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Children's Respiratory Sinus Arrhythmia Reactivity During Sadness-Eliciting Tasks: A Meta-Analytic Review

Edoardo Saija^{1*}, Susanna Pallini², Antonia Lonigro¹, and Roberto Baiocco¹

¹ Department of Psychology of Development and Socialization Processes, Sapienza University of Rome, Rome, Italy

² University of Roma Tre, Rome, Italy

Abstract

Different from other negative emotions, sadness is associated with a slowing of the heart rate, and not its increase, suggesting greater parasympathetic influence that may help children achieve relief and restoration. The current meta-analysis aimed at analyzing respiratory sinus arrhythmia (RSA) reactivity during sadness-eliciting tasks. An inverse relationship between RSA reactivity, emotion regulation, and perceived sadness intensity, as well as a direct relationship between RSA reactivity and internalizing and externalizing symptomatology, were hypothesized. Twenty-two studies met the inclusion criteria, producing 6 independent effect sizes for emotion regulation, 7 for sadness intensity, 15 for internalizing problems, and 13 for externalizing problems. In total, the studies reviewed data on 2,876 individuals. Considering all studies, effect sizes were calculated on the basis of a comparison between the clinical and non-clinical samples. In the non-clinical sample, the mean effect size for emotion regulation was significant but small, and the mean effect size for externalizing problems was not significant. In contrast, in the clinical sample, the mean effect size for externalizing problems was significant but small. The effect sizes for internalizing problems and sadness intensity were not significant, though they were consistent with the predicted directions. Aside from the analysis of internalizing problems in the clinical sample, all analyses were homogeneous, confirming the consistency of the results. No publication bias was found. The paucity of studies on this topic highlights the need for further research on children's reactions to sadness through the lens of polyvagal theory.

Keywords: sadness, emotion regulation, respiratory sinus arrhythmia reactivity, deactivating reaction, children

*Corresponding author.

Edoardo Saija
Department of Psychology of
Development and Socialization Processes,
Sapienza University of Rome,
Rome, Italy
E-mail: edoardo.saija@uniroma1.it
(E. Saija)

Introduction

Sadness in children is a largely unexplored area (Saija et al., 2023; Song et al., 2018), even though this emotion—alongside other negative emotions—constitutes a natural part of child development and actively contributes to the development of adaptive and healthy behaviors (Tuck et al., 2017). Indeed, sadness is a natural emotional reaction to negative life events, and clinicians have recognized that it plays a pivotal role in contributing to social functioning and adjustment in childhood, fostering both prosociality and moral sensibility (Lomas, 2018; Miller et al., 2016; Saija et al., 2024a; Song et al., 2018). Despite these positive outcomes, much attention has been devoted to sadness as a symptom of depressive disorders, rather than as an adaptive response to specific circumstances (Lomas, 2018). Importantly, sadness differs from depression (Horwitz & Wakefield, 2007; Saija et al., 2024b; Zeman et al., 2001), and a better awareness of this difference might allow health professionals, educators, and families to develop more targeted interventions to help children cope with this emotion, rather than avoid it.

Sadness management is a component of the broader process of emotion regulation. According to Thompson (1994), emotion regulation involves both “extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals” (Thompson, 1994, p. 27). Emotions are psychophysiological processes that influence the vagus nerve, thereby indirectly affecting the heart rate (Porges, 2007). Thus, research into the physiological processes associated with emotion regulation could offer new perspectives for understanding and promoting the regulation of sadness (Graziano & Derefinko, 2013; Vasilev et al., 2009).

The Vagal Brake

While sympathetic nervous system (SNS) activity produces an accelerated heart rate, the parasympathetic nervous system (PNS) is responsible for decelerating the heart rhythm (Hamilton & Alloy, 2016). According to Porges’s *polyvagal theory*, this heart rate decrease, known as *cardiac vagal control*, functions as a vagal “brake” (Porges, 2007). The vagal system contributes to maintaining physiological homeostasis, enabling individuals to adopt flexible behavior and therefore use only the necessary resources. Specifically, in a state of rest, if the context is perceived to be safe, the vagal pathway exerts an inhibitory influence on the heart (i.e., the *vagal brake*), reducing the heart rate, blood pressure, and breathing rate, and predisposing the individual to engage in social behavior.

In turn, when individuals are reacting to a challenge or a stressful situation, the vagal brake is withdrawn, thereby activating the SNS. This increases the heart rate and makes physiological and psychological resources available to the individual, facilitating action readiness (Porges, 2007). Finally, when the stressful situation ceases, the SNS is deactivated and the PNS reactivated. This recovers the vagal brake, thereby restoring baseline levels. Thus, vagal brake activity before, during, and after experiences of stress mirrors PNS activity and

is informative of physiological flexibility and the capacity to cope with and regulate emotional challenges (Thayer & Lane, 2000). In other words, the more flexible the parasympathetic regulation, the more adaptive the individual’s emotional regulation (for a meta-analysis, see Pinna & Edwards, 2020).

Traditionally, the vagal brake and its withdrawal have been investigated in studies exploring how individuals react differently to challenging stimuli (Graziano & Derefinko, 2013). Heart rate variability (HRV) is a measure of PNS and SNS activity, and it is considered a biomarker of self-regulation ability (Porges, 2007; Thayer et al., 2012). It is also the basis for the calculation of respiratory sinus arrhythmia (RSA)—an index of the time interval changes between heartbeats, in synchrony with respiration. Specifically, heart rate increase is associated with inspiration, while heart rate decrease is linked to expiration.

Changes in RSA amplitude in response to internal and external stressors support engagement in appropriate social behavior and the management and expression of emotions, fundamental abilities for survival, to carry out actions and initiate or avoid social interactions (Ambron & Foroni, 2015). Thus, RSA provides a reliable index of behavioral and emotional reactivity. When a stressor ceases, the physiological response—or recovery—is evaluated considering the restoration of the baseline RSA (e.g., Santucci et al., 2008). In the context of stressors, adaptive vagal regulation relates to a suppression of RSA, while maladaptive vagal regulation is related to augmented RSA (Graziano & Derefinko, 2013).

RSA Index and Children’s Emotion Regulation

Research addressing the ability to regulate emotion among infants and children has traditionally considered variation in RSA amplitude a significant indicator of self-regulation and adjustment skills, although not all studies converge on this point (Porges, 2007; Sohn et al., 2022). Generally, a higher resting RSA has been shown to be associated with more adaptive outcomes (Davis et al., 2016) and better self-regulation (Graziano & Derefinko, 2013), possibly because a higher RSA at rest may allow individuals to more easily attend to environmental cues and flexibly modify their behavior (Song et al., 2018).

In contrast, evidence of the role of RSA reactivity in self-regulation is less clear. Many studies have documented that a greater RSA decrease in the context of challenging stimuli, as a result of vagal withdrawal and parasympathetic activation, is associated with improved emotion (Calkins & Keane, 2004; Hastings et al., 2008; Kiser et al., 2019), attention (Davis et al., 2016), and behavior regulation skills (Thompson et al., 2008). In other words, an RSA decrease during a challenging task may allow individuals to direct their attention and select appropriate resources to cope with the task (Davis et al., 2016; Porges, 2007). In line with this, several studies have found associations between high RSA withdrawal during cognitive or emotion-eliciting tasks and adaptive outcomes; and low RSA withdrawal during similar tasks and psychopathological outcomes. For example, a study by Gentzler et al. (2009) found a positive association between vagal withdrawal during

the exposition of sad stimuli and emotion regulation skills. Nevertheless, vagal withdrawal negatively predicted depressive symptoms. In line with this finding, Calkins et al. (2007) found less vagal withdrawal in children at risk of externalizing problems, compared to a control group. Again, a systematic review of the literature (Hamilton & Alloy, 2016) and a meta-analysis (Graziano & Derefinko, 2013) produced evidence to support the association between reduced RSA withdrawal and behavioral problems (e.g., internalizing and externalizing problems). However, other evidence has failed to document a relationship between higher RSA withdrawal and adaptive outcomes (Crowell et al., 2005; Eisenberg et al., 2012).

To explain these contradictory results, three variables may be considered: (1) the specific emotion elicited by the task (Obradović et al., 2010), (2) the intensity of RSA reactivity (Pang & Beauchaine, 2012), and (3) the presence of internalizing or externalizing problems. Indeed, regarding the main characteristics of the emotional task, the relation between RSA reactivity and adaptive outcomes appears influenced by the presence of high or low contextual adversity (Obradović et al., 2010), with higher RSA reactivity related to problematic outcomes in highly-adverse contexts yet adaptive outcomes in non-adverse contexts. However, other authors have proposed that the relationship may be contingent on the particular emotion involved in the task. Specifically, Hastings et al. (2014) distinguished fear from sadness, with the former implying increased arousal and the mobilization of resources and the latter implying decreased arousal. In normative samples, sadness has been shown to reduce, rather than increase, the heart rate (i.e., triggering a *deactivating response*) (Kreibig, 2010), suggesting a greater parasympathetic influence (Hastings et al., 2008).

According to Fortunato et al. (2013) and McNaughton and Corr (2004), it is possible to differentiate between two corresponding motivational systems: the approach system, aimed at obtaining a reward or overcoming an obstacle (and involving a behavioral and affective response); and avoidance, aimed at coping with uncertainty or risk related to negative emotions (and involving withdrawal). In both motivational systems, RSA suppression represents an adaptive reaction of the PNS; but when stimuli eliciting sadness are at play, the story is different. Specifically, the association between higher RSA suppression during emotional tasks (eliciting, e.g., fear) and better social outcomes in typically developing infants and children may not hold true for tasks involving sadness. However, greater parasympathetic activity in the context of sadness may help children effectively manage their sadness and distress, thereby fostering their relief and restoration.

Even a reaction to sadness-eliciting tasks may influence RSA reactivity: Kreibig (2010) and Arias et al. (2020) proposed that the physiological activity underpinning sadness may be linked to PNS and SNS co-activation. The author distinguished between two forms of sadness: crying and non-crying. Sympathetic activation is associated with crying sadness, whereas sympathetic-parasympathetic withdrawal is associated with non-crying sadness, partially overlapping with a deactivating sadness response. Such a physiological response—which has been documented in studies using video clips, musical excerpts, and imagery—Involves sympathetic

withdrawal and increased parasympathetic control. Indeed, in non-crying sadness, social withdrawal may orient the individual's attention to their inner state, whereas in crying sadness, the individual may be enacting help-seeking behavior (Kreibig, 2010; Verduyn et al., 2020).

Regarding the intensity of RSA reactivity, moderate levels of RSA withdrawal during challenging situations have been shown to be adaptive and positively related to executive functioning (Marcovitch et al., 2010), better school achievement (Graziano et al., 2007), and better social skills (Blair & Peters, 2003). However, very high levels of RSA withdrawal have been found to be associated with difficulties in self-regulation (Byrd et al., 2020). A moderate decrease in RSA implies competent attention focusing (Davis et al., 2016; Porges, 2007), while disproportionate RSA reactivity suggests emotional lability (Pang & Beauchaine, 2012).

Regarding internalizing and externalizing problems, Fortunato et al. (2013) hypothesized an association with reverse patterns of RSA reactivity. Specifically, the authors observed a relation between internalizing problems and higher RSA withdrawal during a video task eliciting fear and sadness; and between externalizing problems and lower RSA withdrawal during a video task eliciting happiness. Furthermore, in their longitudinal study, Hinnant and El-Sheikh (2013) recorded a higher likelihood of developing moderately elevated internalizing symptoms but low externalizing symptoms among children exhibiting stronger RSA withdrawal during stressful social tasks.

Crowell et al. (2005) found that adolescents engaging in parasuicidal behavior showed greater RSA reactivity in response to negative mood induction, compared to controls. These inconclusive results in the literature may be explained by the heterogeneity among tasks used to induce stress between studies, as observed by Graziano and Derefinko (2013) and Hamilton and Alloy (2016), in their review and meta-analysis, respectively. Indeed, in Beauchaine's (2001; 2012) reviews, high RSA withdrawal in the context of emotion-eliciting stimuli was associated with both externalizing and internalizing problems. Accordingly, the type of emotion elicited by the task and the internalizing or externalizing nature of the psychopathology may interact.

Study Aim

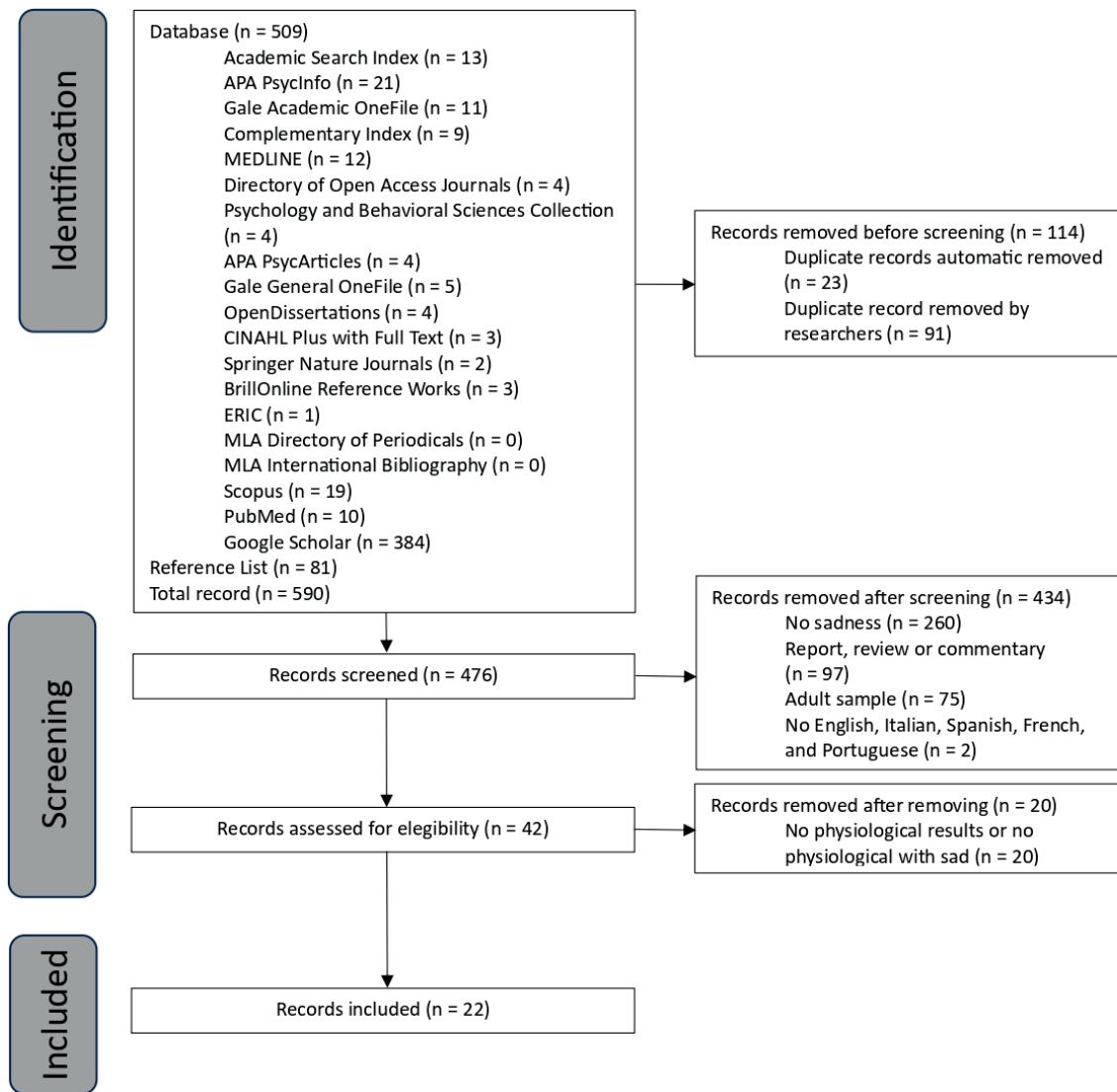
Given the conflicting findings on RSA reactivity depending on the type of emotion involved in the challenging task proposed to children, the present study aimed at reviewing studies focused on the specific emotion of sadness. As described above, sadness—in contrast to other negative emotions—is associated with a deactivating response (Kreibig, 2010) that decelerates the heart rate, thereby quieting and decreasing arousal. This parasympathetic activity may help children regulate sadness and cope with distress, and subsequently prevent overwhelm (Fortunato et al., 2013; McNaughton & Corr, 2004). Consequently, we expected to find an inverse relationship between RSA reactivity and emotion regulation ability, but a positive relationship between RSA reactivity and externalizing or internalizing symptomatology, during sadness-eliciting tasks.

Following Kreibig (2010), we attempted to analyze the specific pattern of RSA reactivity to sadness, with respect to children's: (a) emotion regulation ability; (b) sadness intensity; and (c) normative, internalizing, and externalizing characteristics.

Specifically, the hypotheses were as follows: First, considering evidence that sadness decreases arousal (Hastings et al., 2014) and triggers avoidance (Fortunato et al., 2013; McNaughton & Corr, 2004), we expected that lower RSA reactivity during sadness-eliciting tasks would relate to increased emotion regulation abilities. We did not have clear expectations about whether this pattern of relationship would vary between the non-clinical and clinical samples. Second, given that different reactions to sadness-eliciting tasks have been shown to influence RSA reactivity (Kreibig, 2010), we expected that sadness intensity would relate to RSA reactivity. In more detail, we hypothesized that the higher the intensity of sadness perceived, the higher children's need would be to maintain homeostasis and promote restoration through parasympathetic activation. Consequently, we expected to find an inverse relationship between sadness intensity and RSA reactivity. Third, regarding internalizing and externalizing symptomatology, following Crowell et al. (2005) and Fortunato et al. (2013)

for internalizing problems and Morris et al. (2020) and Pang and Beauchaine (2012) for externalizing problems, as well as the reviews of Beauchaine (2001, 2012) for both internalizing and externalizing symptomatology, we expected to find a positive association between internalizing and externalizing symptomatology and RSA withdrawal during sadness-eliciting tasks. Additionally, we hypothesized that if lower RSA reactivity (and a consequent deactivating reaction) were related to increased emotion regulation abilities during sadness-eliciting tasks, children with internalizing or externalizing problems, characterized by dysregulated emotions (see, e.g., Aldao et al., 2016; Beauchaine, 2012), would have higher RSA reactivity than children with no internalizing or externalizing problems.

Fig. 1. PRISMA diagram summarize search results and studies inclusion



were located in the Academic Search Index, PsycInfo, Gale Academic OneFile, Complementary Index, MEDLINE, Directory of Open Access Journals, Psychology and Behavioral Sciences Collection, PsycArticles, Gale General OneFile, OpenDissertations, CINAHL Plus with Full Text, Springer Nature Journals, BrillOnline Reference Works, ERIC, MLA Directory of Periodicals, MLA International Bibliography, Scopus, PubMed databases. After duplicates were removed, 32 abstracts remained for review. A further 384 records were identified through Google Scholar. Finally, the reference lists of the articles found within the online databases and 81 additional articles were screened to identify further relevant studies. Ultimately, from the 590 initial records, 476 were screened and 22 were selected. Figure 1 describes the characteristics of the included studies (per PRISMA guidelines; Page et al., 2020).

Keywords

The search terms used (considering only study abstracts) included: "skin conductance," "sinus arrhythmia," "cardiac autonomic regulation," "heart rate," "vagal," parasympathetic or sympathetic, "sympathetic flexibility," RMSSD or RSA or psychophys*, "blood pressure," and "autonomic nervous system." These keywords were used in combination with the words: "emotion regulation," or coping or cope; sad*, or unhappiness or sorrow; and child* or adolescent. Considering the breadth of results initially produced by Google Scholar, the keyword string was modified as follows: "respiratory sinus arrhythmia" AND RMSSD AND RSA AND "autonomic nervous system" AND "emotion regulation" OR coping AND sad* AND child* OR adolescent*.

Inclusion Criteria

The study inclusion criteria were as follows: (1) empirical; (2) using indices of HRV entailing respiration (or RSA) in the framework of an emotional induction paradigm (Laborde et al., 2017); (3) reporting RSA at rest and during a challenging task (i.e., estimating RSA reactivity via the difference between RSA during a challenging task and baseline RSA; Thayer et al. 2012); (4) considering RSA reactivity as a parameter of PNS activity (i.e., considering both RSA during the sadness-eliciting task and baseline RSA); (5) using specific stimuli in a

task with a baseline recording that elicits sadness, specifically; (6) comparing RSA scores during the sadness-eliciting task with measures of sadness intensity, emotion regulation, or internalizing/externalizing problems; (7) including only children and adolescents; and (8) written in English, Italian, Spanish, French, or Portuguese.

Coding of Variables and Analysis

The selected studies were coded for demographic, methodological, and publication factors. Demographic features included the number of participants, as well as their average age and age range, gender (% male), ethnicity (% White), country of origin, and socioeconomic status, and the presence of a clinical/at-risk population. Methodological features included the research design (i.e., cross-sectional vs. longitudinal); measures of psychopathology, physiological, emotion regulation, sadness intensity, and RSA reactivity; and stressor task used to derive RSA. Publication characteristics included the author/s, year of publication, and publication status (i.e., published vs. unpublished).

The ProMeta Version 3 software (IDo Statistics-Internovi, Italy) was used to conduct all statistical analyses. First, the effect sizes were calculated for each study. In detail, correlation coefficients were converted into Fisher z scores, to correct for the non-linearity of extreme correlation coefficients. Subsequently, Fisher z scores were transformed into Cohen's d scores, to facilitate interpretation. As conventionally established, Cohen's d values of $|.20|$ were considered small, $|.50|$ were considered medium, and $|.80|$ were considered large (Cohen, 1988).

A random effects model was used for all analyses. Heterogeneity among the studies was evaluated using Q and I^2 . In detail, a significant Q was considered indicative of study heterogeneity, and I^2 quantified this heterogeneity in terms of the percentage of variability across studies (Higgins et al., 2003). A sensitivity analysis was used to verify the robustness of the study findings, observing how the overall effect size changed when each study was removed. Finally, Egger's linear regression was applied and Kendall's tau was computed to estimate publication bias. Due to an insufficient number of studies, the moderator analysis was not computed. Table 1 presents the main characteristics of the included studies.

Tab. 1. Characteristics of the studies and outcomes

Outcome	Authors	Sample type	Clinical features	n	Age range	Age M	Male %	White %	Task used for inducing sadness	Outcome measure
<i>Sadness Intensity</i>										
	Borelli et al., 2014	Non-clinical	/	103	8-12	9.83	51.0	34.0	Vignettes	SAM
	Davis et al., 2016	Non-clinical	/	101	5-6	5.81	45.5	90.1	Film	Self-report of emotions
Fiskum et al., 2019		Non-clinical	/	28	9-13	10.9	50.0	92.8	Film	SAM
		Clinical	INT	32	9-13	10.3	50.0	92.8		
	Gatzke-Kopp et al., 2012	Clinical	EXT	207	5	5	66.0	8.0	Film	Emotional scoring
	Marsh et al., 2008	Mixed	EXT/NC	54	9-13	10.5	100.0	83.3	Film	EBCS

Outcome	Authors	Sample type	Clinical features	n	Age range	Age M	Male %	White %	Task used for inducing sadness	Outcome measure
Emotion regulation	Morris et al., 2020	Non-clinical	/	99	5-13	9.08	56.6	9.3	Film	SAM
		Clinical	EXT	160	5-13	8.78	78.8	9.3		
	Musser et al., 2011	Non-clinical	/	34	7-9	8.12	62.5	71.0	Film	SAM
		Clinical	EXT	32	7-9	7.91	35.3	64.7		
	Fiskum et al., 2019	Non-clinical	/	28	9-13	10.9	50.0	92.8	Film	ERC
		Clinical	INT	32	9-13	10.3	50.0	92.8		
	Gentzler et al., 2009	Non-clinical	/	39	6-13	7.93	53.8	56.9	Film	FMC
		Clinical	INT	26	6-13	7.93	53.8	56.9		
	Kovacs et al., 2016	Clinical	INT	178	11-19	17.01	63.0	93.0	Film	FMC
	Miller et al., 2006	Non-clinical	/	62	4	4	45.0	67.0	Videoclip	Observed behavior Emotion regulation repertoire interview
Internalizing problems	Quiñones-Camacho & Davis, 2018	Non-clinical	/	144	4-9	6.88	48.0	18.0	Disappoint task	
	Vasilev et al., 2009	Mixed	INT/NC	207	8-12	9.8	65.6	62.2	Film	DERS
	Borelli et al., 2014	Non-clinical	/	103	8-12	9.83	51.0	34.0	Vignettes	CDI
	Crowell et al., 2005	Non-clinical	/	23	14-18	15.3	0.0	74.0	Film	CBCL
		Clinical	INT	23	14-18	15.3	0.0	74.0		
	Fiskum et al., 2019	Non-clinical	/	28	9-13	10.3	50.0	92.8	Film	CBCL
		Clinical	INT	32	9-13	10.9	50.0	92.8		
	Fortunato et al., 2013	Mixed	EXT/NC	339	6-10	6.03	64.3	9.0	Film	SDQ
	Gatzke-Kopp et al., 2012	Clinical	EXT	207	5	5	66.0	8.0	Film	SDQ
	Gentzler et al., 2009	Non-clinical	/	39	6-13	7.93	53.8	56.9	Film	CBCL
		Clinical	INT	26	6-13	7.93	53.8	56.9		K-SADS
Externalizing problems	Hastings et al., 2000	Non-clinical	/	26	4-5	4.55	63.0	82.7	Vignettes	CBCL
		Clinical	EXT	31	4-5	4.55	63.0	82.7		
	Hasting et al., 2014	Clinical	INT	171	11-16	15.98	49.1	71.0	Film	YSR
	Kovacs et al., 2016	Clinical	INT	178	11-19	17.01	63.0	93.0	Film	CBCL
		Non-clinical	/	23	9-13	10.5	100.0	83.3		
	Marsh et al., 2008	Clinical	INT Depression	31	9-13	9.8	100.0	83.3	Film	CBCL
		Clinical		31	9-13	9.8	100.0	83.3		
	Miller et al., 2016	Non-clinical	/	180	4-6	Miss	51.4	78.8	Vignettes	CBCL
	Pang & Beauchaine, 2012	Mixed	EXT/NC	207	8-12	9.9	Miss	Miss	Film	CSI
	Ramos, 2019	Clinical	INT Depression	31	3-5	5	42.0	29.0	Film	CBCL
		Clinical	INT Anxiety	31	3-5	5	42.0	29.0		
	Swartz, 2012	Mixed	INT/NC	31	8-12	10.5	45.2	38.7	Film	BASC-2
	Ugarte et al., 2021	Mixed	EXT/NC	180	4-6	5.58	52.7	78.7	Vignettes	CBCL
	Cline, 2013	Clinical	EXT	99	8-11	9.87	48.5	0.0	Videoclip	CASI-4

Outcome	Authors	Sample type	Clinical features	n	Age range	Age M	Male %	White %	Task used for inducing sadness	Outcome measure
	Fortunato et al., 2013	Mixed	EXT/NC	339	6-10	6.03	64.3	9.0	Film	SDQ
	Gatzke-Kopp et al., 2012	Clinical	EXT	207	5	5	66.0	8.0	Film	SDQ
	Hastings et al., 2000	Non-clinical	/	26	4-5	4.55	63.0	82.7	Vignettes	CBCL
	Hastings et al., 2014	Clinical	INT	171	11-16	15.98	49.1	71.0	Film	CBCL
	Marsh et al., 2008	Mixed	EXT/NC	54	9-13	10.1	100.0	83.3	Film	CBCL
	Miller et al., 2006	Non-clinical	/	62	4	4	45	67.0	Videoclip	CBCL
	Miller et al., 2016	Non-clinical	/	180	4-6	Miss	51.4	78.8	Vignettes	CBCL
	Morris et al., 2020	Non-clinical	/	99	5-13	9.08	56.6	9.3	Film	DBDRS
	Musser et al., 2011	Clinical	EXT	160	5-13	8.78	78.8	9.3	Film	K-SADS
	Pang & Beauchaine, 2012	Mixed	EXT/NC	207	8-12	9.9	Miss	Miss	Film	CSI
	Swartz, 2012	Mixed	INT/NC	31	8-12	10.48	45.2	38.7	Film	BASC-2
	Ugarte et al., 2021	Mixed	EXT/NC	180	4-6	5.58	52.7	78.7	Vignettes	CBCL

Note. 1) Definition of the tools coded in the table, divided for outcome: Sadness intensity: Emotional Behavior Coding System (EBCS, Gross, 1996); Self-assessment manikin (SAM, Bradley & Lang, 1994). Emotion regulation: Difficulties in Emotion Regulation Scale (DERS, Gratz & Roemer, 2004); Emotion Regulation Checklist (ERC, Shields & Cicchetti, 1997); Feelings and My Child (parent-rated version of the Feelings and Me Questionnaire, FMC, Kovacs, 2000). Internalizing and externalizing problems: Behavior Assessment System for Children-2nd edition (BASC-2, Reynolds & Kamphaus, 2004); Child and Adolescent Symptom Inventory-4: Parent Checklist (CASI-4, Gadow & Sprafkin, 2002); Child Behavior Checklist and Youth Self-Report (CBCL & YSR, Achenbach, 1991); Children's Depression Inventory (CDI, Kovacs, 1992); Child Symptom Inventory (CSI, Gadow & Sprafkin, 1997); Disruptive Behavior Disorders Rating Scale (DBDRS, Pelham et al., 1992); "Kiddie" Schedule for Affective Disorders and Schizophrenia (KSADS, Kaufman et al., 1997); Strengths and Difficulties Questionnaire (SDQ, Goodman, 1997). 2) EXT = Externalizing; INT = Internalizing; NC = Non-clinical.

Results

Twenty-two studies published between 2000–2024 met the inclusion criteria, providing data for 2,876 participants (mean age 8.12 years; age range 3–18). Of these participants, 1,695 represented clinical or at-risk individuals. All studies explored the relationship between RSA reactivity and sadness. Seven studies investigated the relation with sadness intensity measures, 6 with emotion regulation, 15 with internalizing problems, and 13 with externalizing problems. Across all studies, effect sizes were calculated for the clinical and non-clinical samples. Table 2 presents the condensed results.

RSA Reactivity to Sadness Stimuli

RSA reactivity (i.e., RSA during the sadness-eliciting task minus baseline RSA) as an index of PNS activation was considered, and studies that measured only RSA at baseline were excluded. The tasks used to derive RSA reactivity scores included: watching a sad movie/video clip or reading sad vignettes; and being offered a toy that is broken. Following Gross and Levenson (1995), seven studies (Crowell et al., 2005; Hastings et al., 2014; Kovacs et al., 2016; Marsh et al., 2008; Pang & Beauchaine, 2012; Swartz, 2012; Vasilev et al., 2009) showed a sad scene from the film *The Champ*. Five studies (Fiskum et al., 2019; Fortunato et al., 2013; Gatzke-Kopp et al., 2012; Gentzler et al., 2009;

Ramos, 2019) showed a clip from *The Lion King*. Two studies (Morris et al., 2020; Musser et al., 2011) showed scenes from the film *Homeward Bound* and two studies (Davis et al., 2016; Fiskum et al., 2019) showed clips from *The Land Before Time*. Finally, one study (Hastings et al., 2014) presented clips from the film *Steel Magnolias*. Two studies used more generic video clips to evoke sadness: Cline (2013) showed a video of a child losing its pet bird, whereas Miller et al. (2006) did not specify which video clip was used. Four other studies used vignettes to evoke sadness in children. Among these, three adopted the mood induction framework (Hastings et al., 2000; Miller et al., 2016; Ugarte et al., 2021) and one employed the Laboratory Stressor DV Paradigm (Borelli et al., 2014). Finally, one study (Quiñones-Camacho & Davis, 2018) used a task in which the child was offered a broken toy.

Emotion Regulation

Eight of the 21 studies explored emotion regulation, using several measures. Fiskum et al. (2019) applied the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997); Gentzler et al. (2009) and Kovacs et al. (2016) utilized the Feelings and My Child and Feelings and Me (FMC; Kovacs, 2000); Vasilev et al. (2009) employed the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004); Davis et al. (2016) administered an ad hoc test named

Discrete Emotions and Cognitive Emotion Regulation Strategies; and Swartz (2012) applied the Roberts-2 (Roberts & Gruber, 2005). Finally, Miller et al. (2006) used an observation method, whereas Quiñones-Camacho and Davis (2018) administered interviews to explore children's emotion regulation strategies.

Regarding the non-clinical sample, the results confirmed the first hypothesis. Within this population, the mean effect size for emotion regulation was significant but small, with $d = .29$ (95% CI: .05; .53, $k = 5$, $n = 342$). No significant heterogeneity was found, $Q(4) = 4.48$, $p = .345$; $I^2 = 10.76$. The sensitivity analysis revealed that the mean effect size lost significance when the studies by Vasilev et al. (2009), $d = .21$ (95% CI: -.03; .46, $k = 4$, $n = 273$) and Gentzler et al. (2009), $d = .23$ (95% CI: .00; .46], $k = 4$, $n = 303$) were removed. Conversely, when the study by Quiñones-Camacho and Davis (2018) was removed, the mean effect size increased, $d = .45$ (95% CI: .16; .75, $k = 4$, $n = 198$). Egger's test (*intercept* = 2.15, $t = 1.64$, $p = .199$) and *Kendall's tau* ($Z = .49$, $p = .624$) confirmed no publication bias. In the clinical sample, effect size was not calculated due to an insufficient number of studies ($k = 3$).

Externalizing and Internalizing Problems

Among the selected studies, 14 investigated participants with internalizing problems ($n = 1,830$) and 13 investigated participants with externalizing problems ($n = 1,936$). The Child Behavior Checklist and the Youth Self Report (CBCL, YSR; Achenbach, 1991) were the most frequently used measures to assess internalizing and externalizing problems in the following studies: Crowell et al. (2005), Fiskum et al. (2019), Gentzler et al. (2009), Hastings et al. (2000), Hastings et al. (2014), Marsh et al. (2008), Miller et al. (2006), Miller et al. (2016), Ramos (2019), and Ugarte et al. (2021). Otherwise, Gentzler et al. (2009), Morris et al. (2020), and Musser et al. (2011) applied the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997), and Fortunato et al. (2013) and Gatzke-Kopp et al. (2012) used the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). Finally, five studies employed five other instruments: Borelli et al. (2014) used the Children's Depression Inventory (CDI; Kovacs, 1992); Pang and Beauchaine (2012) applied the Child Symptom Inventory (CSI; Gadow & Sprafkin, 1997); Kovacs et al. (2016) administered the Interview Schedule for Children and Adolescents: Diagnostic version (ISCA-D; Kovacs et al., 2015); Swartz (2012) used the Behavior Assessment System for Children–2nd edition (BASC-2; Reynolds & Kamphaus, 2002); and Cline (2013) applied the Child and Adolescent Symptom Inventory-4: Parent Checklist (CASI-4; Gadow & Sprafkin, 2002).

Externalizing Problems

Regarding the non-clinical sample, the mean effect size for externalizing problems was not significant, with $d = -.02$ (95% CI: -.15; .10, $k = 10$, $n = 1,212$). The studies were homogeneous, as indicated by $Q(9) = 9.95$, $p = .354$, and $I^2 = 9.57$. The sensitivity analysis revealed very consistent results, and Egger's test (*intercept* = .49, $t = 0.58$, $p = 0.581$) and *Kendall's tau* (Z

= .80, $p = .421$) confirmed no publication bias. In the clinical sample, the mean effect size for externalizing problems was significant but small, with $d = .21$ (95% CI: .00; .41, $k = 6$, $n = 672$), and in the hypothesized direction. No significant heterogeneity was found, as shown by $Q(5) = 7.20$, $p = .206$, and $I^2 = 30.57$.

The sensitivity analysis revealed that the mean effect size became insignificant when the following studies were removed: Morris et al. (2020), $d = .14$ (95% CI: -0.06; 0.35, $k = 5$, $n = 512$); Gatzke-Kopp et al. (2012), $d = .16$ (95% CI: -.13; .44], $k = 5$, $n = 465$); and Hastings et al. (2014), $d = .17$ (95% CI: -.11; .44, $k = 5$, $n = 501$). Conversely, the mean effect size increased when the following studies were respectively removed: Cline (2013), $d = .29$ (95% CI: .12; .46, $k = 5$, $n = 573$); and Hastings et al. (2000), $d = .25$ (95% CI: .07; .43, $k = 5$, $n = 641$). *Egger's test* (*intercept* = -1.12, $t = -1.05$, $p = .355$) and *Kendall's tau* ($Z = -.94$, $p = .348$) revealed no publication bias.

Internalizing Problems

Considering internalizing problems, the findings were mixed. Of the 11 studies with non-clinical samples, 7 produced an effect size in the hypothesized direction, with greater RSA reactivity correlating with greater internalizing symptoms. In contrast, three studies failed to find a relationship between RSA reactivity and internalizing symptoms, and one showed an inverse relationship. The mean effect size for internalizing problems was not significant, with $d = .09$ (95% CI: -.05; .23, $k = 11$, $n = 1179$). The results of $Q(10) = 11.98$, $p = .286$, and $I^2 = 16.53$ revealed a homogeneous set of studies. Finally, the sensitivity analysis suggested the stability of the findings, and *Egger's test* (*intercept* = .84, $t = 1.23$, $p = .250$) and *Kendall's tau* ($Z = 1.01$, $p = .312$) confirmed no publication bias.

In the clinical sample, the mean effect size for internalizing problems was not significant, with $d = .12$ (95% CI: -.19; .42, $k = 11$, $n = 792$). Significant heterogeneity was found, as indicated by $Q(10) = 32.17$, $p < .001$, $I^2 = 68.92$. The sensitivity analysis revealed the stability of the study findings, and *Egger's test* (*intercept* = .13, $t = .11$, $p = .916$) and *Kendall's tau* ($Z = .70$, $p = .484$) revealed no publication bias.

Sadness Intensity

Six studies used a specific measure to assess perceived sadness intensity after the presentation of the sad stimuli. Four (Borelli et al., 2014; Fiskum et al., 2019; Morris et al., 2020; Musser et al., 2011) used the Self-Assessment Manikin (SAM; Bradley & Lang, 1994), while one (Marsh et al., 2008) involved child observation by two trained research assistants who coded the presence and intensity of children's sadness using the Emotional Behavior Coding System (EBCS; Gross, 1996). The final study (Davis et al., 2016) used self-report sadness scales.

Six effect sizes in non-clinical samples and four in clinical samples were derived from seven studies. Regarding the non-clinical samples, in four studies, the effect size was in the hypothesized direction (with greater RSA reactivity associated with less intense sadness), and one study found no relationship between these variables (of note, this study used only one

Tab. 2. Meta-analysis results

	Outcome	Random-effects model			95% CI		Heterogeneity	
		k	n	d	LL	UL	Q	I ²
Sadness intensity	Non-clinical sample	6	419	.05	-.14	.25	2.29	.000
	Clinical sample	/	/	/				
Emotion regulation	Non-clinical sample	5	342	.29	.05	.53	4.48	10.76
	Clinical sample	/	/	/				
Internalizing problems	Non-clinical sample	11	1179	.09	-.05	.23	11.98	16.53
	Clinical sample	11	792	.12	-.19	.42	32.17***	68.92
Externalizing problems	Non-clinical sample	10	1212	-.02	-.15	.10	9.95	9.57
	Clinical sample	6	672	.21	.00	.41	7.20	30.57

*** $p < .001$

question to measure children's perceived sadness intensity) (Davis et al., 2016). The overall effect size for sadness intensity measures was not significant, with $d = .05$ (95% CI: $-.14$; $.25$, $k = 6$, $n = 419$). The results of the studies were homogeneous, $Q(5) = 2.29$, $p = .807$; $I^2 = .000$; and consistent, as derived from the sensitivity analysis. *Egger's test (intercept = 1.23, $t = 1.20$, $p = .298$) and Kendall's tau ($Z = 1.69$, $p = .091$) were not significant, suggesting a lack of publication bias.* No effect size was computed for the overall non-clinical sample, due to the inadequate number of studies ($k = 4$).

Discussion

The current review and meta-analysis aimed at analyzing RSA reactivity during sadness-eliciting tasks, hypothesizing an inverse relationship between RSA reactivity, emotion regulation, and perceived sadness intensity; and a direct relationship between RSA reactivity and problem behaviors (e.g., internalizing and externalizing symptomatology). Confirming the first hypothesis, the results showed that, in the non-clinical sample, lower RSA reactivity to sadness-eliciting tasks was associated with increased emotion regulation abilities, with a small but significant effect size.

The correlation between lower RSA reactivity and better emotion regulation implies an ability to activate PNS in order to restore and calm oneself, and thereby gain relief from sadness (Fortunato et al., 2013; Hastings et al., 2014; McNaughton & Corr, 2004). This amounts to a deactivating response (Kreibig, 2010). Such parasympathetic activity may help children effectively manage their sadness and distress, thereby preventing overwhelm (Fortunato et al., 2013; McNaughton & Corr, 2004). In the experimental situations, care- and consolation-seeking behavior was not easily induced; thus, children did not ask for help by crying, but used self-care strategies to cope with the sadness-eliciting tasks (Kreibig, 2010; Verduyn et al., 2020). Of note, the overall effect size for reactivity to sadness-eliciting tasks was not consistent.

Considering emotion regulation, the sensitivity analysis showed that the studies by Vasilev et al. (2009) and Gentzler et al. (2009) carried important weight. When these records were excluded, the effect size lost significance. Furthermore, when the study by Quiñones-Camacho and Davis (2018) was removed, the effect size increased from small to strong. In the remaining four studies, the sadness-eliciting tasks

involved viewing a sad film or video clip; while in the study by Quiñones-Camacho and Davis (2018), the stimulus was quite different, with children being offered a broken toy. It is possible that this task elicited not only sadness in children, but also irritation and annoyance—emotions that are more related to SNS, rather than PNS, activation (Fortunato et al., 2013; McNaughton & Corr, 2004).

Partially confirming the second hypothesis, the results showed that externalizing (but not internalizing) symptoms were associated with higher RSA reactivity to sadness-eliciting tasks. Indeed, children who exhibit externalizing problems are likely to have difficulty regulating their emotions (Aldao et al., 2016; Beauchaine, 2012), as they are typically high in physiological arousal and demonstrate impaired cognitive control over emotional behavior (Matthys et al., 2013). Consequently, enhanced SNS activation would make them highly physiologically reactive to emotional situations (Gatzke-Kopp et al., 2012) and unable to use deactivating strategies during sadness-eliciting tasks. In the present analysis, the effect size was small but significant, but only in the clinical sample. It is possible that the study of this relationship in clinical samples, relative to non-clinical samples, places more emphasis on this relationship.

In the sensitivity analysis for studies exploring externalizing behavior in clinical samples, the overall effect size became non-significant when the study by Morris et al. (2020) was removed. The same result was not found for the study by Musser et al. (2011), whose method was replicated by Morris et al. (2020). Perhaps the different diagnostic criteria used in these studies (i.e., DSM IV vs. DSM 5) and their different sample sizes impacted the mean effect size. Furthermore, the overall effect size increased when the studies by Hastings et al. (2000) and Cline (2013) were respectively removed. However, this increase was very weak, and the mean effect size remained low.

Regarding the third hypothesis (on the relationship between RSA reactivity and children's perceived sadness intensity during sadness-eliciting tasks), the direction of the effect sizes was not significant. This pattern, although not statistically significant, suggests that greater RSA suppression may be associated with lower levels of self-reported sadness. Such a tendency aligns with theoretical frameworks emphasizing the role of autonomic flexibility—reflected in vagal withdrawal—as a physiological marker of emotion regulation capacity (Beauchaine, 2001; Porges, 2007). In this view, children exhibiting greater RSA suppression during sadness-eliciting tasks may be better equipped to modulate their subjective emotional experience,

resulting in attenuated perceptions of sadness. However, all but one study (Davis et al., 2016, which measured perceived sadness using only one question) showed an inverse relationship between sadness intensity and RSA reactivity. The discrepant result reported by Davis et al. (2016) might be attributable to methodological limitations, particularly the use of a single-item measure to assess perceived sadness, which may have lacked the sensitivity to capture individual differences in emotional experience. Taken together, although the findings do not support a robust association at the meta-analytic level, the consistent directionality across most studies and the theoretical plausibility of the inverse association indicate that RSA reactivity could play a functional role in the modulation of perceived sadness intensity in children. Further research employing more refined and reliable measures of self-reported affect is warranted to clarify the nature of this relationship.

The absence of publication bias suggests that the present findings are trustworthy. Moreover, all sets of analyses (except for those regarding internalizing problems in the clinical sample) were homogeneous, confirming the consistency of the results. With regard to internalizing problems in the clinical sample, heterogeneity across studies was found, and the insufficient number of studies prevented us from explaining this heterogeneity using moderator analysis.

The main limitation of the current meta-analysis is the small number of studies considered, which prevented us from testing the potential moderating effects of gender, age, and measures that might better explain the mechanism involved in the relationship between sadness and RSA reactivity. At the same time, the meta-analysis highlights the paucity of studies in this field and underlines the need for further research. Furthermore, the significant (but small) effect sizes represent a limitation of the meta-analysis. This limitation also applies to current physiological research, due to the complexity of measuring, analyzing, and integrating multiple physiological systems simultaneously (Graziano & Derefinko, 2013).

Despite these limitations, the present findings contribute to the growing body of literature suggesting that parasympathetic activity may serve as a physiological marker of sadness regulation in children. These results hold important clinical and developmental implications. As highlighted in the introduction, a deeper understanding of the physiological underpinnings of emotion regulation may open new avenues for identifying children who are at greater risk for dysregulated sadness responses. Furthermore, such knowledge may inform the development of targeted intervention programs that incorporate physiologically based strategies, such as mindfulness (Rowland et al., 2022) or progressive muscle relaxation (Vaudreuil et al., 2024), aimed at enhancing vagal regulation and, in turn, improving emotional functioning in early childhood. Future research integrating physiological monitoring in both typical and clinical populations may provide valuable tools for early diagnosis and the development of personalized prevention strategies.

Even a cautious reading of the present results reveals the necessity of investigating RSA withdrawal with respect to the particular emotion elicited, and the specific need to explore RSA reactivity during tasks eliciting sadness. Hopefully, the present meta-analysis will represent a starting point for

further research on children's reactions to sadness, in the polyvagal framework.

Ethical Approval

Not applicable

Data Availability Statement

The data necessary to reproduce the analyses presented here are not publicly accessible. The analytic code necessary to reproduce the analyses presented here is not publicly accessible. The materials necessary to replicate the findings presented here are not publicly accessible. The analyses presented here were not preregistered.

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Conflict of Interests

All authors declare that they have no conflict of interest.

Author Contributions

Conceptualization: Edoardo Saija, Susanna Pallini, and Roberto Baiocco; methodology: Susanna Pallini and Antonia Lonigro; data collection: Edoardo Saija; validation: Roberto Baiocco; formal analysis and investigation: Antonia Lonigro and Edoardo Saija; writing—original draft preparation: Edoardo Saija and Susanna Pallini; writing—review and editing: Antonia Lonigro; supervision: Roberto Baiocco, Antonia Lonigro, and Susanna Pallini.

Supplementary material

All supplementary materials related to the study are available at the following OSF repository: <https://osf.io/wqxc3/?view-only=59dda07e68bd429f9d9168e9b9a3c2bc>

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