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Authors

Alen, Nicholas V

Shields, Grant S

Nemer, Adele

et al.

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Review article

A systematic review and meta-analysis of the association between parenting and child autonomic nervous system activity

Nicholas V. Alen [a,b], Grant S. Shields [a,c], Adele Nemer [a], Indira A. D'Souza [a], Marcela J. Ohlgart [a], Camelia E. Hostinar [a,*]

[a] Department of Psychology, University of California, Davis, USA
[b] Department of Biological and Clinical Psychology, University of Trier, Germany
[c] Department of Psychological Science, University of Arkansas, Fayetteville, AR 72701, USA

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ABSTRACT

Parental socialization may influence the development of children's autonomic nervous system (ANS), a key stress-response system. However, to date no quantitative synthesis of the literature linking parenting and child ANS physiology has been conducted. To address this gap, we conducted a pre-registered meta-analysis. A systematic review of the literature identified 103 studies (n = 13,044 participants) with available effect sizes describing the association between parenting and either parasympathetic nervous system (PNS) or sympathetic nervous system (SNS) activity in children. The overall analysis revealed non-significant associations between parenting and child ANS physiology on average. However, moderation analyses revealed a positive association between more positive parenting and higher resting PNS activity that was stronger when a study was experimental rather than correlational, and when the sample included children with a clinical condition. In conclusion, well-controlled experimental studies show that positive parenting is associated with the development of higher resting PNS activity, an effect that may be stronger among children who are at elevated developmental risk.

1. Introduction

Parents play a critical role in shaping children's affect and self-regulatory abilities, especially during the earliest years of life (Grusec and Davidov, 2010; Thompson, 2014a). For example, parenting that is characterized by sensitivity, consistency, and developmentally appropriate levels of control is associated with better self-regulation in children (Feldman, 2012; Thompson and Meyer, 2007). One proposed mechanism thought to underpin links between parenting and these beneficial child outcomes is through potential effects on the autonomic nervous system (ANS; Calkins et al., 2013; Miller and Hastings, 2019; Propper and Moore, 2006; Thompson, 2015), a key stress-response system that has been implicated in both health and social-emotional outcomes (Beauchaine et al., 2013; Miller, 2018; Thayer et al., 2010). In support of this idea, a number of studies have found links between parenting and child ANS physiology, and these studies have been the subject of various high-quality narrative reviews (Chiang et al., 2015; Propper and Holochwost, 2013; Quigley and Moore, 2018). However, there is substantial heterogeneity in both results and study methodology in this literature. To address this heterogeneity and provide a quantitative estimate of the overall strength of the association between parenting and child ANS physiology, we conducted a pre-registered meta-analysis.

1.1. The autonomic nervous system

The autonomic nervous system (ANS) is an expansive network of efferent and afferent nerves that work in conjunction with the central nervous and endocrine systems to adaptively respond to changes in the environment and maintain the body in dynamic, context-appropriate homeostasis (Propper and Holochwost, 2013). The ANS is comprised of two branches: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). In general, the SNS is involved in mobilizing the body to confront a threat or challenge (i.e., "fight-or-flight"), such that stressors increase sympathetic output. Conversely, the PNS facilitates return to calm (i.e., "rest-and-digest"). During moments of relative rest, cardiovascular activity is under constant influence by the PNS, which actively reduces heart rate through innervation of the

* Correspondence to: Center for Mind and Brain, University of California-Davis, 202 Cousteau Place, Suite 170, Davis, CA 95618, USA.
E-mail address: cehostinar@ucdavis.edu (C.E. Hostinar).

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sinoatrial node, termed the heart's *pacemaker* (Beauchaine, 2001). As such, the initial physiological response to perceived challenges in the environment is a reduction in parasympathetic modulation of the heart, referred to as parasympathetic, or vagal, withdrawal (Porges, 2007).

Several theoretical models have been proposed to account for observed associations between ANS functioning and well-being. For example, polyvagal theory proposes that the highly myelinated ventral vagus nerve plays a central role in dynamic physiological responding to stimuli, which allows for flexibly attending to subtle social environmental cues among species with relatively high metabolic demands (Porges, 2007). While polyvagal theory has contributed much to our appreciation for the important dynamic interplay between the parasympathetic and sympathetic branches of the nervous system in social situations, its evolutionary and anatomical tenets have been challenged (Grossman and Taylor, 2007). Another theoretical perspective, the neurovisceral integration model, posits that higher and lower order neural and endocrine systems organize the body for goal-directed behavior partially through sympathetic and parasympathetic nerves, which innervate the heart (Thayer and Lane, 2000). According to this perspective, measurements of autonomic modulation of the heart can provide noninvasive insight into individual differences in cognitive and self-regulation abilities (Appelhans and Luecken, 2006; Thayer, 2006). Grossman and Taylor (2007) theorized that respiratory sinus arrhythmia (RSA), a common index of PNS modulation of cardiac activity, can reflect general physiological and neural plasticity to changing environmental needs (i.e., flexibility), as information from multiple bodily systems is integrated to enhance metabolic efficiency.

1.1.1. Measures of the parasympathetic nervous system

Several biomarkers exist that are regularly used to measure PNS activity. Most biomarkers indirectly assess parasympathetic modulation of cardiovascular activity via heart rate variability (HRV), which is an index of the beat-to-beat changes in heart rate over time (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Three commonly used measures of HRV have been well validated to reflect PNS modulation of the heart through the vagus nerve (Laborde et al., 2017). First, high-frequency heart rate variability (HF-HRV) is a measure of variations in HR associated with normative increases and decreases in HR during inhalation and exhalation, respectively (Laborde et al., 2017). When HF-HRV is partitioned into developmentally appropriate frequencies, it is a relatively pure approximation of PNS influence over cardiac activity (Shader et al., 2018). Second, root mean squared successive differences (RMSSD), and third, RSA—derived through the peak-to-valley method—are time-series measures that are highly correlated with HF-HRV (Grossman et al., 1990). A fourth, less common, index of PNS modulation of the heart is the cardiac vagal index (CVI), which has been validated using pharmacological blockade (Toichi et al., 1997).

Resting measures of HRV are positively associated with emotion and self-regulation (Appelhans and Luecken, 2006), executive function (Gillie et al., 2015), and better physical health (Alen et al., 2020c; Thayer et al., 2010). However, some studies have found negligible associations between HRV and psychosocial functioning (Kluttig et al., 2010; Sloan et al., 2017). Some recent evidence suggests the relation between resting HRV and social-emotional outcomes may not be linear (Miller, 2018).

HRV change in reaction to challenge or threat (i.e., vagal withdrawal) represents a physiological mobilization of resources important in attending to changing environmental demands, and it has also been associated with better social and emotional functioning (Calkins and Keane, 2004; Miller et al., 2015; Thompson et al., 2008). However, the appropriateness of vagal withdrawal is context-dependent (Hastings et al., 2014a, 2014b), and in some instances (e.g., normatively non-threatening situations) may reflect reactivity that is ill-suited to situational demands (Beauchaine et al., 2007; Davis et al., 2016; Hastings et al., 2008a, 2008b).

1.1.2. Measures of the sympathetic nervous system

The activity of the sympathetic nervous system can be measured using various indices. Pre-ejection period (PEP) is an index of the mechanical aspects of the heart (i.e., contractility; Sherwood et al., 1990). PEP is derived from cardiac impedance data, with shorter PEP representing more SNS influence over cardiac activity (Schächinger et al., 2001). The SNS can also be indexed through skin conductance level (SCL), which measures electrodermal activity related to sympathetic innervation of sweat glands, and is positively related to SNS activity (Beauchaine, 2001). Additionally, salivary alpha amylase (sAA) has been suggested to reflect primarily sympathetic activity (Nater et al., 2007). However, production of saliva is influenced by both sympathetic and parasympathetic innervation of salivary glands, and therefore sAA levels do not purely index SNS activity (Rohleder and Nater, 2009). Toichi et al. (1997) also developed a validated, though less common, measure of SNS activity, termed the cardiac sympathetic index (CSI).

Increased sympathetic activity at rest is associated with cardiovascular risk factors (e.g., hypertension; Mancia and Grassi, 2013), making resting SNS activity a valuable clinical marker of cardiovascular health and risk of mortality. Research has also linked increased resting sympathetic activity to increased behavioral problems in preschoolers (Esposito et al., 2016), though studies have also found the opposite relation (Beauchaine et al., 2013), or no relation (Nelson et al., 2021). Sympathetic reactivity has been associated with health and psychosocial functioning, with elevated reactivity predicting cardiovascular risk (Treiber et al., 2003) and reduced reactivity predicting increased substance use (Brenner and Beauchaine, 2011), poorer emotion regulation abilities (Stifter et al., 2011), and increased aggression (Posthumus et al., 2009) in children. It should be noted that, like PNS reactivity, sympathetic reactivity must be interpreted in the context of task demands.

1.2. Parental socialization and the autonomic nervous system

Parental socialization of children's affect and self-regulatory abilities is a complex and intricate process that involves several aspects of both the parent's behavior towards the child and the parent-child relationship (Grusec, 2011; Thompson and Meyer, 2007). Established theoretical perspectives suggest that a child's ability to regulate emotional states and adaptively respond to changing environmental demands is most likely influenced by parenting in three domains: *protection*, *teaching*, and *control* (Grusec and Davidov, 2010). More specifically, parents can help facilitate the development of affect and self-regulation through: (1) sensitive responding to distress, (2) socialization of emotional understanding, and (3) consistent and developmentally appropriate discipline (Grusec, 2011; Thompson, 2015). In addition, a child's ability to practice self-regulatory skills early in life depends on internalized conceptualizations of safety and dependability in close relationships (Waters et al., 2010).

The development of affect and self-regulation skills is aided by parental sensitivity and support, as parents engage in efforts to protect infants and children from both internal and external sources of threat (Grusec, 2011). In infancy and early childhood parents act as primary sources of emotional and affective regulation (Thompson, 1994). Without adequately developed cognitive and physiological mechanisms for self-regulation, infants and young children rely on caregivers to help them recover following affective arousal. Through guided socialization processes, parents' efforts to help children manage their internal affective state may over time lead to the entrainment of cognitive strategies and physiological response patterns critical in the development of adaptive emotion and self-regulatory skills (Thompson, 2015).

Through social buffering processes, parental support can help maintain infant and child endocrine and sympathetic responses to internal and external stimuli within moderate, more manageable, levels (Hostinar et al., 2014). At the same time, parental support leads to increased reliance on parasympathetic withdrawal as a physiological

mechanism of engagement (Calkins et al., 2008; Calkins and Keane, 2004), which is thought to be a more adaptive physiological response pattern (Beauchaine, 2001). Over time, parental support may lead to the programming of stress physiology that is more moderate in reactivity, more flexible, and therefore better able to respond appropriately to changing environmental demands (Flannery et al., 2017; Miller et al., 2011). As such, sensitive and supportive parenting may contribute to the development of adaptive physiological regulatory systems. Harsh or abusive parenting, on the other hand, may pose a double risk of (1) an absence of expected caretaker warmth, and (2) an increased level of threat and environmental unpredictability. Children who develop in abusive or unpredictable interpersonal environments may develop physiological reactivity patterns that are evolutionarily adaptive for the short term (e.g., hyper-vigilant), with long term costs to health and well-being (Blair and Raver, 2012; Del Giudice et al., 2011; Repetti et al., 2002).

Child autonomic physiology may also be influenced by parental efforts to teach children emotional understanding. More specifically, a parent's open discussion of, and measured reactions to, their child's and their own emotional expressions can help engender the child's understanding of emotions, and ability to effectively identify and moderate them (Eisenberg et al., 1998). Conversely, parental invalidation of child negative emotions can lead to rigid or avoidant cognitive and physiological emotion response patterns (Crowell et al., 2013). Differences in emotion coaching, or a parent's contribution to a child's understanding of their own affective state, can have substantial influence on the child's ability to regulate their own emotions, which may manifest itself in physiological systems implicated in affect and self-regulation (e.g., ANS physiology; Thompson, 2014b).

Children also benefit from developmentally appropriate levels of control and discipline. Parental behavioral control, that includes active monitoring, clear and realistic expectations, and developmentally appropriate involvement of the child in the decision-making process, guides the child towards practicing self-regulation (Barber, 1996; Grusec, 2011), which may become internalized and reflected at a physiological level (Propper and Holochwost, 2013; Quigley and Moore, 2018). Parental control that is strict, overcontrolling, or that leverages the personal relationship (e.g., psychological control) can undermine autonomy and rob the child of chances to engage in independent self-regulation (Barber and Harmon, 2002; Hastings et al., 2008a, 2008b). Furthermore, harsh or inconsistent discipline can be a source of threat to the child (Morris et al., 2007), which may lead to the canalization of physiological and affective response patterns that are more vigilant (i.e., reactive) and more difficult to regulate (Gunnar and Cheatham, 2003; Repetti et al., 2002).

Lastly, these parental socialization processes share a bidirectional association with the child's style of attachment to the parent. According to attachment theory, infants and children incorporate characteristics of their caregiver's behavior patterns into an internal working model, or a dynamic representation of how their caregiver, and the larger social world, will respond to their emotional signals (Bowlby, 1969; Thompson, 2008a). When a child believes the caregiver will be responsive to distress signals, this facilitates trust in the interpersonal environment and can give rise to a developmentally appropriate balance between interdependence and independence (Thompson, 2008b). Children who are securely attached to their caregiver can thus feel safe using their caregiver as a secure base from which to explore the world and returning to them to seek comfort in moments of distress (Ainsworth et al., 1974; Cassidy, 1994). This means that a secure attachment enables the caregiver to act as an effective buffer in times of acute stress (Hostinar, 2015; Thompson et al., 2008), while also permitting the growing child to practice the self-regulatory processes essential in the development of a flexible physiological approach and avoidance system (e.g., the ANS; Thompson, 2015).

1.3. Moderators

Theoretical and conceptual models, as outlined above, predict parent socialization effects on child ANS physiology. However, narrative reviews of the literature have revealed mixed results (Chiang et al., 2015; Propper and Holochwost, 2013; Quigley and Moore, 2018). It is therefore important to consider participant-level and study-level characteristics that may serve as moderators (see Table 1 for a list of moderators) and may explain this heterogeneity in research findings.

First, *parenting measure valence*, that is whether a parenting measure reflects a positive or a negative parenting behavior, may influence the strength of the association between parenting and child ANS physiology. Negative parenting behaviors (e.g., harshness, aggression) have been proposed to be more influential on child outcomes, as compared to positive parenting behaviors (e.g., warmth; Baumeister et al., 2001). Some evidence also suggests that measures of negative parenting behavior may be more accurate (i.e., have less measurement error). For example, negative parenting behaviors have been found to exhibit stronger correlations between parent-reported and observed measures (Hendriks et al., 2018). Indeed, meta-analyses have found stronger associations between parenting and child behavioral outcomes (e.g., externalizing behavior problems) when the parenting measure is negatively valenced (Hoeve et al., 2009; Johnson et al., 2017). It is therefore possible that the relation between parenting and child ANS physiology will be stronger when a negatively-valenced parenting measure is used.

The strength of the association between parenting and child ANS physiology may also depend on aspects of the general study design, namely, whether a study is *experimental* vs. correlational, or *longitudinal* vs. cross-sectional. Most studies use correlational designs, potentially due to the greater ease and reduced costs. However, true causal effects are more likely to be revealed through experimental designs, whereby random assignment to condition controls for individual differences and unmeasured covariates (Collins et al., 2000). A similar assertion could be made for longitudinal designs, which allow for more precise testing of directional associations. As compared to cross-sectional correlational studies, experimental and longitudinal studies likely require more time, more funding, and more rigorous study planning, and may therefore be better suited to isolate true parenting effects. Based on this, effects of parenting on child ANS physiology may be larger among studies that use experimental, as compared to correlational, and longitudinal, as compared to cross-sectional, designs.

We identified three sample-level characteristics that may moderate the association between parenting and child ANS physiology: *clinical sample* vs. non-clinical sample, *sample age*, and *sample percent female*. Social buffering models propose that positive parenting may be most beneficial within adverse contexts, where parental support can weaken

Table 1
Moderators that may explain variability in links among parenting and child autonomic physiology.

Moderator	Description
1. Parenting measure valence	Whether a parenting measure reflects positive behaviors (e.g., encouragement, warmth) or negative behaviors (e.g., harshness, aggression).
2. Experimental design	Whether the study is experimental (i.e., parenting is intervened on) or correlational (i.e., parenting is studied observationally).
3. Longitudinal design	Whether the correlation for the effect size is drawn from longitudinal or cross-sectional data.
4. Clinical sample	Whether the study was conducted with a clinical sample or non-clinical sample (see main text for list of clinical conditions, e.g. ADHD, prematurity)
5. Sample age	Mean age of the sample for the effect size of interest
6. Sample gender distribution	Percentage of females in the sample
7. Parental presence during the ANS recording	Whether the parent was present during the ANS recording
8. Reactivity formula	Whether ANS reactivity was calculated using residualized change scores or raw change scores

the effects of adversity on child outcomes (Hostinar, 2015). Parenting effects may therefore be stronger among children exposed to higher levels of adversity, such as those with a clinical diagnosis.

Sample age may also moderate the relation between parenting and child ANS physiology. Past findings have revealed consistent developmental trajectories of both PNS and SNS activity. Resting PNS activity tends to increase and resting SNS activity tends to decrease across infancy and childhood, and both reach moderate stability in adolescence (Matthews et al., 2002; Rigterink et al., 2010; Wagner et al., 2021). PNS reactivity decreases from infancy to adolescence, and SNS reactivity increases (Hinnant et al., 2011; Shader et al., 2018). Moderate rank-order stability is found for resting measures, but less so for measures of reactivity, though there may be less stability during early developmental periods (Calkins and Keane, 2004) as compared to later periods (e.g., adolescence; Salomon, 2005). Though it is not clear whether parenting and child ANS physiology will be more strongly correlated among younger or older youths, this is an important moderator to consider.

Gender may also moderate the association between parenting and child ANS physiology. Robust sex differences in ANS activity have been observed, with greater parasympathetic and less sympathetic activity at rest observed in females compared to males (Dart et al., 2002; Koenig and Thayer, 2016). In addition, differences in parenting experienced by boys and girls has been found, with some evidence that girls experience both more support and more control (Van Lissa et al., 2019). One study found that more parental support and less negative interactions with parents were associated with higher resting PNS activity, but only in girls (Van der Graaff et al., 2016), though other research has found no differences between boys and girls in the relation between parenting and ANS physiology (e.g., PNS reactivity; Hastings et al., 2008a, 2008b). Differences in parent socialization effects on behavioral outcomes have also been demonstrated. For example, parent socialization of emotion regulation abilities has been found to be stronger among boys (Rueth et al., 2017), though the opposite has also been observed (Van Lissa et al., 2019). Given the gender/sex differences in both parenting and ANS physiology, and the mixed evidence for moderation by gender, this is another important potential moderator to investigate.

Two additional methodological characteristics are worth considering in explaining heterogeneity in the literature on parenting and child ANS physiology: (1) parent presence during ANS recording, and (2) whether ANS reactivity scores are calculated using residualized change scores or raw change scores. First, having a parent present during the ANS recording may add complexity to our ability to test relations between parenting and child ANS physiology. This is because characteristics of the parenting or parent-child relationship may influence the context in which ANS physiology is measured (e.g., a resting measure in the presence of an aggressive parent may be stressful and therefore not reflect a true 'trait-like' measure). It is therefore possible that the relation between parenting and child ANS physiology will be moderated by parental presence during the ANS recording.

Lastly, it is important to consider how ANS reactivity scores are calculated. Two common methods for calculating ANS reactivity are (1) raw change score (i.e., difference score), which is the simple arithmetic difference between resting levels and task levels, and (2) residualized score, which comes from the residuals of an OLS regression after regressing task levels onto resting levels. Residualized scores, therefore, "correct" for resting levels, whereas raw change scores do not (Burt and Obradović, 2013). This is important when considering the Law of Initial Values (Jamieson, 1995), which describes a common correlation between resting levels and reactivity. This issue can be corrected for by including resting levels as a covariate in multivariate analysis. In addition, the use of latent change or growth methods are now recommended over either raw change or residualized scores (Burt and Obradović, 2013; Miller, 2018). However, for a correlational meta-analysis only unadjusted, bivariate correlations will be used. It is therefore important to test whether the relation between parenting and child ANS reactivity will be moderated by whether a *residualized* or a raw change score was used.

1.4. Current study

Parental socialization theory proposes links between parenting experienced during early life and individual differences in children's affect and self-regulation, which may be reflected in differences in autonomic physiology. In order to synthesize the literature, we conducted a meta-analysis of the association between parenting (or related constructs) and child ANS physiology. In the current study, we had two goals: (1) to quantify the magnitude and direction of the association between parenting experienced during early life and measures of child parasympathetic and sympathetic nervous system physiology, and (2) to investigate sample-level and study-level characteristics that might explain variability in findings across studies. Based on the literature reviewed above, we hypothesized that the relation between parenting and child ANS physiology would be stronger when (1) the parenting measure is negatively valenced, (2) the study is experimental, (3) the study is longitudinal, (4) the participant sample is clinical, (5) the parent is not present during the ANS recording, and (6) reactivity is calculated using residualized change scores, as opposed to raw change scores. In addition, we hypothesized that *study mean age*, and *percent female* would moderate the correlation between parenting and ANS physiology, though notably we did not have a directional hypothesis for these two moderators.

2. Methods

2.1. Literature search and study screening

Search strategy and study methods were pre-registered at the International Prospective Register of Systematic Reviews (PROSPERO; Alen et al., 2020b). Study reporting adheres to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines (Page et al., 2021). To identify possible studies for inclusion we searched for relevant publications through two databases: PubMed and PsychINFO. Searches were conducted initially in April 2020 and then during a second time point in February 2021, using the following search string, with asterisks indicating a wildcard symbol that stands for any one or more characters that may follow the provided word stem:

(parent* OR maternal OR paternal OR mother OR father OR attachment OR maltreatment OR abuse OR neglect) AND ("autonomic nervous system" OR parasympathetic OR sympathetic OR "heart rate variability" OR "respiratory sinus arrhythmia" OR "heart period variability" OR vagal OR vagus OR "pre-ejection period" OR "skin conductance" OR "salivary alpha amylase").

This search resulted in the identification of 2486 studies, from which 307 duplicates were removed (2179 unique studies; see Fig. 1 for PRISMA flow chart). An additional 11 studies were identified through hand searching reference lists from relevant literature reviews and inquiries within our research network. Screening procedures were conducted using the web-based platform Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia). Titles and abstracts for these 2190 studies were screened for relevance twice independently by a team of six trained research assistants (RAs: Cohen's kappa range among RAs:.71–0.93); disagreements, which occurred for 4.8 % of articles, during this process were resolved by the lead author (NVA). Title and abstract screening resulted in the exclusion of 1831 irrelevant studies. The lead author independently reviewed 10 % (k = 183) of these excluded studies and found no errors (i.e., zero incorrectly excluded studies). For the remaining 359 studies, full texts were retrieved, and screening of Methods sections was then conducted by NVA to make inclusion decisions.

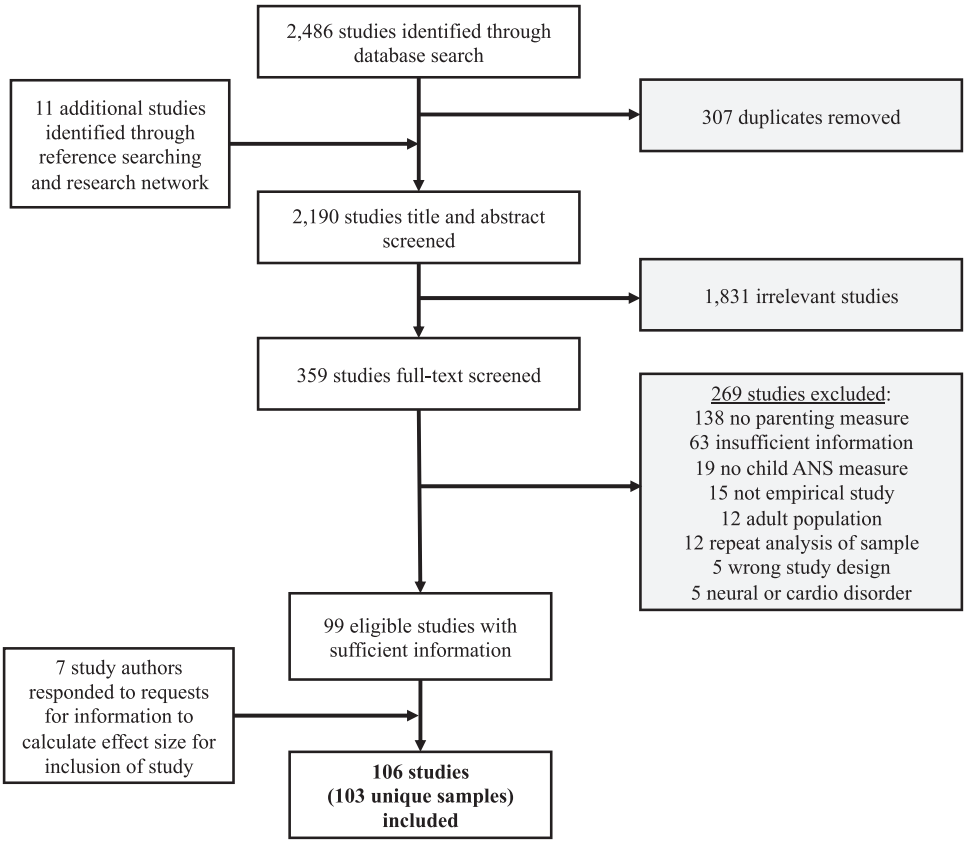

Fig. 1. PRISMA flow chart.

2.2. Inclusion and exclusion criteria

For a study to be included in the analysis the following had to true:

- *The study must be peer reviewed, written in English, and present unique data*. Studies were limited to peer-reviewed articles to increase confidence in the quality of study methodology. Concerns of publication bias were low considering a large portion of studies (63.1 %) were not explicitly designed to test direct relations between parenting and child ANS physiology, and instead collected both variables in order to test a different hypothesis (e.g., 30.1 % of studies tested ANS physiology as a moderator of the relation between parenting and some child outcome). If more than one article described the same sample and data, the article with the most information (e.g., more participants, more measures) was used ($m = 4$). If neither article presented more information, then the earliest published article was used ($m = 8$). If two studies used the same sample and each described different data (e.g., different ANS physiology measures), then all unique data were used and data were grouped together into a single study for analysis ($m = 3$).
- *The study must contain either a measure of parenting, a measure of the parent-child relationship, or a parenting intervention*. Given insufficient prior knowledge on which aspects of parental socialization are most strongly associated with child ANS physiology, the current synthesis adopted an inclusive approach and examined any study that included a measure of parenting or parent-child relationship quality. A parenting measure was considered acceptable if it (1) could be placed conceptually within a spectrum ranging from negative (e.g., harsh, aggressive, abusive) to positive (e.g., warm, sensitive, supportive) parenting, or (2) if it described a parent's socialization of the child's emotions (e.g., emotion coaching), or (3) if it described a manner of parental control or discipline. Documented history of maltreatment was included as a parenting measure if the study explicitly stated that the perpetrators of maltreatment were all parents or primary caregivers. Acceptable measures of the parent-child relationship included measures of attachment security and measures of subjective quality of the relationship. Parenting interventions were only included if they were specific to the parent (i.e., did not involve child-directed intervention).
- *The study must contain at least one of the well-validated single branch (PNS or SNS) measures of the infant/child/adolescent ANS physiology described in the introduction* (e.g., *HRV, PEP, SCL)*. For comprehensiveness, salivary alpha amylase (sAA) was also included, although this measure has been described as reflecting both sympathetic and parasympathetic influence (Rohleder and Nater, 2009). Sensitivity analyses were conducted to test models with and without this additional biomarker.
- *The study article or study authors must provide sufficient information for the calculation of an effect size*. If a study article did not provide sufficient information for the calculation of an effect size, requests for information were sent to the study corresponding author and principal investigator.
- *For studies of ANS reactivity, reactivity must be calculated as either a raw change score* (e.g., *task level minus resting level) or a residualized change score (task level controlling for resting level)*. If a study provided only an effect size for the association between parenting and ANS physiology *during a task* without baseline correction, requests were sent to the corresponding author for effect sizes that use either change scores or residualized change scores.

Studies were excluded if any of the following were true:

- The sample included participants that were older than 18 years of age at the time of ANS physiology measurement.
- The sample included participants with a neurological or cardiovascular disorder.

- The ANS physiology measure was obtained in the hospital shortly after birth (less than 1 week after birth).
- The ANS physiology measure was obtained at an earlier time point than the parenting measure. This exclusion criterion was implemented in order to support the conceptual framework of parenting influencing the development of the child's ANS. If a study presented both effect sizes that met inclusion criteria (e.g., cross-sectional or parenting measured before child ANS) *and* effect sizes that failed to meet inclusion criteria, the study was included but only the acceptable effect sizes were used in meta-analysis.

Full text screening resulted in $m = 153$ eligible studies, of which $m = 99$ studies provided sufficient data for the calculation of an effect size (Abaied et al., 2018; Bell et al., 2018; Blandon et al., 2010; Borelli et al., 2017; Bosquet Enlow et al., 2014; Brown, 2007; Bubier et al., 2009; Burgess et al., 2003; Cai and Tu, 2020; Chen et al., 2015; Cho and Buss, 2017; Clark et al., 2016; Conradt and Ablow, 2010; Creaven et al., 2014; Creavey et al., 2018; Cui et al., 2019; Decarli et al., 2020; Diamond et al., 2012; Dixon-Gordon et al., 2020; Duprey et al., 2021; Eiden et al., 2018; Erath et al., 2009; Fagundes et al., 2012; Feldman, 2015; Feldman et al., 2010, 2013, 2014; Feldman and Eidelman, 2007; Fletcher et al., 2017; Fox et al., 2019; Gilissen et al., 2007, 2008; Giuliano et al., 2015, 2015; Grady and Callan, 2019; Graham et al., 2010; Graham et al., 2017; Hagan et al., 2016; Ham and Tronick, 2009; Han et al., 2020; Hastings et al., 2008a, 2008b, 2014a, 2014b, 2019a, 2019b; Hinnant et al., 2015, 2016; Holochwost et al., 2014, 2018; Huffman et al., 2020; Izard et al., 1991; James et al., 2017; Kaplan et al., 2008; Katz et al., 2020; Katz and Gottman, 1997; Kennedy et al., 2004; Kochanska et al., 2017; Li et al., 2019; McQuade et al., 2021; Miller et al., 2013; Miller-Slough and Dunsmore, 2020; Miskovic et al., 2009; Monti et al., 2014; Nelson et al., 2017; Oshri et al., 2020, 2021; Paret et al., 2015; Partington et al., 2018; Perlman et al., 2008; Perrone et al., 2016; Perry et al., 2012, 2013, 2014, 2018; Pollak et al., 2005; Porges et al., 2019; Quigley et al., 2017; Richardson et al., 2019; Rudd et al., 2017; Scrimgeour et al., 2016; Shakiba et al., 2020; Sharp et al., 2012; Sichko et al., 2018; Skibo et al., 2020; Skowron et al., 2014; Smiley et al., 2020; Stanger et al., 2018; Sturge-Apple et al., 2012; Sweet et al., 1999; Tabachnick et al., 2019, 2021; Taylor et al., 2013, 2015; Tu et al., 2014, 2017; Van der Graaff et al., 2016; Wagner et al., 2018; Welch et al., 2020; West et al., 2021; Willemen et al., 2008, 2009; Zeegers et al., 2018; Zhang et al., 2020).

Requests for necessary data to compute effect sizes were sent to 71 study authors: 54 authors were contacted for all (or any) study effect sizes, in order to include the study; 21 authors were contacted for additional effect sizes, 18 of which were requests for recalculating effect sizes using task level scores into effect sizes using change scores, and 3 were requests for unreported effect sizes. This effort resulted in the inclusion of $m = 7$ additional studies (Del Giudice et al., 2012; Laurent et al., 2012; Mezulis et al., 2015; Noll et al., 2015; Rousseau et al., 2014; Skowron et al., 2011; Tharner et al., 2013a, 2013b) and additional effect sizes from $m = 9$ studies (Bosquet Enlow et al., 2014; Clark et al., 2016; Giuliano et al., 2015; Miller et al., 2013; Partington et al., 2018; Perrone et al., 2016; Rudd et al., 2017; Tabachnick et al., 2019; Willemen et al., 2008). Most studies reported more than one effect size (e.g., due to multiple types of parenting measures or repeated assessments of ANS physiology). All effect sizes were retained for use in meta-analysis with one caveat: if a study contained repeated assessments of the *same* parenting measure, only the first assessment was included. A total of $m = 103$ unique samples, from 106 citations, provided $k = 418$ effect sizes ($n = 13{,}044$ unique participants). A full list and brief description of included studies are presented in Supplemental Table S1. Study data and R script used in analysis, are available on the Open Science Framework (OSF) repository at: osf.io/xs8ku.

2.3. Data extraction

Data for calculating effect sizes, effect size weights, and for coding moderators were extracted by a team of highly trained RAs. Each study was extracted twice independently by RAs, and discrepancies were handled by the lead author. The lead author then conducted an additional check of the information used to calculate effect sizes and weights (i.e., correlation coefficient and sample size) on all studies. Lastly, the principal investigator (CEH) checked 10 % of all extracted data for accuracy.

2.4. Coding of moderators

The following categorical moderators were coded for use in primary analysis: (1) type of ANS physiology measure, (2) parenting measure valence, (3) study design, (4) whether the parent was present or not during the ANS physiology recording, (5) clinical vs. non-clinical sample, and (6) type of change score used. In addition, two continuous moderators were coded (1) mean sample age at time of ANS measurement, and (2) percent female of sample. Continuous moderator variables were mean centered, within each individual model. Lastly, additional moderators were identified post hoc (during study selection and data extraction) as having the potential to explain variability in the correlation between parenting and child ANS physiology. The following moderators were coded for exploratory analysis: (1) relationship/attachment measures vs. parenting measures, (2) the type of parenting measure used (sensitivity/harshness, emotion socialization, control/discipline), (3) the type of report used (parent-report, child-report, observed, composite), (4) country of sample, (5) percent minority sample, and (6) the type of task used during ANS reactivity measurement.

2.4.1. Type of ANS measure

Parasympathetic and sympathetic nervous system measures were tested separately. However, within these separate models, different types of biomarkers were included together. Moderation by biomarker type was therefore tested. For parasympathetic models (resting and reactivity) three biomarkers were used: high-frequency heart rate variability (HF-HRV), root mean squared successive differences (RMSSD), and RSA as derived using the peak-to-valley method. For sympathetic models the following biomarkers were included: skin conductance level (SCL), also known as electrodermal activity, pre-ejection period (PEP), and salivary alpha amylase (sAA).

2.4.2. Parenting measure valence

A dichotomous dummy-coded variable was created to reflect whether a parenting variable measured a *positive* construct or a *negative* construct. Specifically, positive parenting measures reflect measures where higher values or scores on the measure would be theorized to lead to better emotion or self-regulation (e.g., parental warmth, sensitivity, emotional support). Conversely, negative parenting measures reflect measures where higher values or scores on the measure would be theorized to lead to poorer emotion or self-regulation (e.g., parental hostility, harshness, corporal punishment).

2.4.3. Type of reactivity measure

Reactivity effect sizes were calculated and reported as either raw change scores (e.g., task level minus resting level) or as regression-based residualized change scores in source studies. To examine whether this difference in methods influenced the results, we created a dummy coded variable, residualized, such that effect sizes that came from residualized scores = 1, and effect sizes that came from raw change scores = 0.

2.4.4. Study design

Study design was coded into two categorical variables reflecting (1) whether an effect size was experimental (e.g., parenting intervention effect) vs. correlational, and (2) whether an effect size was longitudinal vs. cross-sectional. All experimental effect sizes were longitudinal due to the need for pre- and post-intervention measurement, but correlational studies were either cross-sectional or longitudinal. Experimental studies

identified included parenting intervention programs designed to increase parental sensitivity (Hastings et al., 2019a; Tabachnick, 2019; 2021) and emotion coaching in parents (Katz et al., 2020), as well as programs designed to foster stronger emotional bonds between parent and child (Porges et al., 2019; Welch et al., 2020).

2.4.5. Clinical vs. non-clinical

Studies were coded as clinical if 50 % or more of the sample had a diagnosed clinical disorder or health condition. Clinical studies identified included samples with attention deficit hyperactivity disorder (Bell et al., 2018; McQuade et al., 2021), samples born premature (Brown, 2007; Feldman, 2015; Feldman and Eidelman, 2007; Feldman et al., 2014; Porges et al., 2019; Welch et al., 2020), and samples with a broad range of clinical disorders (internalizing and externalizing disorders, Willemen et al., 2008; Willemen et al., 2009).

2.4.6. Parenting measure type

Due to the large heterogeneity in parenting measures found in our search results, we further coded effect sizes for parenting measure type. We created four dummy coded variables reflecting whether a parenting measure was (1) an *emotion socialization* measure, (2) a measure of a parent's manner of *control or discipline* over the child, (3) a measure of the parent-child *relationship quality*, or (4) a measure of *attachment security*.

2.4.7. Country of sample and percent minority

Due to the distribution of the sample country of origin (i.e., 79 % were USA samples), we tested country of sample as a dichotomous moderator, such that USA samples = 1, and all other country samples = 0. Percent minority reflected the percent of the sample that was reported to belong to a minority racial or ethnic group for the respective country of the sample.

2.4.8. Reactivity task type

ANS reactivity effect sizes were coded into whether they were from ANS reactivity to tasks designed to be challenging (e.g., frustration tasks, conflict tasks, stressors), or tasks not designed to be challenging (e.g., free play, joint interaction). A dummy coded variable "non-challenging" was created such that non-challenging task effect sizes = 1, and challenging task effect sizes = 0.

2.5. Computation and coding of effect sizes

The primary effect size observed was a Pearson's correlation, *r*, describing the association between two continuous variables. If a study reported a different effect size, such as Cohen's *d* ($m = 1$), this was transformed into a Pearson's correlation. If a study reported means, standard deviations (SDs), and sample sizes by group (e.g., groups with different categorical attachment classifications), this was used to first calculate a Cohen's *d*, which was then transformed into a Pearson's correlation ($m = 24$). When a study only reported an F-test ($m = 1$) or t-test ($m = 2$) statistic, this was used in combination with group sample sizes to first calculate a Cohen's *d*, which was then transformed into a Pearson's correlation. If a study only provided information to calculate effect sizes from correlations that were significant (selective reporting), requests were sent to study authors for the non-significant correlations. If study authors did not respond to requests, correlations described as non-significant in study text were imputed as $r = 0$ (this occurred for $k = 7$ effect sizes). This is considered a conservative method for handling selective reporting, as it is unlikely true correlations were exactly zero (Miller et al., 2007).

Due to the heterogeneity in both (1) parenting measures, and (2) type of ANS measures, effect sizes needed to be coded so that they would all reflect the same direction of effect. For resting ANS models, effect sizes were coded such that larger positive effect sizes reflect a greater positive relation between more *positive* parenting and higher levels of the respective resting ANS physiology measure. This was accomplished by multiplying effect sizes with *negative* parenting measures by −1.

For reactivity ANS models, effect sizes were coded such that larger positive effect sizes reflect a stronger positive relation between more *positive* parenting and greater "reactivity". This coding had to take into consideration (1) the type of parenting measure (i.e., *positive* vs. *negative*), (2) the way in which the change scores were calculated (i.e., resting minus task vs. either task minus resting or residualized), and (3) the specific ANS biomarker used. For all included measures of PNS activity, a reduction in levels during a task, known as withdrawal, reflects physiological engagement and/or a fight-or-flight stress response. For SCL or sAA, exposure to a significant stressor should result in increasing levels. However, PEP is inversely related to sympathetic output, and therefore decreases in PEP during threat exposure are expected. In order to have all SNS measures be positively associated with SNS activity, PEP effect sizes were multiplied by −1. A complete description of the effect size coding scheme is available in the appendix. Positive effect sizes in reactivity models subsequently represent positive relations between more *positive* parenting and either greater PNS withdrawal (decreases), or greater SNS augmentation (increases).

Sample size was utilized to calculate variance of the effect sizes. If sample size for each specific correlation was not clearly provided in the article, requests for clarification were sent to study authors. If study authors did not respond, then all available information was used to approximate sample size. We adopted a conservative approach to approximating sample size. For example, if a study only provided a range of missingness across all variables, the maximum missingness of the range was assumed.

2.6. Missing moderator data

When data for moderators were unavailable or unclear in the publication, authors were contacted for clarification. We received responses from eight study authors providing the requested data (Laurent et al., 2012; McQuade et al., 2021; Nelson et al., 2017; Oshri et al., 2020; Perry et al., 2013; Taylor et al., 2015; Willemen et al., 2008). As a result of this effort all moderators in the primary analysis, with the exception of sample percent female, had complete data. Percent female data were missing for $m = 3$ studies. A total of $m = 6$ studies did not have sufficient information on race or ethnicity to calculate percent minority. Considering this low rate of missingness, we ran analysis on available data only. In addition, moderators were tested individually, which means that listwise deletion of studies missing either percent female or percent minority only affected tests of these specific moderators.

2.7. Statistical analysis

All analyses were conducted using Rstudio Version 1.38, running R language Version 4.0.0 (R Core Team, 2020a, 2020b; RStudio Team, 2010). We used random-effects meta-analytic modeling, instead of fixed-effects modeling, due to the high heterogeneity in study design (Hedges and Vevea, 1998). A total of four meta-analytic models were tested looking at the relation between parenting and (1) resting PNS activity, (2) PNS reactivity, (3) resting SNS activity, and (4) SNS reactivity. Pearson's correlations were transformed into Fisher's Z, using the *escalc* function in the *metafor* package (Viechtbauer, 2010), for analysis. This is recommended practice, due to bias introduced when calculating the variance estimate for Pearson's *r* effect sizes (Borenstein and Hedges, 2019). To increase interpretability, pooled effect sizes were back transformed and presented as Pearson's *r* in the text.

Many studies reported more than one effect size per sample, as previously stated. This was most often due to studies collecting multiple parenting measures, but also resulted from repeated measures of ANS physiology. Methods for obtaining a single effect size per study, for example by averaging effect sizes or arbitrarily selecting a single effect size, result in a loss of information (loss of statistical power) and biased

pooled estimates (Assink and Wibbelink, 2016). In order to properly handle this within-study dependency, we employed robust variance estimation (RVE) methods with a correlated effects structure, which produces effect size weights that are corrected for the shared covariance between effect sizes clustered within a given sample (Hedges et al., 2010). Random effects meta-analysis with RVE was conducted using the R package *robumeta* (Version 2.0; Fisher et al., 2017). Unlike alternative methods for handling within-study dependency (i.e., generalized linear modeling), RVE does not require precise knowledge of the covariance structure among study effect sizes, or rho (ρ; Hedges et al., 2010). In the current analysis, rho was set to $\rho = 0.8$, reflecting high covariance among parenting measures and/or repeated measures of ANS physiology. Sensitivity analyses were then conducted that vary rho in increments of.1, ranging from $\rho = 0$ to $\rho = 1$; consistent with the robustness of RVE methods, results were virtually unchanged. We implemented a small sample size bias correction for all models. This correction can reduce bias introduced by small samples, unbalanced moderators, or outliers (Tipton, 2015).

Intercept-only models were initially conducted, which provides a weighted pooled estimate of the effect, across biomarker types. To test for heterogeneity, we calculated the I^2 statistic. I^2 within an RVE model is a measure of total variability (i.e., within study and between study) in effect sizes (Cheung, 2014). This statistic ranges from 0 to 1, with higher values reflecting greater heterogeneity in effect sizes. It has been suggested that an $I^2 < .30$ (less than 30 % of variance attributed to heterogeneity) reflects low heterogeneity, I^2 values of .30 to .50 reflect moderate heterogeneity, and values above .70 reflect high heterogeneity (Deeks et al., 2008). It should be noted that other guides for interpreting the I^2 have been proposed (e.g., Higgins et al., 2003).

We next tested for moderation by biomarker type. Categorical moderation analysis was conducted by running a no-intercept model, with the inclusion of a dummy variable for each biomarker for a given model. For example, the no-intercept model for testing PNS activity biomarker type as a moderator could be written as:

$$Zr_i = B_1(\text{HF HRV}_i) \quad + \quad B_2(\text{RMSSD}_i) \quad + \quad B_3(\text{Peak} - \text{to} - \text{Valley}_i)$$

whereby the resulting estimates B_{1-3} represent the estimated weighted pooled effect size for the association between parenting and HF-HRV, RMSSD, and peak-to-valley, respectively. We used a small sample adjusted F-test, using the Wald test function from the R package *clubSandwich* (Pustejovsky, 2021), to test for significant moderation. Results from this analysis suggested no moderation by biomarker type (see Results section) in any model, we therefore conducted subsequent moderation analysis collapsing across biomarkers.

Moderators were tested individually, in order to avoid suppression effects. Categorical moderators were tested using the small sample adjusted F-test (i.e., Wald test), as described above. Only categorical moderators that had five or more effect sizes per level were included in a given model. Because of this, the list of moderators varied between models. The full list of moderators included: parenting measure valence, experimental study, longitudinal study, parent absent during ANS recording, clinical sample, sample mean age, and sample percent female; an additional moderator, residualized, was included in reactivity models. Experimental study was not tested in the resting SNS model, or the SNS reactivity model, due to too few effect sizes being experimental (k's = 1). Clinical sample was not tested in the resting SNS model, as too few effect sizes were from clinical samples ($k = 4$). In addition, longitudinal study was not tested in the SNS reactivity model because too few effect sizes were longitudinal ($k = 3$).

Publication bias was assessed through visual inspection of contour enhanced funnel plots and through Egger's regression (Egger et al., 1997). Weighted effect sizes were aggregated at the study level before generating funnel plots and testing Egger's regression, a method that has been recommended for testing for publication bias with robust variance estimation methods (Bediou et al., 2018; Tanner-Smith, 2012). The *regtest* function from the *metafor* package in R (Viechtbauer, 2010) was used to run Egger's regression.

3. Results

3.1. Study characteristics

Of the 103 total selected studies, $M = 74$ studies provided $k = 178$ effect sizes for resting PNS activity (HF-HRV, $m = 61$, $k = 142$; peak-to-valley, $m = 8$, $k = 14$; RMSSD, $m = 4$, $k = 18$; CVI, $m = 1$, $k = 4$), $M = 50$ studies provided $k = 137$ effect sizes for PNS reactivity (HF-HRV, $m = 38$, $k = 105$; peak-to-valley, $m = 8$, $k = 18$; RMSSD, $m = 4$, $k = 14$), $M = 25$ studies provided $k = 51$ effect sizes for resting SNS activity (SCL, $m = 13$, $k = 23$; PEP, $m = 6$, $k = 11$; sAA, $m = 5$, $k = 13$; CSI, $m = 1$, $k = 4$), and $M = 27$ studies provided $k = 61$ effect sizes for SNS reactivity (SCL, $m = 17$, $k = 34$; PEP, $m = 9$, $k = 23$; sAA, $m = 1$, $k = 4$). Biomarkers with less than five effect sizes (CVI, CSI, sAA reactivity) were excluded from analysis.

The majority of the included studies were conducted in the United States (77 %), but studies were also obtained from Canada (7 %), the Netherlands (7 %), Israel (5 %), and other countries (4 %). Mean sample age ranged from *term age* (preterm sample) to 16.8 years (mean = 6.6, $SD = 4.8$, years), and on average samples were half female (mean = 50 %, $SD = 8$ %, female). Study sample sizes ranged from $n = 18$ to $n = 450$ (mean sample size = 127, $SD = 95$).

3.2. Publication bias and outliers

Investigation of publication bias did not reveal significant evidence for publication bias. In the resting PNS activity model the Egger's regression only revealed a marginally significant association between *SE* and effect size ($p = .10$). This association was driven by a single outlier (effect size > 4 *SD* above the mean) with a small sample size ($n = 18$). After removing this outlier, the Egger's regression test was non-significant ($p = .26$). Removing this outlier did not change any primary study results; we therefore present the funnel plot with this outlier removed (Fig. 2). Egger's regression was non-significant for all other models (p's > 0.30), suggesting lack of publication bias. In addition, visual inspection of the contour enhanced funnel plots, presented in Fig. 2, revealed a relatively symmetrical distribution of data points on the left and right side of the average effect size, thus there was no evidence for publication bias.

3.3. Pooled estimates

The meta-analysis revealed non-significant overall correlations between parenting and resting PNS activity ($r = 0.01$, $SE = 0.01$, $df = 61.8$, $p = .31$, 95 % CI [−0.01,.04]), PNS reactivity ($r = -0.01$, $SE = 0.01$, $df = 40.6$, $p = .46$, 95 % CI [−0.03,.02]), resting SNS activity ($r = -0.004$, $SE = 0.03$, $df = 19.3$, $p = .90$, 95 % CI [−0.06,.05]), and SNS reactivity ($r = 0.01$, $SE = 0.02$, $df = 20.0$, $p = .59$, 95 % CI [−0.04,.06]). Tests of heterogeneity revealed substantial heterogeneity in the resting SNS model ($I^2 = .54$), moderate heterogeneity in the resting PNS model ($I^2 = .46$) and the SNS reactivity model ($I^2 = .39$), and low heterogeneity in the PNS reactivity model ($I^2 = .28$).

3.4. Moderation analysis

3.4.1. Resting PNS

Results from the resting PNS activity moderation analysis are presented in Table 2. Moderation analysis revealed that the correlation between parenting and resting PNS activity was not moderated by biomarker type ($F(2, 5.1) = 1.2$, $p = .37$). Across all studies, parenting was not significantly correlated with either resting HF-HRV ($r = 0.02$, $SE = 0.01$, $df = 51.9$, $p = .15$, 95 % CI [−0.01,.05]), resting RSA calculated using the peak-to-valley method ($r = -0.03$, $SE = 0.03$, $df =$

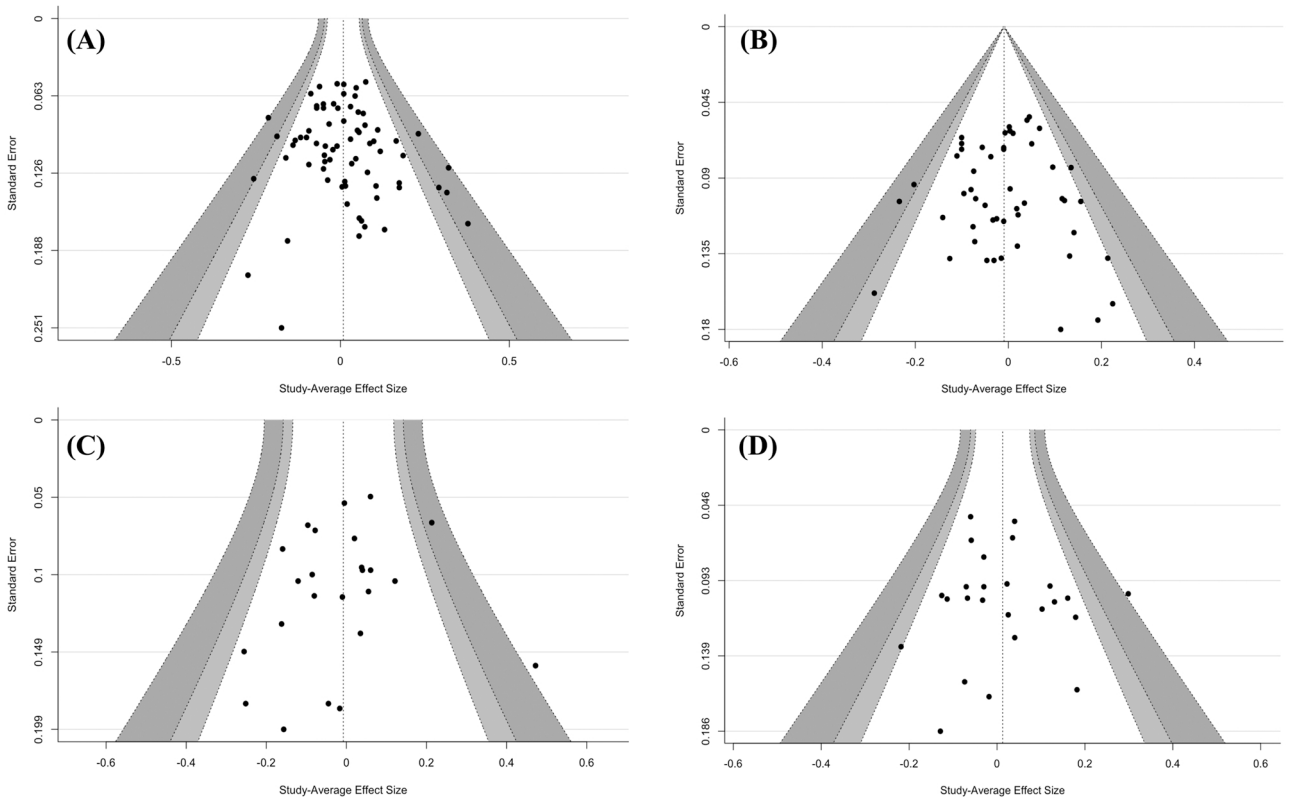

Fig. 2. Contour enhanced funnel plots for visual inspection of publication bias in the included studies on parenting and (A) resting parasympathetic nervous system (PNS) activity, (B) PNS reactivity, (C) resting sympathetic nervous system (SNS) activity, and (D) SNS reactivity. White region represents 95 % CI. The dashed vertical line represents the average effect size.

Table 2
Potential moderators of relation between parenting and baseline PNS activity.

Moderator				
Categorical	*F*-test	*r*	*df*	*p*
ANS biomarker type	1.2		2, 5.1	.37
HF-HRV		.02	51.9	.15
Peak-to-valley RSA		-0.03	6.8	.29
RMSSD		-0.01	2.5[a]	.89
Parenting measure valence	3.55		1, 60.2	.06
Positive		.03	41.5	.07
Negative		-0.01	33.9	.52
Parent presence during ANS measure	0.19		1, 53.7	.67
Present		.02	36.8	.29
Absent		.01	24.5	.75
Study design - type	**18.2**		**1, 3.4**[a]	**.019**
Experimental		**.22**	**3.1**[a]	**.019**
Correlational		.005	58.0	.70
Study design - length	1.22		1, 22.1	.28
Longitudinal		.04	14.4	.18
Cross-sectional		.01	52.7	.67
Participant sample	**12.0**		**1, 7.8**	**.009**
Clinical		**.14**	**6.5**	**.01**
Non-clinical		-0.00	53.6	.98
Continuous	*B*	*SE*	*df*	*p*
Mean age	-0.00	.00	27.9	.31
Percent female	-0.12	.28	4.3	.69

Note. ANS = autonomic nervous system. HF-HRV = high frequency heart rate variability. RSA = respiratory sinus arrhythmia. RMSSD = root mean squared successive differences.
Bolded estimates are significant at the $p < .05$ level.
[a] tests with degrees of freedom < 4 should be interpreted with caution.

6.8, $p = .29$, 95 % CI [−0.10,.04]), or resting RMSSD ($r = -0.01$, $SE = 0.09$, $df = 2.5$, $p = .89$, 95 % CI [−0.31,.29]). Two moderators emerged as significant: experimental study ($F(1, 3.4) = 18.2$, $p = .019$) and clinical sample ($F(1, 7.8) = 12.0$, $p = .009$). The positive correlation between positive parenting and resting PNS activity was greater among experimental studies ($r = 0.22$, $SE = 0.05$, $df = 3.1$, $p = .019$, 95 % CI [.07,.36]) compared to correlational studies ($r = 0.005$, $SE = 0.01$, $df = 58.0$, $p = .70$, 95 % CI [−0.02,.03]). In addition, the positive correlation between positive parenting and resting PNS activity was greater among clinical samples ($r = 0.14$, $SE = 0.04$, $df = 6.5$, $p = .0097$, 95 % CI [.05,.23]) compared to non-clinical samples ($r = -0.00$, $SE = 0.01$, $df = 53.6$, $p = .98$, 95 % CI [−0.03,.03]).

We next ran a model that included all moderators with a p-value less than $p < .10$. This included experimental study, clinical sample, as well as parenting measure valence, which was a non-significant moderator ($F(1, 60.2) = 3.55$, $p = .06$). Both experimental study ($p = .04$) and clinical sample ($p = .03$) remained significant, suggesting independent moderating effects. See Fig. 3 for forest plots of experimental studies and clinical sample studies. Parenting measure valence remained non-significant after controlling for experimental study and clinical sample ($p = .35$). We next ran an intercept model centering both significant moderators at their highest reliably obtained values (i.e., modeling an experimental study with a clinical sample). With these moderators centered positive parenting was positively, and significantly, correlated with resting PNS activity ($r = 0.26$, $SE = 0.05$, $df = 3.2$, $p = .01$, 95 % CI [.11,.41]). To obtain 80 % power to detect this effect would require a sample size of $N = 112$ (i.e., $n = 56$ intervention, $n = 56$ control for a parenting intervention study with a clinical sample).

3.4.2. PNS reactivity

Moderation analysis results for PNS reactivity are presented in Table 3. Biomarker type was not a significant moderator in the PNS reactivity model ($F(2, 5.9) = 1.35$, $p = .33$). Parenting was not significantly correlated with HF-HRV reactivity ($r = 0.001$, $SE = 0.01$, $df = 30.4$, $p = .97$, 95 % CI [−0.03,.03]), RSA as derived from peak-to-valley

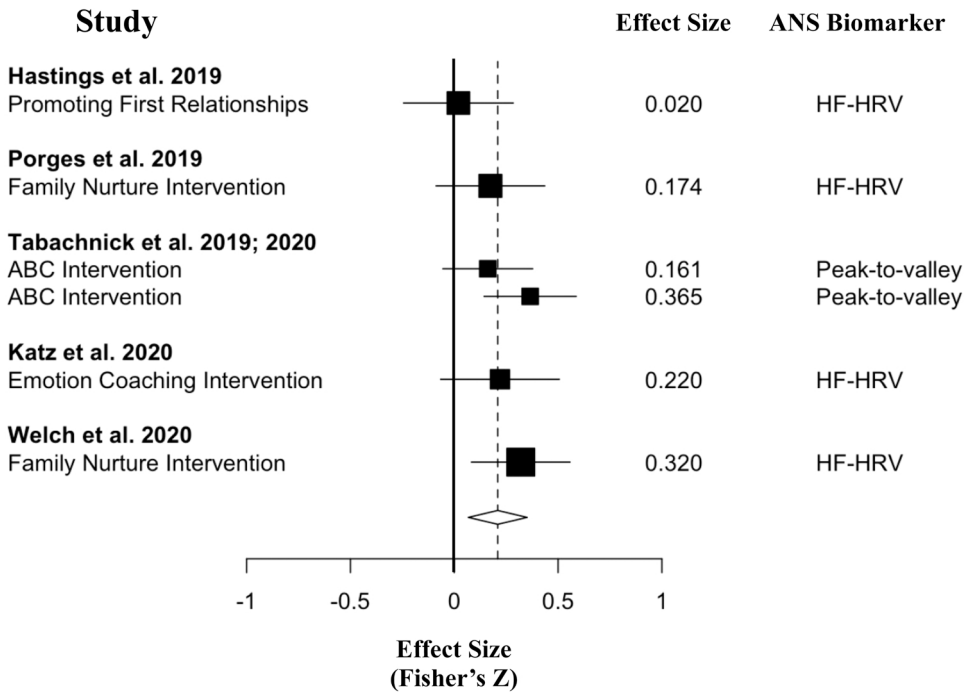

Fig. 3. Effect sizes (Fisher's Z) for relation between parenting and resting PNS activity in parenting intervention studies. HF-HRV = high frequency heart rate variability. Size of squares reflect relative weight of effect. Error bars represent 95 % CI. Diamond and dashed line represent weighted mean effect size.

Table 3
Potential moderators of the relation between parenting and PNS reactivity.

Moderator				
Categorical	*F*-test	*r*	*df*	*p*
ANS biomarker type	1.35		2, 5.9	.33
HF-HRV		.00	30.4	.97
Peak-to-valley RSA		-0.04	5.6	.18
RMSSD		-0.05	2.8[a]	.24
Parenting measure valence	1.55		1, 35.4	.22
Positive		-0.02	29.5	.21
Negative		.01	19.0	.67
Parent presence during ANS measure	0.80		1, 37.2	.38
Present		.00	23.4	.98
Absent		-0.02	16.9	.25
Study design – type	0.02		1, 1.03[a]	.92
Experimental		-0.03	1[a]	.88
Correlational		-0.01	40.0	.48
Study design – length	0.08		1, 11.2	.79
Longitudinal		-0.02	7.5	.57
Cross-sectional		-0.01	35.6	.59
Participant sample	0.72		1, 4.1	.44
Clinical		-0.06	3.6[a]	.40
Non-clinical		-0.01	36.7	.66
Reactivity calculation method	0.51		1, 26	.48
Raw change score		-0.02	26.8	.25
Residualized change score		.01	13.1	.85
Continuous	*B*	*SE*	*df*	*p*
Mean age	-0.00	.00	16.2	.68
Percent female	.13	.16	6.5	.45

Note. ANS = autonomic nervous system. HF-HRV = high frequency heart rate variability. RSA = respiratory sinus arrhythmia. RMSSD = root mean squared successive differences.
Bolded estimates are significant at the $p < .05$ level.
[a] tests with degrees of freedom < 4 should be interpreted with caution.

method reactivity ($r = -0.04$, SE =0.03, df = 5.6, p = .18, 95 % CI [−0.11,.03]), or RMSSD reactivity ($r = -0.05$, SE =0.03, df = 2.8, $p = .24$, 95 % CI [−0.16,.06]). There were no significant moderators of the relation between parenting and PNS reactivity (p's > 0.22).

3.4.3. Resting SNS

Moderation results for resting SNS activity are presented in Table 4. Biomarker type was not a significant moderator in this model ($F(2, 8.6) = 0.79$, $p = .49$). Parenting was not significantly correlated with resting SCL ($r = -0.04$, SE =0.02, df = 10.6, $p = .11$, 95 % CI [−0.09,.01]), resting PEP ($r = 0.02$, SE =0.10, $df = 4.5$, $p = .82$, 95 % CI [−0.23,.28]), or resting sAA ($r = 0.05$, SE =0.06, df = 3.7, $p = .53$, 95 % CI [−0.14,.23]). Only longitudinal design was a significant moderator in this model (F(1, 2.6) = 14.8, $p = .04$), such that among longitudinal studies there was a negative correlation between positive parenting and resting SNS activity ($r = -0.12$, SE =0.02, df = 2.0, $p = .04$, 95 % CI

Table 4
Potential moderators of relation between parenting and baseline SNS activity.

Moderator				
Categorical	*F*-test	*r*	*df*	*p*
ANS biomarker type	0.79		2, 8.6	.49
SCL		-0.04	10.6	.11
PEP		.02	4.5	.82
sAA		.05	3.7[a]	.53
Parenting measure valence	0.09		1, 16.7	.77
Positive		-0.01	9.7	.78
Negative		.00	14.6	.93
Parent presence during ANS measure	0.02		1, 17.7	.90
Present		.00	8.1	.99
Absent		-0.01	10.4	.83
Study design – length	**14.8**		**1, 2.6[a]**	**.04**
Longitudinal		**-0.12**	**2.0[a]**	**.04**
Cross-sectional		.02	16.0	.55
Participant sample	1.03		1, 1.2[a]	.50
Clinical		-0.08	1.0[a]	.48
Non-clinical		.00	17.5	.93
Continuous	*B*	*SE*	*df*	*p*
Mean age	-0.00	.01	7.5	.66
Percent female	.45	.39	2.9[a]	.34

Note. ANS = autonomic nervous system. SCL = skin conductance level. PEP = pre-ejection period. sAA = salivary alpha amylase.
Bolded estimates are significant at the $p < .05$ level.
[a] tests with degrees of freedom < 4 should be interpreted with caution.

[−0.22, −0.02]), that was not observed among cross-sectional studies ($r = 0.02$, $SE = 0.03$, $df = 16.0$, $p = .55$, 95 % CI [−0.04,.08]). However, considering the small degrees of freedom in both the F-test and the estimate among longitudinal studies, likely resulting from there being so few longitudinal studies ($m = 3$ studies, $k = 5$ effect sizes), these results should be interpreted with caution. All other moderators of the relation between parenting and resting SNS activity were non-significant (p's > 0.34).

3.4.4. SNS reactivity

Moderation results for SNS reactivity are presented in Table 5. Biomarker type did not moderate the correlation between parenting and SNS reactivity ($F(1, 11.7) = 0.54$, $p = .48$). Parenting was not significantly correlated with SCL reactivity ($r = 0.02$, $SE = 0.03$, $df = 14.0$, $p = .49$, 95 % CI [−0.04,.09]), or PEP reactivity ($r = -0.01$, $SE = 0.03$, $df = 6.4$, $p = .75$, 95 % CI [−0.08,.06]). Study mean age was the only significant moderator of the relation between parenting and SNS reactivity ($B = -0.01$, $SE = 0.004$, $df = 7.2$, $p = .01$, 95 % CI [−0.02, −0.004]). Visual inspection of the scatterplot between mean age and effect size, presented in Fig. 4, revealed that as sample age increased from childhood to adolescence the correlation between positive parenting and SNS reactivity changed from positive to negative. Figs. 5–7.

3.5. Exploratory moderator analyses

Exploratory analyses were conducted to further investigate study-level and sample-level characteristics, identified during study screening and data extraction, as potential moderators of the relation between parenting and child ANS physiology.

3.5.1. Relationship measures

The moderating effect of attachment and relationship measures was tested by including a categorical variable representing whether a measure was a parenting measure, an attachment measure, or a relationship quality measure. Relationship measure was a significant moderator in the resting PNS model ($F(2, 4.8) = 6.02$, $p = .0496$). Pairwise comparisons revealed significant differences between effect sizes from parenting measures ($r = 0.02$, $SE = 0.01$, $df = 55.1$, $p = .16$, 95 % CI [−0.01,.05]) and those from attachment measures ($r = -0.08$, $SE = 0.03$, $df = 3.99$, $p = .03$, 95 % CI [−0.15, −0.02]). The moderating effect of relationship measure is presented in Fig. 8. Relationship measure was not a significant moderator in the PNS reactivity model ($F(2, 4.5) = 1.0$, $p = .43$), resting SNS model ($F(2, 0.7) = 0.06$, $p = .95$), or SNS reactivity model ($F(2, 0.73) = 0.02$, $p = .98$).

Table 5
Potential moderators of the relation between parenting and SNS reactivity.

Moderator				
Categorical	*F*-test	*r*	*df*	*p*
ANS biomarker type	0.54		1, 11.7	.48
SCL		.02	14.0	.49
PEP		-.01	6.4	.75
Parenting measure valence	0.15		1, 20.0	.71
Positive		.00	13.6	.90
Negative		.02	11.3	.53
Parent presence during ANS measure	0.03		1, 19.4	.87
Present		.01	10.8	.82
Absent		.02	9.1	.57
Participant sample	3.6		1, 3.6[a]	.14
Clinical		-0.09	2.8[a]	.22
Non-clinical		.03	16.5	.29
Reactivity calculation method	0.53		1, 11.3	.48
Raw change score		-0.00	14.0	.98
Residualized change score		.04	5.5	.45
Continuous	*B*	*SE*	*df*	*p*
Mean age	**-0.01**	**.004**	**7.2**	**.01**
Percent female	.01	.26	2.3[a]	.96

Note. ANS = autonomic nervous system. SCL = skin conductance level. PEP = pre-ejection period.
Bolded estimates are significant at the $p < .05$ level.
[a] tests with degrees of freedom < 4 should be interpreted with caution.

3.5.2. Parenting measure type

Within parenting measures, specific type of parenting measure was tested as a moderator by first removing effect sizes for relationship measures, then adding a categorical variable representing whether a parenting measure reflected (1) warmth/aggression, (2) control, or (3) emotion socialization. Parenting measure type was not a significant moderator in the resting PNS model, ($F(2, 13.8) = 1.6$, $p = .25$), the PNS reactivity model, ($F(2, 10.7) = 1.4$, $p = .29$), resting SNS model ($F(2, 4.8) = 0.05$, $p = .95$), or SNS reactivity model ($F(2, 4.8) = 0.84$, $p = .49$).

3.5.3. Type of report

In order to provide information for future researchers regarding differences in report type among correlational studies, we compared parent-reported, child-reported, observed, and composite (e.g., observed and parent-reports combined) parenting measures. First, we removed studies that used either (1) experimental designs, or (2) documented history (i.e., abuse). In addition, only two studies used composite measures in SNS models; composite measure was therefore not tested in those models. Type of report was not a significant moderator in the resting PNS model, ($F(3, 7.9) = 1.5$, $p = .30$), the PNS reactivity model, ($F(3, 7.5) = 0.1$, $p = .93$), resting SNS model ($F(2, 5.0) = 0.11$, $p = .90$), or SNS reactivity model ($F(2, 5.0) = 0.67$, $p = .55$).

3.5.4. Country of sample and percent minority

Country of sample was not a significant moderator in any model (p's > 0.32). Percent minority was also not a significant moderator in any model (p's > 0.24).

3.5.5. Non-challenging task

Non-challenging task was not a significant moderator of the relation between parenting and PNS reactivity ($F(1, 2.8) = 6.0$, $p = .10$). Non-challenging task was not tested as a moderator of the relation between parenting and SNS reactivity because only one SNS reactivity study used a non-challenging task.

3.6. Sensitivity analysis

Sensitivity analysis was conducted to test the robustness of findings to: (1) the exclusion of an outlier in the resting PNS model, (2) exclusion of sAA effect sizes in the resting SNS model, and (3) the exclusion of imputed non-significant effect sizes. The removal of a single outlier with a small sample size in the resting PNS model did not change results. The exclusion of sAA effect sizes in the baseline SNS model ($k = 13$ effect sizes from $m = 5$ studies), only influenced the moderating effect of longitudinal study, which was no longer significant ($p = .20$). When imputed effect sizes were excluded from analysis results were virtually identical and inferences were unchanged.

4. Discussion

In the current meta-analysis, we tested the strength of the correlation between parenting and child ANS physiology. In contrast to expectations, we observed non-significant pooled associations between parenting and child parasympathetic and sympathetic nervous system activity across all studies. These non-significant associations were observed across biomarkers used for measuring PNS and SNS physiology, for both resting and reactivity measures. The autonomic nervous system has been proposed as a biological mediator between parenting experienced in early life and later child health and behavioral outcomes (Propper and Moore, 2006; Repetti et al., 2002). While across studies no

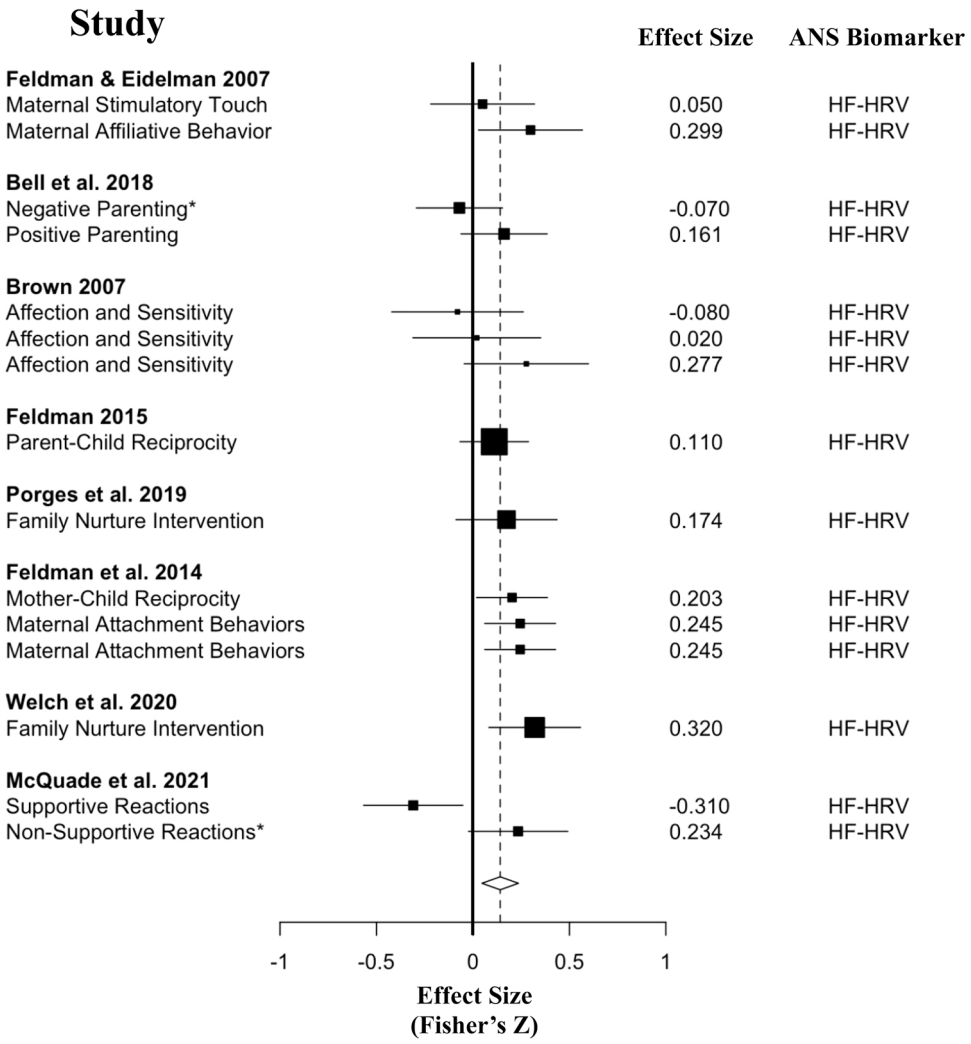

Fig. 4. Effect sizes (Fisher's Z) for relation between parenting and resting PNS activity in clinical sample studies. HF-HRV = high frequency heart rate variability. Size of squares reflect relative weight of effect. Error bars represent 95 % CI. Diamond and dashed line represent weighted mean effect size. * Effect sizes from negative valenced measures were reverse scored.

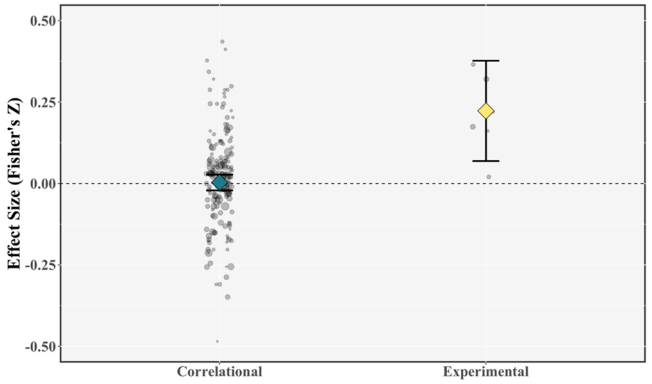

Fig. 5. Moderating effect of experimental study design on the relation between parenting and resting parasympathetic nervous system (PNS) activity. Diamonds represent weighted mean effect size in each subgroup. Size of data points represent relative weight used in calculating mean effect size. Error bars represent 95 % CI.

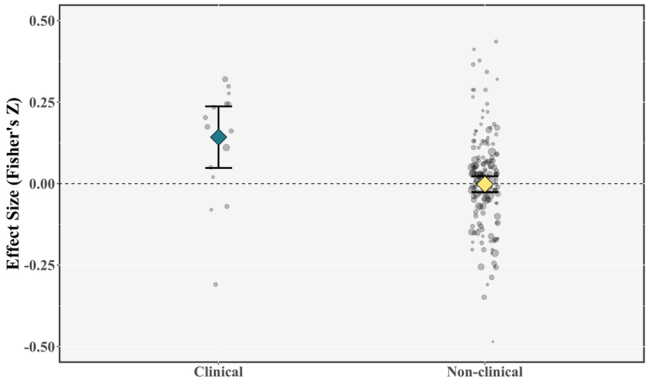

Fig. 6. Moderating effect of clinical sample on the relation between parenting and resting parasympathetic nervous system (PNS) activity. Diamonds represent weighted mean effect size in each subgroup. Size of data points represent relative weight used in calculating mean effect size. Error bars represent 95 % CI.

general association between parenting and child ANS physiology was observed, moderation analysis revealed that experimental interventions aimed at improving parenting resulted in increases in resting PNS activity in children and adolescents, an effect that may be stronger among more at-risk youth.

Importantly, given the comprehensive scope of the current meta-

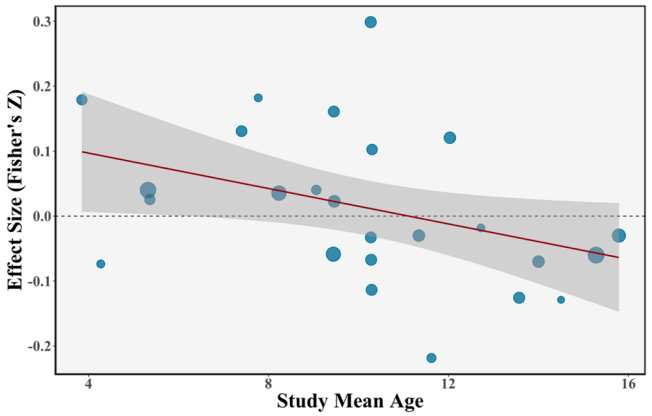

Fig. 7. Moderating effect of sample mean age on relation between parenting and sympathetic nervous system (SNS) reactivity. Negative effect sizes reflect negative relation between positive parenting and increases in SNS (i.e., reactivity). Size of data points reflects relative weight given to each effect size in analysis. Shaded area represents 95 % CI.

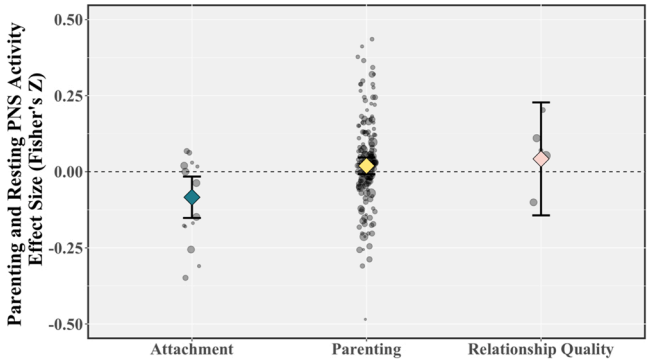

Fig. 8. Moderating effect of relationship measure on relation between parenting and resting parasympathetic nervous system (PNS) activity. Diamonds represent weighted mean effect size in each subgroup. Size of data points represent relative weight used in calculating mean effect size. Error bars represent 95 % CI.

analysis, substantial heterogeneity in both study methods and participant sample were observed in the literature. Mirroring this qualitative characteristic, between-study heterogeneity in effect sizes was also observed, warranting investigation of potential moderators that might explain variability in the correlations between parenting and child ANS physiology. A few significant moderators were observed. First, within the resting PNS model, effect sizes were larger for studies that used experimental designs, specifically parenting intervention studies, as compared to correlational designs. The positive pooled effect size observed among these studies suggests that interventions designed to improve parenting and facilitate the development of a secure attachment between parent and child lead to higher resting PNS activity in children. This is consistent with parent socialization and attachment theory. Developmentally appropriate protection, sensitive responding, and coaching of emotional understanding by parents, as well as secure bonds that facilitate trust between child and caregiver, may influence neural-autonomic systems implicated in self and affect-regulation (Propper and Moore, 2006; Thompson, 2015).

The finding that effects were stronger among intervention studies suggests that accurate approximations of parenting effects on child ANS physiology may be difficult to obtain using correlational data, where we could expect high levels of noise and confounding from unmeasured variables (e.g., physical exercise level, larger family context). In addition, correlational results are complicated by the bidirectional relation between child physiology and parenting. Physiological profiles associated with poorer emotion regulation skills (e.g., low resting HRV) may lead to less supportive, or more overcontrolling, parenting (Hastings, Grady, and Barrieau, 2019; Kennedy et al., 2004). Alternatively, children with poorer emotion regulation abilities may sometimes evoke *more* support during moments of challenge because parents anticipate them requiring it (Planalp et al., 2016). The principle of Goodness-of-fit parenting (Chess and Thomas, 1999) adds further complexity to correlational findings, as certain temperamental or physiological characteristics may influence the magnitude or direction of parent socialization effects (Rubin et al., 2002). In their seminal paper, Collins and colleagues suggested that parenting intervention studies can provide the most convincing evidence for or against parenting influences on child development (Collins et al., 2000). Our results support this suggestion, highlighting the advantages of experimental designs, and suggest that future developmental researchers should increase efforts to utilize experimental, over correlational, designs whenever possible.

For PNS reactivity, no moderating effect of *experimental study* was observed. Notably, only two studies were identified that tested parenting intervention effects on PNS reactivity, and they differed greatly in the length of follow-up. Parenting intervention resulted in reduced PNS withdrawal among toddlers measured 6 months after treatment (Hastings, 2019a), but did not influence PNS withdrawal among children tested 9 years after treatment (Tabachnick et al., 2019). This suggests that parenting intervention effects on PNS reactivity may diminish over time. Future research should aim to continue testing parenting intervention effects on child ANS functioning, stressing the need for additional studies on PNS reactivity, as well as studies on SNS activity. In addition, investigations should be made into the effects of follow-up interventions, whereby parental sensitivity training refreshers could be provided, analogous to vaccine booster shots.

Second, we found that the positive correlation between parenting and resting PNS activity was greater among studies with a clinical sample. This is consistent with recent meta-analytic findings that early-life maltreatment is more strongly associated with resting PNS activity among clinical samples (Sigrist et al., 2021). The types of clinical samples identified varied greatly, including samples with behavioral and mood disorders (e.g., attention deficit hyperactivity disorder, internalizing problems), as well as samples at elevated developmental risk (e.g., premature birth). Stronger positive associations between parenting and resting PNS activity among clinical samples may reflect stronger influences of parenting on children's ANS physiology among these more at-risk youth. Children who are at greater risk of psychopathology or developmental delay may rely more on positive parent socialization efforts, which can directly buffer against, or help entrain positive coping abilities to independently manage the higher levels of adversity to which this group may be exposed (Blair and Raver, 2012; Hostinar et al., 2014). These effects observed were almost entirely correlational, so strong causal inferences should be avoided. Nevertheless, future researchers interested in parent socialization of child ANS physiology could benefit from focusing on clinical samples, or other samples of children at elevated risk of psychopathology (e.g., children living in poverty).

The correlation between parenting and resting PNS activity was also moderated by whether a parenting or attachment measure was utilized. Surprisingly, we observed negative relations between attachment security and resting PNS activity. This is in contrast to findings with parenting measures, and expectations that secure attachment would be linked to higher levels of resting PNS activity. It should be noted that the identified studies that investigated attachment and child ANS physiology exhibited substantial variability. Attachment measures used in primary source studies included observed behavioral (ABC-D classification), narrative, and child-report measures. Unfortunately, due to the limited number of attachment studies identified we were unable to test more nuanced moderation analysis regarding type of attachment measure used. In addition, different forms of insecure attachment (avoidant versus resistant) may exhibit opposing emotion regulation-linked

behavioral reactions to interpersonal challenge such as separation (Cassidy, 1994). It is therefore unlikely that different styles of insecure attachment, or even different methods for measuring attachment insecurity, would relate to physiological systems of affect and stress regulation in a similar manner. The exploratory nature of these results, in addition to the relatively small number of attachment studies, must be taken into consideration. Nevertheless, future research is needed to clarify why attachment security, which is linked to sensitive parenting (Cassidy, 1994), may exhibit differential associations with resting PNS activity as compared to characteristics of parenting.

There was some evidence that the association between parenting and resting SNS activity may be moderated by longitudinal design. Among longitudinal studies more positive parenting was associated with lower resting SNS activity, an association that was not observed among cross-sectional studies. Elevated resting SNS activity has been positively associated with markers of cardiovascular risk (e.g., hypertension; Mancia and Grassi, 2013). If positive parenting predicts lower resting SNS activity this may be one mechanism through which positive parenting leads to better long-term health outcomes (E. Chen et al., 2017). Observing this effect only among longitudinal studies may suggest that directional relations between parenting and resting SNS activity are easier to detect using longitudinal designs. These results should be interpreted with caution considering the small number of resting SNS activity longitudinal studies identified. Nevertheless, considering these preliminary results and the relative gap in the literature, researchers interested in parenting socialization of sympathetic activity should increase efforts to use longitudinal, over cross-sectional, designs.

Lastly, the correlation between parenting and SNS reactivity was moderated by mean age of the sample. As mean age of the sample increased from early childhood to late adolescence, the correlation between positive parenting and SNS reactivity appeared to change from positive to negative. Longitudinal or age-comparison studies of SNS reactivity to challenge have tended to show increases in SNS reactivity from early childhood to adolescence (Hinnant et al., 2011; Quigley and Stifter, 2006). Positive parenting being associated with less reactivity in adolescence may mean that positive parenting leads to more moderate levels of reactivity at an age when reactivity is on average higher. Importantly, determining what is over-reactivity versus an appropriate level of reactivity is difficult; it will depend not only on the task, but also on the range of levels within the sample. For example, if moderate levels of ANS reactivity during challenge are most adaptive (Miller, 2018), then tests of simple linear relations (e.g., correlations) could lead to mixed results: if the range of change scores extend from low to moderate reactivity, then we might expect higher values within the sample to be more adaptive; if scores range from moderate to high, then relatively lower values could be more adaptive. Alternatively, given the lack of a significant pooled estimate, it is possible that the relation between positive parenting and SNS reactivity is always negative, but simply becomes stronger later in youth. This explanation is consistent with recent meta-analytic findings that the negative association between early-life maltreatment and resting PNS activity becomes stronger with age (Sigrist et al., 2021). The effect of negative or harsh parenting may accumulate over time, interacting with other environmental factors (e.g., influencing peer relationships), resulting in greater changes to physiology later in youth. Alternatively, increased attention is being given to adolescence (i.e., puberty) as a potential second *sensitive period* of development (Gunnar et al., 2019). Considering that adolescence may also be a time of increased parent-child conflict (Parra et al., 2015), negative or harsh parenting may be particularly impactful during this developmental period.

Surprisingly, given the large variability in parenting measures, we did not observe any moderating effect of type of parenting measure. Across models, effect sizes did not differ between measures of sensitivity or harshness, measures of emotion socialization, or measures of control or discipline. We also did not find evidence that effect sizes differed between positive and negative measures. This could reflect high correlation among these different parenting domains (i.e., parents who are more sensitive tend to also engage in more emotion coaching, parents who exhibit more positive parenting tend to exhibit less negative parenting). It could also reflect that all of these aspects of parenting are similarly important in shaping physiological mechanisms of emotion and self-regulation (Repetti et al., 2002; Propper and Moore, 2006). Conversely, considering the non-significant pooled correlation between parenting and ANS physiology, this could be explained by a generally minimal effect of normative variation in parenting on the development of the ANS. However, given the significant causal evidence observed within parent-intervention studies, this is not a likely explanation. Instead, the difficulty of identifying associations between parenting and child ANS physiology using correlational designs might be similar between different types of parenting measures.

We also did not observe any moderating effect of type of reactivity measure (raw change scores vs. residualized change scores). This may be due to these different reactivity measures typically being highly correlated (Treadwell et al., 2010). Therefore, how change scores are calculated may not influence the direction or magnitude of the associations.

4.1. Study limitations and future directions

There are several strengths of this study, as the first quantitative synthesis of the literature on parenting and child ANS physiology. First, the study was rather comprehensive. We included a diverse range of parenting and parental socialization measures theorized to influence development through physiological systems of affect and self-regulation. In addition, we assessed well validated single-branch measures of both the PNS and the SNS and included both resting and reactivity measures. Among the ANS physiology literature, substantial heterogeneity of methods exists, posing challenges for the synthesis of evidence (Laborde et al., 2017). Our moderation analysis contributes important information for guiding future researchers interested in parenting and ANS physiology. Lastly, we found no evidence of publication bias, an important finding considering we restricted our analysis to peer-reviewed journal articles.

Despite these strengths, several limitations should be mentioned to help clarify findings and guide future research. First, relatively fewer studies were identified that looked at SNS activity, as compared to PNS activity. This may explain why most significant moderators were found among PNS studies. Second, the study data available were not appropriate for testing moderating effects of child gender. Gender influences on parent socialization have been documented (e.g., Klimes-Dougan et al., 2007). In fact, differential associations between parenting and PNS activity (resting HF-HRV) between boys and girls have been found (Van der Graaff et al., 2016). However, primary source studies identified included half female and half male participants on average, with very little variability. Testing sample percent female as a moderator was therefore unable to reveal any effect. Many studies did not report effect sizes separately for boys and girls, but future efforts to gather separate effect sizes for boys and girls could facilitate testing gender as a moderator. Similarly, we were unable to properly test parent gender as a moderator. This is mainly because of a lack of male parent participants in most primary source studies. Despite the increasing call for addressing the lack of father participation in developmental research, evidence suggests no significant improvements over the past decade (Parent et al., 2017). Future efforts to recruit more fathers, or to test relations between parenting and child ANS physiology separately, could help address this limitation.

Our use of ANS reactivity change scores may also be a limitation. Measuring resting, reactivity, and recovery ANS physiology (referred to as the 3 R's) provides a more comprehensive assessment of ANS functioning (Laborde et al., 2017). Too few studies provided recovery measures to facilitate a meta-analysis of the relation between parenting and ANS recovery. Increased attention to recovery measures is an

important future direction, as this has the potential to clarify the heterogeneous findings with ANS reactivity to challenge. Parent socialization of stress and affect regulation may emerge more in how a child recovers following threat, as compared to how much the child reacts initially (Miller et al., 2013). Developmental psychophysiology researchers have recommended the use of more statistically advanced methods for quantifying change (e.g., growth curve modeling), over the traditional change score (Miller, 2018). However, results using these advanced statistical methods are not easily integrated into a meta-analysis, given that growth curve modeling may differ across studies depending on timing or frequency of measurement, as well as different time-varying covariates. In addition, the focus on the activity of single branches of the ANS may limit the ability to detect associations, if parenting shapes the overall balance of sympathetic versus parasympathetic activity (Quigley and Moore, 2018). We could not identify any studies that used indices of autonomic balance, but measures of autonomic balance (e.g., cardiac autonomic balance) have been implicated in both behavioral (Alen et al., 2021) and health-related outcomes (Alen et al., 2020a; Berntson et al., 2008; Thayer et al., 2010). A clearer understanding of the relation between parenting and autonomic balance is an important future direction for research into the biological underpinnings of parental socialization of affect and self-regulation in children (Quigley and Moore, 2018).

A final limitation of this study is that the scope was limited to exclude child-driven effects on parenting and parental autonomic physiology, although undoubtedly this pathway contributes to parent and child outcomes (see for instance recent evidence by Gao et al., 2022). Future studies should examine bidirectional processes of influence from parents to child physiology, as well as from child behavior to parental physiology, in tandem with patterns of synchrony and mutual behavioral and physiological regulation within the parent-child dyad.

4.2. Conclusion

The current meta-analysis of the association between parenting and child ANS physiology revealed vast variability in both methods and results. The synthesis of these heterogenous studies resulted in an overall lack of evidence for a strong association between parenting and child ANS physiology. However, dissection of this complex literature revealed that the general non-significant results masked some important takeaways. First, experimental manipulation of parenting, through positive parenting intervention, leads to higher resting parasympathetic activity in children. Second, positive parenting is more strongly associated with higher resting PNS activity among clinical samples of youth. Higher resting HRV is consistently associated with better health outcomes across the lifespan and may predict better psychosocial functioning (Beauchaine, 2001; Thayer et al., 2010). Results from this study highlight the ANS as a potential biological mechanism through which positive parenting interventions, particularly among those most at risk, may benefit health and well-being.

Data Availability

All data and code are publicly available as described in the article.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2022.104734.

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