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Review article

Heart rate and skin conductance associations with physical aggression, psychopathy, antisocial personality disorder and conduct disorder: An updated meta-analysis

Peter C. de Looff[a,b,c,*], Liza J.M. Cornet[d,1], Catharina H. de Kogel[e], Belén Fernández-Castilla[f], Petri J.C.M. Embregts[g], Robert Didden[a,c,h], Henk L.I. Nijman[a,b,c,2]

[a] Behavioural Science Institute, Radboud University, Nijmegen, the Netherlands
[b] Fivoor, Science and Treatment Innovation, Den Dolder, the Netherlands
[c] Expertcentre "De Borg", Den Dolder, the Netherlands
[d] Institute of Education and Child Studies, Leiden University, Leiden, the Netherlands
[e] Research and Documentation Centre (WODC), Ministry of Justice and Security, The Hague, the Netherlands
[f] Faculty of Psychology and Educational Sciences and Imec-ITEC, KU Leuven, Belgium
[g] Department of Tranzo, Tilburg School of Social and Behavioral Sciences, Tilburg University, Tilburg, the Netherlands
[h] Trajectum, Specialized and Forensic Care, Zwolle, the Netherlands

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ABSTRACT

The associations between physiological measures (i.e., heart rate and skin conductance) of autonomic nervous system (ANS) activity and severe antisocial spectrum behavior (AB) were meta-analyzed. We used an exhaustive partitioning of variables relevant to the ANS–AB association and investigated four highly relevant questions (on declining effect sizes, psychopathy subscales, moderators, and ANS measures) that are thought to be transformative for future research on AB. We investigated a broad spectrum of physiological measures (e.g., heart rate (variability), pre-ejection period) in relation to AB. The search date for the current meta-analysis was on January 1st, 2020, includes 101 studies and 769 effect sizes. Results indicate that effect sizes are heterogeneous and bidirectional. The careful partitioning of variables sheds light on the complex associations that were obscured in previous meta-analyses. Effects are largest for the most violent offenders and for psychopathy and are dependent on the experimental tasks used, parameters calculated, and analyses run. Understanding the specificity of physiological reactions may be expedient for differentiating between (and within) types of AB.

1. Introduction

In recent decades, many researchers have tried to unravel the biological correlates of antisocial spectrum behavior (AB) in order to better understand its etiology, development and treatment. Varying definitions and sub classifications have been put forward for AB, which, in turn, have proven to be useful for both theory and practice. For instance, AB can form part of diagnostic criteria, such as in the cases of antisocial personality disorder and conduct disorder (American Psychiatric Association, 2013), or it can pertain to the display of physically aggressive behavior, number of violent offences or psychopathy (Lorber, 2004; Ortiz and Raine, 2004), among other classifications (Merk et al., 2005; Raine, 2019). One area of focus is the association between AB and autonomic nervous system (ANS) functioning, which refers to the regulation of largely unconscious bodily activities including, among other things, heart rate (HR), digestion, sweating and respiratory rate (e.g., Beauchaine et al., 2007; Lorber, 2004; Ortiz and Raine, 2004; Portnoy and Farrington, 2015). Research has resulted in a wealth of literature indicating that AB is associated with altered (pathological) ANS functioning, with attenuated levels of resting HR and reduced skin conductance (SC) being among the most predominant findings (Lorber, 2004; Ortiz and Raine, 2004).

Relatively few attempts have been made to systematically review extant literature on the complex relationship between AB and ANS functioning. Two meta-analyses were conducted by Lorber (2004) and Ortiz

* Corresponding author at: Wier, Specialized and Forensic Care, Citroenvlinder 4, 3734 AD, Den Dolder, the Netherlands.
E-mail address: peterdelooff@gmail.com (P.C. de Looff).

[1] Present address: Studio Wetenschap, Amsterdam.

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and Raine (2004), which were followed by a more recent meta-analysis exclusively focused on resting HR and AB by Portnoy and Farrington (2015). Based on their meta-analysis, Ortiz and Raine (2004) concluded that a low resting HR can be considered as "the best-replicated biological correlate to date of antisocial behavior" (p. 1). In fact, both Lorber (2004) and Ortiz and Raine (2004) found small to medium effect sizes (ESs; $d = -0.38$ and $d = -0.44$) between resting HR and AB, thus indicating that individuals with AB are generally characterized by lower resting HR compared to individuals without AB. This finding was subsequently replicated in the meta-analysis by Portnoy and Farrington (2015), albeit with small(er) ESs ($d = -0.15$ – $d = -0.20$). Furthermore, Lorber (2004) concluded that the most important, straightforward and compelling result was the low level of SC among individuals with psychopathy/sociopathy during periods of rest and during tasks (i.e., this also includes *reactivity,* which is typically calculated as the difference between rest and task values), but only for negatively valenced experimental stimuli (i.e., tasks that typically seek to illicit a negative emotion by including negative stimuli such as fear or disgust; Lorber, 2004, p. 540). Although these aforementioned meta-analyses undoubtedly increased extant knowledge about the relationship between AB and ANS, there are nevertheless important aspects that need to be addressed. The aim of the present meta-analysis is to update these previous meta-analyses, while, simultaneously, including a broader range of ANS measures (e.g., heart rate variability). This approach was previously proposed by Lorber (2004), based on the finding that only one study on HR variability was available for his meta-analysis at that time (Umhau et al., 2002).

1.1. Proteus phenomenon

The first goal of the present meta-analysis is to update extant literature with respect to the ANS–AB relationship. Portnoy and Farrington (2015) concluded that the ESs associated with low resting HR and AB have decreased over time. According to the authors, this is because there is a tendency to report significant findings in the early stages of research. Over time, an increasing number of studies have produced either null results or findings that ultimately refuted the initial strong associations reported between low resting HR and AB. This phenomenon has been designated as the "Proteus phenomenon" (Ioannidis and Trikalinos, 2005). We hypothesize that the Proteus phenomenon is present in a broader range of ANS parameters that are studied in the current meta-analysis.

1.2. Psychopathy subscales

The vast majority of studies included in the previous meta-analyses of the ANS–AB association, focused on HR and SC levels. However, the ANS has two branches: the sympathetic branch (popularly referred to as "fight or flight") and the parasympathetic (also referred to as "rest and digest") branch (Appelhans and Luecken, 2006; Beauchaine and Thayer, 2015; Grossman and Taylor, 2007; Oldenhof et al., 2019), which dually innervate the heart (Jarczok et al., 2016; Porges, 2001; Xu et al., 2016). There is a gap in extant literature concerning how this dual innervation of the ANS branches is related to AB (Godfrey & Babcock, 2020).

HR(V) measures primarily derive from sympathetic innervation, parasympathetic innervation or both[3] (see, for instance, Jarczok et al., 2013; Task Force Electrophysiology, 1996), while SC is thought to primarily be the result of sympathetic innervation (Boucsein, 2012). Typically, both branches exert antagonistic control over the heart, resulting in differential associations between sympathetic and parasympathetic branches with respect to behavioral and emotional regulation (Oldenhof et al., 2019). Notwithstanding the antagonistic effects of the ANS, co-inhibition and co-activation of the branches has also been reported as distinguishing between different types of AB (Suurland et al., 2018; Thomson et al., 2019a; Zhang and Gao, 2015). For instance, primary psychopathy has been associated with co-inhibition (i.e., low scores on sympathetic and parasympathetic measures) during a negatively valenced task, while secondary psychopathy has been associated with high parasympathetic reactivity (i.e., high scores on parasympathetic measures; Thomson et al., 2019b).

Hitherto, several studies have posited that specific aspects of ANS functioning might be dependent on different types of AB, which has primarily been investigated in relation to psychopathy subscales (Armenti and Babcock, 2018; Casey et al., 2013; Fanti et al., 2017; Goulter et al., 2019; Hansen et al., 2007; Kavish et al., 2019a; Ling et al., 2018; Verschuere et al., 2005). Therefore, the second goal of the present meta-analysis is to further investigate how these different ANS measures are related to psychopathy, based on the hypothesis that the relationship between ANS and psychopathy is dependent on the subscales of psychopathy questionnaires (i.e., resulting in specific co-inhibitory, co-activation, or antagonistic effects dependent on the subscales).

1.3. Moderators

Ortiz and Raine (2004) tested several covariates that failed to moderate the association between resting HR and AB (i.e., age, gender, type of control group, recording method, recruitment source, research design, source of behavioral assessment, and year of publication). Portnoy and Farrington (2015) performed similar analyses and concluded that "the relationship between low resting HR and high levels of antisocial behavior was confirmed in longitudinal research, was unaffected by sample age, was robust after controlling for covariates, was present in both male and female samples, and was also characteristic of multiple types of antisocial behavior, including psychopathy" (p. 42). The moderators that were used in previous meta-analyses are included in the current study, because they were studied solely in relation to (resting) HR (Ortiz and Raine, 2004; Portnoy and Farrington, 2015), as opposed to also SC and HR variability, or in relation to various other analysis types (i.e., task and reactivity). In addition, the majority of the moderators used in previous meta-analyses were not included by Lorber (2004).

1.4. ANS specificity related to experiments and behavior type

Based on the earlier hypotheses of Lykken (1957); Hare (1968), and Iacono (1991); Lorber (2004) hypothesized that specificity of physiological measures might be used to differentiate between types of AB based on experimental valence (negative vs non-negative), and analysis type (rest, task, and reactivity). This *specificity* hypothesis was, to some extent, also proposed by Lykken (1957), who concluded that primary psychopaths showed blunted patterns of ANS functioning concerning fear conditioning and avoidance learning compared to a control group, thus suggesting at least some degree of specificity of ANS functioning among individuals with psychopathy. While Lykken (1957) used a fear-conditioning experiment, Hare (1968) conducted an orienting response (tones) experiment that showed blunted ANS functioning among individuals with primary and secondary psychopathy. Furthermore, Lorber (2004) concluded that ANS specificity for behavior types might not always exist, and, in the event that they do, might be dependent on both the type of ANS measure (HR or SC), stimulus valence (negative vs non-negative) and analytic measure (rest, task, reactivity) used, and the behavior type (aggression, psychopathy,

[3] The current meta-analysis includes primarily sympathetically innervated measures (SC = Skin Conductance; PEP = Pre-Ejection period), primarily parasympathetically innervated measures (RMSSD = Root Mean Square of Successive Differences; HF = High Frequency; RSA = Respiratory Sinus Arrhythmia) and mixed innervation measures (LF = Low Frequency, IBI = Inter-beat Interval). For an overview of standards of measurement, interpretation, and clinical use, the reader is referred to the guidelines set by both the Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology.

antisocial personality disorder, conduct disorder) studied. However, Lorber (2004) might have had an insufficient volume of data that prohibited him from drawing any firm conclusions, which is evident from, for example, the low number of ESs for both HR variability and antisocial personality disorder. Lorber (2004) highlighted the distinctive patterns in ANS functioning between psychopathy and aggression (for different analysis types), alongside reporting negative associations between psychopathy and SC (for negative stimuli), while aggressive behavior was found to be negatively associated with resting HR and positively associated with HR reactivity (for adults and negative stimuli). Moreover, several significant associations were reported for conduct disorder, while several non-significant ESs were also evident. Notwithstanding the specific associations reported by Lorber (2004); Ortiz and Raine (2004) also reported medium to large ESs for HR reactivity measures.

Although the differentiation in ANS activity provided by both Lorber (2004) and Ortiz and Raine (2004) has proven to be highly expedient for understanding the sources of heterogeneity, the practical and theoretical utility of their respective results are limited. For instance, Lorber's (2004) proposed classification of experiments into negative vs non-negative valenced stimuli results in limited explanatory power. Although receiving shocks, listening to aversive tones or watching unpleasant pictures all involve being presented with a negatively valenced stimulus, they cannot easily be compared to one another. Besides differences between negative vs non-negative stimuli, differences have also been reported for positive and neutral stimuli. In light of this, it is our contention that a rigorous investigation of these different types of experiments will progress the field forward, insofar as these experiments were constructed for different purposes. We hypothesize that an exhaustive partitioning of studies, (valence of) experiments, and types of analysis might prove beneficial for investigating the *specificity* hypothesis.

A similar rationale regarding the limited practical and theoretical utility applies to differentiating between types of behavior. For instance, laboratory aggression often consists of applying noise bursts or mild shocks to study participants who pose as opponents, but this constitutes a wholly different form of AB than, for example, psychopathy or violent offending. Nevertheless, these categories were collapsed within the aggression category in Lorber's (2004) meta-analysis, and, as such, require further partitioning. In addition, laboratory aggression carried out by students and offenders is also different from mental health disorders and aggressive behavior. It is therefore important to disentangle these specific associations with ANS functioning. Related to this point is that antisocial personality disorder and conduct disorder are formal diagnoses in two classification systems for mental health disorders and disease, while psychopathy and aggression are not (American Psychiatric Association, 2013; World Health Organization, 2004). In fact, psychopathy is mentioned as a synonym of antisocial personality disorder in the classification systems (DSM-5, American Psychiatric Association, 2013; ICD-10, World Health Organization, 2004) and as a distinctive aggravated feature (only in DSM-5), which raises questions on including psychopathy as a separate category in the previous meta-analysis (Lorber, 2004). However, psychopathy has been studied extensively as a separate construct in the last years, suggesting that it might be worth investigating the specificity in comparison to antisocial personality disorder. Moreover, physical aggression is a behavior type, while psychopathy, antisocial personality disorder and conduct disorder are not, although they may increase the risk of aggression, they do not necessarily lead to violent behavior. Therefore, in the current study, and in line with Lorber (2004), antisocial spectrum behavior (AB) is used as a term to capture an overarching category that includes the infraction of commonly accepted rules and the violation of rights of others common to psychopathy, aggressive behavior, antisocial personality disorder, and conduct disorder.

Recently, two large-scale longitudinal studies reported that the association between HR and AB was slightly stronger for violent than for non-violent crime among men (Latvala et al., 2015; Murray et al., 2016a, b). Further, based on findings from the Add Health study, Kavish et al. (2019b) suggested that low HR findings among individuals with psychopathy might be due to the inclusion of more overt types (i.e., overt and (physically) aggressive) of AB. Hence, the magnitude of the ANS–AB association might be differentially influenced by how overt and violent the AB is. Although the aforementioned studies were not included in Portnoy and Farrington's (2015) meta-analysis, it is important to note that they found that officially registered offending behavior was not associated with low HR ($d = -.04$). However, they also expressed caution over this conclusion, on the basis that there was a relative dearth of studies investigating this specific association. Having said this, Portnoy and Farrington (2015) did report the largest ES for violent behavior and resting HR ($d = -0.35$).

1.5. Statistical model

Despite including the aforementioned moderators, there remains the likelihood that a clear and concise interpretation of the ANS–AB association is complicated by complex interactions and heterogeneity, both in terms of analysis and reporting. Therefore, the present meta-analysis proposes utilizing a database structure in order to increase the comparability between studies, which can easily be extended to study ESs that were controlled for by covariates (only bivariate measures are used in the present study). In addition, increasingly sophisticated statistical models have been developed to carry out meta-analyses in recent years (Cheung, 2014; Hedges et al., 2010; Van den Noortgate et al., 2013, 2015). This is important, because earlier meta-analyses (Lorber, 2004; Ortiz and Raine, 2004; Portnoy and Farrington, 2015) were precluded from including dependent ESs due to the sophistication of the statistical models at that juncture (Fernández-Castilla et al., 2020). This explains why Portnoy and Farrington (2015) only chose one ES when multiple comparators were available, and opted for the most cited, reliable instrument, as well as aggregated subscale scores, all of which effectively increased the risk for publication bias.

For the purposes of the present meta-analysis, we opted to apply a statistical model that accounts for dependent ESs, thus allowing for the inclusion of manifold ESs for each study, which, in turn, increases the power of the analysis. Another marked difference between the present meta-analysis and Portnoy and Farrington's (2015) study, is that they preferred ESs which were controlled for by covariates, whereas we only included bivariate measures. This decision was made in light of debates over how the use of partially controlled covariates influence the size of the ES (Rothstein & Bushman, 2015), although other authors have subsequently argued that partial correlations can be analyzed if they represent a similar construct (Furuya-Kanamori & Doi, 2016), which could easily be incorporated into the current database format in the future.

1.6. The present study

Based on contemporary research, and in an attempt to move the field forward, we hypothesize that a more rigorous partitioning of studies (e.g., physiological measures and parameters, types of experiments, types of analyses and valence types) will aid the identification of specific ANS measures that can be used to differentiate between different types of AB. More severe types of antisocial and violent behavior are included in the current meta-analysis: physical aggression (which is divided into physical aggression, laboratory aggression, and violent offenses), psychopathy, antisocial personality disorder, and conduct disorder. We also include an extensive range of experiments and questionnaires to target sources of heterogeneity in ESs for the ANS–AB association.

The following research questions are investigated: (1) Is the Proteus phenomenon also evident in other ANS measures besides (resting) HR? (2) Is the relationship between ANS and psychopathy dependent on subscales of psychopathy questionnaires, resulting in specific co-

inhibitory, co-activation, or antagonistic effects? (3) Which covariates moderate the association between ANS and AB? (4) Is there any evidence for the specificity hypothesis in the ANS–AB association based on different experiments and types of analysis?

2. Method

The meta-analysis was pre-registered with PROSPER (CRD42018063990). The database, scripts and analyses can be retrieved from the Open Science Framework (OSF): https://osf.io/5z4u7/?view_only=15fed6193b424b2f8ffd19b8db090f15.

2.1. Literature search

The literature search was conducted in Embase, Medline, PsychInfo, ISI Web of Science and Dissertation Abstracts International, and included studies published between 2002 (due to the fact that Lorber included studies up until 2002) up to and including January 1st 2020. All databases have unique search strings, and thus the search terms were formulated with the help of two librarians from Radboud University. The search strategies used for each database are available as supplements from OSF ("S1 Search strategies.docx"). In total, 3356 references were imported from 3331 studies, and 738 duplicates removed. After screening the title and abstract, 2007 additional studies were excluded, leaving 585 studies eligible for full-text screening of which 437 were excluded (e.g., duplicates, pediatric population, conference proceedings, incorrect comparator or study design). After reviewing the full texts, 135 articles comprising 112 studies and 779 ESs were included. Seven studies provided insufficient information to estimate their ESs. Four studies were subsequently merged into one study, due to the fact that a dissertation and journal article were both available, thus resulting in 101 studies and 769 ESs. An overview of the PRISMA-diagram from Covidence is available from OSF ("S2 PRISMA.docx"). Furthermore, all corresponding authors of the included studies were contacted to include grey literature or additional information for bivariate measures, while non-corresponding authors were contacted if the corresponding author did not reply. Another eight articles were suggested by the contacted authors, of which one was subsequently included in the meta-analysis.

2.2. Inclusion and exclusion criteria PICO

The following inclusion criteria were applied to the studies following the PICO criteria (population, intervention, comparison and outcomes; see for example Pollock and Berge, 2018):

(1) must have been published between 2002 and 2020; (2) must report on AB among (young) both adolescents (aged 12–17) and adults (18+); (3) must report on (validated) behavior type comparators; (4) must report on outcomes of SC and HR variability ; (5) must report on the association between comparators and outcomes (AB–ANS association); (6) must include statistical information to calculate ESs (authors were contacted if the bivariate information was insufficient or unavailable); (7) must be written in English; and (8) all journal articles, book chapters, dissertations, conference proceedings, and unpublished research obtained from the contacted authors were potentially eligible for inclusion, if sufficient information could be extracted. There were no restrictions placed on the timing of the follow up and the type of setting (e.g., laboratory, applied, etc.). The following exclusion criteria were applied: (1) studies that include animals, and (2) studies that include participants with a mean sample age below 12 years old.

The included samples consisted of adolescents and adults, who were exposed to aggression-eliciting experimental tasks, subjected to stimuli, or measured when resting. The comparators consisted of several behavior types that measure physical aggression (including violent offenders and laboratory aggression), psychopathy, antisocial personality disorder and conduct disorder. In contrast to Lorber (2004), we only included studies that used a formal diagnosis of antisocial personality disorder or conduct disorder following the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2013). The outcomes were ANS measures of SC and HR variability, and their derived parameters (see Jarczok et al., 2016; Kamath et al., 2016).

2.3. Data coding

Screening, eligibility, full-text screening and data extraction were all carried out in Covidence. At all stages of the meta-analysis, the studies and ESs were selected by three independent reviewers (PdL, LJMC, CHdK). Data extraction was conducted via the use of a customized code sheet that was piloted beforehand on five studies. Besides PICO items, additional data was coded for the variables, before subsequently being added to a database that is available from OSF ("S3 Database.csv"). The description of the variables in the database is also available from OSF ("S4 Method variables.docx").

2.4. Data extraction

Two types of ESs were first coded and then subsequently calculated. These ESs were then transformed to a common index, Hedges' *g* (Borenstein et al., 2009). First, 36 % of the studies reported means, standard deviations and sample sizes for both the control and target groups, which meant that the ES Hedges' *g* could be directly derived. Second, 64 % of the studies reported Pearson correlations between the comparators and outcomes. These correlations were transformed to Hedges' *g* (Cooper et al., 2009, p. 221–235) with R (Harrer et al., 2019; R Core Team, 2014). A positive ES indicated that the experimental group (i.e., the AB–group) scored higher in the outcome of interest (ANS outcomes) than the control group. In the event that the studies reported insufficient information through which to calculate ESs, the ES was then coded as zero, which is in accordance with Lorber's (2004) conservative approach.

2.5. Interrater agreement

Interrater agreement (McHugh, 2012) was .87 for abstract screening, .86 for the review of the full texts, and .81 for extraction of the ESs. After holding several meetings to establish a consensus, in which all raters had to agree, full agreement was ultimately achieved for each stage of the meta-analysis. All the studies were coded by the first author, and double coded by either the second or third author (both authors completed half of the studies).

One specific problem with the current meta-analysis concerns dependency. For the majority of the studies, multiple ESs were able to be calculated. Several reasons for dependency were evident, both within and between studies: (1) the use of multiple scales to measure AB, (2) multiple available analyses for several ANS outcomes, including in longitudinal studies as well (i.e., Popma et al., 2006 and de Wied et al., 2012), (3) multiple independent tasks being administered to a single sample (Pfabigan et al., 2015; Seidel et al., 2013), on different occasions, using separate control groups (Rosenberger et al., 2019; personal communication Claus Lamm, 21-7-2020), (4) experiments having a dependent structure (Benning et al., 2005), but including separate analyses for the various stages in the task, and/or (5) concurrently reporting rest, task and reactivity measures (e.g., Beauchaine, 2002a,b). Although, in the absence of task measures, rest measures can be considered as between-subject comparisons, there is arguably a dependency (e.g., auto–correlation) between measures when both rest and task measures are used to calculate a reactivity measure, and this correlational structure is preferably modeled (Hox et al., 2017). Therefore, a statistical model was required to account for this dependency.

2.6. Statistical model

First, we considered applying a three-level model to take into

account the dependency between ESs belonging to the same study (Cheung, 2014; Van den Noortgate et al., 2013, 2015). This three-level model assumes that ESs (Level 1) are nested within outcome types (Level 2), and outcome types are nested within studies (Level 3). However, we detected other variables that influenced the pooled ES and that could be introduced in the model as random effects: the type of experiment conducted, the type of questionnaire used to measure psychopathy, as well as the samples from different articles, but the same study. ESs were obtained from a range of experiments (e.g., affective responsiveness, baseline, picture viewing, posture challenge task, competitive reaction time tasks, fear conditioning, and so on and so forth), while, simultaneously, ESs were derived from different questionnaires/subscales of psychopathy (e.g., psychopathy checklist – short version (PCL-SV), Youth Psychopathic Traits Inventory – Short Version (YPI-SV), etc.). Across a total of 769 ESs (please note that 10 of the 779 ESs were subsequently excluded due to insufficient information), there were 185 different experiments, 106 different questionnaires and subscales of questionnaires used to measure AB reported across 101 studies. Therefore, ESs (Level 1) were nested within outcomes (Level 2) and outcomes were cross-classified within studies, experiments and questionnaires (Level 3; please note that samples from different articles were not included due to insufficient variance). To analyze this cross-classified data structure, we used a cross-classified random effects model (CCREM; Fernández-Castilla et al., 2019a,b). By properly modeling this cross-classified data structure, inflated Type I error rates were avoided. Some studies either did not include questionnaires or experiments or failed to provide information on the questionnaires or experiments they used, and were labeled as 'missing', which constitutes another category of the random variable.

To verify whether the variance parameters were different from zero, log-likelihood ratio tests were performed to compare the full model with several models that excluded one of the variance parameters (i.e., the between-outcomes variance, or the between-studies variance, or the between-questionnaires variance, or the between-experiments variances) each time. If relevant variability was observed, then the moderator variables were entered separately to see whether they could explain the observed variability across all the mentioned levels. For this purpose, we performed separate CCREMs for each category of the qualitative moderators. The reason for proceeding in this fashion was that, in most cases, there was a large difference in the variability of the pooled ES estimated for each category of the moderator. For instance, the total variance for RMSSD[4] (Root Mean Square of Successive Differences) outcome was 0.43, whereas the total variance of the outcome HF (High Frequency) was 0.02. If a meta-regression was carried out (using any meta-analytic method: Robust Variance Estimation method, multi-level modeling, etc.), then one would assume that the total variance should be the same for all the categories of the moderator, which was not true in this case and, as such, could lead to biased estimates (Fernández-Castilla et al., 2020). In those cases where the number of observations were smaller or equal to 10, a standard random effects meta-analysis that used restricted maximum likelihood (REML) as the estimation method was carried out.

Analyses were conducted in R using the *metafor* package (Viechtbauer, 2010), with REML as the estimation method. To investigate the influence of both outlying ESs and influential studies on the overall ES estimate, we calculated the standardized deleted residuals of all the effects (Viechtbauer and Cheung, 2010). ESs with standardized deleted residuals either above or below ± 1.96 were deemed to be potential outliers, with analyses then being performed again without these observations. To detect for the influence of influential studies, we calculated the cook distance for each study (Viechtbauer and Cheung, 2010). Studies that showed a cook distance larger than 0.45[5] were considered to be potentially influential studies, and the analyses were carried out again without these studies. Finally, the possible presence of publication bias was examined through both visually inspecting funnel plots (Light and Pillemer, 1984) and through a cross-classified version of the Egger regression test (Egger et al., 1997). However, the multilevel version of the Egger regression test has been shown to occasionally lead to inflated Type I error rates (Fernández-Castilla et al., 2019b). It also does not provide an adjusted estimate of the pooled ESs corrected for publication bias. Therefore, in addition to this, sensitivity analyses were also performed, using the selection method of Vevea and Woods (2005). This selection method estimates an adjusted overall ES based on a weight function (specified by the researcher), in which probabilities (of being published) are assigned to ESs depending on the *p*-value associated with them. If the adjusted estimate is radically different from the unadjusted estimate, then publication bias could potentially exist. A two-tailed selection pattern was specified for both moderate and severe publication bias, using the weights recommended by Vevea and Woods (2005). Given that this selection method does not allow for dependent ESs, one ES was randomly selected from each study (therefore, with the R code, the reader could recreate the analysis and obtain slightly different results).

The Winner's curse (see Button et al., 2013) is a well-known problem that results in inflated estimates of ESs if research only focuses on statistical significance without taking into account statistical power and sample size. In addition, several papers have recommended the need to abandon the common and arbitrary cutoffs proposed by Cohen (1998) to interpret Cohen's *d* effect size, and assess ESs in relation to other Cohen's *d* values observed in specific research fields (e.g., Kraft, 2020; Lakens, 2013; Vacha-Haase and Thompson, 2004).

Portnoy and Farrington (2015) reported that the ES for resting HR varied between −0.15 and −0.20, while Ortiz and Raine (2004) purported that a low resting HR was the best biological correlate. Therefore, we used a magnitude of 0.15 as a cut-off criterion to investigate the relevance of an ES. However, besides magnitude, we also need to take the precision of the overall effect into account, which depends directly on the number of studies synthesized. An overall effect is assumed to be precise and reliable if it is based on a substantial number of studies although clear criteria are currently lacking on the number of studies needed. Cooper et al. (2019) discuss the minimum number of studies required for a category of a nominal moderator variable: "*Unfortunately, it is not possible to quantify the terms small and relatively large. The definition of these terms depends on analytic choices, such as model choice, the within-study sample sizes, though the number of studies is more important, and on the spread of the covariates" (pp. 137).* In addition, Deeks et al. (2019) recommend to include at least 10 studies for each covariate in a meta-regression, and Schmid et al. (2004) indicate that at least 10 studies are required to detect effects in a meta-regression.

Therefore, in line with the recommendations, we decided to use previous ESs as a reference to determine whether observed overall effects are small, medium or large (the magnitude of an effect), but also take the sample size and significance into account. This resulted in the following four criteria, which were used to determine what the most valid and reliable results were:

Criterion 1) magnitude exceeding, $k > 10$, and significant.

[4] RMSSD is a measure of HRV. Other measures that were used in the present meta-analysis were HR = Heart rate; IBI = Inter Beat Interval; HF = High Frequency; LF = Low Frequency; PEP = Pre-Ejection period; RSA = Respiratory Sinus Arrhythmia. For an overview of the standards of measurement, interpretation, and clinical use, the reader is referred to the guidelines set out by both the Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology.

[5] To interpret the cook distance, we calculated the value of the chi-square statistic corresponding to the percentile 50 with 1 degree of freedom (degrees of freedom = number of predictors + 1, and this analysis was carried out on the null model without predictors), and used this value (0.45) as the cut-off to decide whether a study was influential or otherwise.

Criterion 2) magnitude exceeding, $k > 10$, and *non*-significant.
Criterion 3.1) magnitude exceeding, $k <= 10$, and significant.
Criterion 3.2) magnitude exceeding, $k <= 10$, and *non*-significant.

Please note that the overall ES within each category was estimated with a CCREM if the number of ESs were larger than 10 ($k > 10$), whereas univariate analyses were conducted if the number of ESs were smaller or equal to 10 ($k <= 10$; i.e., "uni" in Tables 3 and 4).

Criterion 1 and 2 are considered to be the *most reliable* (i.e., the extent to which a result can be reproduced) results, and, hence, are reported in the text. Criterion 3.1 and 3.2 are considered to be *promising* results at best and need to be interpreted cautiously. Criterion 3.1 and 3.2 results can be found in the relevant tables.

3. Results

An overview of the included studies and the variables that were collected is provided in Table 1 (the analyses that produced Table 1 can be found in "S5 Table 1.R"). A textual analysis of Table 1 is provided in "S6 Table 1 analysis.docx ". The results from the meta-regression "CCREM" are shown in Table 2 (analysis available in "S7 Table 2.R"), which indicated that the overall ES is -0.075 [SE = 0.037, 95 % CI = (-0.147, -0.003)]. While this effect is statistically significantly different from zero, it does not take into account the various functions of the ANS outcomes (e.g., HR and SC). The main source of variability occurred between studies (0.077), followed by within studies (or between-outcomes) (0.029), between questionnaires (0.009), and between experiments (0.006). Given that all variances were significantly different from zero, moderator variables were thus introduced one by one in the CCREM.

3.1. Proteus phenomenon

First, the results indicate that the ES is −0.17 for the rest HR–AB association, which is similar to Portnoy and Farrington's (2015) estimate. In order to test whether the Proteus phenomenon was evident in the data, we ran a regression analysis with resting HR as an outcome and the year of publication as a moderator (available in "S8 Table 3. R"). The results indicate that the inclusion of the year of publication as a moderator was non-significant (b = 0.02, $p = .104$), which is to say that the ES for resting HR was relatively stable across time, while for HR–rest, the Proteus phenomenon was not replicated (Portnoy and Farrington, 2015).

We further tested the effect of year of publication on both the types of outcomes and types of analyses (rest, task, and reactivity) for which more than 10 ESs were available: HR–reactivity, b = −0.01, $p = .438$, $k = 79$; HR–task, b = 0.04, $p = .047$, $k = 125$; RMSSD–task, b = −0.06, $p = .01$, $k = 18$; RSA (respiratory sinus arrhythmia)–reactivity, b = −0.04, $p = .58$, $k = 11$; RSA–rest, b = 0.01, $p = .706$, $k = 21$; SCL (skin conductance level)–reactivity, b = 0.01, $p = .888$, $k = 46$; SCL–rest, b = 0.02, $p = .185$, $k = 30$; SCL–task, b = 0.04, $p = .15$, $k = 16$; SCR (skin conductance reactivity)–reactivity, b = 0.03, $p = .034$, $k = 188$; SCR–task, b = 0.04, $p = .637$, $k = .59$.

In short, the Proteus phenomenon was evident in HR–task and SCR–reactivity, insofar as these regressed to zero across time. Although the phenomenon was also evident in RMSSD–task, this was only measured for two years. The other variables were non-significant, thus indicating that these were relatively stable across time.

3.2. Psychopathy subscales

The second research question concerned whether the relationship between ANS and psychopathy was dependent on subscales of psychopathy questionnaires. A CCREM interaction model ("S9 Psychopathy subscale.R") with a psychopathy (sub)-scale as a moderator indicated that the estimate for Factor 1 of the psychopathy subscale (ES = −0.10, $p = .076$, $k = 115$), Factor 2 (ES = −0.03, $p = .673$, $k = 113$) and the Total psychopathy scale (ES = −0.05, $p = .184$, $k = 168$) did not exceed the preset magnitude criterion threshold. Further exploratory analyses demonstrated that not all of the outcome categories included sufficiently large samples. Criterion 2 estimates were obtained for both HR–Factor 1 (ES = −0.21, $p = .059$, $k = 39$) and HR–Factor 2 (ES = −0.16, $p = .017$, $k = 38$). With respect to SCR, both Factor 1 (ES = -0.09, $p = .436$, k = 52) and Factor 2 (ES = −0.09, $p = .408$, $k = 51$) were negative, but did not exceed the preset magnitude criterion threshold. To be able to conclude that specific co-inhibitory, co-activation, or antagonistic effects were evident, we would need to have observed the presence of at least some bidirectional effects within the different subscales. However, the results suggest that for both HR and SCR, specificity of physiological reactions were not directly evident when taking into account different subscales of psychopathy questionnaires. To determine if this was also the case for the other ANS parameters (outcome types), further studies are required.

3.3. Moderators

The third research question investigated which covariates moderated the association between ANS and AB. The results are presented in Table 3 (analyses available in "S8 Table 3.R", and an Excel file ("Table 3. xlsx") is available as well to quickly filter the results that the reader is interested in). The pooled ES estimates for *method of assessment* showed that Criterion 1 estimates were obtained for HR that is measured without traditional electrocardiography (ECG; ES = -0.29, $p = .021$, $k = 58$).

Analysis type resulted in a significant effect for rest measures, but the magnitude was not large enough to be interpreted (ES = −0.11, $p = .001$, $k = 180$).

The *outcome type* variables were mainly non-significant. Criterion 2 ESs were PEP (Pre–Ejection Period; ES = 0.15, $p = .206$, $k = 20$), RMSSD (ES = 0.37, $p = .370$, $k = 26$), and RSA (ES = −0.16, $p = .226$, $k = 42$).

In accordance with our hypothesis, the *behavior type* variable resulted in a Criterion 1 estimate for the violent offender group (ES = −0.33, $p = .037$, $k = 95$). Although a Criterion 2 estimate was found for laboratory aggression (ES = 0.20, $p = .227$, $k = 26$), this was in the opposite direction, which means that the control group displayed, on average, a higher response than the laboratory aggression group.

The *sample description variable* resulted in a Criterion 1 estimate for the mixed sample (ES = −0.18, $p = .020$, $k = 190$). The *recruitment source variable* resulted in a Criterion 1 estimate for the mixed recruitments (ES = −0.22, $p = .040$, $k = 151$).

The *experiment variable* showed various significant ESs, which exceeded the magnitude threshold. First, Criterion 1 experiments comprised the anticipation of noise task (ES = -0.15, $p = .049$, $k = 54$) and cognitive tasks (ES = 0.19, $p = <.001$, $k = 15$). Second, Criterion 2 estimates were obtained via both competitive reaction time tasks (ES = 0.36, $p = .117$, $k = 31$) and fear conditioning tasks (ES = −0.21, $p = .242$, $k = 53$).

It is important to note here that some experiments are designed to elicit a greater ES during a particular stage of the experiment. For instance, in a competitive reaction time task, a baseline or pre-aggression period is typically included along with shocks or pneumatic pressure. The theoretically-informed prediction here is that the ES will be largest during the negatively valenced shocks and pneumatic pressure conditions. A similar rationale applies to emotional picture experiments, whereby it is predicted that the ES will be largest when negatively valenced pictures are shown, as opposed to neutral or positive pictures. To test for this effect, we used a selection of experiments for which we had sufficient ESs and multiple valence categories. Indeed, the ESs varied for theorized vs non-theorized (available in "S10 Theory dummy.R") in relation to competitive reaction time (0.54 vs 0.04), fear conditioning (−0.31 vs −0.25), picture viewing (−0.23 vs −0.09), public speaking (−0.09 vs 0.20), anticipation of noise (−0.18 vs. 0.10), and emotional video tasks (−0.22 vs 0.01). All ESs were in the expected direction, with the exception of the public speaking task. Further analysis of public speaking revealed that the direction of HR (ES = 0.20, $p =$

Table 1
Study characteristics of included studies.

study	name	group	sample	recruitment	comparison	design	country	age	sd	female	assessment	N	outcome	analysis	experiment	behavior	questionnaire
#5173 - Armenti 2018	(Armenti & Babcock, 2018)	IPV men	AB	community	none (one group)	c	usa	32.02	10.21	0	sc electrode; hr ecg	135	SCR; HR	reactivity	conflict	psychopathy	PPI-SF Factor 1 Fearless Dominance
#64 - Armstrong 2009	(Armstrong et al., 2009)	students	students	community	none (one group)	c	usa	21.43	5.7	45	hr other	105	HR	rest	other	physical aggression	self-report questionnaire
#5003 - Armstrong 2017	(Armstrong et al., 2017)	violent offenders	AB	criminal justice	high vs low heart rate	c	usa	31.63	11.2	0	hr other	90	HR	rest	other	physical aggression - violent offenders	arrests for violent crimes
#72 - Arriaga 2008	(Arriaga et al., 2008)	students	students	community	none (one group)	c	portugal	23	3.21	55.4	hr ecg	138	HR	reactivity	competitive reaction time	physical aggression - lab aggression	
#84 - Assaad 2006	(Assaad et al., 2003, 2006; Assaad, 2002)	adults	non_AB	community	high vs low HR responders	l	canada	20.84	0.7	0	hr ecg	74	HR	reactivity	competitive reaction time	physical aggression - lab aggression	
#99 - Babcock 2004	(Babcock et al., 2004; Babcock et al., 2005)	IPV non-violent; IPV low-level violent; IPV clinical-level violent	non_AB; AB	community	none (one group)	c	usa	32	9.6	0	hr ecg	30; 22; 50	HR	rest; reactivity	other; conflict	psychopathy; physical aggression	SRP-2 full scale; Generality of Violence questionnaire
#5111 - Babcock 2019	(Babcock & Michonski, 2019)	IPV men	AB	community	none (one group)	c	usa	29.9	8	0	sc electrode	79	SCR	reactivity	emotion picture	physical aggression; psychopathy	CTS-2; PPI-SF Factor 1 Fearless Dominance; PPI-SF Factor 2 Impulsive Antisociality
#8168 - Babcock 2020 and #1801 - Potthoff 2016	(Babcock & Potthoff, 2020; Potthoff, 2016)	IPV-men and non-violent control	mix	community	high vs low	c	usa	31.7	9.4	0	hr ecg; sc electrode	114	HR; RSA; SCL	rest; task	other; anger induction	physical aggression	CTS-2
#107 - Babel 2016	(Babel et al., 2016)	adolescents	AB	mental health	high vs low DBD	c	netherlands	15.4	1.1	100	hr ecg	44	HR; PEP; RSA	rest	other	conduct disorder	DISC-4
#145 - Bare 2004	(Bare et al., 2004)	students	students	community	none (one group)	c	usa	18.9	3	78	hr ecg; sc electrode	76	HR; SCR	reactivity	guided imagery	psychopathy	SRP-2 Factor 1 emotional detachment; SRP-2 Factor 2 deviant behavior
#191 - Beauchaine 2002	(Beauchaine, 2002b, 2002a)	adolescents	non_AB	community	CD present vs absent	c	usa	13.6	1.5	0	hr ecg	42	HR; PEP; RSA	rest; task	other	conduct disorder	ASI, DSM4
#212 - Benning 2005	(Benning et al., 2005)	twins	non_AB	community	high vs low	l	usa	20	NA	0	sc electrode	70	SCR	reactivity	emotion picture	psychopathy	PPI Factor 1 Fearless Dominance; PPI Factor 2 Impulsive antisociality
#224 - Betensky 2010	(Betensky & Contrada, 2010)	students	students	community	none (one group)	c	usa	19.38	4.2	100	hr other	63	HR	rest; reactivity	other; public speaking	physical aggression	BPAQ - physical aggression
#237 - Birbaumer 2005	(Birbaumer et al., 2005)	psychopaths and controls	mix	criminal justice and community	criminal psychopaths vs matched controls	c	germany	33.08	7.14	0	sc electrode	12	SCR	reactivity	fear conditioning	psychopathy	PCL-R total
#252 - Bobadilla 2007	(Bobadilla & Taylor, 2007)	students	students	community	none (one group)	c	usa	20.8	4.15	59	sc electrode	107	SCR	reactivity	anticipation-of-noise	aspd	SIDP-4
#249 - Bobadilla 2008 (dissertation)	(Bobadilla, 2008)	students	students	community	high vs low	c	usa	19.26	1.1	52	sc electrode	103	SCR; SCL	reactivity; task	anticipation-of-noise	psychopathy	PPI total
#5037 - Bolinger 2018 (dissertation)	(Bolinger, 2018)	students; high psychopathy group students	students	community	high vs low; none (one group)	c	usa	19.15	1.4	0	sc electrode	84; 52	SCR	reactivity; rest	emotion picture; other	psychopathy	PPI-SF total; LSRP Factor 1 primary callous manipulative; LSRP Factor 2 secondary impulse control; PPI-SF Factor 1 Fearless Dominance; PPI-SF Factor 2 Impulsive Antisociality
#291 - Broom 2012 (dissertation)	(Broom, 2012)	students; violent offenders; incarcerated offenders	students; AB	community; criminal justice	none (one group)	c	canada	18.87; 33.22	1.78; 10.17	71; 0	sc electrode; hr ecg arm	65; 67	SCR; HR	task	iowa gambling task	psychopathy	PCL-SV total; PCL-R Total; PCL-SV Facet 1 Interpersonal; PCL-SV Facet 2 Affective; PCL-SV Facet 3 Lifestyle; PCL-SV Facet 4 Antisocial; PCL-R Facet 1 Interpersonal; PCL-R Facet 2 Affective; PCL-R Facet 3 Lifestyle; PCL-R Facet 4 Antisocial

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Table 1 (*continued*)

#297 - Brown 2015 (dissertation)	(Brown, 2015)	adults	non_AB	community	none (one group)	c	usa	NA	NA	50	hr ecg	4	RSA; PEP	task	other	physical aggression	BPAQ - physical aggression
#352 - Casey 2013	(Casey et al., 2013)	violent offenders	AB	criminal justice	high vs low; none (one group)	c	uk	40.5	11.11	0	hr other	62; 61	HR	task	emotion picture	psychopathy	PCL-R total; PCL-R Factor 1 Interpersonal Affective; PCL-R Factor 2 Lifestyle Antisocial
#358 - Cauffman 2005	(Cauffman et al., 2005)	violent offenders and student controls	mix	criminal justice and community	violent offenders vs student controls	c	usa	16.19	1.31	53	hr other	183	HR	rest	other	physical aggression - violent offenders	-
#474 - daSilva 2014	(da Silva et al., 2014)	students	students	community	high vs low median split	c	brazil	13.84	1.46	45	sc electrode	40	SCR	task	other	physical aggression	Olweus Bully Victim Questionnaire physical scale
#485 - deBarros 2013	(de Barros et al., 2013)	incarcerated offenders	AB	criminal justice	none (one group)	c	brazil	18	NA	0	sc electrode	30	SCR	reactivity	emotion picture	psychopathy	PCL-R Factor 1 Interpersonal Affective; PCL-R Factor 2 Lifestyle Antisocial
#499 - deVries-Bouw 2012	(De Vries-Bouw et al., 2011; de Vries-Bouw et al., 2012; Popma et al., 2006)	offenders and matched controls	mix	criminal justice and community	DBD+ vs matched controls	l	netherlands	18.26	0.93	0	hr ecg	31	HR; HF	rest; task	public speaking	conduct disorder	DISC
#505 - deWied 2012	(de Wied et al., 2012)	DBD+ and matched controls	mix	community	DBD+ vs matched controls	c	netherlands	13.67	0.75	0	hr ecg	63	HR; RSA	rest; reactivity	other; emotion video	conduct disorder	DISC-4 and APSD
#554 - Dindo 2011	(Dindo & Fowles, 2011; Dindo & Fowles, 2008; Dindo, 2008)	students	students	community	none (one group)	c	usa	19	NA	0	sc electrode	115	SCL	reactivity	public speaking; anticipation-of-noise	psychopathy	PPI Factor 1 Fearless Dominance; PPI Factor 2 Impulsive Antisociality; PPI total
#659 - Fanti 2017	(Fanti et al., 2017)	students and community	students	community	none (one group)	c	cyprus	19.92	0.99	50	hr ecg arm; sc electrode	99	HR; SCL	rest; reactivity	other; emotion video	psychopathy	YPI-SV callous unemotional; YPI-SV impulsie irresponsible; YPI-SV grandiose manipulative; YPI-SV total
#5025 - Florez 2017	(Florez et al., 2017)	incarcerated offenders	AB	criminal justice	high vs low	c	spain	40.9	11.2	14	hr ecg	185	HRV; LF; HF	task	other; iowa gambling task	psychopathy	PCL-R total
#735 - Fung 2005	(Fung et al., 2005; Loeber et al., 2007)	PYS	non_AB	community	high vs low	c	usa	15.98	0.81	0	sc electrode	130	SCL; SCR	rest; task	other; anticipation-of-noise	psychopathy	CPS
#5028 - Galan 2017	(Galan et al., 2017)	adolescents	non_AB	community	none (one group)	l	usa	12	NA	0	hr ecg	160	HR	rest	other	physical aggression - violent offenders; physical aggression	arrests for violent crimes; SRD - self report; SRD - peer report
#765 - Gao 2012	(Gao et al., 2012)	adults	non_AB	community	high vs low	c	usa	35.52	10.2	0	hr ecg; sc electrode	89	HR; SCL	rest; task	public speaking	psychopathy	PCL-R total and 10 collateral sources
#802 - Gerra 2003	(Gerra et al., 2003)	aspd and no-aspd	mix	mental health	aspd vs no-aspd	c	italy	26.7	7.92	0		32	HR	rest	other	aspd	SIDP-4
#5099 - Goulter 2019	(Goulter et al., 2019)	students	students	community	none (one group)	c	australia	19.02	1.5	100	hr ecg	101	RMSSD	rest; task	other; public speaking; competitive reaction time	psychopathy ; physical aggression - lab aggression	PPI-SV Factor 1 affective interpersonal; PPI-SV Factor 2 impulsivity; PPI-SV total;
#923 - Hansen 2007	(Hansen et al., 2007)	incarcerated offenders	AB	criminal justice	none (one group)	c	norway	32.07	7	0	hr ecg	53	RMSSD; HR	rest; task	other; continuous performance ; working memory	psychopathy	PCL-R Facet 1 Interpersonal; PCL-R Facet 2 Affective; PCL-R Facet 3 Lifestyle; PCL-R Facet 4 Antisocial
#932 - Hasan 2013	(Hasan et al., 2013)	students	students	community	none (one group)	c	france	20.1	3.1	83	hr other	77	RSA	task	competitive reaction time	physical aggression - lab aggression	-
#5156 - Hong 2018	(Hong et al., 2018)	students	students	community	high vs low	c	korea	NA	NA	45	hr other	40	HR	task	concealed information	psychopathy	LSRP total
#1024 - Humphreys 2012	(Humphreys et al., 2012)	adults	non_AB	other	aspd vs no-aspd	c	usa	43.28	3.86	0	hr ecg arm; sc electrode	287	HR; SCL	reactivity	other	aspd	SCID
#1028 - Ibanez 2016	(Ibanez et al., 2016)	students	students	community	none (one group)	c	spain	NA	NA	65	hr ecg arm; sc electrode	110	IBI; SCR	reactivity	trust game	psychopathy	LSRP total
#5193 - Im 2019	(Im et al., 2018, 2019)	students	students	community	none (one group)	c	korea	18.3	1.2	57	hr ecg	70	HR	task	other; emotion video	physical aggression	BPAQ - physical aggression
#5108 - Iria 2019	(Iria et al., 2020)	offenders and controls	mix	community	aspd vs no-aspd	c	usa	38.36	10.51	0	sc electrode	74	SCR	reactivity	emotion picture	aspd	SCID-2
#1035 - Isen 2012	(Isen et al., 2012)	MTFS	non_AB	community	none (one group)	c	usa	17.83	0.25	51	sc electrode	2129; 2065	SCR	reactivity	other	physical aggression	MPQ (aggression scale and control scales)

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Table 1 (*continued*)

#1089 - Jennings 2013	(W. G. Jennings et al., 2013)	CSDD	AB	community	violent vs non-violent offenders	l	uk	18	NA	0	hr other	386	HR	rest	other	physical aggression - violent offenders	total conviction frequency (Ages 10-50)
#1090 - Jennings 2017	(Jennings et al., 2017, 2019)	PYS	non_AB	community	none (one group)	c	usa	32	0.9	0	hr ecg	296	HR	rest; reactivity	other; mental arithmetic	physical aggression	BPAQ - physical aggression
#1140 - Kavish 2017	(Kavish et al., 2017)	students	students	community	none (one group)	c	usa	12.15	0.87	0; 100	hr other	69; 58	HR	rest	other	psychopathy	YPI total; YPI grandiose manipulative; YPI callous unemotional; YPI impulsive irresponsible
#8158 - Kavish 2019	(Kavish, Bergstrøm, et al., 2019)	CSDD	mix	community	none (one group)	l	uk	18	NA	0	hr other	292	HR	rest	other	psychopathy	PCL-SV total; PCL-SV Factor 1 Affective; PCL-SV Factor 2 Antisocial
#5089 - Kavish 2019	(Kavish, Boisvert, et al., 2019)	students	students	community	none (one group)	c	usa	20.21	2.85	100; 0	hr other; sc electrode	308; 145	HR; SCL; SCR	rest; reactivity	other; public speaking	psychopathy	LSRP total; LSRP Factor 1 primary callous manipulative; LSRP Factor 2 secondary impulse control
#1173 - Kirsch 2009 (dissertation)	(Kirsch, 2009)	students	students	community	high vs low	c	usa	18.7	0.5	0	hr ecg arm	31	HR	task	emotion video	psychopathy	PPI-R total; PPI-R Factor 1 Fearless Dominance; PPI-R Factor 2 Self-Centered Impulsivity; PPI-R Factor 3 Coldheartedness
#5078 - Koegl 2018	(Koegl et al., 2018)	incarcerated offenders	AB	criminal justice	none (one group)	c	canada	36.2	12.2	0		333	HR	rest	other	physical aggression - violent offenders	
#1244 - Kulper 2016 (dissertation)	(Kulper, 2016)	students	students	community	none (one group)	c	usa	22.1	4.51	65.8		79	HR; SCR; HF	reactivity	competitive reaction time; other	physical aggression - lab aggression; physical aggression	; BPAQ - physical aggression
#1256 - Kyranides 2017	(Kyranides et al., 2017)	students	students	community	none (one group)	l	cyprus	19.92	0.99	50	hr ecg arm; sc electrode	88	HR; SCL	rest; reactivity	other; emotion video	psychopathy	tripm boldness; tripm disinhibition; tripm meanness
#1291 - Latvala 2015	(Latvala et al., 2015; Latvala et al., 2016)	violent offenders and non-criminals	mix	community	violent offenders vs non-criminals	l	sweden	18.2	0.5	0	other	710264	HR	rest	other	physical aggression - violent offenders	
#5046 - Ling 2018	(Ling et al., 2018)	adults	non_AB	community	none (one group)	c	usa	35.72	8.61	0	hr ecg; sc electrode	156	HR; SCL	task	public speaking	psychopathy	PCL-R total; PCL-R Factor 1 Interpersonal Affective; PCL-R Factor 2 Lifestyle Antisocial
#5038 - Lishak 2018 (dissertation)	(Lishak, 2018)	Domestic violence	mix	criminal justice and community	DV vs no-DV	c	canada	37.23	9.21	0	hr ecg; sc electrode	208	HR; SCR; HF	rest; reactivity	other; emotion audio	physical aggression - violent offenders	
# 1349 Lobbestael 2009 AND #1346 - Lobbestael 2010	(Lobbestael et al., 2009; Lobbestael & Arntz, 2010)	patients and controls	mix	mental health and criminal justice, community	aspd vs no-aspd	c	netherlands	34.43	10.98	43	hr ecg; sc electrode	56	HR; SCL; SCR	rest; reactivity	other; emotion video	aspd	SCID
#1377 - Lotze 2007	(Lotze et al., 2007)	adults	non_AB	community	none (one group)	c	germany	28.6	6.5	0	sc electrode	14	SCR	reactivity	competitive reaction time	physical aggression - lab aggression	
#5101 MacDougall 2019 and dissertation #1405	(MacDougall, 2016; MacDougall et al., 2019)	incarcerated offenders	AB	criminal justice	none (one group)	c	usa	15.92	1.31	0	hr ecg; sc electrode	56; 32	HR; SCR; SCL; RSA	rest; task; reactivity	other; anticipation-of-noise	psychopathy	PCL-YV total; PCL-YV Grandiose Manipulative; PCL-YV Callous Unemotional; PCL-YV Daring Impulsive; PCL-YV Antisocial; PCL-YV Total
#1515 - Molapour 2016	(Molapour et al., 2016)	healthy volunteers	non_AB	community	none (one group)	c	sweden	22.57	3.33	61	sc electrode	23	SCR	reactivity	fear conditioning	physical aggression - lab aggression	
#1548 - Munoz 2008	(Munoz, 2005; Munoz et al., 2008b, 2008a)	incarcerated offenders	AB	criminal justice	none (one group)	c	usa	15.53	1.28	0	sc electrode	85	SCL; SCR	reactivity	other; competitive reaction time	physical aggression	PCS-C - proactive overt; PCS-C - reactive overt
#1557 - Murray 2016	(Murray, Hallal, Mielke, Raine, Wehrmeister, Anselmi, et al., 2016; Murray, Hallal, Mielke, Raine, Wehrmeister, & Barros, 2016)Murray 2016	PBH	mix	community hospital (at birth)	violent offenders vs control	l	brazil	15; 18	NA	0; 100	hr other	1607; 1753; 1742; 1770	HR	rest	other	physical aggression - violent offenders	

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Table 1 (*continued*)

#1647 - Northover 2015	(Northover, 2015; Northover et al., 2015)	adolescents	AB	mental health	cd/adhd vs adhd	c	uk	13.98	1.79	0	sc electrode	183	SCL	reactivity	other	conduct disorder	DAWBA
#8128 - Oldenhof 2019	(Oldenhof et al., 2019)	adolescents	mix	mental health and community	CD present vs absent	c	europe (Germany, Greece, Hungary, Netherlands, Spain, Switzerland, United Kingdom)	14.33; 14.01	2.33; 2.60	100; 0	hr ecg	659; 351	HR; RSA; PEP	rest	other	conduct disorder	K-SADS-PL and DSM-5/4-TR
#1680 - Osumi 2007	(Osumi et al., 2007)	students	students	community	detached/antisocial vs controls	c	japan	NA	1	42.5	hr ecg arm	17	HR	reactivity	emotion video; emotion picture	psychopathy	PSPS
#1679 - Osumi 2010	(Osumi & Ohira, 2010)	students	students	community	high vs low	c	japan	19.1	1.01	46	sc electrode	28	SCR	reactivity	ultimatum game	psychopathy	PSPS
#8105 - Osumi 2019	(Osumi, 2019)	students	students	community	none (one group)	c	japan	19.23	1.22	69	sc electrode	35	SCR	reactivity	ultimatum game; other	psychopathy	LSRP Factor 1 primary callous manipulative; LSRP Factor 2 secondary impulse control
#1706 - Pastor 2003	(Pastor et al., 2003)	incarcerated offenders	AB	criminal justice	high vs low	c	spain	30.67	NA	0	sc electrode; hr ecg	28	SCR; HR	reactivity	emotion picture	psychopathy	PCL-R total
#1747 - Pfabigan 2015	(Pfabigan et al., 2015)	incarcerated offenders and matched community controls	mix	criminal justice and community	high psychopathy vs controls; incarcerated offenders vs controls	c	austria	37; 35.6	11.31; 11.58	0	sc electrode	29; 45	SCR	reactivity	empathy for pain	psychopathy; physical aggression - violent offenders	PCL-R and PPI-R; convictions
#1781 - Popma 2006	(Popma et al., 2006)	offenders and matched controls	mix	criminal justice and community	DBD+ vs matched controls	l	netherlands	13.44	0.71	0	hr ecg; sc electrode	52	HR; SCL	rest; task	public speaking	conduct disorder	DISC
#1796 - Portnoy 2014	(Portnoy, 2015; Portnoy et al., 2014)	PYS	mix	community	none (one group)	c	usa	16.15	0.89	0	hr ecg	335	HR	rest; task	other; continuous performance; public speaking	psychopathy; physical aggression - violent offenders	CPS; SRD
#5493 - Puhalla 2019	(Puhalla et al., 2020)	students	students	community	none (one group)	c	usa	22.02	4.46	68	hr ecg	81	HR; HF; LF	rest	competitive reaction time	physical aggression - lab aggression	NA
#1824 - Ragsdale 2013	(Ragsdale et al., 2013)	students	students	community	none (one group)	c	usa	20.41	4.83	50	sc electrode	54	SCL	reactivity	emotion picture	psychopathy; physical aggression	PPI-R total; PPI-R Factor 2 Self-Centered Impulsivity; PPI-R Factor 1 Fearless Dominance; PAI physical aggression
#1828 - Raine 2003	(Raine et al., 2003)	adults	non_AB	community	high vs low	c	usa	29.85	6.6	0		40	SCL; HR	task	public speaking	aspd and Psychopathy	SCID-2 and PCL-R
#1845 - Raine 2014	(Raine et al., 2014)	adolescents	non_AB	community	none (one group)	c	hong kong	13.22	1.19	42	hr other	334	HR	rest	other	psychopathy	APSD total; APSD Impulsivity; APSD Callous-unemotional; APSD Narcissism
#8052 - Rinnewitz 2019	(Rinnewitz et al., 2019)	adolescents	non_AB	community	aggression vs no-aggression	c	germany	15.15	1.08	100	hr other	38	HR	task	competitive reaction time	physical aggression - lab aggression	
#1913 - Romero-Martinez 2013	(Romero-Martínez et al., 2013)	IPV men	mix	criminal justice and community	IPV vs controls	c	spain	36.95	8.59	0	sc electrode	39	SCR	rest; reactivity	other; public speaking	physical aggression - violent offenders	first conviction
#1915 - Romero-Martinez 2014	(Romero-Martinez et al., 2014)	IPV men	mix	criminal justice and community	IPV vs controls	c	spain	35.85	2.32	0	hr ecg	34	HR	rest; task	other; public speaking	physical aggression - violent offenders	first conviction
#8091 - Rosenberger 2019	(Rosenberger et al., 2019)	incarcerated offenders and community controls	mix	criminal justice and community	violent offenders vs community controls	c	austria	35.05	10.06	0	sc electrode	48	SCR	reactivity	trust game	physical aggression - violent offenders	
#1922 - Rothemund 2012	(Rothemund et al., 2012)	offenders	mix	criminal justice and community	offenders vs controls	c	germany	29.5	6.58	0	sc electrode; hr ecg	22	SCR; HR	reactivity	fear conditioning	psychopathy	PCL-R total
#2003 - Schug 2007	(Schug et al., 2007)	adults	non_AB	community	sspd/aspd vs no-aspd controls; aspd vs no-aspd controls	c	usa	30.76	6.63	15	sc electrode	56; 62	SCR	reactivity	other	aspd	SCID-2
#2016 - Scott 2013 (dissertation)	(Scott, 2013)	adolescents	non_AB	community	none (one group)	c	usa	13.88	1.95	51	hr ecg	68	HF	rest; task; reactivity	other; mental arithmetic	physical aggression	PCS-CP - reactive overt; PCS-CP - proactive overt
#2020 - Seidel 2013	(Seidel et al., 2013)	incarcerated offenders and matched community controls	mix	criminal justice and community	violent offenders vs matched controls	c	austria	35.2	11.32	0	sc electrode	60	SCR	reactivity	emotion picture; other	physical aggression - violent offenders	
#2027 - Serafim 2009	(Serafim et al., 2009)	incarcerated offenders and community controls	mix	criminal justice and community; criminal justice	violent offenders vs non-psychopathic non-criminals; none (one group)	c	brazil	31.8	NA	0	hr other	110; 38	HR	rest; task	other; emotion picture	physical aggression - violent offenders	PCL-R total; PCL-R Factor 1 Interpersonal Affective

(*continued on next page*)

Table 1 (*continued*)

#2082 - Sijtsema 2011	(Sijtsema et al., 2011)	adolescents	non_AB	community	none (one group)	c	netherlands	12.47	1.95	100	hr ecg; sc electrode	115	HR; RSA; SCL	reactivity	other	physical aggression	CSBS-T (physical aggression scale)
#2080 - Sijtsema 2015	(Sijtsema, et al., 2015)	adolescents	non_AB	community	none (one group)	c	netherlands	16.14	0.37	50.8	hr ecg	624	PEP; RSA	rest; task; reactivity	other; public speaking	physical aggression	ASBQ
#2131 - Sorman 2016	(Sorman et al., 2016)	diverse sample	students	community	none (one group)	c	sweden	31.7	17.6	0	hr ecg; sc electrode	26; 61	HR; SCR	reactivity	empathy for pain	psychopathy	PPI-R Factor 1 Fearless Dominance; PPI-R Factor 2 Self-Centered Impulsivity; PPI-R Factor 3 Coldheartednes s
#2145 - Stanger 2012	(Stanger et al., 2012)	students	students	community	none (one group)	c	uk	19.95	1.61	39	hr ecg	66	HR	reactivity	emotion picture	psychopathy	SRP-3
#2203 - Sylvers 2010	(Sylvers et al., 2008, 2010)	students	students	community	none (one group)	c	usa	NA	NA	50	sc electrode; hr ecg	100	SCL; PEP; RSA	reactivity	anticipation-of-noise; emotion picture	psychopathy ; aspd	PPI-SF total; SCID-2
#5081 - Thomson 2019	(Thomson, Aboutanos, et al., 2019)	students	students	community	none (one group)	c	uk	19.65	1.22	69	hr ecg; sc electrode	103	RSA; SCL	reactivity	VR horror movie	psychopathy	SRP-4 Factor 1 interpersonal affective; SRP-4 Factor 2 impulsive-antisocial
#8060 - Thomson 2019	(Thomson & Beauchaine, 2019)	students	students	community	none (one group)	c	uk	19.85	1.17	83	hr ecg	104	RSA	rest	other	physical aggression	SRD - interpersonal violence subscale
#8043 - Thomson 2019	(Thomson, Kiehl, et al., 2019)	students	students	community	none (one group)	c	uk	19.57	1.1	100	hr ecg	83	RSA	rest	other	psychopathy ; physical aggression	SRP-4 total; SRP-4 Interpersonal; SRP-4 Affective; SRP-4 Lifestyle; SRP-4 Antisocial; SRD -interpersonal violence subscale
#8152 Trahan 2019 and #2252 - Trahan 2014	(Trahan, 2014; Trahan & Babcock, 2019)	IPV men	AB	community	aspd vs no-aspd	c	usa	31	10.25	0	hr ecg; sc electrode	116	HR; RSA; SCL	reactivity	conflict	aspd	SCID-2 and CTS-2
#2286 - Umhau 2002	(Umhau et al., 2002)	Domestic violence	mix	community	DV vs no-DV	c	usa	37	8.33	16		31	RSA	rest	other	physical aggression	interview recent physical aggression
#2304 - vanGoozen 2016	(van Goozen et al., 2016)	adolescents	mix	mental health and community	none (one group)	c	uk	13.95	1.82	0	sc electrode	113	SCR	task	fear conditioning	conduct disorder	DAWBA
#2335 - Veit 2013	(Veit et al., 2013)	psychiatric patients	AB	criminal justice	none (one group)	c	germany	43.14	11.52	0	sc electrode	11	SCR	task	fear conditioning	physical aggression - violent offenders	PCL-R total; PCL-R Factor 1 Interpersonal Affective; PCL-R Factor 2 Lifestyle Antisocial
#2343 - Verona 2008	(Verona & Sullivan, 2008)	students	students	community	aggression vs non-aggression trials	c	usa	21	5.7	49	hr ecg arm	110	HR	task	other	physical aggression - lab aggression	
#2352 - Verschuere 2005	(Verschuere et al., 2005)	Prisoners	AB	criminal justice	none (one group)	c	belgium	39	11	0	sc electrode; hr ecg	37	SCR; HR	reactivity	concealed information	psychopathy	PPI Factor 1 Fearless Dominance; PPI Factor 2 Impulsive Antisociality
#2357 - Verschuere 2007	(Verschuere et al., 2007)	Prisoners and community controls	mix	criminal justice and community	prisoners vs controls	c	belgium	36.78	10.38	0	hr ecg; sc electrode	79	HR; SCR	reactivity	concealed information	physical aggression - violent offenders	offenses
#5167 - Vitoria-Estruch 2018	(Vitoria-Estruch et al., 2018)	IPV men	mix	criminal justice and community	IPV vs controls	c	spain	40.75	10.94	0	hr ecg; sc electrode	95	HR; SCL; PEP; HF; RSA	rest; task	other; cognitive	physical aggression - violent offenders	CTS-2 and conviction
#2397 - Wahlund 2010	(Wahlund et al., 2010)	psychiatric patients and community control	mix	criminal justice and community	ASPD vs community control; ASPD vs non-ASPD psychiatric control	c	sweden	33.6; 34.94	7.91; 11.5	0	sc electrode	36; 41	SCR	reactivity	emotion picture	aspd	SCID-1
#2476 - Wininger 2016 (dissertation)	(Wininger, 2016)	students	students	community	none (one group)	c	usa	22.33	6.37	68	sc electrode	125	SCR	reactivity	other	psychopathy	PPI-R total
#5008 - Zhan 2017	(Zhan et al., 2017)	students	students	community	none (one group)	c	china	20.76	1.73	66	sc electrode	180	SCL	task	competitive reaction time	physical aggression - lab aggression	-
#2524 - Zimak 2014	(Zimak, 2012; Zimak et al., 2014)	students	students	community	high psychopathy vs low psychopathy	c	usa	19.32	1.54	0	sc electrode	76	SCL; SCR	rest; reactivity	emotion picture; iowa gambling task	psychopathy	PPI-SF total

Note. (M)TAP = (Modified) Taylor Aggression Paradigm; AB = Antisocial Behavior; APSD = Antisocial Process Screening Device; ASBQ = Antisocial Behavior Questionnaire; ASI = Adolescent Symptom Inventory; ASPD = Antisocial Personality Disorder; ATSS = Articulated Thoughts in Simulated Situations; BDHI = Buss Durkee Hostility Inventory; BPAQ = Buss Perry Aggression Questionnaire (only the physical aggression scale is used in this meta-analysis); BRFI = Brief Reflective Function Interview; CANTAB = Cambridge Neuropsychological Test Automated Battery; CIT = Concealed Information Test; CPS = Child Psychopathy Scale; CPT – CalCAP = Continuous Performance Test – California Computerized Assessment Package abbreviated version; CPT = Continuous Performance Task; CRTT = Competitive Reaction Time Task; CSBS – T = Children's Social Behavior Scale – Teacher Report; CSDD = Cambridge Study in Delinquent Development; CTS-2 = Conflicts Tactics Scale; DAWBA = Development and Well Being Assessment structured interview; DBD = Disruptive Behavior Disorder; DISC – 4 = Diagnostic Interview Schedule for Children Version IV; DSM – 4 = Diagnostic and Statistical Manual of Mental Disorders Version 4; DSM – 5 = Diagnostic and Statistical Manual of Mental

Disorders Version 5; ECG = Electrocardiography; FCE = Fear Conditioning Experiment; GSST = Groningen Social Stress Task; HF = High Frequency; HR = Heart rate; HRV = Total Power (in this meta-analysis); IADS = International Affective Digitized Sounds; IAPS = International Affective Picture System; IAT = Implicit Association Task; IBI = Inter Beat Interval; ICG = Impedance Cardio Graphy; IGT = Iowa Gambling Task; IPV = Intimate Partner Violence; K – SADS – PL = Schedule for Affective Disorders and Schizophrenia for School – Age Children – Present and Lifetime Version; LF = low Frequency; LPS = Levenson Psychopathy Scales; LSRP = Levenson's Self – Reported Psychopathy Scale; Mix = Mixed Sample; MMPI-2 = Minnesota Multiphasic Personality Inventory 2; MPQ = Multidimensional Personality Questionnaire; MPT = Manual Precision Task; MTFS = Minnesota Twin Family study; PAI = Personality assessment inventory – aggression scale(PAI – AGG); PBH = 1993 Pelotas Birth Cohort; PCL – R = Psychopathy Checklist – Revised; PCL – SV = Psychopathy Checklist – Screening Version; PCS = Peer Conflict Scale; PCS – C = Peer Conflict Scale Youth Self-Report; PCS – P = Peer Conflict Scale Parent – Report; PDG = Prisoners Dilemma Game; PEP = Pre Ejection Period; PPI = Psychopathic Personality Inventory; PPI – R = Psychopathic Personality Inventory – Revised; PPI – SF = Psychopathic Personality Inventory – Short Form; PSPS = Primary and Secondary Psychopathy Scales; PST = Public Speaking Test; PTSD = Posttraumatic Stress Disorder; PYS = Pittsburgh Youth Study; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SC = Skin Conductance; SCID = Structured Clinical Interview for Diagnostic and Statistical Manual; SCL = Skin Conductance Level; SCR = Skin Conductance Response; SIDP-4 = Structured Interview for DSM – IV Personality; SRD = Self – Reported Delinquency scale; SRP – 2 = Self – Report Psychopathy 2; SRP – 3 = Self – Report Psychopathy 3; SRP-4 = Self – Report Psychopathy 4; SSPD = Schizophrenia – Spectrum Personality Disorder; SSS = Sexual Strategies Scale; SUD = Substance Use Disorder; TRAILS = Tracking Adolescents' Individual Lives Survey; TRIPM = Triarchic Psychopathy Measure; TSST = Trier Social Stress Test; WMT = Working Memory Test; YPI – SV = Youth Psychopathic Traits Inventory – Short Version.
Studies without all necessary information (Colasante & Malti, 2017; Fairchild et al., 2010; Fairchild, Van Goozen, et al., 2008; Fairchild, van Goozen, et al., 2008; Flor et al., 2002; Gallant et al., 2018; Hoaken et al., 2003; Klimecki et al., 2016).
References of Table 1: Armstrong et al., 2009, 2017; Arriaga et al., 2008; Assaad et al., 2003, 2006; Assaad, 2002; Babcock et al., 2004, 2005; Babcock and Michonski, 2019; Babcock and Potthoff, 2020; Potthoff, 2016; Babel et al., 2016; Bare et al., 2004; Beauchaine, 2002a, 2002b; Benning et al., 2005; Betensky and Contrada, 2010; Birbaumer et al., 2005; Bobadilla and Taylor, 2007; Bobadilla, 2008; Broom, 2012; Brown, 2015; Casey et al., 2013; Cauffman et al., 2005; da Silva et al., 2014; de Barros et al., 2013; De Vries-Bouw et al., 2011, 2012; Popma et al., 2006; de Wied et al., 2012; Dindo and Fowles, 2011, 2008; Dindo, 2008; Fanti et al., 2017; Florez et al., 2017; Fung et al., 2005; Loeber et al., 2007; Galan et al., 2017; Gao et al., 2012; Gerra et al., 2003; Goulter et al., 2019; Hansen et al., 2007; Hasan et al., 2013; Hong et al., 2018; Humphreys et al., 2012; Ibanez et al., 2016; Im et al., 2018, 2019; Iria et al., 2020; Isen et al., 2012; Jennings et al., 2013; Jennings et al., 2017, 2019; Kavish et al., 2017; Kavish, Bergstrøm, et al., 2019; Kirsch, 2009; Koegl et al., 2018; Kulper, 2016; Kyranides et al., 2017; Latvala et al., 2015, 2016; Ling et al., 2018; Lishak, 2018; Lobbestael et al., 2009; Lobbestael and Arntz, 2010; Lotze et al., 2007; MacDougall, 2016; MacDougall et al., 2019; Molapour et al., 2016; Munoz, 2005; Muñoz et al., 2008a, 2008b; Murray et al., 2016a,b; Northover, 2015; Northover et al., 2015; Oldenhof et al., 2019; Osumi et al., 2007; Osumi and Ohira, 2010; Osumi, 2019; Pastor et al., 2003; Pfabigan et al., 2015; Popma et al., 2006; Portnoy, 2015; Portnoy et al., 2014; Puhalla et al., 2020; Ragsdale et al., 2013; Raine et al., 2003, 2014; Rinnewitz et al., 2019; Romero-Martínez et al., 2013; Romero-Martinez et al., 2014; Rosenberger et al., 2019; Rothemund et al., 2012; Schug et al., 2007; Scott, 2013; Seidel et al., 2013; Serafim et al., 2009; Sijtsema et al., 2011, 2015; Sorman et al., 2016; Stanger et al., 2012; Sylvers et al., 2008, 2010; Thomson and Beauchaine, 2019; Thomson, Kiehl, et al., 2019; Trahan, 2014; Trahan and Babcock, 2019; Umhau et al., 2002; van Goozen et al., 2016; Veit et al., 2013; Verona and Sullivan, 2008; Verschuere et al., 2005; Verschuere et al., 2007; Vitoria-Estruch et al., 2018; Wahlund et al., 2010; Wininger, 2016; Zhan et al., 2017; Zimak, 2012; Zimak et al., 2014; Bolinger, 2018.

.385, $k = 17$) differed from SC (ES = −0.24, $p < .001$, $k = 7$) for the non-theorized ES, while, as one can see in supplement 10, both HR (ES = −0.11, $p = .145$, $k = 34$), and SC (ES = −0.15, $p = .248$, $k = 21$) were negative for the theorized effect.

The *valence variable* did not result in Criterion 1 ESs (Table 3), although the neutral and positive valence resulted in Criterion 2 estimates: neutral (ES = −0.18, $p = .206$, $k = 29$), and positive (ES = −0.17, $p = .323$, $k = 37$). It is notable that the negative valence was not significant (ES = .02, $p = .708$, $k = 206$). Further valence analyses revealed (available in "S11 Valence.R") that this may be due, in part, to the inclusion of both HR(V) and SC analyses. For instance, with the emotional pictures, the estimates for HR (ES = 0.34, $p < .001$, $k = 7$) were higher than for SC (ES = 0.01, $p = .950$, $k = 27$), which might have influenced the results.

The *Countries* with level 2 estimates were Austria (ES = −0.19, $p = .578$, $k = 26$), Brazil (ES = −0.23, $p = .685$, $k = 24$), Cyprus (ES = −0.22, $p = .064$, $k = 42$), and Japan (ES = −0.39, $p = .056$, $k = 18$), respectively.

Table 2
Log-likelihood ratio tests and variance parameter estimates.

	Estimates	Deviance	LRT	p-value
k	769	−350.977		
Pooled effect	−0.075			
Standard Error	0.037			
95 % CIs	(−.147, −.003)			
Variance of random effects				
Between-outcomes variance (number of outcomes)	0.029 (769)	−377.460	52.966	<.0001
Between-experiment variance (number of experiments)	0.006 (184)	−353.803	5.651	.018
Between-questionnaire variance (number of questionnaires)	0.009 (105)	−354.328	6.701	.0096
Between-studies variance (number of studies)	0.077 (101)	−402.248	102.541	<.0001

Note. k = number of effect sizes, LRT = Likelihood ratio test.

Overall, it is notable that several covariates did not moderate the association, such as ECG, (more) homogeneous samples, different types of AB and (the negative valence of) some experiments, which appears to indicate that there is no interaction effect for the ANS–AB association. Other covariates, however, did moderate the association, such as non–traditional ECG, various HR variability measures (PEP, RMSSD, RSA), violent offender groups and laboratory aggression, more heterogeneous (mixed) samples, and some (i.e., positively and neutral valenced) experiments. In addition, anticipation of noise tasks, cognitive tasks, competitive reaction time tasks, and fear conditioning tasks, including theorized effects, resulted in either larger ESs or *bidirectional* effects depending on the type of outcome (e.g., HR and SC in the public speaking tasks), thus indicating that there is an interaction effect for the ANS–AB association. Notably, moderator variables reduced the observed variability across all levels (studies, experiments, questionnaires and outcomes) with respect to the variances observed in the null model (Table 2). This denotes that the moderators accounted for differences among studies, and/or outcomes, and/or questionnaires, and/or experiments, despite the fact that some of the moderator variables did not reach the preset criterion cut-off for statistical relevance.

3.4. ANS specificity in experiments and for behavior type

The fourth and final research question involved examining if certain experiments and types of analysis could be used to specifically differentiate between types of AB (Table 4, analyses available in "S12 Interactions.R", and an Excel file ("Table 4.xlsx") is available as well to quickly filter the results that the reader is interested in). The low frequency (LF; $k = 6$), inter-beat interval (IBI; $k = 2$) and HR variability ($k = 3$) outcomes could not be considered for the interactions due to the small number of estimates.

Table 3
Meta-regression of study variables.

name	*test*	*pooled estimate*	*se*	*z*	*p*	*study*	*outcome*	*experiment*	*questionnaire*	*total variance*	*N study*	N outcome	*N experiment*	*N questionnaire*	*criterion*
						Variance of random effects					Number of random effects				
Method assessment															
Hr ecg	CCREM	0	0,03	0,07	*0,943*	0,01	0,08	0	0	0,08	42	*269*	88	60	
Hr ecg arm	CCREM	−0,14	0,08	−1,82	*0,069*	0	0	0,05	0,03	0,08	8	*80*	20	25	
Hr other	CCREM	−0,29	0,13	−2,3	*0,021*	0,19	0	0,01	0	0,2	15	*58*	17	22	1
Sc electrode	CCREM	−0,06	0,05	−1,21	*0,227*	0,11	0	0	0,01	0,12	55	*348*	138	66	
Analysis															
Reactivity	CCREM	−0,05	0,04	−1,15	*0,25*	0,05	0,03	0,01	0,01	0,09	55	*338*	122	59	
Rest	CCREM	−0,11	0,03	−3,23	*0,001*	0,03	0,03	0	0	0,06	45	*180*	22	70	
Task	CCREM	−0,06	0,08	−0,74	*0,461*	0,17	0,03	0	0,01	0,21	32	*251*	65	44	
Design															
C	CCREM	−0,07	0,04	−1,85	*0,065*	0,08	0,03	0	0,01	0,13	91	*701*	176	95	
L	CCREM	−0,06	0,08	−0,83	*0,408*	0,02	0	0,02	0	0,04	10	*68*	15	14	
Age															
	CCREM	0	0	−0,9	*0,369*	0,09	0,02	0	0,01	0,12	96	*744*	177	105	
Age sd															
	CCREM	−0,01	0,01	−0,96	*0,338*	0,06	0,02	0	0,01	0,1	88	*698*	178	99	
Female percentage															
	CCREM	0	0	1,12	*0,264*	0,08	0,03	0,01	0,01	0,12	97	*751*	180	104	
Sample size															
	CCREM	0	0	0,1	*0,92*	0,08	0,03	0,01	0,01	0,12	101	*769*	184	105	
Outcome type															
Hr	CCREM	−0,08	0,05	−1,69	*0,091*	0,08	0,03	0,01	0,01	0,13	71	*422*	116	93	
Sc	CCREM	−0,06	0,05	−1,24	*0,215*	0,11	0	0	0,01	0,12	57	*347*	139	68	
Outcome type															
Hf	CCREM	−0,03	0,06	−0,46	*0,649*	0	0	0,01	0,01	0,02	7	*29*	22	7	
Hr	CCREM	−0,08	0,05	−1,58	*0,113*	0,09	0	0,01	0,01	0,12	59	*294*	100	76	
Hrv	uni	−0,06	0,09	−0,68	*0,499*					0	0	*3*	0	0	
Ibi	uni	−0,68	0,14	−4,93	*0*					0	0	*2*	0	0	3.1
Lf	uni	−0,04	0,09	−0,48	*0,632*					0,02	0	*6*	0	0	
Pep	CCREM	0,15	0,12	1,27	*0,206*	0	0	0,07	0,03	0,09	7	*20*	11	8	2
Rmssd	CCREM	0,37	0,41	0,9	*0,37*	0,3	0	0	0,13	0,43	2	*26*	6	8	2
Rsa	CCREM	−0,16	0,13	−1,21	*0,226*	0,21	0	0	0,06	0,27	17	*42*	20	26	2
Scl	CCREM	−0,02	0,07	−0,24	*0,81*	0,08	0	0,01	0	0,09	24	*92*	37	42	
Scr	CCREM	−0,09	0,07	−1,3	*0,194*	0,12	0	0	0,02	0,14	40	*255*	111	52	
Outcome type															
Mixed	uni	−0,18	0,13	−1,45	*0,148*					0,09	0	*8*	0	0	3.2
Sympathetic	CCREM	−0,04	0,05	−0,88	*0,378*	0,11	0	0,01	0,01	0,13	62	*367*	144	72	
Undefined	CCREM	−0,08	0,05	−1,58	*0,113*	0,09	0	0,01	0,01	0,12	59	*294*	100	76	
Vagal	CCREM	−0,03	0,1	−0,26	*0,792*	0,15	0	0	0,08	0,23	25	*100*	41	37	
Behavior type															
Aspd	CCREM	−0,04	0,15	−0,27	*0,787*	0,13	0,13	0,01	0	0,27	9	*32*	15	5	
Aspd and psychopathy	uni	−0,82	0,24	−3,41	*0,001*					0	0	*2*	0	0	3.1
Conduct disorder	CCREM	−0,09	0,06	−1,51	*0,13*	0,01	0,03	0	0	0,05	8	*54*	17	6	
Physical aggression	CCREM	−0,07	0,09	−0,83	*0,405*	0,12	0	0	0	0,13	20	*71*	30	17	
Physical aggression - lab aggression	CCREM	0,2	0,16	1,21	*0,227*	0,23	0,01	0	0	0,24	11	*26*	15	1	2
Physical aggression - violent offenders	CCREM	−0,33	0,16	−2,08	*0,037*	0,07	0,01	0	0,19	0,27	18	*95*	48	11	1
Psychopathy	CCREM	−0,07	0,04	−1,93	*0,054*	0,02	0,03	0,01	0,01	0,07	44	*489*	95	68	
Comparison															
Correlation	CCREM	−0,06	0,04	−1,48	*0,138*	0,06	0,04	0,01	0,01	0,11	54	*494*	85	85	
Difference	CCREM	−0,1	0,06	−1,72	*0,085*	0,13	0,01	0	0	0,15	50	*275*	116	36	
Risk of bias															
High	CCREM	−0,05	0,04	−1,38	*0,166*	0,01	0,04	0	0,02	0,08	27	*286*	54	60	

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Table 3 (*continued*)

name	test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	criterion
						Variance of random effects					Number of random effects				
Low	CCREM	−0,09	0,06	−1,51	*0,132*	0,03	0,03	0	0,02	0,08	24	*154*	64	24	
Medium	CCREM	−0,13	0,08	−1,64	*0,1*	0,12	0,01	0,02	0	0,15	24	*142*	39	23	
Very high	CCREM	0,05	0,08	0,62	*0,537*	0,02	0,03	0	0,01	0,06	7	*85*	14	16	
Very low	CCREM	−0,13	0,12	−1,06	*0,288*	0,26	0,01	0	0	0,27	19	*102*	45	19	
Sample description															
Ab	CCREM	−0,02	0,03	−0,66	*0,509*	0	0,08	0	0,01	0,09	19	*215*	35	29	
Mix	CCREM	−0,18	0,08	−2,32	*0,02*	0,15	0,01	0	0	0,16	27	*190*	87	23	1
Non ab	CCREM	−0,08	0,06	−1,24	*0,216*	0,04	0	0	0,03	0,07	21	*90*	35	27	
Students	CCREM	0,02	0,07	0,28	*0,778*	0,09	0,01	0,02	0,02	0,14	36	*274*	59	51	
Recruitment source															
Community	CCREM	−0,02	0,04	−0,44	*0,66*	0,07	0,01	0,01	0,02	0,11	69	*434*	107	82	
Criminal justice	CCREM	−0,14	0,09	−1,56	*0,119*	0,07	0,11	0	0	0,18	13	*179*	27	20	
Mental health	uni	−0,2	0,19	−1,06	*0,288*					0,1	0	*5*	0	0	3.2
Mix	CCREM	−0,22	0,11	−2,05	*0,04*	0,15	0,02	0	0,02	0,19	18	*151*	72	12	1
Year															
	CCREM	0,01	0,01	1,9	*0,057*	0,07	0,03	0,01	0,01	0,12	101	*769*	184	105	
Experiment															
Anger induction	uni	−0,14	0,14	−1	*0,32*					0,02	0	*3*	0	0	
Anticipation-of-noise	CCREM	−0,15	0,08	−1,97	*0,049*	0,01	0	0	0,02	0,03	6	*54*	7	13	1
Cognitive	CCREM	0,19	0,06	3,51	*0*	0	0	0	0	0	1	*15*	3	1	1
Competitive reaction time	CCREM	0,36	0,23	1,57	*0,117*	0,23	0,01	0	0,08	0,32	10	*31*	15	6	2
Concealed information	uni	−0,29	0,15	−2,03	*0,042*					0,08	0	*8*	0	0	3.1
Conflict	CCREM	0	0,12	0,01	*0,991*	0	0,07	0	0	0,07	3	*11*	1	4	
Continuous performance	uni	−0,02	0,22	−0,11	*0,912*					0,44	0	*10*	0	0	
Emotion audio	uni	−0,23	0,13	−1,74	*0,081*					0,03	0	*3*	0	0	3.2
Emotion picture	CCREM	−0,05	0,15	−0,34	*0,735*	0,28	0,03	0	0,03	0,33	15	*111*	31	20	
Emotion video	CCREM	−0,07	0,1	−0,73	*0,463*	0,03	0	0,02	0	0,05	7	*47*	10	15	
Empathy for pain	uni	−0,24	0,13	−1,81	*0,071*					0,04	0	*8*	0	0	3.2
Fear conditioning	CCREM	−0,21	0,18	−1,17	*0,242*	0	0,33	0	0,05	0,38	5	*53*	27	5	2
Guided imagery	CCREM	−0,13	0,08	−1,67	*0,096*	0	0	0	0,01	0,01	1	*16*	5	2	
Iowa gambling task	CCREM	0	0,04	0,03	*0,976*	0	0,03	0	0	0,04	3	*84*	5	12	
Mental arithmetic	uni	−0,2	0,08	−2,35	*0,019*					0	0	*5*	0	0	3.1
Other	CCREM	−0,1	0,04	−2,85	*0,004*	0,04	0,03	0	0	0,07	55	*198*	35	76	
Public speaking	CCREM	−0,06	0,08	−0,69	*0,488*	0,04	0	0	0,04	0,08	13	*79*	20	20	
Trust game	uni	0,16	0,17	0,93	*0,352*					0,23	0	*10*	0	0	3.2
Ultimatum game	uni	−0,55	0,13	−4,16	*0*					0	0	*7*	0	0	3.1
Vr horror movie	uni	0,15	0,07	2,22	*0,027*					0	0	*8*	0	0	3.1
Working memory	uni	0,12	0,26	0,46	*0,649*					0,44	0	*8*	0	0	
Country															
Australia	CCREM	−0,04	0,24	−0,16	*0,869*	0	0	0,01	0,22	0,22	1	*14*	4	4	
Austria	CCREM	−0,19	0,34	−0,56	*0,578*	0,32	0	0	0	0,32	3	*26*	25	3	2
Belgium	uni	−0,41	0,16	−2,6	*0,009*					0,06	0	*6*	0	0	3.1
Brazil	CCREM	−0,23	0,58	−0,41	*0,685*	1,15	0,09	0	0,13	1,37	4	*24*	7	5	2
Canada	CCREM	0,07	0,11	0,67	*0,504*	0,02	0,04	0	0	0,07	4	*88*	8	11	
China	uni	0,81	0,16	5,21	*0*					0	0	*1*	0	0	3.1
Cyprus	CCREM	−0,22	0,12	−1,85	*0,064*	0	0	0,05	0	0,05	2	*42*	4	7	2
Europe	uni	0,05	0,06	0,9	*0,37*					0,01	0	*6*	0	0	
France	uni	−0,74	0,24	−3,14	*0,002*					0	0	*1*	0	0	3.1
Germany	CCREM	0	0,31	0,01	*0,989*	0	0,27	0	0,29	0,56	5	*56*	30	4	
Hong kong	uni	−0,24	0,08	−2,96	*0,003*					0,01	0	*4*	0	0	3.1
Italy	uni	−0,99	0,38	−2,61	*0,009*					0	0	*1*	0	0	3.1
Japan	CCREM	−0,39	0,2	−1,91	*0,056*	0,07	0	0	0,01	0,07	3	*18*	12	3	2
Korea	uni	0,29	0,12	2,43	*0,015*					0	0	*5*	0	0	3.1
Netherlands	CCREM	−0,08	0,07	−1,21	*0,226*	0,02	0	0	0	0,03	7	*51*	16	6	

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Table 3 (*continued*)

name	test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	criterion
						Variance of random effects					Number of random effects				
Norway	CCREM	0,05	0,17	0,31	0,76	0	0,57	0	0	0,57	1	24	3	4	
Portugal	uni	0,2	0,17	1,17	0,241					0	0	1	0	0	3.2
Spain	CCREM	0,05	0,08	0,63	0,526	0	0,03	0	0,02	0,05	6	48	15	4	
Sweden	CCREM	−0,06	0,17	−0,32	0,747	0,09	0	0	0	0,09	4	12	5	5	
Uk	CCREM	−0,1	0,2	−0,51	0,607	0,23	0	0,03	0,04	0,31	9	29	9	17	
Usa	CCREM	−0,06	0,04	−1,46	0,145	0,04	0	0,01	0,01	0,06	42	312	67	54	
Valence															
Negative	CCREM	0,02	0,05	0,37	0,708	0,07	0,01	0	0,02	0,11	52	206	67	54	
Neutral	CCREM	−0,18	0,14	−1,26	0,206	0,15	0,06	0	0	0,21	12	29	6	18	2
Pleasant	CCREM	0,06	0,06	1,09	0,274	0	0	0	0	0	2	14	1	7	
Positive	CCREM	−0,17	0,17	−0,99	0,323	0,28	0	0	0,04	0,33	12	37	11	17	2

Note. AB = Antisocial Behavior; ASPD = Antisocial Personality Disorder; Criterion = Criterion 1) magnitude exceeding, k > 10, and significant, Criterion 2) magnitude exceeding, k > 10, and non – significant, Criterion 3.1) magnitude exceeding, k < 10, and significant, Criterion 3.2) magnitude exceeding, k < 10, and non – significant; C = Crossectional; CCREM = cross-classified random effects model; HF = High Frequency; HR = Heart rate; ECG = Electrocardiography; HRV = Heart Rate Variability; IBI = Inter Beat Interval; L = Longitudinal; LF = low Frequency; PEP = Pre Ejection period; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SE = Standard error; SC = Skin Conductance; SCL = Skin Conductance Level; SCR = Skin Conductance Response; SD = Standard Deviation; UK = United Kingdom; UNI = Univariate analysis; USA = United States of America; Z = z-value.

3.4.1. ANS measures and experiments

The ‘other experiment’ category was not considered because of the large heterogeneity of the experiments included within this category, as well as the small number of ESs for the different types of experiments in that category. A likelihood ratio test showed that the interaction between experiment type and outcome was significant ($\chi2_{(31)} = 215.99$, $p < .0001$). That is to say, the relationship between the outcome type and the ESs differed depending on the type of experiment conducted. Criterion 1 estimates were obtained for HR–Fear conditioning task (ES = 0.33, p = .005, k = 14) and HR–Iowa gambling task (ES = −0.2, p = .027, k = 40). Criterion 2 estimates were obtained for HR–competitive reaction time task (ES = 0.2, p = .400, k = 11), HR–emotion picture (ES = -0.18, p = .651, k = 19), and SCL–public speaking task (ES = −0.22, p = .226, k = 18). For a considerable number of experiments, we obtained both Criterion 3.1 and 3.2 estimates. According to our own preset criteria, these are currently promising at best, and need to be interpreted with caution (Table 4).

3.4.2. Analysis and outcome type

A likelihood ratio test showed that the interaction effect between the type of analysis and outcome type was significant ($\chi2_{(13)} = 75.65$, p < .0001). In other words, the relationship between the outcome type and ESs was different depending on the type of analysis used (Fig. 1). The analysis variable showed various significant ESs, which exceeded the magnitude threshold. Criterion 1 estimates were obtained for HR–rest (ES = −0.17, p < .001, k = 90). Criterion 2 estimates included RMSSD–task (ES = 0.36, p = .331, k = 18), RSA–rest (ES = −0.16, p = .2, k = 21), and SCL–rest (ES = −0.15, p = .086, k = 30).

3.4.3. Behavior type and outcome

A likelihood ratio test showed that the interaction between behavior type and outcome was significant ($\chi2_{(16)} = 84.41$, $p < .0001$). That is to say, the relationship between the behavior type and the ESs was dependent on the outcome used (Fig. 2). Criterion 2 estimates included HR–laboratory aggression (ES = 0.16, p = .397, k = 12), HR–violent offenders (ES = −0.21, p = .101, k = 33), RMSSD–psychopathy (ES = 0.44, p = .194, k = 24), RSA–physical aggression (ES = −0.24, p = .337, k = 12), SCR–physical aggression (ES = 0.17, p = .45, k = 15), and SCR–violent offenders (ES = −0.36, p = .109, k = 44).

3.4.4. Behavior type and analysis

A likelihood ratio test showed that the interaction effect between the variables behavior type and type of analysis was not significant ($\chi2_{(12)}$ = 14.78, p = .097). Consequently, the relationship between the behavior type and the ESs was not dependent on the analysis used, and, hence, was not interpreted (the analysis can be run using the supplement “S12 Interactions.R”).

3.4.5. Behavior type, analysis and outcome

A likelihood ratio test indicated that the triple interaction effect between behavior type, analysis type and outcome type was significant ($\chi2_{(62)} = 35.04$, p = .020). That is to say, the relationship between the behavior type, analysis type and ESs was dependent on the outcome used (Fig. 3). Criterion 1 estimates for the triple interaction were HR–psychopathy–rest (ES = -0.26, p < .001, k = 43). Criterion 2 estimates included HR–psychopathy–task (ES = −0.15, p = .065, k = 93), RMSSD–psychopathy–task (ES = 0.41, p = .225, k = 17), HR–violent offenders for both rest (ES = −0.19, p = .083, k = 17) and task (ES = −0.35, p = .413, k = 14), SCR–violent offenders–reactivity (ES = −0.37, p = .181, k = 31), and SCR–violent offenders–task (ES = −0.32, p = .37, k = 12). Finally, SCR–antisocial personality disorder–reactivity (ES = −0.15, p = .544, k = 13) was a Criterion 2 estimate.

3.5. Publication bias

Publication bias analyses were first conducted on the complete

Table 4
Interaction analyses of study variables.

outcome	Experiment	test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	Criterion
							Variance of random effects					Number of random effects				
Hf	cognitive	uni	0,21	0,12	1,74	*0,082*					0	0	*3*	0	0	3.2
Hf	competitive reaction time	uni	0,06	0,11	0,49	*0,621*					0	0	*4*	0	0	
Hf	emotion audio	uni	−0,45	0,14	−3,22	*0,001*					0	0	*1*	0	0	3.1
Hf	iowa gambling task	uni	0	0,15	0	*1*					0	0	*1*	0	0	
Hf	mental arithmetic	uni	−0,19	0,12	−1,6	*0,11*					0	0	*4*	0	0	3.2
Hf	other	uni	−0,03	0,07	−0,47	*0,636*					0	0	*9*	0	0	
Hf	public speaking	uni	0,06	0,14	0,45	*0,652*					0	0	*7*	0	0	
Hr	anger induction	uni	0	0,19	0	*1*					0	0	*1*	0	0	
Hr	anticipation-of-noise	CCREM	−0,03	0,09	−0,27	*0,788*	0	0	0	0,03	0,03	1	*20*	4	6	
Hr	cognitive	uni	0,04	0,12	0,36	*0,722*					0	0	*3*	0	0	
Hr	competitive reaction time	CCREM	0,2	0,23	0,84	*0,4*	0,24	0	0	0	0,24	5	*11*	11	1	2
Hr	concealed information	uni	−0,16	0,13	−1,22	*0,223*					0	0	*5*	0	0	3.2
Hr	conflict	uni	0,02	0,15	0,12	*0,904*					0,08	0	*8*	0	0	
Hr	continuous performance	uni	−0,46	0,16	−2,83	*0,005*					0,11	0	*6*	0	0	3.1
Hr	emotion audio	uni	0	0,14	0	*1*					0	0	*1*	0	0	
Hr	emotion picture	CCREM	−0,18	0,39	−0,45	*0,651*	0,68	0	0,03	0	0,71	5	*19*	6	5	2
Hr	emotion video	CCREM	−0,09	0,12	−0,73	*0,466*	0	0	0,08	0,01	0,09	7	*31*	10	15	
Hr	empathy for pain	uni	−0,05	0,23	−0,2	*0,842*					0	0	*3*	0	0	
Hr	fear conditioning	CCREM	0,33	0,12	2,83	*0,005*	0	0	0	0	0	1	*14*	14	1	1
Hr	guided imagery	uni	0,05	0,08	0,64	*0,522*					0	0	*8*	0	0	
Hr	iowa gambling task	CCREM	−0,2	0,09	−2,21	*0,027*	0	0	0,01	0,04	0,05	1	*40*	4	10	1
Hr	mental arithmetic	uni	−0,2	0,12	−1,72	*0,086*					0	0	*1*	0	0	3.2
Hr	other	CCREM	−0,14	0,05	−2,77	*0,006*	0,06	0,01	0	0	0,07	36	*85*	15	52	
Hr	public speaking	CCREM	−0,12	0,05	−2,23	*0,026*	0	0	0,02	0	0,02	9	*34*	17	13	
Hr	working memory	uni	−0,51	0,14	−3,63	*0*					0	0	*4*	0	0	3.1
Pep	anticipation-of-noise	uni	−0,33	0,14	−2,33	*0,02*					0	0	*2*	0	0	3.1
Pep	cognitive	uni	0,12	0,12	1,01	*0,314*					0	0	*3*	0	0	
Pep	emotion picture	uni	0,18	0,29	0,61	*0,539*					0,28	0	*4*	0	0	3.2
Pep	other	uni	0,14	0,05	3,07	*0,002*					0	0	*9*	0	0	
Pep	public speaking	uni	0,12	0,06	2,11	*0,035*					0	0	*2*	0	0	
Rmssd	competitive reaction time	uni	−0,17	0,2	−0,83	*0,407*					0,16	0	*5*	0	0	3.2
Rmssd	continuous performance	uni	0,73	0,14	5,11	*0*					0	0	*4*	0	0	3.1
Rmssd	other	uni	0,54	0,2	2,77	*0,006*					0,21	0	*7*	0	0	3.1
Rmssd	public speaking	uni	0,08	0,18	0,44	*0,66*					0,16	0	*6*	0	0	
Rmssd	working memory	uni	0,75	0,14	5,27	*0*					0	0	*4*	0	0	3.1
Rsa	anger induction	uni	0	0,19	0	*1*					0	0	*1*	0	0	
Rsa	cognitive	uni	0,15	0,12	1,18	*0,237*					0	0	*3*	0	0	3.2
Rsa	competitive reaction time	uni	−0,74	0,24	−3,14	*0,002*					0	0	*1*	0	0	3.1

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Table 4 (*continued*)

outcome	Experiment	test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	Criterion
							Variance of random effects					Number of random effects				
Rsa	conflict	uni	−0,52	0,26	−2,01	*0,045*					0	0	*1*	0	0	3.1
Rsa	emotion picture	uni	0,72	0,1	6,95	*0*					0	0	*4*	0	0	3.1
Rsa	other	CCREM	−0,22	0,15	−1,5	*0,133*	0,17	0	0	0,07	0,25	13	*26*	9	20	2
Rsa	public speaking	uni	0,23	0,11	2,07	*0,038*					0,02	0	*2*	0	0	3.1
Rsa	VR horror movie	uni	0,15	0,11	1,34	*0,181*					0,01	0	*4*	0	0	3.2
Scl	anger induction	uni	−0,42	0,19	−2,2	*0,028*					0	0	*1*	0	0	3.1
Scl	anticipation-of-noise	uni	−0,27	0,08	−3,43	*0,001*					0	0	*6*	0	0	3.1
Scl	cognitive	uni	0,45	0,12	3,6	*0*					0	0	*3*	0	0	3.1
Scl	competitive reaction time	uni	0,81	0,16	5,21	*0*					0	0	*1*	0	0	3.1
Scl	conflict	uni	0,28	0,26	1,1	*0,271*					0	0	*1*	0	0	3.2
Scl	emotion picture	CCREM	0,08	0,29	0,28	*0,776*	0,15	0	0	0	0,15	2	*14*	5	5	
Scl	emotion video	CCREM	−0,06	0,07	−0,81	*0,418*	0	0	0	0	0	3	*15*	3	8	
Scl	other	CCREM	−0,06	0,08	−0,8	*0,423*	0,05	0	0	0	0,05	12	*29*	9	23	
Scl	public speaking	CCREM	−0,22	0,19	−1,21	*0,226*	0,13	0	0	0,03	0,16	5	*18*	11	9	2
Scl	VR horror movie	uni	0,16	0,1	1,67	*0,095*					0	0	*4*	0	0	3.2
Scr	anticipation-of-noise	CCREM	−0,04	0,12	−0,36	*0,718*	0,02	0	0	0,02	0,05	4	*26*	7	8	
Scr	competitive reaction time	uni	0,07	0,1	0,71	*0,479*					0	0	*6*	0	0	
Scr	concealed information	uni	−0,53	0,3	−1,79	*0,073*					0,17	0	*3*	0	0	3.2
Scr	conflict	uni	0,1	0,17	0,58	*0,563*					0	0	*1*	0	0	
Scr	emotion audio	uni	−0,23	0,14	−1,67	*0,094*					0	0	*1*	0	0	3.2
Scr	emotion picture	CCREM	−0,07	0,11	−0,65	*0,518*	0,04	0	0	0,08	0,12	9	*70*	27	14	
Scr	emotion video	uni	−0,34	0,28	−1,22	*0,224*					0	0	*1*	0	0	3.2
Scr	empathy for pain	uni	−0,34	0,19	−1,74	*0,082*					0,1	0	*5*	0	0	3.2
Scr	fear conditioning	CCREM	−0,11	0,33	−0,34	*0,737*	0,21	0,03	0	0,23	0,47	5	*39*	27	5	
Scr	guided imagery	uni	−0,31	0,08	−3,84	*0*					0	0	*8*	0	0	3.1
Scr	iowa gambling task	CCREM	0,04	0,23	0,16	*0,873*	0,08	0	0,01	0,01	0,1	2	*41*	5	11	
Scr	other	CCREM	0,04	0,13	0,31	*0,754*	0,15	0	0	0	0,16	11	*29*	16	14	
Scr	public speaking	uni	0,07	0,05	1,46	*0,145*					0	0	*10*	0	0	
Scr	trust game	uni	0,42	0,09	4,65	*0*					0	0	*8*	0	0	3.1
Scr	ultimatum game	uni	−0,55	0,13	−4,16	*0*					0	0	*7*	0	0	3.1
	Analysis															
Hf	reactivity	uni	−0,31	0,09	−3,48	*0*					0	0	*5*	0	0	3.1
Hf	rest		0,1	0,06	1,5	*0,134*	0	0	0	0	0	0	*0*	0	0	
Hf	task	uni	−0,02	0,07	−0,32	*0,746*					0,01	0	*10*	0	0	
Hr	reactivity	CCREM	0,01	0,07	0,14	*0,887*	0,06	0	0,01	0	0,07	24	*79*	45	30	
Hr	rest	CCREM	−0,17	0,05	−3,68	*0*	0,05	0	0	0	0,05	35	*90*	16	53	1
Hr	task	CCREM	−0,13	0,11	−1,21	*0,226*	0,19	0	0	0,03	0,22	20	*125*	46	35	
Pep	reactivity	uni	0,02	0,17	0,1	*0,921*					0,17	0	*7*	0	0	
Pep	rest	uni	0,11	0,05	2,42	*0,015*					0	0	*6*	0	0	
Pep	task	uni	0,2	0,06	3,1	*0,002*					0	0	*7*	0	0	3.1
Rmssd	rest	uni	0,39	0,23	1,69	*0,092*					0,37	0	*8*	0	0	3.2
Rmssd	task	CCREM	0,36	0,38	0,97	*0,331*	0,25	0	0	0,1	0,35	2	*18*	5	8	2
Rsa	reactivity	CCREM	0,12	0,19	0,64	*0,524*	0,14	0	0	0,02	0,17	5	*11*	7	7	
Rsa	rest	CCREM	−0,16	0,12	−1,28	*0,2*	0,06	0,12	0	0	0,17	11	*21*	4	18	2
Rsa	task	uni	−0,12	0,14	−0,87	*0,386*					0,14	0	*10*	0	0	
Scl	reactivity	CCREM	0,1	0,06	1,79	*0,074*	0,01	0	0,02	0	0,03	12	*46*	16	24	

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Table 4 (*continued*)

outcome			test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	Criterion
	Experiment							Variance of random effects					Number of random effects				
Scl	rest		CCREM	−0,15	0,09	−1,72	*0,086*	0,06	0	0	0	0,06	11	*30*	11	22	2
Scl	task		CCREM	−0,13	0,19	−0,71	*0,48*	0,25	0	0	0	0,25	8	*16*	13	10	
Scr	reactivity		CCREM	−0,13	0,07	−1,72	*0,086*	0,12	0	0	0,03	0,15	35	*188*	96	39	
Scr	rest		uni	0,18	0,09	2	*0,045*					0	0	*8*	0	0	3.1
Scr	task		CCREM	0,07	0,23	0,3	*0,763*	0,22	0	0,01	0,01	0,24	5	*59*	15	16	
	Behavior type																
Hf	cd		uni	0,06	0,14	0,45	*0,652*					0	0	*7*	0	0	
Hf	pa		uni	−0,08	0,08	−1,05	*0,294*					0	0	*9*	0	0	
Hf	pa laboratory		uni	0,06	0,11	0,49	*0,621*					0	0	*4*	0	0	
Hf	pa violent		uni	0,05	0,12	0,38	*0,701*					0,06	0	*6*	0	0	
Hf	psychopathy		uni	−0,13	0,09	−1,48	*0,14*					0	0	*3*	0	0	
Hr	aspd		uni	0,01	0,27	0,04	*0,97*					0,38	0	*6*	0	0	
Hr	cd		CCREM	−0,06	0,07	−0,86	*0,389*	0	0	0,03	0	0,03	6	*24*	14	5	
Hr	pa		CCREM	−0,07	0,07	−0,89	*0,375*	0,03	0	0	0	0,03	9	*22*	11	7	
Hr	pa laboratory		CCREM	0,16	0,19	0,85	*0,397*	0,19	0	0	0	0,19	6	*12*	12	1	2
Hr	pa violent		CCREM	−0,21	0,13	−1,64	*0,101*	0,19	0,01	0	0	0,2	13	*33*	17	9	2
Hr	psychopathy		CCREM	−0,11	0,05	−2,16	*0,03*	0,02	0	0,02	0,02	0,06	24	*196*	59	53	
Pep	aspd		uni	−0,15	0,19	−0,79	*0,428*					0,06	0	*3*	0	0	3.2
Pep	cd		uni	0,27	0,17	1,57	*0,117*					0,11	0	*6*	0	0	3.2
Pep	pa		uni	0,11	0,05	2,3	*0,022*					0	0	*4*	0	0	
Pep	pa violent		uni	0,11	0,11	1,06	*0,29*					0	0	*4*	0	0	
Pep	psychopathy		uni	0,16	0,4	0,4	*0,689*					0,45	0	*3*	0	0	3.2
Rmssd	pa laboratory		uni	−0,5	0,2	−2,52	*0,012*					0,04	0	*2*	0	0	3.1
Rmssd	psychopathy		CCREM	0,44	0,34	1,3	*0,194*	0,19	0	0	0,11	0,31	2	*24*	6	7	2
Rsa	aspd		uni	0,39	0,44	0,88	*0,378*					0,54	0	*3*	0	0	3.2
Rsa	cd		uni	−0,32	0,12	−2,56	*0,01*					0,05	0	*7*	0	0	3.1
Rsa	pa		CCREM	−0,24	0,25	−0,96	*0,337*	0,36	0	0	0	0,36	7	*12*	8	6	2
Rsa	pa laboratory		uni	−0,74	0,24	−3,14	*0,002*					0	0	*1*	0	0	3.1
Rsa	pa violent		uni	0,11	0,11	1,01	*0,312*					0	0	*4*	0	0	
Rsa	psychopathy		CCREM	0,12	0,11	1,04	*0,299*	0	0	0	0,11	0,11	4	*15*	5	12	
Scl	aspd		uni	0,06	0,11	0,6	*0,548*					0,03	0	*6*	0	0	
Scl	cd		uni	−0,31	0,09	−3,57	*0*					0	0	*8*	0	0	3.1
Scl	pa		uni	−0,13	0,1	−1,32	*0,188*					0,04	0	*9*	0	0	
Scl	pa laboratory		uni	0,81	0,16	5,21	*0*					0	0	*1*	0	0	3.1
Scl	pa violent		uni	0,43	0,11	4,01	*0*					0	0	*4*	0	0	3.1
Scl	psychopathy		CCREM	−0,01	0,08	−0,14	*0,887*	0,04	0	0,02	0	0,06	13	*63*	16	29	
Scr	aspd		CCREM	−0,08	0,24	−0,33	*0,741*	0,22	0,09	0	0	0,31	5	*14*	9	4	
Scr	cd		uni	−0,22	0,34	−0,64	*0,52*					0,19	0	*2*	0	0	3.2
Scr	pa		CCREM	0,17	0,22	0,76	*0,45*	0,22	0	0	0	0,22	5	*15*	12	6	2
Scr	pa laboratory		uni	0,43	0,19	2,3	*0,022*					0	0	*3*	0	0	3.1
Scr	pa violent		CCREM	−0,36	0,22	−1,6	*0,109*	0,06	0	0	0,19	0,26	7	*44*	36	7	2
Scr	psychopathy		CCREM	−0,12	0,08	−1,45	*0,148*	0,1	0	0	0,02	0,13	22	*177*	62	37	
	Behavior type	**Analysis**															
Hf	cd	rest	uni	0,12	0,16	0,76	*0,446*					0	0	*5*	0	0	
Hf	cd	task	uni	−0,09	0,26	−0,36	*0,717*					0	0	*2*	0	0	
Hf	pa laboratory	reactivity	uni	−0,04	0,22	−0,18	*0,86*					0	0	*1*	0	0	
Hf	pa laboratory	rest	uni	0,09	0,13	0,67	*0,501*					0	0	*3*	0	0	
Hf	pa violent	reactivity	uni	−0,45	0,14	−3,22	*0,001*					0	0	*1*	0	0	3.1
Hf	pa violent	rest	uni	0,12	0,12	1,04	*0,299*					0	0	*2*	0	0	
Hf	pa violent	task	uni	0,21	0,12	1,74	*0,082*					0	0	*3*	0	0	3.2
Hf	pa	reactivity	uni	−0,28	0,14	−2,07	*0,039*					0	0	*3*	0	0	3.1
Hf	pa	rest	uni	0,06	0,12	0,53	*0,596*					0	0	*4*	0	0	

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Table 4 (*continued*)

outcome			test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	Criterion
	Experiment							Variance of random effects					Number of random effects				
Hf	pa	task	uni	−0,07	0,23	−0,31	*0,76*					0,05	0	*2*	0	0	
Hf	psychopathy	task	uni	−0,13	0,09	−1,48	*0,14*					0	0	*3*	0	0	
Hr	aspd	reactivity	uni	0,18	0,32	0,55	*0,584*					0,36	0	*4*	0	0	3.2
Hr	aspd	rest	uni	−0,38	0,58	−0,65	*0,518*					0,57	0	*2*	0	0	3.2
Hr	cd	reactivity	uni	−0,39	0,28	−1,42	*0,157*					0,17	0	*3*	0	0	3.2
Hr	cd	rest	0	0,08	0,05	1,59	*0,111*	0	0	0	0	0	0	*0*	0	0	
Hr	cd	task	uni	−0,14	0,13	−1,07	*0,284*					0	0	*6*	0	0	
Hr	pa laboratory	reactivity	uni	0,18	0,35	0,51	*0,611*					0,32	0	*3*	0	0	3.2
Hr	pa laboratory	rest	uni	−0,21	0,13	−1,62	*0,106*					0	0	*3*	0	0	3.2
Hr	pa laboratory	task	uni	0,51	0,17	2,99	*0,003*					0,08	0	*6*	0	0	3.1
Hr	pa violent	reactivity	uni	−0,15	0,19	−0,78	*0,433*					0,04	0	*2*	0	0	3.2
Hr	pa violent	rest	CCREM	−0,19	0,11	−1,74	*0,083*	0,03	0,02	0	0,03	0,08	12	*17*	4	7	2
Hr	pa violent	task	CCREM	−0,35	0,43	−0,82	*0,413*	0,71	0	0	0	0,72	4	*14*	11	5	2
Hr	pa	reactivity	uni	−0,11	0,08	−1,46	*0,143*					0	0	*7*	0	0	
Hr	pa	rest	uni	−0,15	0,08	−1,78	*0,075*					0,03	0	*10*	0	0	3.2
Hr	pa	task	uni	0,18	0,1	1,83	*0,068*					0	0	*5*	0	0	3.2
Hr	psychopathy	reactivity	CCREM	0,03	0,06	0,52	*0,6*	0,01	0	0,01	0	0,02	12	*60*	31	22	
Hr	psychopathy	rest	CCREM	−0,26	0,07	−3,51	*0*	0,05	0	0	0	0,05	11	*43*	6	33	1
Hr	psychopathy	task	CCREM	−0,15	0,08	−1,85	*0,065*	0,03	0	0	0,03	0,06	9	*93*	22	26	2
Pep	aspd	reactivity	uni	−0,15	0,19	−0,79	*0,428*					0,06	0	*3*	0	0	3.2
Pep	cd	rest	uni	0,13	0,07	1,71	*0,087*					0	0	*4*	0	0	
Pep	cd	task	uni	0,77	0,23	3,42	*0,001*					0	0	*2*	0	0	3.1
Pep	pa violent	rest	uni	0,08	0,21	0,37	*0,709*					0	0	*1*	0	0	
Pep	pa violent	task	uni	0,12	0,12	1,01	*0,314*					0	0	*3*	0	0	
Pep	pa	reactivity	uni	0,08	0,08	0,99	*0,321*					0	0	*1*	0	0	
Pep	pa	rest	uni	0,08	0,08	1	*0,317*					0	0	*1*	0	0	
Pep	pa	task	uni	0,16	0,08	1,99	*0,047*					0	0	*2*	0	0	3.1
Pep	psychopathy	reactivity	uni	0,16	0,4	0,4	*0,689*					0,45	0	*3*	0	0	3.2
Rmssd	pa laboratory	rest	uni	−0,69	0,2	−3,39	*0,001*					0	0	*1*	0	0	3.1
Rmssd	pa laboratory	task	uni	−0,3	0,2	−1,5	*0,133*					0	0	*1*	0	0	3.2
Rmssd	psychopathy	rest	uni	0,54	0,2	2,77	*0,006*					0,21	0	*7*	0	0	3.1
Rmssd	psychopathy	task	CCREM	0,41	0,34	1,21	*0,225*	0,19	0	0	0,1	0,29	2	*17*	5	7	2
Rsa	aspd	reactivity	uni	0,39	0,44	0,88	*0,378*					0,54	0	*3*	0	0	3.2
Rsa	cd	rest	uni	−0,14	0,06	−2,31	*0,021*					0	0	*5*	0	0	
Rsa	cd	task	uni	−0,78	0,23	−3,42	*0,001*					0	0	*2*	0	0	3.1
Rsa	pa laboratory	task	uni	−0,74	0,24	−3,14	*0,002*					0	0	*1*	0	0	3.1
Rsa	pa violent	rest	uni	−0,01	0,21	−0,02	*0,98*					0	0	*1*	0	0	
Rsa	pa violent	task	uni	0,15	0,12	1,18	*0,237*					0	0	*3*	0	0	3.2
Rsa	pa	reactivity	uni	0,09	0,07	1,17	*0,241*					0	0	*2*	0	0	
Rsa	pa	rest	uni	−0,24	0,28	−0,84	*0,401*					0,41	0	*6*	0	0	3.2
Rsa	pa	task	uni	0,15	0,13	1,11	*0,266*					0,03	0	*4*	0	0	3.2
Rsa	psychopathy	reactivity	uni	0,3	0,12	2,49	*0,013*					0,05	0	*6*	0	0	3.1
Rsa	psychopathy	rest	uni	0,04	0,14	0,27	*0,784*					0,11	0	*9*	0	0	
Scl	aspd	reactivity	uni	0,12	0,1	1,27	*0,203*					0,01	0	*5*	0	0	
Scl	aspd	rest	uni	−0,35	0,28	−1,27	*0,202*					0	0	*1*	0	0	3.2
Scl	cd	reactivity	uni	0,04	0,15	0,27	*0,784*					0	0	*1*	0	0	
Scl	cd	rest	uni	−0,48	0,13	−3,78	*0*					0	0	*5*	0	0	3.1
Scl	cd	task	uni	−0,54	0,2	−2,66	*0,008*					0	0	*2*	0	0	3.1
Scl	pa laboratory	task	uni	0,81	0,16	5,21	*0*					0	0	*1*	0	0	3.1
Scl	pa violent	rest	uni	0,38	0,21	1,79	*0,074*					0	0	*1*	0	0	3.2
Scl	pa violent	task	uni	0,45	0,12	3,6	*0*					0	0	*3*	0	0	3.1
Scl	pa	reactivity	uni	0,05	0,09	0,51	*0,611*					0	0	*6*	0	0	
Scl	pa	rest	uni	−0,45	0,19	−2,37	*0,018*					0	0	*1*	0	0	3.1

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Table 4 (*continued*)

outcome	Experiment		test	pooled estimate	se	z	p	study	outcome	experiment	questionnaire	total variance	N study	N outcome	N experiment	N questionnaire	Criterion
								Variance of random effects					Number of random effects				
Scl	pa	task	uni	−0,42	0,13	−3,12	*0,002*					0	0	*2*	0	0	3.1
Scl	psychopathy	reactivity	CCREM	0,12	0,09	1,4	*0,162*	0,01	0	0,04	0	0,05	6	*34*	9	16	
Scl	psychopathy	rest	CCREM	−0,11	0,1	−1,14	*0,253*	0,05	0	0	0	0,05	7	*22*	5	18	
Scl	psychopathy	task	uni	−0,26	0,08	−3,17	*0,002*					0,01	0	*7*	0	0	3.1
Scr	aspd	reactivity	CCREM	−0,15	0,24	−0,61	*0,544*	0,23	0,06	0	0	0,29	5	*13*	8	4	2
Scr	aspd	rest	uni	0,52	0,28	1,84	*0,066*					0	0	*1*	0	0	3.2
Scr	cd	task	uni	−0,22	0,34	−0,64	*0,52*					0,19	0	*2*	0	0	3.2
Scr	pa laboratory	reactivity	uni	0,43	0,19	2,3	*0,022*					0	0	*3*	0	0	3.1
Scr	pa violent	reactivity	CCREM	−0,37	0,28	−1,34	*0,181*	0,08	0	0	0,21	0,29	6	*31*	31	4	2
Scr	pa violent	rest	uni	0	0,32	0	*1*					0	0	*1*	0	0	
Scr	pa violent	task	CCREM	−0,32	0,35	−0,9	*0,37*	0	0	0	0,27	0,27	1	*12*	4	3	2
Scr	pa	reactivity	CCREM	−0,06	0,11	−0,61	*0,543*	0,03	0	0	0	0,03	4	*14*	11	5	
Scr	pa	task	uni	1,21	0,34	3,5	*0*					0	0	*1*	0	0	3.1
Scr	psychopathy	reactivity	CCREM	−0,14	0,09	−1,49	*0,137*	0,11	0	0	0,04	0,15	20	*127*	55	26	
Scr	psychopathy	rest	uni	0,15	0,1	1,57	*0,117*					0	0	*6*	0	0	3.2
Scr	psychopathy	task	CCREM	0,01	0,22	0,03	*0,973*	0,08	0	0,01	0,01	0,1	2	*44*	8	11	

Note. AB = Antisocial Behavior; ASPD = Antisocial Personality Disorder; CCREM = cross-classified random effects model; CD = Conduct Disorder; Criterion = Criterion 1) magnitude exceeding, k > 10, and significant, Criterion 2) magnitude exceeding, k > 10, and non – significant, Criterion 3.1) magnitude exceeding, k < 10, and significant, Criterion 3.2) magnitude exceeding, k < 10, and non – significant; HF = High Frequency; HR = Heart rate; PA = Physical aggression; PEP = Pre Ejection period; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SC = Skin Conductance; SCL = Skin Conductance Level; SCR = Skin Conductance Response; SE = Standard error; Uni = Univariate analysis; Z = z-value.

dataset, and then being carried out for each outcome type, analysis type, behavior type, and experiment type. If any of the categories included ten or less ESs, then none of the analyses were performed. Analyses were conducted in R, using the *metafor* (Viechtbauer, 2010) and the *weightr* package (Coburn and Vevea, 2016). As aforementioned, the presence of publication bias was investigated by carrying out visual inspections of funnel plots, conducting cross-classified Egger regression tests, as well as via the use of selection methods. (available in “S13 Publication bias analysis.R”. Sensitivity analyses were subsequently conducted to check for outlying effects and influential studies (available in “S14 Sensitivity analysis moderators and interactions.R”). The following section presents a summary of these analyses, but an extensive analysis is available in “S15 Publication bias analysis and sensitivity analysis.docx”.

The publication bias analyses indicated that moderate publication bias might be present, due to the fact that highly negative ESs were more likely to be published than highly positive ESs. The funnel plot containing all the ESs was relatively symmetrical, although there were “slightly” more highly negative ESs than there were highly positive ESs. To statistically assess the symmetry of the funnel plots, (cross-classified) Egger regression tests were applied. Significant associations between the ESs and their standard errors were observed for RMSSD and SCR, for reactivity analysis, for antisocial personality disorder, and for the experimental anticipation of noise tasks, cognitive tasks, and guided-imagery tasks. Therefore, it is wholly possible that the overall ESs for these categories are somewhat inflated. Finally, the selection method showed that the largest differences between the observed ESs and those ESs corrected for publication bias (assuming a moderate level of bias) were observed for RSA outcome, in which the corrected overall ESs were reduced from −0.25 to −0.22, and for violent offenders and psychopathy, where the corrected overall ESs were reduced from 0.21 to 0.18 and from −0.18 to −0.16, respectively.

3.6. *Sensitivity analyses*

Sensitivity analyses were conducted to check for outlying effects and influential studies. The standardized deleted residuals (SDR) detected two ESs as potential outliers. The first of these belonged to Serafim et al.’s (2009) study, and had an SDR of −2.05 and an ES of −2.12. The second outlying ES came from Veit’s (2013) study, and had an SDR of −2.06 and an ES of −2.14. Although all the analyses were subsequently repeated without these two ESs, none of the results changed significantly in light of their omission.

Although no study was found to have a Cook distance larger than the cut-off (0.45), there was one study that had a much larger Cook distance compared to all the other studies. Specifically, Serafim et al.’s (2009) study, which included seven ESs, had a Cook distance of 0.18, whereas the average Cook distance for all the other studies was 0.05. In accordance with Cheung and Viechbauer’s (2010) recommendations, we examined the influence of this study but found that it was in line with the established cut-off. All the analyses were subsequently repeated excluding Serafim et al.’s (2009) study, which led to a substantial decrease in some of the ESs (Analyses available in “S14 Sensitivity analysis moderators and interactions.R”). The overall ES reduced from −0.075 to −0.059. Furthermore, it did not result in lower moderator estimates for HR (HR changed from −0.08 to −0.04) during both rest (analysis–rest changed from −0.11 to −0.10) and task (analysis–task changed from −0.06 to −0.01). However, it did change for violent offenders (violent offenders changed from −0.33 (Criterion 1) to −0.19 (Criterion 2) from Brazil (changed from −0.23 to 0.15) in neutral (neutral changed from −0.18 (Criterion 2) to −0.05 (no Criterion)) and positively (positive changed from −0.17 (Criterion 2) to −0.04 (no Criterion)) valenced emotional picture tasks recorded with a pulse oximeter (HR–other changed from −0.29 (Criterion 1) to −0.16 (also Criterion 1)) including a mixed sample (mixed sample changed from −0.18 (Criterion 1) to −0.12 (no Criterion)), and mixed recruitment source (changed from −0.22 (Criterion 1) to −0.13 (no Criterion)).

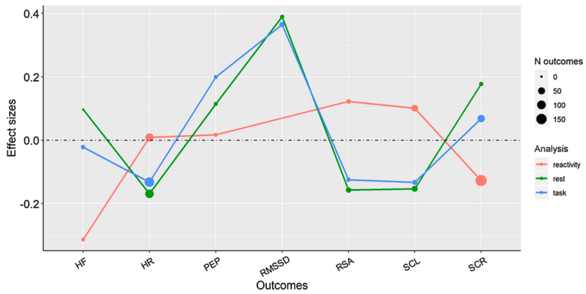

Fig. 1. Interaction between type of analysis and outcome.
Note. Analysis – rest = autonomic measures obtained during a rest phase; Analysis – reactivity = autonomic measures obtained during a rest and task phase and typically calculated as the difference from rest to task; Analysis – task = autonomic measures obtained during a task; HF = High Frequency; HR = Heart rate; N outcomes = Number of outcomes; PEP = Pre Ejection period; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SC = Skin Conductance; SCL = Skin Conductance Level; SCR = Skin Conductance Response.

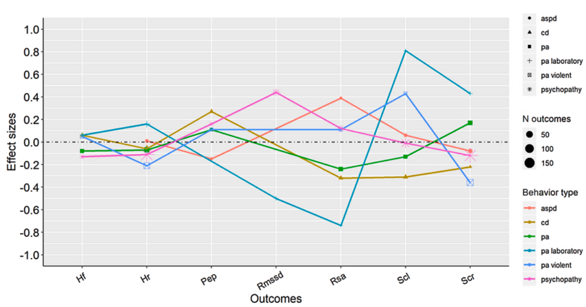

Fig. 2. Interaction between behavior type and outcome.
Note. ASPD = Antisocial Personality Disorder; CD = Conduct Disorder; HF = High Frequency; HR = Heart rate; N outcomes = Number of outcomes; PA = Physical aggression; PA laboratory = Physical aggression in the laboratory; PA violent = physical aggression measured as violent behavior or offenses; PEP = Pre Ejection period; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SC = Skin Conductance; SCL = Skin Conductance Level; SCR = Skin Conductance Response.

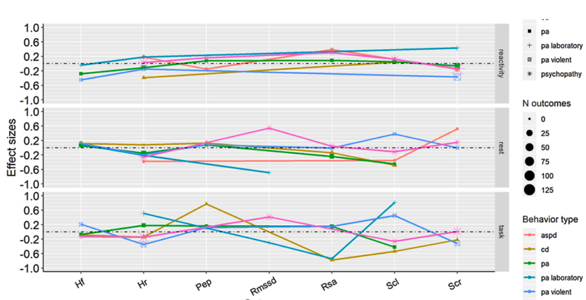

Fig. 3. Triple interaction.
Note. Analysis – rest = autonomic measures obtained during a rest phase; Analysis – reactivity = autonomic measures obtained during a rest and task phase and typically calculated as the difference from rest to task; Analysis – task = autonomic measures obtained during a task; ASPD = Antisocial Personality Disorder; CD = Conduct Disorder; HF = High Frequency; HR = Heart rate; N outcomes = Number of outcomes; PA = Physical aggression; PA laboratory = Physical aggression in the laboratory; PA violent = physical aggression measured as violent behavior or offenses; PEP = Pre Ejection period; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SC = Skin Conductance; SCL = Skin Conductance Level; SCR = Skin Conductance Response.

In accordance with the moderator analyses, the interaction analyses that changed were HR–violent offenders (changed from −0.21 (Criterion 2) to −0.08 (no Criterion)), HR–violent offenders–rest (changed from −0.19 (Criterion 2) to −0.14 (no Criterion)), and HR–violent offenders–task (changed from −0.35 (Criterion 2) to −0.01 (no Criterion)). Notably, HR–rest changed from −0.17 (Criterion 1) to −0.15 (also Criterion 1), which appears to indicate that even with the omission of the outlier, HR–rest is a Criterion 1 ES. Consequently, those outcomes that include any of the categories that markedly changed must be interpreted with care. Finally, Latvala et al.'s study (2015) had an extremely large sample size, and therefore had an important weight on the results of the meta-analysis. Hence, all analyses were carried out again without this study, but this had almost no effect on the results.

Overall, when taking into consideration the adjusted outcomes from Serafim et al.'s (2009) study, the adjusted estimates obtained through the selection method of Vevea and Woods (2005) demonstrated that although the conclusions from the current meta-analysis remained the same, the magnitude of the ESs may have been slightly inflated.

4. Discussion

The meta-analysis presented in this paper updates the earlier seminal meta-analyses of the association between physiological measures of autonomic nervous system (ANS) functioning and severe antisocial spectrum behavior (AB). More specifically, we investigated whether (1) the Proteus phenomenon was evident in resting HR and other ANS measures, (2) whether the relationship between ANS and psychopathy was dependent on subscales of psychopathy questionnaires, (3) which covariates moderate the association between AB and ANS, and (4) whether there was any evidence for the specificity hypothesis in the ANS–AB association, based on different experiments and analysis types. For the purposes of addressing these four research questions and the findings of the meta-analysis, we first discuss the most important results vis-à-vis the specificity hypothesis and moderators, before proceeding to explore the Proteus phenomenon and the psychopathy subscales. Subsequently, the limitations, clinical implications, directions for future research and concluding remarks are delineated.

4.1. Specificity hypothesis

Most importantly, the interaction analyses showed reliable evidence of specific ANS functioning in psychopathy, violent offenders, and to a lesser extent in physical aggression, laboratory aggression and antisocial personality disorder, depending on the type of experiment and analysis (to reiterate, both Criterion 1 and 2 estimates were considered to be reliable results, while Criterion 3.1 and 3.2 were considered to be promising results). Moreover, promising results were found for all behavior types, including for conduct disorder. Specifically, the interaction analyses resulted in bidirectional effect sizes (ESs), which were dependent on experimental task, analysis types, and physiological outcome. Consequently, the specificity hypothesis thus can be said to be relatively tenable, which, in turn, means that physiological measures can potentially be used to differentiate between types of AB, based on experimental task and analysis type (rest, task, and reactivity). The variability in ANS measures appears to be accounted for, at least in part, by the available number of studies, outcomes, experimental tasks and questionnaires. The variability of ANS measures within types of AB indicates that these measures are not always consistently generalizable across types of AB. In accordance with Lorber (2004); Ortiz and Raine (2004) and Portnoy and Farrington (2015), ESs were found to be in the small to medium range, and as being dependent on the interactions studied. Evidence for both publication bias and outlier influence was evident for several variables and covariates, and, as such, warrants further consideration.

A major meta-analysis showed that the ESs for eleven important risk domains for life-course persistent offending varied from .2 to .76 (Assink

et al., 2015). Criminal history, aggression, and alcohol and drug use were all considered to have a large effect on life-course persistent offending (ESs varied between .43–.76) while family, neurocognitive and attitudinal domains were considered to have a relatively small effect (ESs varied between .20–.25). With regards to the present meta-analysis, HR (heart rate) and SCL (skin conductance level) ESs for psychopathy were in some cases higher than the small ESs reported in Assink et al.'s (2015) meta-analysis. RMSSD (root mean square of successive differences) for psychopathy and HR and SCR (skin conductance response) for violent offenders even went so far as to approach the relatively larger ESs observed in Assink et al.'s (2015) study. Comparing the effect sizes obtained in the current study to several other (psychosocial) risk factors often found in the literature, the current meta-analysis stresses the importance of considering ANS measures in AB as well.

For psychopathy, HR (rest and task) and RMSSD (task) were the most reliable predictors. A negative association between psychopathy and resting HR was obtained in the present meta-analysis, thus appearing to indicate that, generally speaking, individuals with psychopathy showed reduced HR in resting conditions. This is in accordance with Portnoy and Farrington's (2015) study in which the psychopathy–rest HR association resulted in the second highest negative ES. Lorber (2004) reported a non-significant result, which was probably due to assigning a zero to several ESs for this particular association. During tasks, in general, individuals with psychopathy showed reduced HR and increased RMSSD. These HR (variability) results appear to be in contrast to the attenuated SC findings found by Lorber (2004) under resting, task and reactivity measures in psychopathy. In fact, Lorber (2004) concluded that SC attenuation was the most simple and compelling result, insofar as most ESs were available for psychopathy.

In the current meta-analysis however, psychopathy–task SCL resulted in a negative association, which, from the perspective of our preset criteria, was a promising result. One potential explanation for this particular finding is that Lorber (2004) categorized electrodermal activity as a whole, whereas the present meta-analysis broke this down further into levels and responses of electrodermal activity. Indeed, the triple interactions displayed an almost perfect inverse pattern for both SCL and SCR in psychopathy when measured during rest, task, and reactivity. This is potentially due to the parameters representing tonic and phasic components, respectively (Boucsein, 2012). Based on these results, we hypothesize that a combination of ANS outcomes and analysis types might be expedient for differentiating individuals with psychopathy from those without psychopathy.

For violent offenders, HR and SCR were most reliable. The hypothesized negative association between ANS and violent offenders was also evident from the moderator analysis. This is, at least in part, in line with the results from Portnoy and Farrington (2015), who reported the largest ESs for resting HR and violent behavior. Lorber (2004) already posited that ANS differentiation was likely to be more straightforward with respect to aggression and psychopathy. Surprisingly, we found similar results, albeit with partly reversed ANS measures for the aggression and psychopathy groups. It is important to note here that we found reliable HR–psychopathy and SCR–violent offender (but also HR–violent offender) associations, whereas Lorber (2004) reported significant HR–aggression and SC–psychopathy associations. The difference between our respective findings may derive, in part, from how Lorber (2004) operationalized aggression, namely as an aggregation including physical aggression, laboratory aggression, and family-related aggression, which is something we specifically sought to disentangle in the present meta-analysis.

Furthermore, Lorber (2004) concluded that HR and SC were non-specific risk factors for aggression and psychopathy, whereas our results indicate that specificity is plausible based on an exhaustive partitioning of (the valence of) experimental tasks and analyses. In addition, Portnoy and Farrington (2015), based on their HR–rest moderator analysis, concluded that the association between HR and AB was unlikely to differ substantially between behavior types. Conversely, our interaction analyses suggests that HR is differentially associated with types of AB, depending on the type of analysis and experiment that is used. Based on these results, we firstly concur with Lorber (2004) that a degree of specificity exists, and secondly provide evidence that it can also be reliably studied across a range of behavior types. Having said, we readily concede that additional research is necessary, and, in this respect, it remains to be established if the specificity of physiological measures for experiments and analysis type is sufficient for distinguishing between behavior types.

This is the first meta-analysis that showed that SCR reactivity resulted in a reliable estimate of antisocial personality disorder, and that we have sufficient data on an ANS outcome for antisocial personality disorder, which was identified as a shortcoming in Lorber's (2004) study. Antisocial personality disorder is still not investigated as frequently as other types of AB, which might stem from the sheer amount of time and effort required to validly assess antisocial personality disorder based on the Diagnostic and Statistical Manual for Mental Disorders criteria (DSM-5, American Psychiatric Association, 2013). The specificity hypothesis might not only be useful to distinguish between, but also within, behavior types. This is illustrated by the differential ANS associations reported for psychopathy and antisocial behavior disorder. These behavior types are represented as synonyms in the classification systems for (mental) health disorders (American Psychiatric Association, 2013; ICD-10, World Health Organization, 2004), but might be physiologically different based on the differential ESs obtained in the current meta-analysis, assuming that the questionnaires used to assess psychopathy are valid. Overall, the careful partitioning of study variables provided useful insights into complex relationships of the ANS-AB association that were obscured from previous meta-analysis.

4.2. Moderators

The moderator analyses indicated that several covariates are important to consider in the ANS–AB association, including method of assessment, outcome type, behavior type, sample description, recruitment source, experiment type, valence type, and country. However, not all moderators exceeded the preset ES magnitude criterion. Notably, analysis type, age, gender, year of publication, design, sample size, comparison type, risk of bias, were found not to moderate the ANS–AB association.

Studies that included a mixture of AB and non-AB samples obtained the highest ESs estimates. It was observed that ESs diminished when studies included more equivalent samples. However, this effect tapered off after excluding an outlier study by Serafim et al. (2009), which serves to illustrate the relative impact of large ESs in the meta-analysis.

The moderator analyses indicated that anticipation of noise tasks, cognitive tasks, competitive reaction time tasks, and fear conditioning tasks resulted in the most reliable ESs between individuals in both AB and non–AB samples. Although other experiments were also shown to induce small to medium ESs (e.g., concealed information, empathy for pain, mental arithmetic, ultimatum game, trust game and VR horror movie), these require further study. Furthermore, the interaction analyses suggested that particular combinations of ANS outcomes and specific experiments resulted in larger differences than others. For instance, the observed differences for fear conditioning tasks, Iowa gambling tasks, and competitive reaction time tasks were most reliable for HR. For SCL, the public speaking tasks resulted in the most reliable estimates. For HR variability measures, the results were mostly promising, but largely dependent on the experimental task, and, as such, requires further investigation.

The moderator analyses indicated that it is reasonable to conclude that HR variability is useful for ES moderation, but that this ultimately requires further examination. For instance, IBI (inter-beat interval) was significant, but this was based on only two estimates. The HR and SC (both SCL and SCR) variables did not moderate the ESs, which is most likely a consequence of both the heterogeneity in the parameters and

specificity found in the interaction analyses.

It is also worthwhile to highlight here that some variables did not moderate the ANS–AB association, namely gender, age and comparison type variables. These findings are in accordance with both Ortiz and Raine (2004) and Portnoy and Farrington's (2015) studies, who revealed that the ANS–AB association was similar for both genders. However, ESs were larger for all male samples compared to samples that included females for HR (and also SCL), but surprisingly not for SCR. The latter might thus be an important predictor for distinguishing between males and females. The age moderator analysis showed that the negative association for HR and SCR was larger in samples with a mean age above 18, while the reverse (i.e., positive association) was true for SCL. This suggests that adolescents with conduct disorder show an attenuated general sympathetic tone. Generally speaking, the insignificant, small estimates found for all moderator analyses might stem from heterogeneity, depending on the specific comparison that is made, which was evident from the interaction analyses.

In the moderator analysis, the analysis type (rest, task, reactivity) variable did not meet the preset criterion. Closer inspection of the interaction analysis suggested that this was probably due to heterogeneity in ESs, insofar as both HR–reactivity and HR–task failed to meet the preset magnitude criterion. This is in contradistinction to Ortiz and Raine's (2004) study, which, for instance, reported a relatively large ES (d = -0.76) for HR–reactivity. One possible explanation for this is that Ortiz and Raine (2004) only included 10 studies on HR–reactivity, whereas 79 outcomes were available in the present meta-analysis. However, the interaction analyses also revealed that reliable estimates can be obtained if the triple interactions are considered.

Interestingly, there were no significant or preset magnitude criterion differences between low- and high-quality studies. The fact that there were neither significant values nor values which exceeded the magnitude threshold is an altogether curious finding, which appears to suggest that the ANS–AB association is also found in less rigorous experiments.

It is notable that some countries with a larger number of estimates obtained relatively small ESs (e.g., USA, Canada, Germany, the Netherlands); this might be the result of a greater range of experiments and ANS measures, which, in turn, balance out the positive and negative estimates. Another explanation could be that more non-significant findings are published in these particular countries.

4.3. Proteus phenomenon

The Proteus effect (i.e., the tendency to report large ESs for significant findings in the initial stages of research) was evident in several ANS parameters (i.e., HR–task and SCR–reactivity), but was not present for resting HR. The moderator analysis suggested that the relationship between AB and resting HR was now relatively stable, based on the 18-year inclusion range within the present meta-analysis. The size of the estimate was confirmed by the moderator analysis, which showed that sample size was not a significant source of moderation, while omitting the study with the large sample size (Latvala et al., 2015) failed to alter the main conclusions of the resting HR results. It is important to note here that in Latvala et al.'s (2015) original study, the authors used a Cox regression with HR groups being categorized into quintiles in order to study the risk of a low HR for AB. Considering sample size, number of estimates, significance, and magnitude, it is therefore reasonable to state that the negative resting–HR estimate remains the best replicated biological correlate for AB (Ortiz and Raine, 2004).

4.4. Psychopathy subscales

Recent research indicates that specificity might be plausible in psychopathy and psychopathology (Babcock and Michonski, 2019; Thomson et al., 2019a; Thomson and Beauchaine, 2019), as well as in specific experiments (Dindo and Fowles, 2011; Fanti et al., 2017; Osumi et al., 2007). Based on the prior finding that primary psychopathy was predicted by co-inhibition while secondary psychopathy was predicted by high parasympathetic reactivity (Thomson et al., 2019b), we expected this to be evident in consistent, criterion exceeding, and bidirectional effect sizes. Ultimately, there was a lack of compelling evidence that the relationship between ANS and psychopathy was dependent on subscales of psychopathy questionnaires. However, we should also note that we were unable to include a sufficient number of ANS parameters for most analyses.

4.5. Limitations

Although the current meta-analysis produced an exhaustive and high-quality investigation of the literature, it is by no means without limitations. First, we were unable to retrieve ESs for several studies, which may have impacted upon the results. Moreover, the number of studies on HR variability and the derived HR variability measures was somewhat limited and thus requires follow-up research. In addition to this, a recent meta-analysis suggested that HR variability and conduct disorder can be used to distinguish between subtypes of conduct disorder, and, in fact, that one can even redefine diagnoses based on these profiles (Fanti, 2018). Unfortunately, we were only able to include a limited number of studies on conduct disorder within our meta-analysis. We did however found differential ESs in psychopathy and antisocial personality disorder which are usually represented as synonyms (American Psychiatric Association, 2013; World Health Organization, 2004).

Furthermore, although it is established that the ANS–AB association can be influenced by several moderators and covariates (Oldenhof et al., 2019), we opted to include bivariate measures in the present meta-analysis. One problem associated with including analyses with covariate-controlled correlates is that they often use different phases of an experiment or control for different covariates. For example, a repeated measures analysis with two habituation trials, two acquisition trials, and two extinction trials is not readily comparable to an analysis that uses only one phase for each. To overcome this problem, two additional columns could be added to the current database to differentiate between the covariates included in the analysis and the number of repeated measures.

In terms of ESs it is also important to address the interpretation of the magnitude of the ESs. An ES of 0.44 (e.g., RMSSD - psychopathy) for instance, effectively means that there is an 83 % overlap between the distributions of the AB group with the control group and only a 62 % chance that a randomly picked individual from the AB group has a higher score than the individual from the non-AB group (Lakens, 2013). The ESs should thus be interpreted cautiously, and illustrates the need to be careful in what conclusions can be derived from the individual studies as even the largest effect sizes show significant overlap between groups.

We are also aware that the classification of categories in the current study is easier for ANS measures than for behavior type. ANS measures consist of formulas that can be applied to the data, while descriptions of AB are typically guided by descriptions and expressed in questionnaires and interviews. We have updated the previous meta-analyses using the search terms from the previous studies with some adjustments to the behavior type categories that we thought were essential to move the field further or needed clarification. A limitation of this strategy is that we did not include other potentially interesting antisocial spectrum behaviors such as theft or pedophilia. Future research could focus on these behaviors and disorders as well.

The inclusion of laboratory aggression as a sub category in the previous meta-analysis (Lorber, 2004) as well as the current meta-analysis is not recommended for future systematic reviews in light of the samples that were included. Only community samples were included in the current meta-analysis for laboratory aggression and it is unclear how community samples are associated to antisocial spectrum behavior. Would future studies include participants with antisocial spectrum behavior in laboratory aggression tasks, this recommendation could be

reconsidered.

The further partitioning of experiments, behavioral tasks, and ANS measures was relatively straightforward, also for aggressive behavior. However, it was not as straightforward for antisocial personality disorder and conduct disorder, as these are formal diagnoses. As we set out to only include validated questionnaires for all included behavior types, we decided to include antisocial personality disorder and conduct disorder only if a formal diagnosis was provided with which we effectively lost information on the more general categories of antisocial and conduct problems. This strategy was not viable for psychopathy as this is not a formal diagnosis, and we were therefore confined to include psychopathy based on validated questionnaires. Future studies could however include the more general categories of antisocial and conduct problems, as this was a limitation in the current study.

Related to this problem is the exclusion of child samples. As most of the authors of the current study work in forensic psychiatry, our main interest was in adolescent and adult samples. However, if future studies could include child samples it might be feasible to map the entire trajectories of ANS-AB development, which was not possible with the current study. In addition, it would also be interesting to test the associations that were found between SCL and different behavior types among children, particularly in light of our recommendation to include longitudinal studies across the lifespan to investigate changes in biological measures.

The publication bias analyses indicated that the ESs for psychopathy and violent behavior were reliable. Even if moderate publication bias was present, the results were still rendered valid, which is to say that the effect sizes met the preset magnitude and significance criteria. The triple interactions indicated that ANS functioning measured during rest and task with HR, SCR, and RMSSD were the strongest predictors for both psychopathy and violent offenders. However, one should be cognizant that the limited number of studies on RMSSD might result in insignificant findings as and when new research emerges. Moreover, both anticipation of noise and cognitive tasks showed inflated standard errors in the Egger test and, as such, must be interpreted with care, insofar as the overall effect sizes for these categories could be somewhat inflated. The conclusions are therefore not equally valid and reliable for all the variables within the database.

4.6. Clinical implications

The results of this meta-analysis clearly indicate a relationship between ANS functioning and AB. Based on these findings, the present meta-analysis has several clinical implications for diagnosis, (dynamic) risk assessment, and treatment interventions. Firstly, as has been pointed out previously in a variety of ways by Iacono (1991), Hare (1969), Lykken (1957); Lorber (2004) and others, ANS measures might be useful for categorizing behavior types, and, arguably, could even be used in differential diagnosis (Campbell et al., 2019). However, not all outcomes, analysis types or experiments are of equal utility for differentiating between types of AB based on ANS specificity (Koenig et al., 2016, 2017). Rather, the results suggest that ANS measures are specifically expedient for distinguishing between psychopaths, violent offenders, physical aggression, laboratory aggression, antisocial personality disorder on the one hand and control samples on the other hand depending on the analysis type and experiment used. A careful consideration of parameters is thus of vital importance for differentiation. For instance, regarding the direction of the ESs, HR was the only positive ES for laboratory aggression irrespective of the analysis type and experiment used, while PEP was the only negative ES for antisocial personality disorder. In addition, for HR reactivity, both antisocial personality disorder and laboratory aggression resulted in positive ESs, while conduct disorder and violent offenders resulted in negative ESs, with no effects being evident for physical aggression and psychopathy. Only fear conditioning tasks and competitive reaction time tasks resulted in positive ESs for HR, while the other experiments either resulted in negative ESs or no effect for HR. These are only a few examples of the different and bidirectional effects for types of AB (see Table 4), and for most HR variability ESs only a limited number of studies were available. However, what it does illustrate is the important distinctions in effect directions. Interestingly, some measures have been studied extensively, and failed to produce an effect. This information in itself can be used for the purposes of differentiating between behavior. In this respect, this meta-analysis provides a first step in terms of targeting the outcomes, analysis types, and experiments that might prove useful for behavior categorization.

In terms of (dynamic) risk assessment, the present results might prove useful in terms of producing an objective and non-intrusive estimate for the risk of future violence. Based on the results, it seems preferable for AB samples to be contrasted with non–AB samples in order to obtain the largest estimates. Interestingly, in light of these results, recent studies indicate that HR and SC might also be useful for dynamic risk assessment in real-life (Looff et al., 2019). The multilevel models used by Looff et al. (2019) showed that HR and SC levels rose significantly in the 20-minute period immediately preceding aggressive behavior. This may potentially provide further opportunities to both design and implement interventions that precede imminent aggressive and violent behavior, which are either aimed at cueing staff members of imminent aggression (Goodwin et al., 2019; Greer et al., 2019) or providing a person displaying AB with real-time information of the bodily signals that precede aggressive behavior (Derks et al., 2019). Overall, based on the current results, it is worthwhile to explore unobtrusive ANS measures as additional risk assessment tools.

Finally, ANS measures might be expedient for determining both if various forms of treatment interventions are suitable for AB and if individuals objectively benefit from treatment. With respect to the latter, HR variability is seen as a marker of physical and mental health (Campbell et al., 2019; Jarczok et al., 2019). Hence, assuming that treatment aims to enhance the cognitive, psychological, and social capacities of persons who display AB, as is the case in forensic psychiatric treatments for instance, then we would expect HR variability indices to improve over the course of treatment. Another important point to consider here is how ANS information can be used to determine which individuals might benefit from treatment interventions. Indeed, Ortiz and Raine (2004) already noted that some individuals might benefit to a larger extent from treatment than others, based on their psychophysiology. The rationale for this is that a low awareness of stress is caused by dysregulation of the ANS. This dysregulation, in turn, effects a person's ability to process emotional cues and physiological stimulation of ANS responses (such as the fight or flight response), which has consequences for the beneficial effects of treatment via punishment and reward (Goozen and Fairchild, 2008). The attenuated ANS responses observed in several ANS parameters can perhaps be used to optimize treatment. We propose that integrating psychosocial and biological information could prove to be useful in determining treatment strategies for individuals with AB (Lorber, 2004; Ortiz and Raine, 2004; Portnoy and Farrington, 2015).

4.7. Directions for future research

Several recommendations for future research are important to consider in relation to ANS measures and behavior types, including longitudinal studies, rigorous reporting, power analysis, (valence of) experiments, and the use of wearables.

Over the course of conducting the present meta-analysis, it became apparent that additional longitudinal research on HR variability measures, antisocial personality disorder, conduct disorder, physical aggression, and laboratory aggression is sorely needed. This was, in part, already pointed out by Lorber (2004) for antisocial personality disorder and HR variability, and the recommendation remains valid all these years later. Longitudinal studies might prove useful for investigating both which ANS profiles in youths increase the risk of AB in adulthood,

and if these ANS profiles are stable or change over time, insofar as changes in ANS functioning have been observed in youths (Harteveld et al., 2021) and adults as well (Jarczok et al., 2019). For instance, Bergstrøm and Farrington (2018) reported that HR at age 18 predicted psychopathy at age 48, while HR at 48 was found not to be a predictor of psychopathy at age 48. It is unclear how these predictors change over time, and what their associations with psychosocial risk factors are. In that regard, it is notable that we were able to include ten longitudinal studies, but this still represents only a fraction of the studies compared to the 91 cross-sectional studies included in the present meta-analysis. This suggests that the results of the current meta-analysis can thus not be used for long term prediction of AB, as most of the studies included were cross-sectional in nature.

Furthermore, it is becoming increasingly more straightforward to report multiple parameters on HR (variability) (Rodríguez-Liñares et al., 2011; Tarvainen et al., 2014) and SC (Bach, 2014; Taylor et al., 2015), which is important given that these estimates can have a significant impact on the conclusions that are drawn. Indeed, in the present meta-analysis, there were several instances in which we were forced to include a zero estimate because the comparison was either not reported or reported as being non-significant. After contacting the authors of the study we were often able to also include the non-significant values, which resulted in ES estimates above the 0.15 magnitude criterion. This effectively means that the current estimates can be considered conservative, which is a problem that was also underscored by Portnoy and Farrington (2015). Arguably, this has a large impact on the estimates included in the present meta-analysis, and testifies to the importance of reporting as much information as possible, as it directly affects the conclusions that can be drawn.

No covariates were included in the present meta-analysis due to methodological considerations, although ESs including covariate corrected measures could be easily implemented within the current database. Several parameters known to influence the ANS–AB association are, among other things, age, respiration rate, sports, smoking (Portnoy and Farrington, 2015), alcohol use (Hu et al., 2017), substance use (Isen et al., 2013) BMI, SES, medication, sports, and IQ (Oldenhof et al., 2019). Extensive reporting on these covariates might provide further insight into the specific dynamics of the ANS–AB association[6]. Currently, the relative dearth of covariate-controlled analyses in various studies prohibited us from including these ESs in the present meta-analysis.

Another important aspect pertaining to reporting the available information is the power of the included studies, which also relates to the replication crisis in the fields of psychology and neuroscience (Botvinik-Nezer et al., 2020; Button et al., 2013; Elliott et al., 2020; Ioannidis and Trikalinos, 2005). The vast majority of the included studies included small samples and did not report power analyses. This has important implications for replication, but also in terms of targeting the specific and precise associations between ANS and AB.

Due to the limited amount of ESs available for several experiments, we were unable to test the interaction effects for all of the included experiments. The moderator analyses indicated that the anticipation of noise and cognitive tasks both exceeded the magnitude threshold. Although non-significant, competitive reaction time tasks, fear conditioning and the trust game also exceeded the magnitude threshold. This suggests that a very specific set of experiments might be most suitable for differentiating between various behavior types on the basis of ANS functioning. Surprisingly, we did not find effects from public speaking tasks, emotional picture tasks, and emotional video tasks in the moderator analyses. Further interaction analyses did reveal however, that theorized effects (i.e., theorized effects in the current meta-analysis are in line with the rationale that experiments are designed to elicit a greater ES during a particular phase of the experiment; see 3.3 Moderator section) resulted in higher ES estimates. The reason for detecting differential effects in the first place might lie in the fact that certain experimental tasks pose differentiating demands on cognitive, emotional, psychological factors that are related to differential brain and physiological connections in the body (Everly (Jr.) et al., 2019). Developmental disorders and trauma can cause dysregulation of the ANS (Goozen and Fairchild, 2008) which might also be visible in the physiological reactions of HR and SC during certain tasks, as our analyses indicated. Future research could focus on including test batteries with large samples to disentangle the specific effects between tasks and AB as the current number of samples for a large number of tasks was rather limited and as a result the analysis of triple interactions with behavior types could not be carried out.

Further interaction analyses indicated that only a small number of studies were available to test the valence hypothesis (supplement 9) across different experiments. One indication that heterogeneity in the outcome type may be responsible for the non-significant results comes from the emotional picture experiments, insofar as the HR estimate for negatively valenced pictures was much larger than the SC outcome estimate. In conclusion, further research is thus needed to test the specific interactions that might be used to support behavior classification. In this respect, the present meta-analysis constitutes only the first step toward developing a pathophysiology taxonomy approach (Spellman and Liston, 2020; Williams, 2016), which might prove useful for future psychopathology classification.

The present meta-analysis provided estimates that showed that cheaper, less sophisticated devices can also be used to capture the ANS–AB association, and, in fact, resulted in the largest estimates. Although we concur with the notion that less sophisticated and often cheaper devices (e.g., wearables in which HR is measured on the body with a PPG sensor) can be used to obtain significant results, the most rigorous and robust devices should be used to obtain the ground truth. It appears that there is a trade-off between usability and reliability that needs to be taken into consideration (Schuurmans et al., 2020; van Lier et al., 2019). Recent studies have shown that cheaper, wearable devices can be used to obtain SC and HR variability measures, but in comparison to the gold standard (de Geus et al., 1995), can only currently be used to obtain fairly similar estimates under resting conditions (Schuurmans et al., 2020), and, moreover, cannot be easily applied to real-life situations in which significant artifacts can arise. However, wearables are increasingly being used in research, and carry great potential to advance neuroscience (Johnson and Picard, 2020), these devices might also provide an opportunity to better understand the relationship between ANS activity and AB.

4.8. Conclusion

The present study updates previous landmark meta-analyses of the association between physiological measures of autonomic nervous system activity and severe antisocial spectrum behavior. It is the first meta-analysis to include a quantitative analysis of HR variability measures, as well as encompassing an exhaustive partitioning of variables relevant to the ANS–AB association, including various experimental tasks. Results indicate that effect sizes are heterogeneous and bidirectional. The careful partitioning of relevant factors sheds light on the complex associations that were obscured in previous meta-analyses. There are three major conclusions to be drawn from this meta-analysis:

(1) Resting heart rate remains the best replicated biological correlate of AB.
(2) The results indicate that the specificity hypothesis is currently tenable, which is to say that physiological measures might be used to differentiate between (and within) types of AB based on

[6] For instance, the Latvala studies (Latvala et al., 2015, 2016) reported very small bivariate ESs for the ANS–AB association. However, the original study used a survival analysis which resulted in larger estimates, while the associations were stronger if covariates were included.

the experimental task and analysis type (rest, task, and reactivity). The variability in ANS measures appears to be accounted for, in part, by the available number of studies, outcomes, experimental tasks and questionnaires. The variability of ANS measures within types of AB indicates that these measures are not always consistently generalizable. It remains to be seen if the specificity of physiological measures for experiments and analysis type is of sufficient magnitude to distinguish between behavior types. Specificity is currently mostly evident in psychopathic and violent offender samples, as well as in physical aggression, laboratory aggression and antisocial personality disorder. The differential effects found for psychopathy and antisocial personality disorder also point to differential physiological functioning in AB that is typically used as a synonym.

(3) The data suggests that ANS measures might be useful for a future taxonomy of specific types of pathophysiology. In particular, we hypothesize that ANS measures might be expedient for classification of psychopathology based on pathophysiology. Integrating rigorous laboratory research with real-life measures might also provide us with additional opportunities for further theory development, diagnosis, (dynamic) risk assessment, and treatment of AB.

Overall, the results of the present meta-analysis indicate that in addition to other psychosocial risk factors for AB, it is also important to consider ANS measures. Furthermore, the present study addresses several issues related to the complexity of research examining the relationship between ANS and AB, and demonstrates that we need a collection of specific types of experiments, sample comparisons, analysis types, and ANS outcomes to optimize the models that describe the pathophysiology of types of AB.

Author note

All data is available at the Open Science Framework (OSF): https://osf.io/5z4u7/?view_only=15fed6193b424b2f8ffd19b8db090f15.

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Declaration of Competing Interest

The authors have no conflict of interest to declare.

Appendix A

Supplements, databases, scripts and analyses can be retrieved from the Open Science Framework (OSF): https://osf.io/5z4u7/?view_only=15fed6193b424b2f8ffd19b8db090f15.

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