A Meta-Analysis on the Autonomic Nervous System Correlates of Human Emotional Crying

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All data and syntax are available at: https://osf.io/4j8cu/

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Abstract

Various theories have tried to make sense of human emotional crying, predicting different psychophysiological correlates. In the present paper, we systematically review empirical studies on this relationship. Our preregistered meta-analysis identified 230 effect sizes across 12 articles (*N*=1,017), focusing on 18 measures reflecting cardiovascular, respiratory, electrodermal, and temperature-related activity. We find evidence of increased sympathetic activity at crying onset transforming into sympathetic withdrawal post crying. We also find weaker evidence for increased parasympathetic activity after crying. We conclude that none of the specific theories, but rather a combination of them best accounts for the observed results – emotional crying seems to fulfill an intraindividual function regulating autonomous nervous system activity. Importantly, different operationalizations and methods complicated comparisons, calling for standardization.

Keywords: emotional crying; ANS activity; sympathetic activity; parasympathetic activity; meta-analysis

A Meta-Analysis on the Autonomic Nervous System Correlates of Human Emotional Crying

Emotional crying is considered a uniquely human phenomenon (Vingerhoets, 2013). People cry for a multitude of reasons including experiences of grief or sadness, such as when losing a dear person, but also moments of intense happiness, such as observing an achievement of one's own child (Denckla et al., 2014; Zickfeld, Seibt, et al., 2020). While the functions of crying have been recently studied more systematically (Bylsma et al., 2008, 2011; Zickfeld et al., 2021), there is little consensus on *why* humans actually cry (Gračanin et al., 2018).

The three most influential theories of emotional crying have argued that people cry because they feel aroused or distressed (*arousal account,* Averill, 1968; Gross et al., 1994), because it promotes homeostasis by restoring the bodily balance (*catharsis account,* Bindra, 1972; Efran & Spangler, 1979; Labott & Martin, 1987), or because people feel vulnerable or helpless (*helplessness account,* Miceli & Castelfranchi, 2003; Vingerhoets & Bylsma, 2016). Each of these theories assume different autonomic nervous system (ANS) activity changes during crying. For example, the *arousal account* predicts an activation of the sympathetic nervous system when people cry. On the other hand, the *catharsis* and *helplessness* account expect higher activation of antagonistic processes – the parasympathetic nervous system. These opposing predictions are both supported by empirical findings, suggesting that crying is associated with high sympathetic activity (e.g., Gross et al., 1994) and high parasympathetic activity (e.g., Rottenberg et al., 2003). In order to resolve this empirical paradox, researchers have argued that crying is accompanied by both high sympathetic and parasympathetic activation sequentially (Hendriks et al., 2007).

In the present research, we provide a first systematic quantitative review of the autonomic nervous system correlates of emotional crying. Our findings shed light on the empirical validity

of different theories of emotional crying focusing on intraindividual processes and provide insight on whether crying is associated with increased sympathetic or parasympathetic activity or relies on the activation of both systems.

Emotional Crying

We focus on human emotional crying, broadly defined as an emotional response characterized by the secretion of (visual) tears that can be accompanied by changes in facial muscle contractions and vocalizations. Thereby, we focus on both acoustic aspects such as wailing or sobbing and visual aspects including moist eyes or tears.[1] Our focus is on emotional crying or tears, thereby excluding so-called *basal tears*, that are intended to keep the cornea lubricated, and *reflex tears* that are shed because of irritation to the eye (e.g., dust, sunlight; Madison & Dutton, 2019).

Functions of Emotional Crying

Researchers have argued that the persistence of emotional crying into adulthood presents a riddle (Vingerhoets & Bylsma, 2016). While (vocal) crying in infants and children serves the ultimate purposes to alert the caregivers attention and secure survival, its function across adults have been widely debated. Research typically distinguishes between a focus on the *intra*- and the *interpersonal* functions of crying, so the effects of crying for the individual and its possible communicative functions (Gračanin et al., 2018). Research on the interpersonal function has provided evidence that emotional crying and tears communicate the need for help and affiliation (Balsters et al., 2013; Hendriks et al., 2008; MacArthur & Shields, 2019; Van de Ven et al., 2017; Zickfeld et al., 2021). Criers are not only perceived as warmer and more trustworthy, but people

[1] Some researchers have separated *weeping*, as only focusing on visual characteristics, and crying, focusing on acoustic characteristics (Bellieni, 2017). We focus on both expressions as aspects of human emotional crying and will consider possible differences between these expressions in our results.

across different cultures also report increased support intentions when tears are visible (Zickfeld et al., 2021). The authors argue that crying has evolved as some type of *social glue* holding people together and securing help by others when people are in need.

Findings have been more mixed when focusing on the intrapersonal domain. Different studies have provided evidence that people typically consider crying to help them with their mood regulation and that they feel better after shedding a tear when asked in retrospective. However, when reporting their psychological state right after a crying episode their mood is similar or worse in comparison with non-criers (Cornelius, 2004; Rottenberg et al., 2008; Bylsma et al., 2008, 2011; Gračanin et al., 2014). A recent study suggests that possible cathartic effects of crying might take longer than typically assumed and are, hence, not captured by previous reports (Gračanin et al., 2015). In addition, different individual, contextual, and cultural variables moderate the effects of emotional crying on mood effects (Rottenberg et al., 2008).

The present review, focus primarily on the question how emotional crying is associated with specific physiological or psychological changes within the crier, thereby considering possible intrapersonal functions of emotional crying. Nevertheless, some of the reviewed theories highlight an interpersonal function and consider how changes in the environment might influence ANS responses during emotional crying. Before turning to specific theories of emotional crying and their predictions with regard to ANS activity, we first need to consider the physiological basis of human emotional crying.

Anatomy & Physiology of the Lacrimal Gland

What are the biological systems that control emotional crying in humans? An in-depth treatise of the different systems involved in crying is presented by Bylsma et al. (2019) and Dartt (2009). In the following, we focus on the most basic processes involved in tear secretion.

Emotional tearing is caused by stimulation of the lacrimal gland that is situated in the upper lateral quadrant of the ocular orbits. The same gland is also involved in producing reflex tears, but not basal tears (which are produced by the accessory lacrimal glands under the eyelids). The main neurotransmitters regulating the activation of the secretory cells include acetylcholine and vasoactive intestinal peptide (parasympathetic), as well as norepinephrine and neuropeptide Y (sympathetic). In general, the lacrimal gland is innervated by both sympathetic and parasympathetic nerves, though the parasympathetic processes play a more important role in triggering a secretion process (Bylsma et al., 2019). Innervation of the parasympathetic responses is supplied by the seventh cranial nerve (CN VII) and begins in the lacrimal nuclei located in the brainstem, passing the geniculate ganglion and terminating in the pterygopalatine ganglion, before becoming part of the greater superficial petrosal nerve and the vidian nerve. From the pterygopalatine ganglion, axons innervate the lacrimal gland. According to Bylsma and colleagues (2019) the main neural circuits that are involved in emotional crying represent the central autonomic network (CAN; Benarroch, 1993). The CAN represents a network of brain areas including the diencephalon, telencephalon, and brainstem. The CAN and the limbic system influence the lacrimal nuclei, which in turn triggers the lacrimal gland. However, direct evidence on the specific neurophysiological correlates of emotional crying in human is sparse (Bylsma et al., 2019).

In the present review, we focus specifically on autonomic nervous system activity (ANS), a part of the peripheral nervous system that is divided into the sympathetic, the parasympathetic, and the enteric nervous system (McCorry, 2007). We focus on the former two. The sympathetic nervous system (SNS) is considered to control *fight-or-flight* responses, while the parasympathetic nervous system (PNS) focuses on antagonistic *rest-and-digest* responses. High

SNS activity is typically associated with increased (emotional) arousal (Kreibig, 2010). There exists no perfect measure to assess SNS activity, although studies commonly consider changes in cardiovascular, electrodermal, and respiratory responses indicative of SNS changes (Cacioppo et al., 2000). For instance, increased heart rate (HR), skin conductance level or responses (SCL; SCR), and respiratory rate (RR) are often used as indicators of increased SNS activity (Kreibig, 2010; Siegel et al., 2018). Parasympathetic activity is commonly measured focusing on respiratory sinus arrythmia (RSA), an indicator of heart rate variability, assessing the variation between successive heart beats linked to respiratory activity (Laborde et al., 2017; Shaffer & Ginsberg, 2017).

To investigate the relationship between emotional crying and ANS activity, we will briefly review different theories of emotional crying and assess what specific prediction they make with regard to ANS responses in emotional crying.

Theories of Emotional Crying & ANS Activity

Based on the specific functions of emotional crying, researchers have suggested at least three different theoretical models of why people cry in everyday life, the *arousal*, *catharsis*, and *helplessness* account. These theories map differently on the specific functions of emotional crying, either highlighting intra- (*catharsis*) or interpersonal aspects (*arousal, helplessness*). In addition, they either consider changes in ANS activity as central (*arousal, catharsis*) or focus mainly on specific subjective feelings and interpretations (*helplessness*). Previous studies have emphasized the importance of when ANS responses are assessed in emotional crying (e.g., Hendriks et al., 2007; Gračanin et al., 2015), and theories make different predictions about changes before, during, and after a crying episode.

Arousal View. The idea that emotional crying is associated with high arousal has already been put forward several decades ago (Averill, 1968; Tomkins, 1963). In this view, crying is considered a generally aversive state that is related to distress. Ultimately, emotional crying is proposed to foster social cohesion because both criers and bystanders want to end the tears, thereby strengthening their bonds (Averill, 1968; Gross et al., 1994). Therefore, this theoretical view proposes that the main function of emotional crying is primarily interpersonal in nature. Notably, most of the theorizing has been based on emotional crying with regard to negative states such as sadness (Gross et al., 1994; Tomkins, 1963). It is not clear whether the arousal account assumes the same mechanism when people shed positive tears of joy (see Ishii & Shinya, 2021).

The arousal view has been supported by several psychophysiological studies (Gross et al., 1994; Kraemer & Hastrup, 1988; Sharman et al., 2019). In a systematic attempt to test the arousal against the catharsis view, Gross et al. (1994) showed a sad film to 150 participants and then compared criers with non-criers. They observed increased autonomic nervous system activity during crying with higher responses on cardiovascular, electrodermal, and respiratory measures.

Specifically, the arousal view predicts increased sympathetic activity during crying and crying onset (see Table 1). While the theory is not completely explicit on the causal direction of the relationship between emotional crying and arousal, it can be assumed that increased sympathetic activity is also expected at pre-crying. There is no specific prediction for post-crying but, Gross et al. (1994) acknowledge that crying might result in autonomic recovery after more time has passed than what has been measured in their experiment. Therefore, sympathetic withdrawal at post-crying seems a plausible prediction.

Catharsis View. The catharsis view (also *recovery view*) suggests that tears promote autonomic recovery after increased SNS activity, with the goal of physiological homeostasis (Bindra, 1972; Efran & Spangler, 1979; Frey & Langseth, 1985; Labott & Martin, 1987). That is, in stark contrast to the arousal view, tears are assumed to be related to a *catharsis* instead of high distress. There are four different main propositions how crying can be cathartic, focusing on the idea that emotional crying reduces negative affect or stabilizes mood (Bylsma et al., 2011), releases toxins (Frey & Langseth, 1985), stimulates the releases of specific hormones (Bellieni, 2017), or decreases sympathetic activity and increases parasympathetic activity (Bindra, 1972; Efran & Spangler, 1979).

First, considering the reduction of negative affect, people indicate that emotional crying improves their mood and reduces negative emotions when asked in hindsight, but not when immediately after the crying episode (Bylsma et al., 2008, 2011). Findings by Gračanin and colleagues (2015) further suggest that such positive recovery effects of crying might take several minutes to unfold. Right after watching a sad film, participants in their study reported more negative affect when they also cried (in contrast to non-criers), these differences were negligible after 20 minutes, and criers even reported less negative affect at a 90-minute follow-up. Similarly, it has been argued that crying might act as a self-soothing behavior, reflecting a homeostatic regulation of subjective and bodily responses (Gračanin et al., 2014). Further supporting the function of emotional crying in regulating mood, participants given a mild antidepressant were less likely to weep during emotional films than a placebo control group (van der Veen et al., 2012).

Second, another idea has proposed that emotional tears represent a way to release bodily toxins and thereby reduce stress and instigate recovery (Frey & Langseth, 1985). However, while

this proposition often seems to be treated as a scientific fact in newspaper and online publications (Govender, n.d.; Newhouse, 2021; Pfeifer Watson, 2017), there is no systematic evidence backing this claim up for humans to our knowledge. One study comparing emotional with reflex tears found higher concentration of proteins in the former (Frey et al., 1981), which can be at most interpreted as evidence that these types of tears differ in their composition (Gross et al., 1994).

Third, Bellieni (2017) theorized that tears flowing down the cheeks during emotional crying might stimulate skin massage and release endorphins. While (social) touch has been associated with an increased release of endorphins (Dunbar, 2010), there is no systematic evidence testing the relationship between emotional crying (with and without tears) and endorphin release to our knowledge.

Finally, other theories have proposed that emotional crying facilitates recovery because it increases parasympathetic activity (Bindra, 1972; Efran & Spangler, 1979). Based on the idea that the lacrimal gland is innervated by parasympathetic fibers (Bylsma et al., 2019), this view suggests that emotional crying transforms sympathetic into parasympathetic activation. There are some studies showing that parasympathetic activity indeed increases after emotional crying (Hendriks et al., 2007; Rottenberg et al., 2003). In one study (Rottenberg et al., 2003), respiratory sinus arrythmia, typically considered a measure of parasympathetic activity, increased for non-clinically depressed criers after watching a sad film in contrast to non-criers.

Specifically, the catharsis view suggests increased PNS activity during crying and at post-crying. In order to induce an autonomic recovery and homeostasis, we assume that this view would consider increased SNS activity at pre-crying (see Table 1).

Arousal-Catharsis View. Empirical evidence at first glance suggests support for both the arousal and the catharsis view. Recently, Hendriks et al. (2007) attempted to integrate the conflicting evidence. The authors propose that emotional crying features high arousal and increased sympathetic activity early on at pre-crying, which, over time, shifts to sympathetic withdrawal and increased parasympathetic activity inducing an autonomic recovery (Table 1). This proposition, that we term the *arousal-catharsis* view, was partly supported in their study and they emphasize that conflicting findings in the literature might have arisen because studies record their physiological measures at different time points of emotional crying.

Helplessness View. The third view suggests that emotional crying is associated with a high degree of perceived helplessness and powerlessness (Vingerhoets, 2013; Vingerhoets & Bylsma, 2016). Based on this view, situations in which people feel reduced agency and control are likely to induce emotional crying, whether they are negative or positive in nature. There is indirect evidence for this proposition in that perceiving tears facilitates inferences of helplessness (Gračanin et al., 2021; Zickfeld et al., 2021). Moreover, in a small survey study participants relatively often reported the combination of certain emotions with helplessness when thinking of crying eliciting events (Vingerhoets et al., 1997). However, to our knowledge there exists no systematic test whether emotional crying and feelings of helplessness are positively associated across different contexts. The helplessness view suggests that crying's main function lies in the interpersonal context by communicating the crier's state of helplessness to other individuals and possibly triggering social support (Zickfeld et al., 2021).

Although this view does not directly assume specific autonomic nervous system changes, we can draw on studies testing the psychophysiological correlates of states of sadness or helplessness. Previous research has distinguished *activating* from *deactivating* sadness, with the

former typically being accompanied by crying (Kreibig, 2010; Kreibig et al., 2007). In general, deactivating sadness has been characterized by sympathetic withdrawal (Kreibig et al., 2007). On the other hand, feelings of helplessness might arise for high sympathetic arousal, because the distress becomes *too much to handle*, as suggested by studies on activating sadness. Similarly, it has been speculated that helplessness is associated with increased PNS activity (Vingerhoets, 1985). Therefore, we would expect the helplessness view to expect an interaction of sympathetic and parasympathetic activity (Table 1).

Table 1. Overview of different theoretical crying models and their prediction with regard to associated ANS activity.

Theoretical Model	Functional Focus	Sympathetic Activity			Parasympathetic Activity		
		Pre	Onset	Post	Pre	Onset	Post
Arousal View	Inter	+	+	-[?]			
Catharsis View	Intra	+[?]				+	+
Arousal-Catharsis View	Inter & Intra	+	+	-			+
Helplessness View	Inter		+			+	

Note. [?]Indicates that the theory is not perfectly clear about its predictions but these could be derived from other assumptions.

Emotional Crying & the Autonomic Nervous System

According to the overview, there seems to be no one clear ANS response pattern theoretically associated with emotional crying. While the lacrimal gland, which is responsible for secreting tears, is mainly innervated by parasympathetic fibers, different theories of emotional crying make different predictions when it comes to the interplay of sympathetic and

parasympathetic responses. Similarly, empirical evidence fails to favor one specific theoretical explanation and system as can be seen from the overview in Table 1.

Unfortunately, the theories are not explicit on the causal direction of the effects. Does high arousal cause crying, does crying cause high arousal, or is the relationship bidirectional (Gross et al., 1994)? As Bylsma and colleagues (2019, p. 67) have already noted: "unpacking the peripheral psychophysiology of crying is a complex issue given that it is a complex behavior with multiple components". Studies have employed different methodologies to induce and measure emotional crying, have focused on different physiological measurements, and have considered different temporal solutions. Most importantly, it is extremely difficult to determine the exact onset of emotional crying, not to mention its offset. Studies have either focused on self-reported crying behavior, by having participants pressing a button when they feel that they start to cry or provide their ratings after watching a crying-inducing stimulus (e.g., Wassiliwizky et al., 2017) or a more objective, observational method, by recording the participants' face and letting coders determine the onset and offset of the crying response (e.g., Gross et al., 1994). Further, emotional crying might be accompanied by several (a)specific facial, respiratory (e.g., sobbing), and/or other non-verbal expressions that might influence ANS activity, thereby resulting in contradicting findings.

The Present Meta-Analysis

In the present paper, we attempt to systematically investigate the ANS correlates of emotional crying by focusing on changes in several physiological measures and their temporal relationship with emotional crying, more specifically on possible changes before, during and after a crying episode. While conducting the meta-analysis, we tried to adhere to general principles of transparency and reproducibility of meta-analyses (Lakens et al., 2016). Our

protocol, coding scheme, and analysis plan were pre-registered (https://osf.io/uq34g) and deviations are explicitly noted, all studies were coded by two independent researchers, we adhere to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Moher et al., 2009), and all data are openly available at the OSF project website (https://osf.io/4j8cu/), facilitating reproducibility.

Method

Literature Search

Database Search.

We conducted a database search from 15th to the 17th of February 2021. Based on previous theoretical and empirical studies, we employed the Boolean search:

((crying OR "emotional crying" OR tears OR "emotional tears"
OR lacrimation OR weeping)
AND
(psychophysiology OR "autonomic nervous system" OR "sympathetic
activity" OR "parasympathetic activity" OR "autonomic correlates"))

We conducted the search using two different databases. First, we searched Web of Science (n = 105) including all years and ordering articles based on relevance. Second, we employed GoogleScholar (n = 200), restricting the search to the first 20 pages based on relevance. GoogleScholar was used complementarily, to include potential unpublished reports or preprints that might not be indexed in Web of Science. Third, we also included all articles (n = 275) that cited at least one of three papers that we identified as *gold standard of physiological*

research on emotional crying a-priori, namely Gross et al. (1994), Hendriks et al. (2007), and Kraemer & Hastrup (1988). These papers were chosen because they represent influential sources assessing the ANS correlates of emotional crying from different decades. Fourth, we issued a call for published and unpublished data via various associations and mailing lists (n = 1), including the Society for Affective Science (SAS), the International Society for the Research on Emotions (ISRE), and the European Association for Social Psychology (EASP). In addition, we also contacted researchers directly who have published articles on the psychophysiology of emotional crying and asked for unpublished reports or data (n = 1). Fifth, we included one article that was published during the screening process and fit our inclusion criteria. In total, our search resulted in 583 articles.

Inclusion Criteria.

We included studies based on three different criteria. First, focusing on the independent variable, we only included studies manipulating the occurrence of emotional crying by either experimental or quasi-experimental designs. This could be achieved by comparing participants reporting crying in response to a specific stimulus or stimuli to participants reporting no tears or by comparing reactions to stimuli intended to evoke tears with a control condition. We included crying in response to both positive and negative situations and focused on emotional crying as operationalized by emotional tears, but also vocal crying. Second, we only included studies assessing actual psychophysiological variables, thereby excluding self-reported changes in physiological activity. We focused on ANS activity as summarized by Siegel et al. (2018), therefore excluding central nervous system activity measures such as fMRI or EEG or other indices not focusing on ANS activity, such as muscle contractions as assessed by EMG. Third,

we only included studies focusing on samples of non-clinical adults. If a study included both clinical and non-clinical samples, we focused exclusively on the non-clinical participants.

Emotional Crying Manipulations.

For the independent variable, we focused on different designs either experimentally or quasi-experimentally manipulating emotional crying behavior. We categorized them into three different general paradigms:

a. One-stimulus-matching: Participants are exposed to one stimulus intended to elicit crying and criers are compared to non-criers (e.g., Gross et al., 1994; Hendriks et al., 2007).
b. Multiple-stimulus-matching: Participants are shown several stimuli and criers are compared to non-criers or reactions to self-selected stimuli are compared to experimenter-selected stimuli (e.g., Mori & Iwanaga, 2017; Wassiliwizky et al., 2017).
c. Control-stimulus: Participants are exposed to a stimulus/stimuli known to elicit emotional crying and one or more stimuli intended as a control stimulus (e.g., Zickfeld, Arriaga, et al., 2020).

ANS Measures

For the dependent variable, we focused on objective measurements of ANS activity. These measurements were grouped into the following five distinct systems (Siegel et al., 2018):

a. The Cardiovascular System focuses on heart-related changes in ANS activity, including the following measures: heart rate (HR), heart rate variability (HRV), interbeat interval (IBI), cardiac output (CO), diastolic blood pressure (DBP), finger pulse amplitude (FPA), finger pulse volume (FPV), mean arterial pressure (MAP),

pulse transit time (PTT), pre-ejection period (PEP), stroke volume (SV), systolic blood pressure (SBP), and total peripheral resistance (TPR)

b. The Electrodermal System focuses on electrodermal changes including the following measures: skin conductance amplitude (SCA), skin conductance magnitude (SCM), skin conductance level (SCL), and skin conductance responses (SCRs)
c. The Respiratory System with the focus on changes related to the respiratory cycle, including the following measures: expiratory time (TE), inspiratory time (TI), respiratory rate (RR) and tidal volume (TV) or respiratory depth (RD).
d. Temperature Related changes, in particular changes related to body core or skin/ surface temperature.
e. Others, which include measures not captured by the other four systems, such as gastrointestinal changes.

An overview of all measures, their specific measurement system, and a description are provided in the Supplementary Material (Supplementary Table 1) or in Siegel et al. (2018).

Article Screening & Coding

Screening Procedure.

We performed the main screening procedure using the *revtools* package in *R* (version 0.4.1; Westgate, 2019). First, we identified possible duplicates using exact matching and fuzzy matching procedures. Possible duplicates were verified and excluded by hand. This resulted in 437 articles. The titles of these articles were then independently screened by the two authors. The second author screened 70% of all articles and the first author the remaining share. Thirty percent of all articles were screened by both authors. For the title screening, agreement between

the authors was fair (κ = .50, overall agreement 83.3%). A total of 76 articles remained after title screening. Using the same distribution proportion as for the title screening, the authors then screened the abstracts of the remaining articles for eligibility. Agreement between the authors for the abstract screening was good (κ = .66, overall agreement 85%). We retained 22 articles for the final inclusion after the abstract screening. An overview of the screening process is provided in Figure 1.

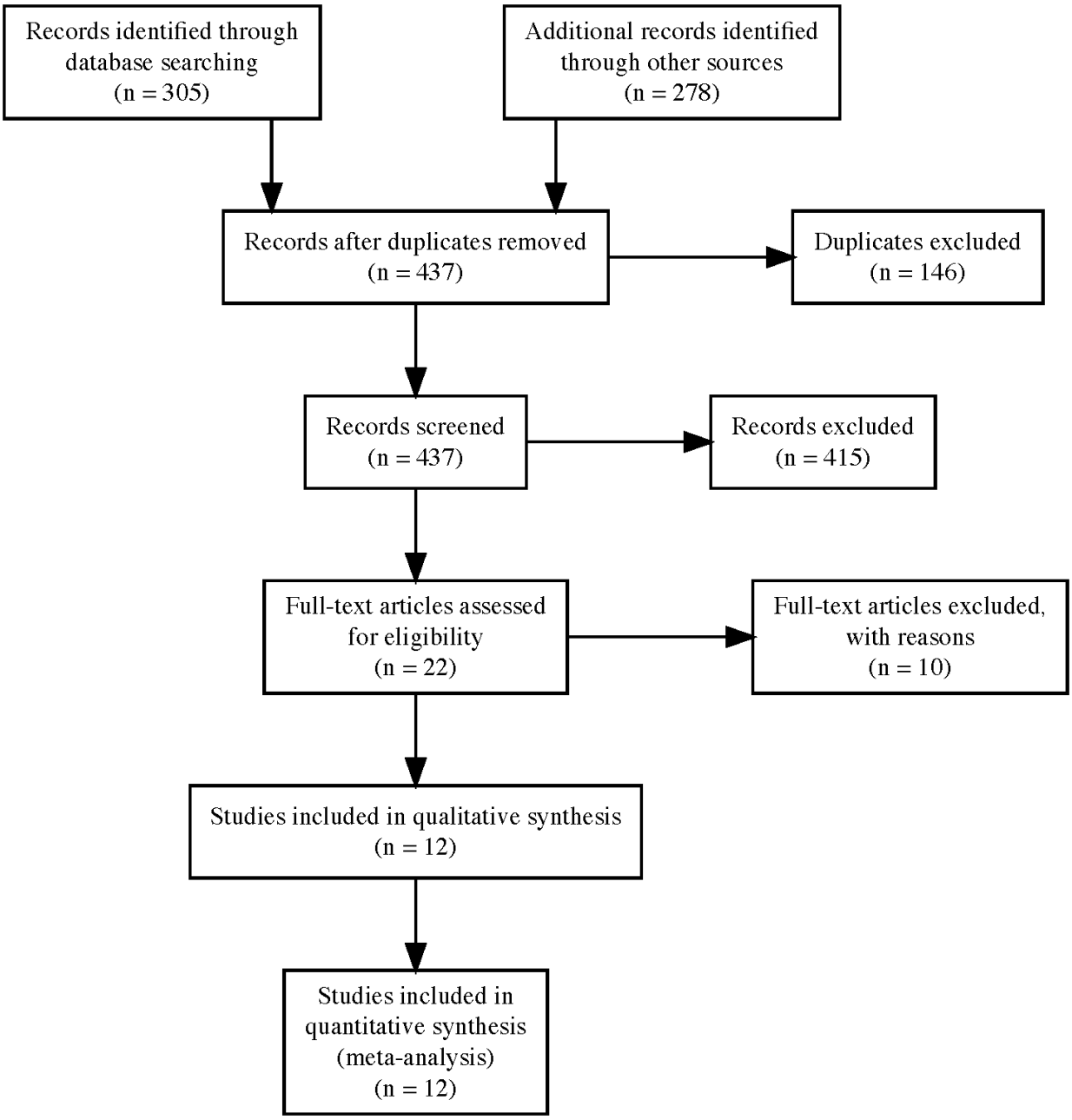

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) flow diagram illustrating the literature search, article screening, and final inclusion.

Coding Procedure.

For the final coding procedure, we only employed the 22 full-text articles. Of these, five were coded by both authors (complete agreements ranging between 64.5 and 86.8%). Differences

in codings were resolved by discussion among the authors. Ten articles were excluded after full-text screening due to not meeting one or more of the inclusion criteria, resulting in a final number of 12 articles (see Table 2 for the final included articles and Supplementary Table 2 for reasons of exclusion). We compared the crying conditions with control conditions (i.e., non-criers or control stimuli) and extracted effect sizes for each measure separately. If an article reported several measures (e.g., HR, SCL, RR), each measure was recorded as an individual effect size. In addition, we focused on the different time periods, a measurement was taken: pre-crying onset, crying onset, and post-crying onset. These periods were operationalized differently by each article. For example, crying onset was sometimes operationalized by self-reported button presses or by coding video material. Some articles reported several pre-cry or post-cry periods (e.g., measurements taken 120s and 60s before/after crying onset). We included all of these periods as individual effect sizes. Possible post periods were not included if additional manipulations or measures were included in between (e.g., filler tasks, additional dependent variables) that might affect ANS responses.

In order to derive effect sizes, we coded the mean, standard deviation, and cell size for the experimental (e.g., criers) and control (e.g., non-criers) conditions. If information for any of these metrics were not available, we took the following steps (in the specified order): (1) we checked whether the information was available in the Appendix or Supplementary Material of the article; (2) we employed algebraic information in order to derive missing information (Weir et al., 2018); (3) we checked whether the respective data was openly available; (4) we contacted the corresponding author(s) to retrieve the missing information; (5) we employed approximate algebraic methods to derive the missing information (see e.g., Hozo et al., 2005; Weir et al., 2018); and (6) we excluded the article if all previous steps were unsuccessful. Altogether, we

contacted authors in six cases and received a response in 85.7% of the cases. Two articles were excluded because authors did not reply or because the authors indicated that they had no access to the data.

Moderators.

An overview of all codes and their specific measurement is provided in Supplementary Table 3. For each article, we assessed the article's meta-data including information on author, title, journal, doi, publication year, and publication status. In addition, we focused on sample-specific information including the country in which the study was conducted, the type of population that was used, whether the sample used a student sample, the total sample size, the number of female participants, and the mean age and its standard deviation. We also coded the location of the study (i.e., online, lab, field), the type of compensation and the total length of the study. We added several codes related to the independent variable (emotional crying manipulation) and the dependent variable (ANS activity). For the crying manipulation, we coded the type of crying (i.e., tears, vocal crying, other), the type of manipulation, the type of stimuli, whether stimuli were self- or experimenter-selected, the valence of the crying episode (i.e., negative, positive, mixed), the mean duration of the crying stimulus or stimuli, how the crying onset was assessed (e.g., self-report), whether the crying occurred in private, the number of bystanders, the type of crying period, and the length of the specific measurement period. For the ANS activity, we coded the type of measure, the type of ANS system, the length of the baseline recording, the type of equipment, the location of the recording, the specific type of the measure, and the number of missing responses.

Table 2. Overview of final articles included in the meta-analysis.

ID	Author(s)	Title	Year	Publication	# Studies	# Effects	Country	*N*	Measures	Stimuli
1	Gross et al.	The Psychophysiology of Crying	1994	Psychophysiology	2*	14	USA	150	IBI, SCL, FPA, Temp (finger), RR, RD, PTT	Video
18	Mori & Iwanaga	Two types of peak emotional responses to music: The psychophysiology of chills and tears	2017	Scientific Reports	1	10	Japan	34	HR, SCL, SCR, RR, RD	Music
21	Zickfeld et al.	Tears of joy, aesthetic chills and heartwarming feelings: Physiological correlates of Kama Muta	2020	Psychophysiology	2^	13	Norway, Portugal	144	HR, HRV (RMSSD, lnHF), RR, RD, SCL, SCR, Temp (chest)	Videos
75	Shirai & Suzuki	Is Sadness Only One Emotion? Psychological and Physiological Responses to Sadness Induced by Two	2017	Frontiers in Psychology	1	30	Japan	74	HR, HRV (HF), SCL, DBP, SBP	Vignettes

		Different Situations: ”Loss of Someone” and ”Failure to Achieve Goal”								
112	Rottenberg et al	Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals	2003	Psychophysiology	1	12	USA	56	HRV (lnHF), HR, RR, RD	Video
117	Hendriks et al.	Can the distress-signal and arousal-reduction views of crying be reconciled? Evidence from the cardiovascular system.	2007	Emotion	1	21	Netherlands	60	HR, DBP, SBP, SCL, RR, HRV (P2T), PEP	Video
118	Rottenberg et al	Crying threshold and intensity in major depressive disorder.	2002	Journal of Abnormal Psychology	1	10	USA	104+	HR, SCL, SCR, Temp (finger), RR	Video
134	Sharman et al	Using crying to cope: Physiological responses to stress following tears of sadness.	2019	Emotion	1	4	Australia	197	HR, RR	Video

145	Mori & Iwanaga	Being emotionally moved is associated with phasic physiological calming during tonic physiological arousal from pleasant tears	2021	International Journal of Psychophysiology	1	6	Japan	34	HR, SCR, RR	Music
581	Baker	Blood, sweat and tears: The intra- and interindividual function of adult emotional weeping	2019	Unpublished (Dissertation)	2	72	UK	80	HR, HRV (RMSSD, SDNN, HF), SCL, RR, Temp (nose, forehead, maxillary, cheek, chin, preorbital)	Videos
582	Zupčić	Effects of crying on changes in negative mood and ANS activation	2013	Unpublished (Master's Thesis)	1	30	Croatia	72	FPA, SCL, SCR, IBI, HRV (lnHF)	Video
583	Ishii & Shinya	Positive emotions have different impacts on mood and sympathetic changes in crying from negative emotions	2021	Motivation & Emotion	1	8	Japan	52	SCL, HRV (lnHf, CSAB), HR	Videos

Note. *The paper originally reports two different studies using the same stimuli and setup that we treat as one study in the present analyses. ^The paper reports two studies on different populations using the same materials but different physiological measurements. +We focused only on the non-clinical sample.

Results

The analyses were performed in *R* (version 4.0.3; R Core Team, 2019), using the following packages: metafor (version 2.4-0; Viechtbauer, 2010), dplyr (version 1.0.2; Wickham et al., 2015), ggpubr (version 0.4.0; Kassambara, 2017), PublicationBias (version 2.2.0; Mathur & VanderWeele, 2020), and MetaForest (version 0.1.3; van Lissa, 2020).

Effect Size Calculations

We employed effect sizes Hedge's *g*, a standardized mean difference measure correcting for small sample size bias (Hedges & Olkin, 2014). For larger sample sizes ($n > 20$), *g* is basically equivalent to the more commonly employed Cohen's *d* measure. We calculated the standardized mean difference with the help of the *metafor* package, by focusing on the mean, standard deviation, and cell size of the experimental and control groups. In the current case, a positive effect size reflects higher activity of the specific measures for emotional crying (compared to non-criers or a control). For all instances, we calculated *g* by using the pooled standard deviation based on the two groups design and correcting for small sample sizes (Goulet-Pelletier & Cousineau, 2018; Westfall, 2016). This was done because repeated measures designs require the cross-measurement correlation, which was often not provided in the papers. Further, it has been suggested that the between-groups calculation method can be used for within-group designs (Westfall, 2016), resulting in possibly more conservative estimates. Similarly, most studies reported aggregate scores over several seconds. As the minority of studies employed multilevel models accounting for time as a random factor (Judd et al., 2012), we might have lost some power by focusing on the aggregate estimates. However, in order to calculate effect sizes from multilevel models we would have needed access to the raw data, which was not feasible in most cases and might have increased exclusions excessively. However, we note that caution

should be applied when focusing on the precision of individual estimates. We focus on overall estimates, which partly mitigates this problem.

Analysis Strategy

Because of the nested structure of the final data, we employed three-level-meta-analytic models (also known as *multilevel meta-analysis*) in *metafor*. As random effects we specified individual study and article, controlling for the fact that effect sizes might come from the same study and that effect sizes might be derived from the same article. Notably, since we focused on each type of measure and period separately, it was uncommon that several effects were derived from the same study. We fitted separate meta-analysis for each type of measure and period (pre, onset, post). We only performed a meta-analytical model if at least two independent effect sizes were available for the measure and period combination. Measures of HR and IBI were merged into one category, as IBI represents the inverse score of HR. For HRV, we merged different measures including time-domain measures such as the root mean square of successive differences (RMSSD), and frequency-domain measures, such as high frequency (HF), into the same category (categorization based on Pham et al., 2021). In an exploratory analysis, we also differentiated between these different types of HRV measures. A final overview of the number of effects per measure period combination is provided in Table 3.

Table 3. Overview of effect sizes per measure and crying period.

Measure	Abbrev.	Cry Period			Total *k*
		Pre	Onset	Post	
Diastolic Blood Pressure	**DBP**	1	**2**	**6**	9
Systolic Blood Pressure	**SBP**	1	**2**	**6**	9
Finger Pulse Amplitude	**FPA**	**4**	**2**	**2**	8

Measure	Abbrev.	Cry Period			Total *k*
Heart Rate	**HR**	**13**	**15**	**10**	35
Heart Rate	*HR*	9	13	8	
Inter-beat Interval	*IBI*	*4*	*2*	*2*	
Heart Rate Variability	**HRV**	**11**	**16**	**11**	38
Time-Domain					
Standard Deviation of NN intervals	*SDNN*	*2*	*1*	*1*	
Root mean square of successive NN interval differences	*RMSSD*	*3*	*4*	*1*	
Frequency-Domain					
Power spectrum (0.15-0.4Hz)	*HF*	*1*	*2*	*5*	
Natural logarithm of HF	*lnHF*	*4*	*6*	*3*	
Balance Index of ANS	*CSAB*		*2*		
Respiratory Sinus Arrhythmia					
Peak-to-through algorithm	*P2T*	*1*	*1*	*1*	
Pre-Ejection Period	PEP	1	1	1	3
Pulse Transit Time	PTT	1	1		2
Respiratory Depth	**RD**	**3**	**4**	1	6
Respiration Rate	**RR**	**10**	**10**	**3**	19
Skin Conductance Level	**SCL**	**10**	**12**	**9**	29
Skin Conductance Responses	**SCRs**	**6**	**6**	**2**	10
Temperature	**TEMP**	**20**	**15**	**12**	45
	Temp Cheek	3	2	2	
	Temp Chest		1		
	Temp Chin	3	2	2	
	Temp Finger	2	2		

Measure	Abbrev.	Cry Period		Total *k*
	Temp Forehead	3	2	2
	Temp Maxillary	3	2	2
	Temp Nose	3	2	2
	Temp Preorbital	3	2	2

Note. Measures in **bold** are included in the final meta-analysis. IBI and HR were merged into one measure of HR as registered.

Based on the number of individual effects, we conducted a meta-analysis on HR, HRV; FPA, RR, SCL, SCRs, and TEMP measures for all periods, on RD for the pre-period and onset, and on DBP, and SBP for the onset and post-period.

Quantifying Publication Bias

Publication bias, also labeled the file-drawer problem, arises if, holding all other influences equal, non-affirmative studies have a smaller probability of entering the literature than affirmative studies (e.g., Dickersin, 1990). Because the majority of investigated combinations showed a small number of individual effect sizes ($n < 10$), validly quantifying and controlling publication bias for this set of studies was difficult. A problem with applying methods trying to correct for publication bias on such small samples of reviews is a reduced power of the test (Dalton et al., 2016). We still apply these methods to combinations including a small number of effects, but these should be interpreted with caution. Different methods have been proposed to control for publication bias and their validity and effectiveness has been discussed (Carter et al., 2019; Dickersin, 1990; Duval & Tweedie, 2000; Mathur & VanderWeele, 2020b; McShane et al., 2016). We employed two different methods to adjust for publication bias. First, we employed a trim-and-fill procedure (Duval & Tweedie, 2000), which attempts to restore symmetry in a

funnel plot of effect sizes by imputing possible missing studies. Second, we conducted a sensitivity analysis using the *PublicationBias* package in *R* (Mathur & VanderWeele, 2020b). This approach focuses on the idea how much more likely affirmative results would need to be published to change the observed effect size. We set *eta* at 3.51, following a previous review observing that affirmative studies are three and a half times more likely to enter the literature (Mathur & VanderWeele, 2020a).

Moderator Analysis

We then followed up to check which variables could explain heterogeneity in our main findings by employing a random forest algorithm using the *MetaForest* package in *R* (van Lissa, 2020). This algorithm represents a supervised machine learning method and has various advantages compared to traditional regression approaches (IJzerman et al., 2018). Random forests can handle issues with multicollinearity, while focusing on higher-order interactions and are non-parametric. From a simplistic viewpoint, random forests include many different regression models that plan a *tree*, which ultimately make up a forest. For a detailed treatise of random forests and their advantages, we refer to Breiman (2001) and Strobl et al. (2009). The MetaForest approach identifies the importance of each variable and how much it contributes in explaining the observed variation among effects. Contrary to our preregistration, we only applied the moderator analysis on measures of HR, HRV, SCL, RR, and TEMP, as the remaining measures included too few observations per measurement crying period combination, which makes the application of random forest not advisable and can involve serious issues with regard to the power of the procedure (van Lissa, 2020).

Following van Lissa (2020) we first checked for the convergence of the random forest model and at what number of trees it converged. A model typically converges of the mean

squared out-of-bag prediction error stabilizes. Then, we selected variables for which the 50% percentile interval did not include zero and determined the optimal tuning parameters using a 10-fold clustered cross-validation. We then applied the model and explored the variable importance of each included variable. We included all variables in the moderator analysis from our coding scheme that were not redundant (e.g., number and percentage of females in sample). We only included variables showing no missing values, as the MetaForest algorithm cannot handle missing values.

Overview of Effects

The present literature review identified 230 different effect sizes based on 12 articles, 14 studies, and a total of 1,017 participants (ranging from 34-197, $M = 84.75$, $SD = 52.81$). Of the final sample, studies included 833 female participants (81.91%) and 80.33% of all participants were undergraduates. The average of mean ages across studies was also rather young ($M = 23.20$, $SD = 5.06$). An overview of all studies is provided in Table 2. The majority of studies were conducted in Japan ($n = 4$) and the US ($n = 3$), with the remaining studies including participants from Australia, Croatia, the Netherlands, Norway, Portugal, and the UK (each $n = 1$). Thus, the majority of studies were conducted in Northern America and Europe. Publication years ranged from 1994 to 2021 (*median* = 2017) and the majority of studies were published after peer review ($n = 10$, 83.33%). All studies focused on emotional tears. An overview of individual effects per specific crying measure and crying period is provided in Table 3. The majority of effects measured TEMP ($n = 45$), HRV ($n = 38$), HR ($n = 35$), and SCL ($n = 29$). Notably, HRV was measured by different indices representing either time- ($n = 12$) or frequency-domain measures ($n = 23$).

Preregistered Analyses

ANS Activity Across Periods.

We first focused on the results of the different ANS measures per type of crying period. An overview of all results is provided in Table 4 and Figure 2 and 3.

Cardiovascular response.

We observed a positive effect for HR (suggesting higher activity for crying than non-crying/control) that increased from pre-crying to crying onset and again decreased post-crying. Heterogeneity for the HR effect at crying onset was high. While the obtained meta-analytical effect was high (g = .57 [-.02, 1.16]), its wide confidence interval suggests low precision and substantial variability across studies and measures. We observed a nearly opposite pattern for HRV, with a small positive effect pre-crying, which decreased at crying onset to a small negative effect and again increased to a positive effect post-crying. None of the HRV effects were statistically significant and we observed again high heterogeneity, specifically for the effect at crying onset. In addition, we included a handful of studies focusing on systolic and diastolic blood pressure. The overall pattern fit the other cardiovascular responses. We observed a small positive effect for both measures at crying onset, which then decreased post-crying, observed heterogeneity was low. Finally, we also included a small number of studies for FPA at each measurement period. We observed that the effect increased over time for emotional crying. Drops in pulse amplitude are considered indicators of vasoconstriction and autonomic arousal (Wesseling et al., 1985). Thus, our FPA findings suggest a decrease in autonomic arousal from onset to post-period as indicated by the other cardiovascular measures. None of the FPA effects were statistically significant.

Respiratory response.

We found small positive effects for RD pre-crying and for the crying onset. Effects increased slightly at crying onset. None of the effects was statistically significant. For RR, we observed a small negative effect pre-crying which decreased further at crying onset and even more post-crying. The crying onset effect showed high heterogeneity. In general, breathing seemed to deaccelerate from pre-crying to crying onset and post-crying.

Electrodermal response.

We observed an increase in effect size from pre-crying to crying onset for SCL. This effect then decreased again at post-crying. The effect at crying onset was characterized by high heterogeneity. For SCR, we observed a similar pattern, which was more pronounced at post-crying. The effect was small and negative pre-crying and increased for crying onset and again decreased post-crying.

Temperature Related.

We observed a small positive effect for temperature at pre-crying and crying onset, which increased to a strong positive effect post-crying. There was high heterogeneity and low precision for the effects pre-crying and at the crying onset.

We found evidence for an overall mild publication bias when applying a trim-and-fill and sensitivity analysis (see Supplementary Table 4 for an overview). In general, the trim-and-fill procedure often indicated stronger effects compared to the non-corrected effect, while the sensitivity analysis resulted in reduced effects. The procedures suggested that effect sizes were possibly overestimated for HR, HRV, RR and underestimated for RD and Temp. Importantly, overall patterns of change remained similar to the non-corrected effect sizes.

Table 4. Overview of meta-analytic effects per measure and crying period.

Measure	Crying Period					
	Pre		Onset		Post	
			g [95% CI] *Q(df), p*			
DBP			.23 [-.17, .62]	.74(1), .39	-.04 [-.83, .75]	8.78(5), .12
SBP			.28 [-.12, .67]	.22(1), .64	.01 [-.43, .45]	3.74(5), .59
FPA	-.19 [-.45, .06]	.12(3), .989	-.19 [-.51, .13]	.03(1), .872	.01 [-.41, .43]	.38(1), .536
HR	.19 [.04, .34]	9.51(12), .658	.57 [-.02, 1.16]	275.03(14), <.001	.19 [.004, .38]	1.57(10), .997
HRV	.11 [-.07, .29]	3.35(10), .972	-.19 [-.78, .41]	185.57(15), <.001	.20 [-.06, .47]	9.06(10), .526
RD	.13 [-.14, .40]	.87(2), .647	.18 [-.06, .42]	3.65(3), .302		
RR	-.12 [-.36, .12]	15.65(9), .074	-.27 [-.64, .10]	43.60(9), <.001	-.78 [-1.12, -.45]	.75(2), .686
SCL	.10 [-.22, .42]	19.97(9), .018	.33 [-.09, .74]	90.63(11), <.001	.001 [-.39, .39]	13.10(8), .108
SCR	-.03 [-.35, .30]	6.44(5), .266	.14 [-.03, .31]	2.05(5), .842	-.47 [-.87, -.08]	.02(1), .887
Temp	.17 [-.23, .57]	36.53(19), .009	.14 [-.31, .59]	32.01(14), .004	.92 [.73, 1.11]	8.51(11), .667

Note. Meta-analytic effect only provided if $k >= 2$. To get k per combination, use $df + 1$.

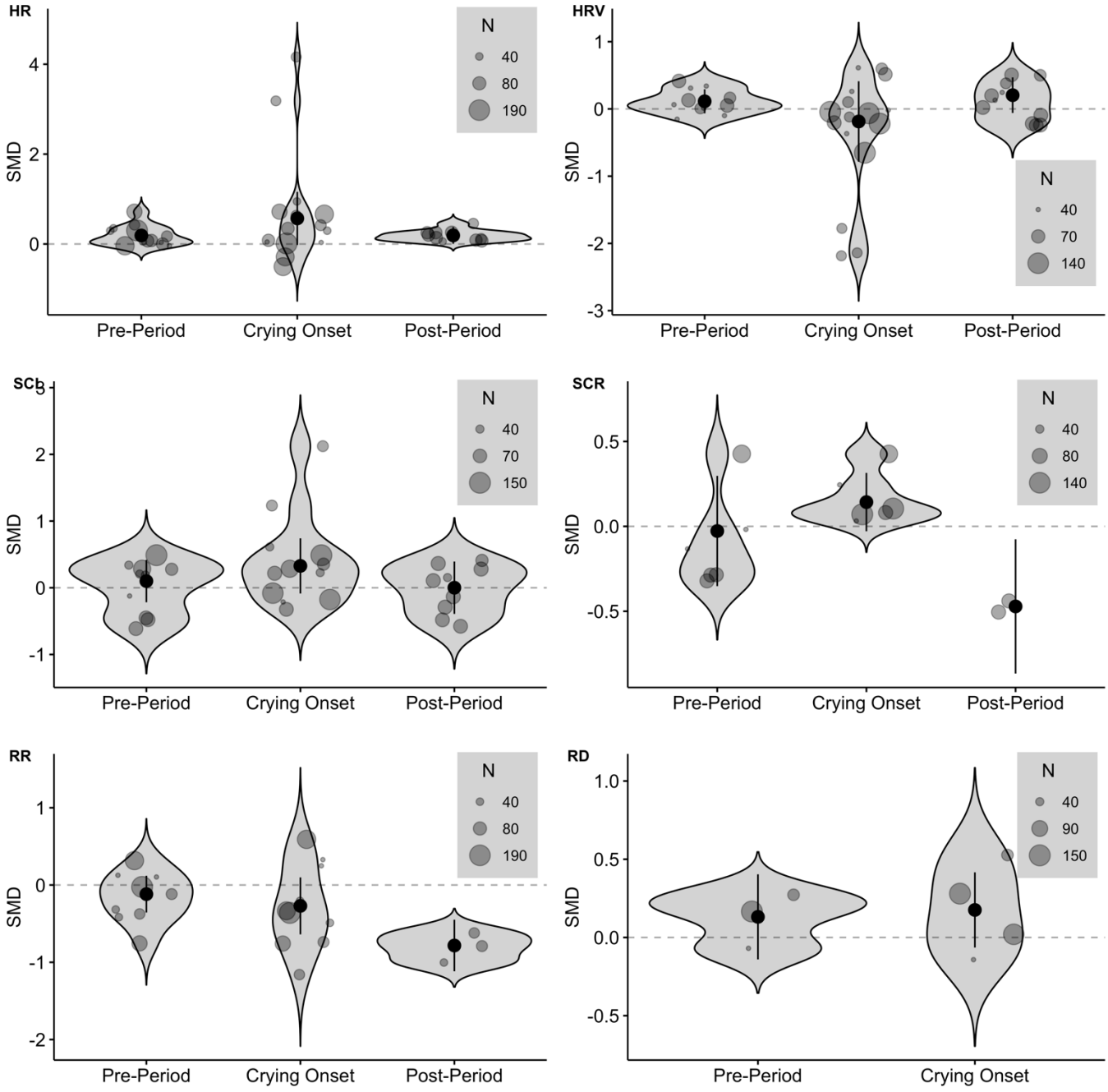

Figure 2. Overview of meta-analytic models for the different measures and crying periods. Error bars represent 95%-confidence intervals.

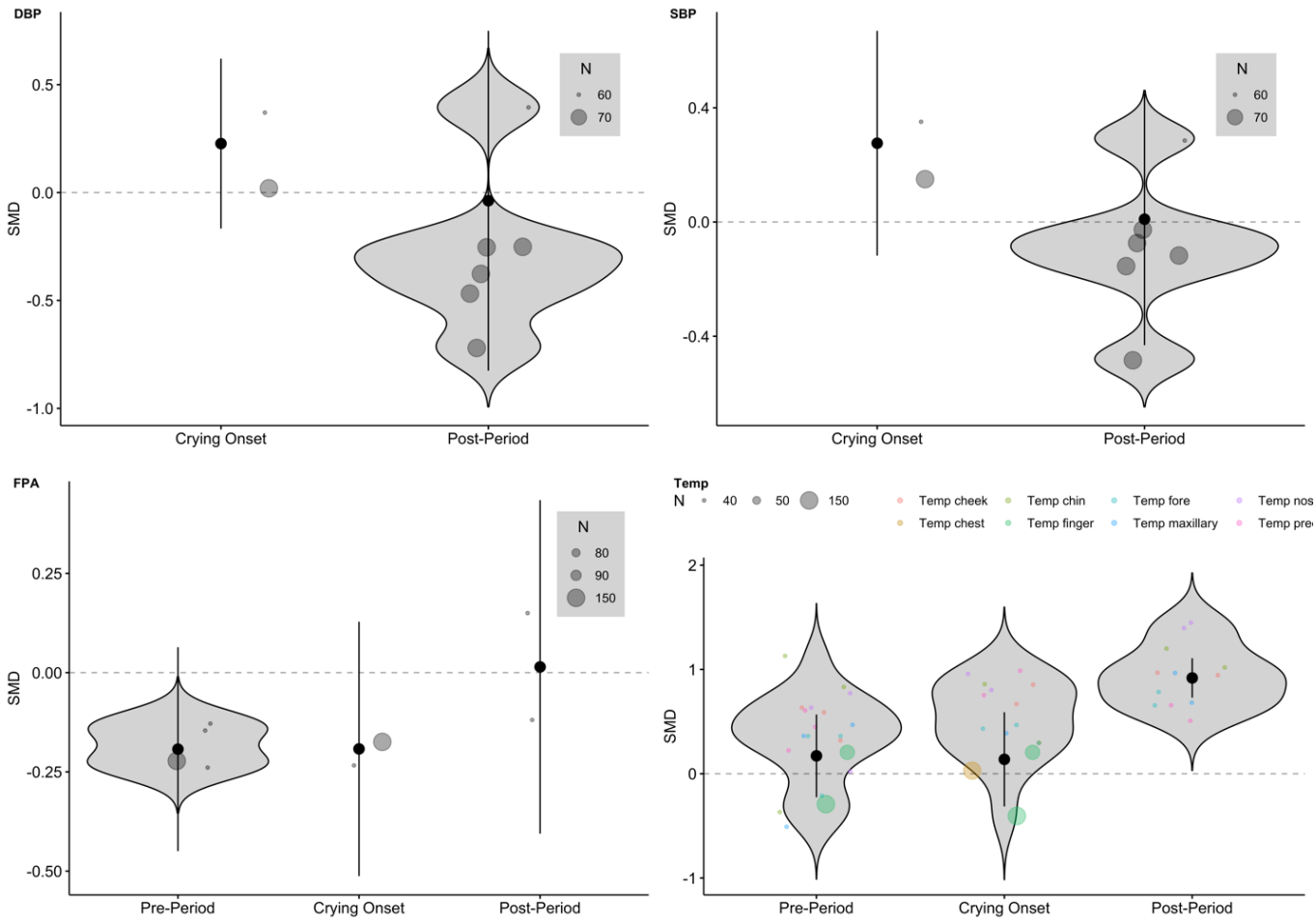

Figure 3. Overview of different meta-analytical models for the different crying periods. Error bars represent 95%-confidence intervals.

Moderator Analysis.

We explored possible variables explaining the observed heterogeneity for the different measures and periods. Due to the random forest algorithm, we only focused on combinations including at least five studies. In addition, we only studied possible moderating variables for combinations that showed a statistical significant heterogeneity in the previous analysis. We were able to only apply the random forest algorithm for HR onset, HRV onset, and SCL onset because

the remaining combinations either contained too few effect sizes or because the different variables showed too small variable importance to be able to explain heterogeneity.

For HR at crying onset, we found that mean age of participants, the type of physiological measure, the crying stimulus, and the total number of participants per study showed a high variable importance. Number of participants accounted for 6.15% of the observed heterogeneity, while the other three variables accounted for 0%. Effect sizes decreased for the increasing number of total participants. For HRV at crying onset, we observed that total duration of the crying stimulus (15.72%), the year of publication (17.62%), the type of measure (0%), the type of manipulation (9.92%), and the type of HRV measure possibly moderated the effect sizes. Effect sizes increased with increasing cry duration. On the other hand, effect sizes decreased for increasing publication year. Effect size were slightly smaller for studies standardizing measures instead of studies using raw scores and smaller for studies using several stimuli. Effects were slightly stronger for time-domain measures (RMSSD, SDNN) than for frequency-domain measures. For SCL at crying onset, we observed that the length of the specific measurement period or bin (27.47%) possibly moderated the effects. Effect sizes decreased with increasing length of the measurement period.

Exploratory Analyses

Differences in HRV Results.

We explored whether different type of HRV measures produced similar results. Therefore, we repeated the meta-analysis comparing time-domain (RMSSD, SDNN) with frequency-domain measures (HF, lnHF, CSAB). An overview of the effects is provided in Supplementary Figure 1. Effects were positive and stronger for frequency-domain measures at pre-crying (g = .17 [-.09, .43] vs. g = .03 [-.26, .31]), stronger in the positive direction for time-

domain at post-crying (g = .19 [-.26, .63] vs. g = .15 [-.23, .52]), and stronger in the negative direction for frequency-domain measures at crying onset (g = -.49 [-1.18, .21] vs. g = .07 [-.23, .36]). Ninety-five percent confidence intervals for these effects overlapped at all measurement periods. Nevertheless, for both types of measures effects increased from crying onset to post-crying, suggesting increased parasympathetic activity after crying.

Time Series Results.

Our main results focus on pre-crying, crying onset, and post-crying, but they suffer from differences in measurement periods. While some studies reported an average measure for 30s after crying onset, others used 60s or 90s intervals. The same is true for the pre- and post-crying periods. Similarly, there is no standardized operationalization of the crying onset. Therefore, a post-crying period might start at 30s after crying onset, 60s, 90s, or even later. This reduces comparability of individual effect sizes of emotional crying across the different studies.

In order to get a better temporal overview of ANS activity during emotional crying, we explored the time specific results. Unfortunately, we were only able to retain average measures for different time bins and as mentioned earlier, these differed in length across studies. To summarize the results of different studies, we assumed that the effect size represented the valid measurement across the complete time bin period. For instance, if we found an effect of g = .20 at crying onset with a time bin length of 60s, a value of .20 was assumed from 0s to 60s. Importantly, this assumption neglects variation of activity within the period and only reflects the average activity across the period. In an optimal setting, we would have been able to obtain the actual ANS activity at the 1s solution for each study. However, this might have resulted in a high number of exclusions. We averaged all values across each second to derive the final curve. We focused on responses from 600s prior to crying onset to 600s post crying onset.

An overview of results is provided in Figure 4. Results were similar to the main results. We found that HR showed only a small difference from between crying and non-crying prior to crying onset. At crying onset, HR activity increased abruptly for emotional crying and continued to rise until around 300s, at which point it dropped again showing only a small difference compared to the control. SCL results followed a similar pattern showing an increase at crying onset and a decrease at around 300s. For HRV, activity decreased prior to crying onset until around 300s, meaning that HRV activity was higher for non-crying compared to crying. This effect then returned to around null at approximately 300s.

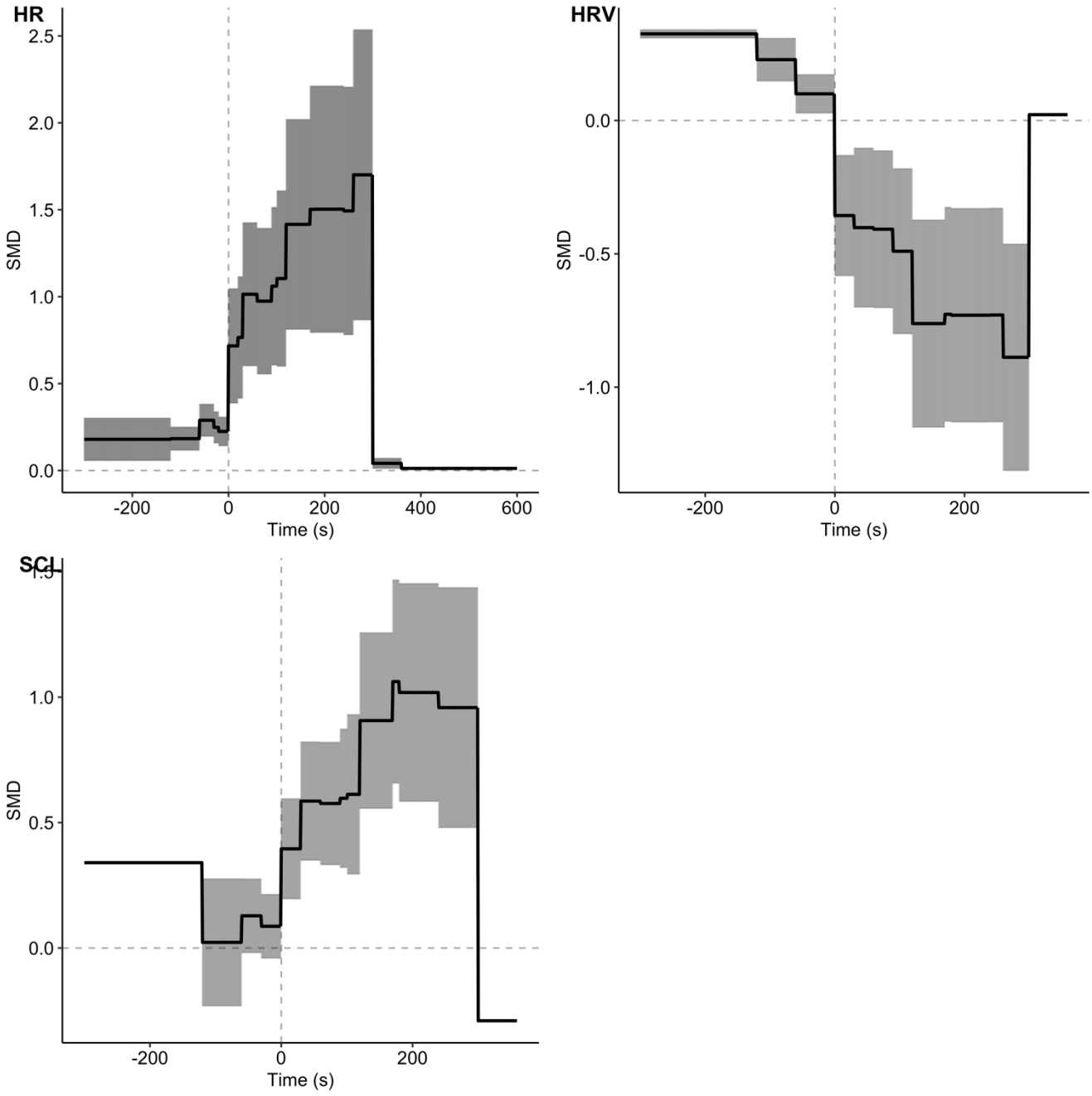

Figure 4. Time series overview of standardized mean difference scores for heart rate, heart rate variability, and skin conductance level. Time zero denotes crying onset. Positive scores indicate higher activity for crying (in contrast to non-crying). Grey area indicates standard error.

Raw Changes in Experimental Scores. In the main analyses, we compared physiological changes in criers to non-criers or during a crying episode versus a control condition. Therefore, a positive effect signifies that criers exhibited higher physiological reactions than non-criers for a comparable period and a negative effect lower physiological reactions. Nevertheless, temporal differences in changes might still increase or decrease regardless of the comparison condition. That is, criers might increase their HR from crying onset to post-crying. However, if the control condition or non-criers shows a steeper slope, we might still observe a negative effect (and reduced activity) for criers at post-crying (although activity increased from onset to post-crying). In order to control for this possibility, we focused on the actual experimental scores (i.e., physiological reactions of criers/crying condition) across the different periods. Unfortunately, different studies employed different ways of assessing these reactions either presenting raw scores or baseline corrected scores (either difference or standardized). To be able to compare different studies, we were forced to focus on studies presenting standardized estimates, which resulted in a low number of studies for the different measures. An overview is presented in Supplementary Figure 2. Overall, we observed a small number of studies reporting standardized or baseline-corrected estimates. We found increases in sympathetic activity from pre-crying to crying onset based on HR, SCL, SCR, and RR responses. In addition, we also observed some evidence for sympathetic withdrawal based on HR responses. These findings seem to support the main pattern of our analyses, but they should be interpreted with caution based on the small number of estimates, especially for the post-crying period.

Discussion

We conducted a systematic quantitative review of the psychophysiological correlates of emotional crying. Focusing on 12 articles and 230 effect sizes from 1,017 participants, we

explored differences between emotional crying and specific control conditions before, at, and after crying onset for measures representing the cardiovascular, respiratory, electrodermal and temperature-related system. We focused on measures reflecting sympathetic and parasympathetic activity and set out to test three major theories predicting different changes in ANS activity in response to emotional crying.

The Psychophysiology of Emotional Crying

We found consistent evidence for increases in sympathetic activity at crying onset. Different measures that are thought to represent sympathetic activity, including HR, DBP/SBP, SCL, and SCR showed a stronger activity at crying onset for criers compared to non-criers. Similarly, HRV activity showed the opposite pattern at crying onset further supporting the idea that the onset of emotional crying is associated with increased sympathetic activity. Notably, we did not observe this pattern for RR, which is often considered as an indicator of sympathetic activity as well (Gross et al., 1994). As previously discussed (Bylsma et al., 2019), breathing instead slowed down at crying onset for criers in contrast to non-criers.

In addition, we found evidence for sympathetic withdrawal after emotional crying. For HR, SCR, and SCL, the effects were smaller for post-crying than for the crying-onset. For SCR and RR, the effect was also negative at post-crying, suggesting a reduced activity in criers compared to non-criers. The exploratory time series analysis suggested that this withdrawal effect occurs at around 300s post-crying onset. As descriptive support, we found a non-significant effect for systolic and diastolic blood pressure at post-crying and a decrease of the effect from crying onset.

Our findings further suggest increased parasympathetic activity at post-crying. HRV showed a positive, although non-significant, effect post crying. This might be related to the fact

that there were only few studies for these measurements and periods and also the fact that post periods were operationalized differently across individual studies. Exploring the time series of HRV responses, we observed an increase in the effect at around 300s post crying. Importantly, this effect was around zero, suggesting similar HRV activity for crying and non-crying.

We did not find any systematic effects at the pre-crying period. All measures showed non-significant pre-crying effects and most of the time they showed a small positive effect. In general, this suggests that the physiological activity before the onset of crying is similar to the physiological activity of non-criers in a comparable time frame. This might be related to the measurement periods employed in the different studies. Changes for criers at pre-crying might be more fast paced than what could be detected based on the different operationalizations or there might be no physiological differences before crying (compared to non-criers or a control condition).

Finally, temperature increased post crying, showing higher temperature for emotional crying compared to non-crying. Importantly, the included studies measured temperature at different locations of the body, but based on our results these locations showed low heterogeneity at post-crying. Increases in face skin temperature might be related to the production of tears (Ioannou et al., 2016), whereas increases in finger temperature have been associated with reduced SNS activity (Boudewyns, 1976; Merla & Romani, 2007), supporting the overall pattern of the other ANS measures. We were not able to disentangle specific locations on the body showing increased temperature, limited by the small number of studies on the topic.

Importantly, we found some evidence suggesting that these overall patterns were similar when focusing on physiological changes across the different periods in criers only. Sympathetic activity increased at crying onset based on HR, SCL, SCR, and RR and we observed evidence

for sympathetic withdrawal at post-crying for HR. Notably, the limited amount of comparable standardized scores minimizes the conclusive power of these findings.

Theories on Emotional Crying

Our results support the *arousal view* and *helplessness view* at crying onset, as we showed increased sympathetic activity. However, the observed pattern of increased parasympathetic activity and sympathetic withdrawal at post-crying does not necessarily support either of these theories. Similarly, the pattern of reduced parasympathetic activity at crying onset does not support the *catharsis view*, but instead suggesting the exact opposite processes. Similarly, we did not observe strong evidence for the arousal view at pre-crying, as this theory typically assumes that high arousal triggers emotional crying (see Gross et al., 1994). Across the different studies, sympathetic activity was only somewhat increased prior to the emotional crying episode and most changes occurred at or after crying onset.

The data of the present review fit the *arousal-catharsis view* best (Hendriks et al., 2007). The fact that sympathetic activity was found to increase during crying supports the arousal part of this theory (and the arousal view). Similarly, the finding that sympathetic withdrawal sets in and parasympathetic activity increases after crying supports the catharsis part (and the catharsis view). Importantly, we found evidence that this recovery process, which is in contrast to the arousal account, might take some time to set in (Gračanin et al., 2015). This fits previous findings, showing that mood repairing or enhancing effects might take several minutes after crying to properly set in. Based on our results, it seems that it could take up to around five minutes for homeostatic processes to set in after emotional crying. While increases in sympathetic activity seem to occur in a more fast-paced manner in earlier stages of crying, sympathetic withdrawal and parasympathetic increases seem to occur slower in later stages. This

pattern can account for seemingly contradicting findings that people report a worsened mood state when asked right after an emotional crying response (Bylsma et al., 2008, 2011; Gračanin et al., 2015). That is, the present meta-analysis provides the empirical support for Hendriks and colleagues' (2007) idea reconciling theories of arousal and catharsis, which have long been believed to express opposing views on emotional crying.

Functions of Emotional Crying

Altogether, we found weak but consistent evidence that emotional crying fulfills an intrapersonal function by restoring homeostasis. Emotional crying seems associated with increased sympathetic activity and the organism seems to respond by triggering sympathetic withdrawal and increasing parasympathetic activity. The question whether emotional crying is the result of heightened arousal or whether crying triggers arousal in the first place cannot be easily resolved with the current data. While the data suggest that sympathetic activity increases at crying onset and not before, how crying onset was defined differed considerably across the different individual studies. At the same time, the time bins of the pre-crying periods were variable sometimes including 60s periods or more. It is conceivable that rapid changes in sympathetic activity that could trigger emotional crying only occur within a few seconds before crying onset, something that the present analysis could not capture. Therefore, the question remains whether emotional crying is the product or cause of increased sympathetic activity.

What seems better supported is the finding that sympathetic activity decreases and parasympathetic activity at least partly increases after (or during) emotional crying. This evidence thus suggests that emotional crying fulfills the function of regulating bodily homeostasis (Bylsma et al., 2008, 2019; Gračanin et al., 2018; Rottenberg et al., 2008). Exactly how does emotional crying regulate these bodily processes? As reviewed earlier, the lacrimal

gland is mainly innervated by parasympathetic nerves (Bylsma et al., 2019). Increased production of tears by the lacrimal gland, might therefore increase parasympathetic activity (and also trigger sympathetic withdrawal). On a different note, we observed that respiratory rate decreased at crying onset and post-crying. Breathing patterns associated with emotional crying might in fact facilitate the downregulation of increased arousal (see also Bylsma et al., 2019; Sharman et al., 2019). Slower breathing observed in criers (in comparison with non-criers) could modulate RSA activity and thereby regulate bodily homeostasis. One particular aspect of emotional crying is sobbing, which represents repetitive and convulsive breathing and the inhalation of cold air (Gračanin et al., 2014). Previous research has theorized that sobbing might be related to increased PNS activity and suggested that the repetitiveness of sobbing can act as a calming response (Gračanin et al., 2014). Future research could manipulate the effect of different breathing patterns during emotional crying on ANS activity to test a possible importance of the respiratory system directly or compare ANS activity in response to tearing up and sobbing.

Recent empirical research presented increasing evidence that human emotional crying fulfills also an interpersonal function, by attracting social support and helping behavior from others (Hasson, 2009; Reed et al., 2019; Van de Ven et al., 2017; Zickfeld et al., 2021). The question arises how the intra- and interpersonal function of emotional crying interact. For instance, does the interpersonal outcome in the form of social support behavior facilitates regulation of bodily processes in the crier? The reviewed studies focused exclusively on crying in private. Future research could systematically manipulate whether criers are supported by bystanders or not, while their ANS activity is monitored. Indeed, some studies suggest that receiving social support might induce homeostasis more quickly in contrast to no consolation

(Goodyke et al., 2021). Such findings might at least in part explain why emotional tears contain such a universal communicative function (Zickfeld et al., 2021).

Limitations & Future Research

There are several aspects that minimize the conclusive quality of the current meta-analysis and point at important avenues for future research.

First, the majority of studies in the current meta-analysis focused on physiological responses to emotional tears of young female undergraduates in private, most often as a response to negative videos. Since more than three fourth of the total sample were female, it is questionable whether the current results generalize to emotional crying in men. Social norms and attitudes about crying have been found to differ strongly between women and men (Sharman et al., 2019; van Hemert et al., 2011), which might also influence possible regulatory effects of emotional crying. We were not able to disentangle these effects in the present analysis, as the studies did not report findings for females and males individually. Similarly, recovery processes might occur faster if possible bystanders console or support the crier (Bylsma et al., 2008; Zickfeld et al., 2021) instead of crying in private. Different regulation strategies might enhance possible recovery effects. In an extreme case, recovery functions might even only occur when the organism shuts down (e.g., crying oneself to sleep). An experimental study observed lower physiological arousal in criers instructed to express their tears in contrast to participants instructed to inhibit and *swallow their tears* around possible crying onset (Labott & Teleha, 1996), suggesting that the homeostatic function could also depend on successful emotion regulation strategies. Finally, a recent study suggested different physiological correlates of crying in response to negative and positive emotional videos (Ishii & Shinya, 2021). Sympathetic activity was higher in response to negative videos, suggesting that different types of emotional

crying might induce different degrees of distress (that in turn needs to be regulated). Self-report evidence further suggests that mental and physical states are improved after shedding positive tears instead of negative ones (Bylsma et al., 2008). We did only include a small number of studies focusing on positive emotional crying and this factor did not emerge when exploring possible moderators. Following previous discussions, whether emotional crying results in homeostasis and recovery and at what time point it might be subject to moderation by various individual, situational, and cultural variables (Bylsma et al., 2008, 2011; Rottenberg et al., 2008). Considering that the majority of studies investigated emotional crying under *non-optimal* conditions (i.e., negative reasons, in private), regulatory effects and changes in ANS activity might be even stronger in what has been suggested as more *optimal* settings (i.e., positive tears, with possibility of support; see Rottenberg et al., 2008).

Second, related to the previous point all reviewed studies investigated physiological responses to emotional tears. We did not find any study focusing on vocal characteristics of emotional crying or specific behaviors such as sobbing that fit our inclusion criteria. It is possible that some participants in the current studies also showed other characteristics of emotional crying such as screaming, sobbing, or specific facial expressions. Few of the reviewed studies controlled for these aspects. As one exception, Gross et al. (1994) coded behavioral concomitants of crying and found that criers showed among others increased facial expressions or movements and more face touching. Previous studies argued that specific behavior such as sobbing might be helpful in reducing arousal (Gračanin et al., 2014) or that tearing has a different effect on physiological activity than vocal crying (Bellieni, 2017). We were not able to test these propositions in the present meta-analysis and future studies would need to systematically manipulate different aspects of emotional crying or directly control for them.

Third, we only identified a small number of studies and effect sizes. This resulted in the caveat that for many combinations the number of effects was too small to derive any precise and valid conclusions. Nevertheless, we obtained enough studies for the most common measures of ANS activity (HR, HRV, SCL, SCR) to formulate at least some preliminary conclusions.

Fourth, we observed some heterogeneity and low precision for many of the combinations studied. Specifically, the effects at crying onset showed high variability and were sometimes biased by few *influential* effects. Such variability might reflect some general variation in stimuli, measurement equipment, procedure of measurement, and other situational variables, similar to previous studies focusing on psychophysiological responses (e.g., Kreibig, 2010; Siegel et al., 2018). On the other hand, it is also possible that these findings are based on the different operationalization of the specific time periods. We did not observe systematic moderating variables related to the different study designs. For some measures, the type of manipulation or the length of the crying stimuli explained some of the variation, but this was only applicable for specific measures and specific time points. In addition, we should note that the reviewed studies probably overestimated effect sizes in specific contexts, especially for measures at crying onset. This fact might again be related to different operationalizations in the exact time of crying onset and its length.

Fifth, these variations in when precisely ANS activity was recorded in temporal relation to the crying response make proper comparisons across the different studies difficult. Crying onset was defined based on different methods, using self-report or observational method, and most studies did not define a proper crying offset based on such reports, but rather focused on fixed time intervals. Further, different study designs chose different time intervals. For instance, Study A might have defined the crying onset by having participants pressing a button when they

cried and then averaged the ANS responses for the 60s from this onset (crying onset) and the 60s before (pre-crying) and after (post-crying) this period (without further checking the actual crying offset). In contrast, Study B might have focused on defining crying onset by coding video material of the participants and taking 120s time intervals from this onset, as well as before and after. Thus, the time period of the crying onset (0-120s) of Study B overlaps with the crying onset (0-60s) and post-crying period (61-120s) of Study A. Raw data at the 1s solution might have mitigated this comparison problem, but such data were not available in many cases. Moreover, some studies collected post-crying measurements while participants were still exposed to the emotional stimulus or stimuli, whereas other studies defined the end of the stimulus as the end of the crying period.

This also begs for a different question: how valid can the crying onset (and offset) be defined by the existing methods? Does the subjective feeling when someone is crying differ across individuals? When does a response actually qualify for an emotional crying episode? How should emotional crying be coded by observers? Are moist eyes enough to start a crying episode or do tears need to run down the cheek? This debate is not new (see Vingerhoets, 2013) and there is no easy solution to it. The key lies in the specific definition of emotional crying and creating valid methods to assess emotional crying (and its onset and offset). For example, thermal imaging has shown some promises (Ioannou et al., 2014, 2016). Future studies would need to address this point in order to increase the comparability of psychophysiological studies on emotional crying.

Conclusion

We systematically reviewed the empirical literature on the autonomic nervous system correlates of emotional crying. Across 12 articles and 230 effect sizes, we found evidence that

emotional crying is associated with increased sympathetic activity around crying onset that transforms to sympathetic withdrawal after some time. We also found evidence for increased parasympathetic activity at several minutes after the crying onset. These findings fit a combination of the arousal and catharsis or recovery views best and highlight that emotional crying might in general fulfill an important intraindividual function in restoring homeostasis.

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