular identified risk factors. The current thesis focused on whether there was evidence for offending trajectories that are distinct in terms of time path from early adolescence (age 12) into early adulthood (Moffitt, 1993), and addressed the typological prediction that offending trajectories are distinct in terms of frequency and type of offending (Moffitt, 1993). In addition, this thesis addressed the theoretical assumption that males, minorities, and children from disadvantaged neighborhoods are at increased risk of showing persistent delinquent behavior (Moffitt, 1993). In order to address its first aim, the current thesis used official registration data on offense frequency, type of offending, mortality, and criminal sanctions over an extended period of time on children included in the *Dutch Childhood Arrestees Study* – a longitudinal study on over 700 children registered by the police for the first time because of an alleged

offense below age 12 (Geluk et al., 2014; van Domburgh, Vermeiren, et al., 2009).[1,2]

The second aim was to improve our understanding of variation in delinquent development by combining theoretical insights stemming from different scholarly traditions (i.e., sociological, biosocial, and developmental criminology) on risk exposure in multiple life domains (i.e., individual, familial, peers, school, and neighborhood). This is important because biosocial (Monroe & Simons, 1991; Zuckerman, 1999) and developmental criminology (Caspi et al., 2014; Farrington & Welsh, 2008; Loeber, Stouthamer-Loeber, et al., 2008; Moffitt, 1993, 2006) suggest that risk factors of offending do not operate in isolation but rather co-occur and are mutually reinforcing. Biosocial and developmental criminological theorists argue that specific, relatively stable individual characteristics develop early in the life-course, and influence delinquent development over an extended period of time. Importantly, stable individual characteristics are also assumed to render some individuals more vulnerable or susceptible to changes in their social environment than others. As a result, changes in social life domains with age are thought to especially contribute to the likelihood of persistent offending in individuals with specific individual characteristics. The current thesis therefore adopted a holistic view on risk exposure, and examined associations between the interaction between and co-occurrence of risk in distinct life domains – focusing on biological, psychological, and social correlates of offending – and official as well as self-reported frequency of offending. In order to address this second aim, the official registration data on the *Childhood Arrestees Sample* was combined with rich survey data, containing information on problems from individual, familial, peer, school, and neighborhood life domains, as well as self-reported delinquency, measured using standardized instruments at baseline (N = 348), and one (n = 295), and two (n = 266) years (n = 134) follow-up.

In this final chapter of the thesis, findings from four separate studies are summarized in Section 6.2. Subsequently, findings are related to criminological theory in Section 6.3. In Section 6.4, strengths and limitations of the current thesis are addressed, and suggestions for future research are provided. Lastly, practical implications are offered in Section 6.5.

1 This study was approved by the Dutch Ministry of Justice.

2 In order to include children registered for displaying behavior that could be prosecuted if displayed by someone aged twelve years and older, children were selected from local registrations systems in three police districts in the Netherlands (Rotterdam-Rijnmond, Gelderland-Midden, and Utrecht).

6.2 Summary

In addressing questions on whether the co-occurrence of risk in distinct life domains can help explain delinquent development, the current thesis started out with providing an overview of literature on interactions between biological and social/environmental correlates of antisocial behavior (Chapter 2). Based on a total of 50 studies (documented in 66 publications), it was shown that children suffering from biological vulnerability – measured as either peri/prenatal risk exposure or extreme psychophysiological functioning – are at increased risk of developing antisocial behavior when they are also exposed to adverse social or environmental circumstances. In addition, biosocial interaction was found to be mostly associated with more severe, violent, and persistent types of antisocial behavior, and play a more significant role in antisocial development in males. Regarding the second aim of the current thesis, Chapter 2 thus highlighted the importance of including both biological and social/environmental explanatory factors in studies aimed at increasing our understanding of antisocial behaviors.

Chapter 3, addressing the first general aim of this thesis, examined the extent to which distinct offending trajectories can be found in a sample of early onset offenders that differ both in shape and nature of offending from early adolescence (age 12) into early adulthood, and whether singular identified risk factors (i.e., gender, ethnicity, and neighborhood factors) can help characterize early onset offenders following distinct offending trajectories. Using multitrajectory modeling, it was shown that – next to an a-priori defined non-recidivist group (55%) – five trajectory subgroups could be identified in the data: sporadic recidivists (25%), and low-rate (8%), moderate-rate (10%), high-rate adult peaked (3%), and high-rate adolescence peaked recidivists (3%). Offenders were overall shown to be versatile in their offending behavior. Early onset offenders assigned to either of the high-rate trajectory subgroups committed a relatively large amount of property crimes, and violent crimes made up an increasing proportion of crimes committed by the high-rate adolescence peaked trajectory-subgroup. Multinomial regression analysis revealed that males, and non-Western participants were more likely to be classified as low-rate offenders than to abstain from crime during follow-up than their counterparts. Residing in a low socioeconomic neighborhood as a child substantially increased the chances of being assigned to high-rate trajectory-subgroups over the non-recidivist subgroup.

While Chapter 3 focused on associations between singular identified risk factors and offense patterns in early onset offenders, Chapter 4 addressed the second general aim of this thesis by studying the extent to which exposure to specific combinations of risk in childhood increases the risk of following specific offending trajectories into early adulthood. Trajectory modeling led to the identification of four trajectory-subgroups next to an a-priori defined group of non-recidivists (55%): low-rate desisting (14%), low-rate persisting

(18%), high-rate desisting (5%), and high-rate persisting (8%). Data on risk exposure revealed that the *Childhood Arrestees Sample* overall constitutes a biologically vulnerable, cognitively challenged and somewhat hyperactive group, experiencing emotional problems and peer victimization, while growing up in disadvantaged neighborhoods (see Chapter 4 for more detailed information on levels of risk in the *Childhood Arrestees Sample*). Latent profile models however revealed heterogeneity in exposure to clusters of risk from multiple life domains, as three risk profiles were found in the data: a low-problem/impulsive (31%), cognitive- and neighborhood-problem (48%), and multi-problem group (21%). Subsequently, and relevant for the second aim of the current thesis, Chapter 4 revealed that children suffering from specific combinations of problems are at risk of following distinct offending trajectories. Multinomial regression analysis showed that children with low levels of problems across life domains were least likely to display persistent offending behavior. Children with impaired cognitive abilities, growing up in disadvantaged neighborhoods were at risk of displaying high-rate persistent offending behavior, whereas children suffering from combinations of internalizing and externalizing problems displayed low-rate persistent offending into early adolescence.

Finally, Chapter 5 continued to address the second general aim of this thesis by examining whether changes in social influences affect individual offending behavior over time, and whether these effects depend on biological vulnerability towards delinquent development. By doing so, Chapter 5 adopted a dynamic approach to both offending behavior, as well as risk exposure in key social life domains (i.e., family, peers, school). As Chapter 2 of the current thesis had revealed that individuals suffering from peri/prenatal risk and social/environmental risk were most likely to display antisocial behavior, Chapter 5 specifically accounted for biological risk resulting from peri/prenatal problems, while studying the effects of changes in social relationships on individual delinquent involvement. Using data gathered during the first, second, and third measurement waves of the *Dutch Childhood Arrestees Study*, hybrid random effects models revealed associations between changes in social relationships over time and changes in self-reported offending behavior. While not all social bonds that were expected to play a role in variation in delinquent involvement were found to exert an influence (i.e., parental bonds), findings showed that an increase in the proportion of delinquent peers proved to be an important offending-inducing change in social circumstances. In addition, Chapter 5 showed that the impact of changes in the social environment depend on biological vulnerability, as a decrease in bonds with school only increased concurrent offense frequency in biologically vulnerable children.

In conclusion, with respect to its first aim, the current thesis found that delinquent development in the *Childhood Arrestees Sample* was often discontinuous. Half of the early onset offenders abstained from crime during follow-up according to official registration data, and the early onset offenders that did

re-offend, mostly did so at a low-rate (i.e., a little over 30% of the sample was registered for an offense every three to five years). Only a small group of early onset offenders– less than 14% of the sample – continued to display frequent and persistent offending behavior according to police crime records. In addition, delinquent development in the *Childhood Arrestees Sample* was found to be highly heterogeneous, as four to five distinct offending trajectories were identified in Chapters 3 and 4. Regarding associations between singular identified risk factors and offending trajectories, it was shown that males, participants of non-Westerns ethnic background, and children residing in low SES neighborhoods below age 12 were more likely to populate the trajectory-subgroups than the non-recidivist group. Unfortunately, the singular identified risk factors were less helpful in differentiating between offenders following distinct re-offending trajectories.

With respect to its second aim, findings presented in the current thesis highlight the relevance of accounting for the co-occurrence of problems in distinct life domains. Risk exposure in various life domains was found to shape the development of delinquent behavior over time. Importantly, the additive effect of risk factors from distinct domains was shown to be more complicated than simply the sum of risk, as specific combinations of risk factors were found to improve our understanding of heterogeneity in longitudinal offending patterns in early onset offenders. In addition, the impact of change in the social environment on offense frequency was found to depend on risk exposure in the individual life domain, as associations between change in social bonds and offense frequency depended on individuals' biological characteristics. Findings from the current thesis therefore stress the importance of examining combinations of problems in distinct life domains when aiming to explain between- and within-individual variation in offending over time.

6.3 Theoretical reflection

The following paragraph discusses the implications of the aforementioned findings on theoretical assumptions regarding delinquent development and associated singular identified risk factors, as well as associated risk exposure across life domains.

6.3.1 Delinquent development and associated singular identified risk factors

The results presented in the current thesis to some extent provide support for assumptions from the most prominent framework used to explain delinquent development in early onset offenders: Moffitt's (1993, 2006) developmental taxonomy (Moffitt, 1993, 2006). First, in line with theoretical expectations (Moffitt, 1993, 2006), the current thesis identified an early onset group display-

ing continuously high offending rates, as well as a group displaying low yet persistent levels of offending across adolescence. Second, findings on the distribution of types of crime across offending trajectories were largely in line with theory (see Moffitt, 1993), as Chapter 3 showed that persistent offenders were versatile in their offending behavior, and were increasingly inclined to commit violent crimes toward early adulthood. Third, results regarding associations between offending trajectories and demographic and childhood neighborhood characteristics were in accordance with the Moffitt-taxonomy (Moffitt, 1993, 2006), as findings revealed that males, non-Western participants, and participants residing in low SES neighborhoods below age 12 were likely to populate the more frequent re-offending pathways.

However, two main findings presented in the current thesis seem to contradict expectations from typological theory. The first contradiction refers to the expected level of persistence in early onset offenders. Typological theory expects all early onset offenders to continuously engage in crime during – at least – adolescence (Moffitt, 1993, 2006). Official registration data revealed however that delinquent behavior in the *Childhood Arrestees Sample* was often discontinuous. Specifically, over half of the sample did not come into contact with the police during follow-up. This finding indicates that a first police registration in childhood is not always followed by a persistent offense pattern into early adulthood. It should be noted however that the *Childhood Arrestees Sample* was still found to be at increased risk of showing persistent delinquent behavior, as the prevalence of offending in the current sample was still three times higher than that of the general Dutch population (Blokland et al., 2010).

The second contradiction refers to the expected heterogeneity in offending trajectories in early onset offenders. Typological theory expects early onset offenders to either offend at a high rate during adolescence (i.e., high-rate chronic offenders), or at a lower, yet persistent, rate into early adulthood (i.e., low-rate chronic offenders) (Moffitt, 2006). However, the current thesis found that at least five offender subgroups could be distinguished in the *Childhood Arrestees Sample*. While this finding is in line with previously found heterogeneity in short-term re-offense patterns in children in contact with the law (van Domburgh, Vermeiren, et al., 2009), it reveals that heterogeneity in offending patterns exceeds theoretical expectations on early onset offenders.

6.3.2 Delinquent development and associated risk exposure across life domains

Findings presented in the current thesis overall highlight the importance of accounting for risk exposure across life domains when studying between- and within-individual variation in delinquent behavior (over time). As such, findings corroborate with theoretical expectations originating from biosocial

(Monroe & Simons, 1991; Zuckerman, 1999), and developmental (Moffitt, 1993; Wright et al., 2001) perspectives on offending.

Regarding between-individual variation in offending, the current thesis supports the theoretical notion that accounting for particular combinations of adverse circumstances in multiple life domains can increase our understanding of long-term delinquent pathways (Caspi et al., 2014; Moffitt, 1993, 2006; Stouthamer-Loeber et al., 2002). Specifically, and in line with the Moffitt-taxonomy, high-level persistent offenders were found to suffer from a combination of cognitive- and neighborhood-related problems, whereas low-level persistent offenders were found to experience a combination of individual (i.e., internalizing as well as externalizing), familial, and peer problems (Moffitt, 2006). Contradicting typological theory, however, is the finding that a substantial part of the sample developed relatively well in most life domains. The fact that the current thesis also identified distinct risk profiles based on level differences in risk exposure is however in line with prior studies identifying risk profiles in offender populations (e.g., T. Brennan et al., 2008; Lopez-Romero et al., 2019; Mulder et al., 2010; Schwalbe et al., 2008).

Lastly, the current thesis also confirmed the theoretical notion that accounting for the co-occurrence of risk in distinct life domains can help increase our understanding of within-individual variation in offending over time (Moffitt, 1993; Wright et al., 2001). In line with theory (Moffitt, 1993; Wright et al., 2001), the current thesis showed that the impact of time-varying social influences is modified by stable individual characteristic, i.e., biological vulnerability in the case of the present thesis. Specifically, in Chapters 2 and 5, it was shown that biologically vulnerable individuals are more likely to display antisocial behaviors when they are also exposed to social adversity. Findings therefore support the notion that individuals differing in levels of biological vulnerability respond in varied ways to similar social environments. While confirming the importance of social influences on offending behavior, results from this thesis do, however, challenge the theoretical assumption from sociological criminology that changes in social circumstances affect all offenders equally (Akers, 1973; Hirschi, 1969; Sutherland, 1947). Rather, findings from the current thesis highlight the relevance of taking offenders' biological traits into account when studying the effects of changes in social relationships on individual offending behavior over time. It therefore seems of great importance to integrate biological, sociological, and developmental perspectives on crime in order to arrive at a more comprehensive explanation of variation in offending behavior.

6.4 Strengths, limitations, and directions for future research

By studying delinquent development in children with a police contact/arrest and its correlates from multiple life domains, the current thesis made an important step in the field of developmental criminology. Specifically, three

unique qualities of this thesis are worth highlighting. First and foremost, the empirical chapters in this thesis are based on a rich dataset consisting of several registration databases, and information from questionnaires with childhood arrestees and their parents. Using this rich dataset made it possible to study delinquent development in this specific high-risk population over an extended period of the life-course, distinguish between a broad range of re-offense patterns, and study assumptions on a broad range of risk factors from multiple life domains. Second, the current thesis applied theoretical insights stemming from different scholarly traditions. By combining insights from sociological, biosocial, and developmental perspectives on offending, the current thesis progressed our understanding of possible mutually reinforcing factors from distinct life domains. This makes the current thesis innovative and interdisciplinary. Third, the use of advanced analytical strategies helped account for possible interaction effects between and clustering of risk factors for offending.

Despite the advancements made in the current thesis, there are a number of limitations that are worth mentioning, as well as some matters that could be addressed in future studies in the field of developmental criminology. First, conclusions drawn in this thesis on delinquent development are based on the specific offender group of childhood arrestees. For future research an essential avenue would be to examine the generalizability of current findings to other samples of early onset offenders – for example, defined as displaying prolonged antisocial behavior at home and at school prior to the age of 12 (see for example Moffitt et al., 1996).

Second, the reliance on police registrations as a measure of offending might have influenced the shapes of the delinquent trajectories identified in Chapters 3 and 4. Defining offending trajectories based on registered crimes rather than self-reported crime might have resulted in an underestimation of the total number of delinquent acts, as we lack information on delinquent behavior unknown to the police. For comparison, prior work comparing conviction data and self-reported offenses revealed that individuals reported 22 offenses for every conviction (Theobald, Farrington, Loeber, Pardini, & Piquero, 2014). Furthermore, underestimations of offending in official registration data might be selective, as chances of being arrested by the police are not equal for all offenders, for example due to selective monitoring. On the other hand, the use of police registrations might have also resulted in an overestimation of offending behavior, as police registrations indicate the number of times a person was identified as a suspect in a criminal case, as opposed to the number of times a person was convicted. Besides these disadvantages of police registrations, the use of official data has important advantages as well. Compared to self-report data, official registration data are more reliable regarding the timing of offenses, and less likely to be disturbed by memory problems (van de Rakt, 2011).

Third, it is important to note that the trajectories identified in Chapter 3 and 4 are not meant to represent 'true types of offenders'. Trajectory modelling is exploratory in nature, and will extract a number of distinct trajectories in most datasets (Morizot, 2019). In addition, trajectory modelling is not accommodating to outliers (Liu & Bushway, 2019), and may force offenders with extreme offending trajectories into larger trajectory subgroups. Trajectories can however be useful in simplifying the complex reality of trajectories of offending behavior (Nagin & Tremblay, 2005), as it is fair to argue that distinguishing between quite opposite courses of offending will increase our understanding of delinquent development with age.

Fourth, even though the current thesis had access to a considerable number of registration databases, information on noncriminal justice interventions during the observation period were not retrieved, and would perhaps have been beneficial. To the extent that parents, schools, child protection services, and other professionals were actively trying to curb participants' delinquent development, the identified trajectories could have evolved either because of or despite such efforts. Future research could strive to include information on noncriminal justice interventions, in order to reveal possible effects of prevention and intervention efforts aimed at reducing recidivism in early onset offenders.

Fifth, the current thesis used a specific definition of biological vulnerability in its empirical chapters; exposure to peri/prenatal problems. As findings from the current thesis showed that simultaneously studying biological and social/environmental correlates of delinquency can contribute to our understanding of the etiology of delinquent development, future research could strive to use other biological parameters (i.e., genetics, brain abnormalities, neuropsychology, psychophysiology, neurotransmitters, and hormones, see F. R. Chen et al., 2015; Raine, 2002a; Rudo-Hutt, 2011; Yang et al., 2014) to explore whether current findings can be replicated when different definitions of biological vulnerability are used.

On a final note, there are several meaningful ways for future research to build on the findings presented in the current thesis. First, future research could aim to study risk exposure on the level of the individual, in order to develop more personalized risk prediction models. Such studies may help transfer research findings based on groups of individuals to the individual level, as well as improve the applicability of research findings to individual treatment plans. Second, it would be useful to identify risk profiles of distinct demographic groups of offenders, as there are indications that concurrent risk factors of offending may differ between such groups (see for example DeLisi et al., 2017; Rhoades, Leve, Eddy, & Chamberlain, 2016). Lastly, as an early onset of offending is theorized to be associated with a range of adverse adolescent and adult outcomes (Moffitt, 1993), it would be interesting to explore whether the use of risk profiles could also improve our understanding of

outcomes such as drug and/or alcohol abuse, young parenthood, and unemployment.

6.5 Practical implications

Findings from the current thesis offer three main implications for prevention and intervention efforts aimed at curbing offense patterns in childhood arrestees. First, the discontinuity of offending in a large share of the *Childhood Arrestees Sample* indicates that intervening at a young age may be unnecessary for a substantial group of early onset offenders known to the police. Rather, generally healthy developing children would benefit most from being diverted away from intervention programs, as prior work revealed that targeting offenders at low risk of re-offending may actually lead to an increase in their re-offending behavior (Lowenkamp & Latessa, 2002). When the police refers childhood arrestees to Child Welfare Services Youth, short interventions may thus be offered when problems in the child and the family are found to be limited.

Second, by revealing heterogeneity in offense patterns among early onset recidivists, Chapters 3 and 4 showed that a small proportion of childhood arrestees continues to display persistent offending behavior and inflicts substantial harm on others. In this small group of childhood arrestees, it is therefore important to prevent the progression along persistent offending pathways. As Chapter 4 showed that children experiencing problems across life domains are at increased risk of continuing their delinquent behavior, it is important for Child Welfare Services Youth to refer those children and their family to a care institution and offer them required help. Findings reported in Chapter 4 suggest that offering help to prevent the escalation of offending behavior would be most viable when aimed at children residing in neighborhoods characterized by low socioeconomic status, especially if children also suffer from low intelligence levels, because, as a group, such children were found to be at increased risk of continuously engaging in crime at a high-rate.

Third, besides pointing towards groups of childhood arrestees at risk of displaying persistent offending behavior, the current thesis also offers insight into the types of problems interventions could target. Chapter 5 showed that several malleable social influences are related to change in individual offending behavior, and these social factors may therefore be the focus of intervention efforts (for an overview of multifaceted interventions for juvenile offenders, see Boxer & Goldstein, 2012). Specifically, findings from the current thesis indicate that it is important for practitioners and clinicians to focus on minimizing contact with delinquent peers, as an increase in affiliation with delinquent peers was associated with an increase in offense frequency over time. In addition, current findings suggest that it is beneficial to address early signs of school disengagement, as skipping class has the potential to facilitate an

increase in individual offense rates. Thus, findings from the current thesis overall suggest that it is most effective for interventions to prevent the hindrance of conventional developmental pathways. Interventions should focus on creating a wide support system to help facilitate positive development in order to decrease the risk of continued delinquent involvement.

Samenvatting (Dutch summary)

Een onderzoek naar de aard en verklaring van de langetermijnontwikkeling van delinquent gedrag van kinderen met een politiecontact

Achtergrond en doel

Het criminele gedrag van de meeste personen is slechts van korte duur. Echter, een kleine groep daders vertoont frequent en langdurig crimineel gedrag. Dergelijke persistente daders beginnen vaak al op jonge leeftijd met het vertonen van delictgedrag. Tot op heden ontbreekt echter adequate kennis over de langetermijnontwikkeling van delictgedrag van kinderen die op jonge leeftijd in contact komen met politie, vanwege een gebrek aan justitiële gegevens over zeer jonge kinderen, en het ontbreken van longitudinale gegevens om die kinderen prospectief voor een lange periode te volgen. Om die reden resteert de vraag of kinderen met een politiecontact voorbestemd zijn om persistent delinquente gedragspatronen te ontwikkelen. Of kunnen zelfs de jongste kinderen in contact met politie opgroeien tot sociaal en conformerende volwassenen? En als sprake is van heterogeniteit in de delinquente ontwikkeling, welke kinderen stoppen dan met het plegen van delicten? En welke kinderen zetten het delictgedrag met een bepaalde frequentie voort? Antwoord op deze vragen is van belang om op juiste wijze te kunnen reageren op kinderen die vanwege delictgedrag in contact komen met politie, om zo een potentieel lange criminele carrière te voorkomen.

Dit proefschrift beoogt inzicht te geven in de langetermijnontwikkeling van delinquent gedrag van kinderen met een politiecontact. Een deel van dit proefschrift is gericht op de beschrijving van ontwikkelingspaden van delinquent gedrag van deze kinderen en de rol van specifieke risicofactoren. Het andere deel van dit proefschrift heeft ten doel om variatie in ontwikkelingspaden van delinquent gedrag te verklaren op basis van mogelijke clusters en wederzijds versterkende effecten van risicofactoren uit verschillende levensdomeinen, waaronder individu, familie, leeftijdsgenoten, school en buurt.

In de empirische hoofdstukken is gebruik gemaakt van data van de *Dutch Childhood Arrestees Study* (Hoofdstuk 3, 4, en 5). Deze data bevatten informatie over ruim 700 kinderen die tussen 2000 en 2006 vanwege het vertonen van delictgedrag onder de twaalf jaar voor het eerst door de politie zijn geregistreerd. Een belangrijk voordeel is dat er informatie aanwezig is over delict-

gedrag van kindertijd tot in de vroege volwassenheid, evenals gedetailleerde gegevens over blootstelling aan risicofactoren in de verschillende levensdomeinen. Deze unieke data zijn uitermate geschikt om de langetermijnontwikkeling van kinderen met een politiecontact in kaart te brengen, evenals de rol van risicofactoren uit verschillende levensdomeinen te bestuderen.

Resultaten

Aangezien het samenspel van risicofactoren in verschillende levensdomeinen centraal staat in dit proefschrift, geeft het eerste inhoudelijke hoofdstuk (Hoofdstuk 2) een overzicht van literatuur over interacties tussen biologische factoren en sociale- en omgevingsinvloeden gerelateerd aan antisociaal gedrag. In dit hoofdstuk is aandacht besteed aan twee biologische factoren, te weten peri- en prenataal risico (waaronder prenatale blootstelling aan alcohol en drugs) en psychofysiologisch functioneren (de intensiteit waarmee het lichaam reageert op stressvolle situaties). De resultaten van 50 geïncludeerde wetenschappelijke onderzoeken tonen samen aan dat biologisch kwetsbare kinderen vooral een verhoogd risico hebben om antisociaal gedrag te ontwikkelen als zij worden blootgesteld aan risicovolle sociale- of omgevingsinvloeden, waaronder blootstelling aan ouders met psychische problemen en het wonen in achterstandswijken. Daarnaast blijkt dat deze biosociale interactie met name verband houdt met ernstig, gewelddadig en persistent antisociaal gedrag. Ten slotte toont Hoofdstuk 2 aan dat dit met name geldt voor mannen.

In Hoofdstuk 3 is de langetermijnontwikkeling van delinquent gedrag van kinderen met een politiecontact in kaart gebracht. In aanvulling daarop is onderzocht in hoeverre specifieke socio-demografische en buurtkenmerken verband houden met delinquente ontwikkelingspaden. De volgende zes groepen konden onderscheiden worden op basis van politieregistratiegegevens over pleegfrequentie en type delict gepleegd in de leeftijd van 12 tot 25: 1) *niet-recidivisten* (51%), 2) *sporadische recidivisten* (25%) met slechts één of een enkele registratie tussen de leeftijd van 12 en 25, 3) *laag-frequente recidivisten* (8%) met gemiddeld één registratie per twee à drie jaar en een piek in delinquente activiteit rond de leeftijd van 18 jaar, 4) *gemiddeld-frequente recidivisten* (10%) met gemiddeld één registratie per jaar en meer registraties voor het plegen van geweldsmisdrijven dan de sporadische recidivisten, 5) *hoog-frequente laat-piekende recidivisten* (3%) met bijna twee registraties per jaar en een piek in pleegfrequentie rond de leeftijd van 22 jaar, en 6) *hoog-frequente vroeg-piekende recidivisten* (3%) met gemiddeld ruim twee registraties per jaar, en een piek in delinquente activiteit rond de leeftijd van 18 jaar, waarna deze groep relatief vaak geweldsmisdrijven gaat plegen. Vergeleken met de niet-recidivisten, zijn de laag-frequente recidivisten vaker man en niet-westers, en de hoog-frequente recidivisten vaker afkomstig uit buurten met een lage sociaaleconomische status.

In Hoofdstuk 4 is achterhaald in hoeverre het verloop van delinquent gedrag van kinderen met een politiecontact verklaard kan worden op basis van blootstelling aan combinaties van risicofactoren in de kindertijd. Op basis

van politiegegevens omtrent pleegfrequentie kunnen vijf groepen onderscheiden worden: niet-recidivisten (55%), laag-frequente recidivisten met een afnemend delictpatroon (14%), laag-frequente recidivisten met een persistent delictpatroon (18%), hoog-frequente recidivisten met een afnemend delictpatroon (5%), en hoog-frequent recidivisten met een persistent delictpatroon (8%). Op basis van blootstelling aan combinaties van risicofactoren konden drie risicoprofielen onderscheidden worden: 1) een impulsieve groep met relatief weinig andere problemen (*impulsieve groep*, 31%), 2) een cognitief beperkte groep wonende in achterstandswijken en relatief weinig problemen in het gezin en met leeftijdsgenoten (*cognitieve- en buurt-probleem groep*, 48%), en 3) een groep met problemen in alle levensdomeinen (*multi-probleem groep*, 21%). De impulsieve groep is het minst geneigd om persistent delinquent gedrag te vertonen. De cognitieve- en buurt-probleem groep ontwikkelt het vaakst hoog-frequent en persistent delictgedrag, terwijl de multi-probleem groep het vaakst laag-frequent en persistent delictgedrag vertoont.

In Hoofdstuk 5 is onderzocht in hoeverre veranderingen in relaties met familie, leeftijdsgenoten en school verband houden met veranderingen in de frequentie van zelfgerapporteerd delictgedrag van kinderen met een politiecontact. In Hoofdstuk 5 is tevens nagegaan of het verband tussen veranderingen in sociale invloeden en pleegfrequentie afhankelijk is van blootstelling aan peri- en prenataal risico. De resultaten toonden aan dat verandering in relaties met ouders geen verband houdt met verandering in de mate van delictgedrag. Verdere analyses wezen uit dat kinderen vaker delinquent gedrag vertonen in de jaren dat zij meer delinquente vrienden hebben. Ten slotte toonde Hoofdstuk 5 aan dat een afname van betrokkenheid bij school verband houdt met een toename in delictgedrag bij biologisch kwetsbare kinderen, terwijl dit voor biologisch niet-kwetsbare kinderen niet het geval is.

Conclusie

Concluderend blijkt dat de meeste kinderen met een politiecontact niet terugkomen in de politiestatistieken gedurende de follow-up. Deze bevinding toont aan dat kinderen met een politiecontact niet voorbestemd zijn om een persistent delinquent ontwikkelingspad te volgen. In tegenstelling, de meeste kinderen komen niet meer in aanraking met de politie. Bovendien is de delinquente ontwikkeling van de recidivisten zeer heterogeen, variërend van een groep die sporadisch en met afnemende mate delictgedrag vertoont tot een groep die zeer frequent en persistent delictgedrag vertoont tot in de vroege volwassenheid. Jongens, kinderen met een niet-westerse achtergrond, en kinderen uit buurten met een lage sociaaleconomische status behoren vaker tot de recidivisten dan tot de niet-recidivisten. De specifieke factoren sekse, etniciteit, en buurtkenmerken kunnen echter niet verklaren in welk ontwikkelingspad de recidivisten terecht komen. Daarvoor blijkt het bestuderen van

combinaties van factoren noodzakelijk. De bevindingen van dit proefschrift benadrukken dan ook het belang van het includeren van risicofactoren uit verschillende levensdomeinen in onderzoek naar de langetermijnontwikkeling van delinquent gedraging. Het gezamenlijke effect van blootstelling aan risico in verschillende domeinen is complexer gebleken dan enkel de som van risicofactoren. Uit de resultaten komt namelijk naar voren dat combinaties van risicofactoren verschillen in het verloop van delictgedrag tussen personen kunnen verklaren, evenals veranderingen in pleegfrequentie binnen personen over tijd. Zo tonen de resultaten aan dat kinderen met een laag intelligentieniveau afkomstig uit achterstandswijken vaker hoog-frequent en persistent delictgedrag vertonen dan kinderen met relatief weinig problemen in alle levensdomeinen.

Beleids/Praktijkaanbevelingen

Uit huidig onderzoek is gebleken dat kinderen met een politiecontact worden gekenmerkt door verschillende risicoprofielen die om een aparte aanpak vragen. De heterogeniteit aan problemen en risico op recidive zoals gerapporteerd in dit proefschrift benadrukken dan ook het belang van het kunnen bieden van interventies die passen bij de specifieke problemen van het kind, zodat per kind op de meest passende wijze gereageerd kan worden op een eerste politiecontact.

Het bepalen van een passende interventie dient gebaseerd te zijn op een waardering van blootstelling aan risico in verschillende levensdomeinen. De mate van risico in verschillende levensdomeinen blijkt namelijk verband te houden met variatie in delinquente gedragspatronen tot in de vroege volwassenheid. Zo blijkt dat kinderen met relatief weinig problemen in alle levensdomeinen het delictgedrag vermoedelijk niet voortzetten. Voor deze groep kinderen zou een korte groepsinterventie op school of in de buurt volstaan. Tegelijkertijd laten de bevindingen ook zien dat kinderen met problemen in meerdere levensdomeinen mogelijk persistent delictgedrag ontwikkelen. Deze bevinding benadrukt het belang van vroegtijdig ingrijpen om te voorkomen dat jonge, nog niet strafbare kinderen zich ontwikkelen tot persistente daders. Als kinderen met een politiecontact multiproblematiek ervaren, wordt kind en ouders/verzorgers idealiter een multimodale interventie aangeboden, waarbij aan de hand van 1-op-1 begeleiding gewerkt kan worden aan problemen van het kind, binnen de familie, met leeftijdsgenoten en op school. Dergelijke interventies dienen nadrukkelijk toegankelijk te zijn voor kinderen met een licht verstandelijke beperking, aangezien de resultaten wijzen op verhoogd risico op persistent delictgedrag bij de groep kinderen met een laag IQ, wonende in achterstandswijken. Aangezien dit proefschrift verder heeft aangetoond dat de frequentie van delictgedrag mogelijk toeneemt bij een toename in spijbelgedrag en vriendschappen met delinquente leeftijdsgenoten, lijkt het

zinvol om in de klinische praktijk te proberen de band met school en prosociale leeftijdsgenoten te versterken.

Aanbevelingen voor toekomstig onderzoek

Met dit proefschrift is een belangrijke stap gezet in het onderzoek binnen de ontwikkelingscriminologie door de langetermijnontwikkeling van delictgedrag van kinderen met een politiecontact te bestuderen, en bijbehorende verklarende clusters van risicofactoren te onderzoeken. Echter, belangrijke onderzoeksvragen resteren die aandacht behoeven in toekomstig onderzoek. Hieronder volgen enkele aanwijzingen voor vervolgonderzoek die voortvloeien uit de studies gepresenteerd in dit proefschrift.

Een aanbeveling voor toekomstig onderzoek is om informatie over (buiten)strafrechtelijke interventies te verzamelen. Bevindingen gepresenteerd in dit proefschrift tonen aan dat de meeste kinderen met een politiecontact een waaier aan problemen ervaren. Het is daarom aannemelijk dat ouders, scholen, de Raad voor de Kinderbescherming en andere professionals hebben geprobeerd de delinquente ontwikkeling van het kind zoveel mogelijk te beperken. De delinquente ontwikkelingspaden zoals gepresenteerd in dit proefschrift zijn mogelijk mede het gevolg van dergelijke inspanningen. Door informatie over interventies te verzamelen kan meer inzicht worden verkregen in de redenen waarom kinderen bepaalde delinquente ontwikkelingspaden volgen. Tevens is het includeren van beschermende factoren, zoals vriendschappen en liefdesrelaties met niet-delinquente leeftijdsgenoten, aan te bevelen. Dit kan leiden tot een betere duiding van de relatie tussen sociale- en omgevingsinvloeden en delinquente gedragspatronen.

Vervolgonderzoek is ook nodig om enkele nieuwe onderzoeksvragen te adresseren. Zo zou toekomstig onderzoek gericht kunnen zijn op het ontwikkelen van modellen die toekomstig delictgedrag op individueel niveau voorspellen in plaats van op groepsniveau, wat de relevantie van onderzoeksresultaten voor individuele behandelplannen zou vergroten. Daarnaast zou het interessant zijn om te onderzoeken of het gebruik van risicoprofielen ook kan helpen bij het verklaren van de ontwikkeling van drugs- en alcoholmisbruik, aangezien delinquent gedrag in de kindertijd verband houdt met een reeks deviante gedragingen in de adolescentie en volwassenheid.

Hoewel vervolgonderzoek aan te bevelen is, kan op basis van dit proefschrift in ieder geval worden vastgesteld dat kinderen met een politiecontact aanzienlijke heterogeniteit vertonen in hun recidivepatronen, en dat het bestuderen van combinaties van risicofactoren uit meerdere levensdomeinen ons begrip van de heterogene ontwikkeling van delinquent gedrag op de lange termijn vergroot. Het is daarom van groot belang om kennis vanuit de sociologische, biosociale, en ontwikkelingscriminologie te integreren om tot een meer

omvattende verklaring van (variatie in) de ontwikkeling van delinquent gedrag te komen.

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Babette, maart 2021

Curriculum vitae

Barbara (Babette) Catharina Maria van Hazebroek was born on the 7th of September 1988 in Haarlem, the Netherlands. In 2011 she obtained two master's degrees at the VU Amsterdam University: one in Life Course Criminology and one in Developmental Psychology (cum laude). After graduating, Babette worked as a lecturer at the Department of Psychology at VU Amsterdam University (2011-2013), and at the Department of Criminology at Leiden University (2013-2016). From 2011 until 2013, she also worked as a social worker for Spirit at 'Beter met Thuis', supervising children who were (temporarily) unable to live with their parents. From February 2016 until May 2020, Babette worked on her doctoral research on the long-term delinquent development of children with a police registration at Leiden University. During her doctoral research, she contributed to the data collection of the *Childhood Arrestees Project*, and was schooled in diverse forms of longitudinal data analysis at the Utrecht Summer School of 2018. As of June 2020, Babette is working as an Assistant Professor at the Institute of Criminal Law and Criminology at Leiden University.

In the range of books published by the Meijers Research Institute and Graduate School of Leiden Law School, Leiden University, the following titles were published in 2020 and 2021

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E.M. MEIJERS INSTITUUT

INSTITUUT VOOR RECHTSWETENSCHAPPELIJK ONDERZOEK

We know that most persistent offenders who cause considerable damage to society showed delinquent behavior in childhood. However, the long-term development of childhood arrestees is not well understood as longitudinal data are largely lacking. Do these high-risk children develop long-term offense patterns? Or are even the youngest children with a police contact capable of growing into law abiding adults? Or is it both? And, in case of the latter, which childhood arrestees stop showing delinquent behavior, and which children persist in crime into early adulthood? By providing insight into the long-term development of offending of childhood arrestees, and uncovering its explanatory factors, the current thesis improves our understanding of the delinquent development in this high-risk offender group.

The current thesis reveals that, in contrast to popular belief, childhood arrestees are not predestined to develop persistent delinquent behavior, as most children are not re-arrested between the age of 12 and 25. Recidivists display heterogeneity in their re-offense patterns, with only a small group of children developing into persistent offenders. Accounting for simultaneous risk exposure across life domains proved necessary to explain why childhood arrestees follow one trajectory over another. Problems in multiple life domains were found to predict persistent offense patterns.

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