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

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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).

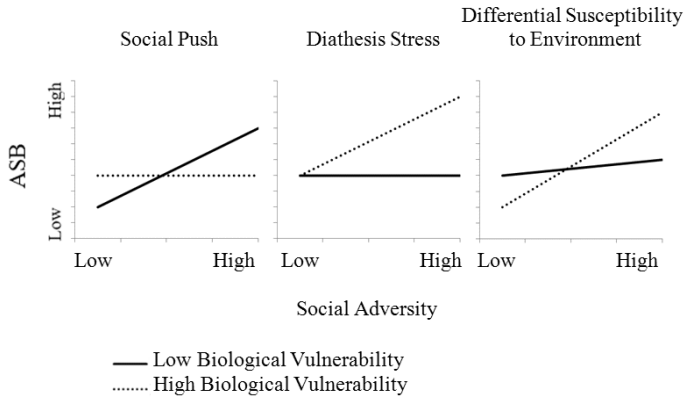
This last-mentioned theoretical perspective has been extended to encompass the idea that individuals with biological vulnerabilities have the lowest levels of ASB in privileged social environments (Belsky, 1997; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009; Boyce & Ellis, 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