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Experimental induction of peritraumatic dissociation: The role of negative affect and pain and their psychophysiological and neural correlates

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ABSTRACT

While research has elucidated processes underlying dissociative symptoms in patients with posttraumatic stress disorder, little is known about the circumstances under which trauma-related dissociation initially arises. To experimentally investigate causes and concomitants of peritraumatic dissociation, we subjected sixty-nine healthy women to aversive-audiovisual and painful-electrical stimulation in a 2(aversive/neutral film) x 2 (pain/no pain) within-subject design while recording psychophysiological and fMRI-BOLD responses. Afterwards, participants rated negative-affect, pain, and dissociation for each condition. Using Bayesian multilevel regression models, we examined (1) whether aversive-audiovisual and painful-electrical stimulation elicit higher dissociation-levels than control conditions and (2) whether stronger negative-affect and pain responses (operationalized via self-report, psychophysiological, and neural markers) correlate with higher dissociation-levels. Several key findings emerged: Both aversive-audiovisual and painful-electrical stimulation elicited dissociation. Dissociation was linked to higher self-reported negative-affect, but we did not find enough evidence linking it to psychophysiological and neural negative-affect markers. However, dissociation was associated with higher levels of self-reported pain, a skin-conductance-response-based pain marker, and the fMRI-BOLD-based Neurologic-Pain-Signature. Results indicate that both aversive-audiovisual and painful stimuli can independently cause dissociation. Critically, pain responses captured via self-report, psychophysiological, and neural markers were consistently linked to higher dissociation-levels suggesting a specific, evolutionary meaningful, contribution of pain to the rise of dissociation.

1. Introduction

Many individuals with Posttraumatic Stress Disorder (PTSD) suffer from dissociative symptoms like depersonalization and derealization (White et al., 2022), which has prompted the introduction of a dissociative PTSD subtype in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). However, while considerable effort has been undertaken to understand psychological and neurobiological processes underlying posttraumatic dissociative states (Harnett & Lebois, 2022; Lanius et al., 2010, 2018; Lebois, Harnett, et al., 2022; Lebois, Kumar, et al., 2022; Mertens et al., 2022; Nicholson et al., 2020; Roydeva & Reinders, 2021; Wolf et al., 2022), little is known about the circumstances under which trauma-related dissociation initially arises, i.e., the causes of and mechanisms behind peritraumatic dissociation.

According to evolutionary accounts, traumatic events initially cause strong negative feelings like negative-affect and pain which, when reaching a certain threshold, elicit dissociation (Danböck et al., 2021; Schauer & Elbert, 2010) which might manifest in various ways including psychological (e.g., depersonalization, derealisation) and somatoform (e.g., loss of motor control, sensory loss) dissociative phenomena (Marmar et al., 1997; Nijenhuis & Van der Hart, 1998; van der Hart et al., 2008). However, once dissociation intensity has reached a certain threshold, it is assumed to attenuate these negative feelings and by these means facilitate remaining still and increase probability of survival. Hence, peritraumatic negative-affect, pain, and dissociation appear to be part of a complex bidirectional interplay. To empirically examine causes of and mechanisms behind the initial rise of peritraumatic dissociation, tailored studies are needed that allow rigorous testing of factors giving rise to dissociation. Specifically, studies are needed that

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(1) experimentally manipulate trauma features and examine effects on dissociation and (2) investigate relationships between peritraumatic negative-affect and pain with dissociation. Here we aim to address these questions using a novel trauma-analogue paradigm incorporating aversive-audiovisual and painful-electrical stimulation combined with a multidimensional assessment of negative-affect and pain.

1.1. Trauma features eliciting dissociation

Traumatic events are defined as exposure to actual or threatened death, serious injury or sexual violence (American Psychiatric Association, 2013). However, per definition, exposure can happen in manifold ways, including directly experiencing the event as a victim, witnessing the event as it occurs to others, learning that the event occurred to a close relative or friend, or being repeatedly or extremely exposed to aversive details of the event (American Psychiatric Association, 2013). Evolutionary models postulate that dissociation especially arises during traumatic events which are characterized by close proximity to a superior perpetrator and violation of one's own physical integrity (Kozlowska et al., 2015; Schauer & Elbert, 2010), hence during traumatic events happening directly to oneself and including painful stimulation. This is in line with retrospective reports of trauma-survivors indicating that peritraumatic dissociation is more prevalent during directly experiencing interpersonal violence than during witnessing or learning from the sudden unexpected death of a loved one (Hetzel-Riggin & Roby, 2013). Yet, retrospective reports of peritraumatic dissociation have been shown to be influenced by current psychopathology levels (Zoellner et al., 2001), which might have biased these initial findings. Moreover, traumatic events usually differ in more than one feature (e.g., experiencing vs. witnessing the event, interpersonal vs. accidental threat) and experimental manipulation of trauma features is not feasible in real-life making it impossible to study causal effects of trauma features in real-life.

Experimental studies thus have employed analogue designs to systematically manipulate event features and assess effects on dissociation. One line of research had healthy participants watch films displaying scenes of the aftermath of road traffic accidents and rate the intensity of evoked dissociation. Participants reported higher dissociation after aversive film-viewing compared to pre-film (Chou et al., 2014; Holmes et al., 2004, 2006) and post-film (Chou et al., 2014) baselines, implying that witnessing aversive-audiovisual scenes (without being physically involved in the situation) can already evoke dissociation. Other studies had healthy participants hold their arm into pain-eliciting ice water. Participants reported higher dissociation after the task compared to a pre-task baseline (Giesbrecht et al., 2008; Gómez-Pérez et al., 2013; Horowitz & Telch, 2007), suggesting that painful stimulation (without simultaneously witnessing aversive-audiovisual material) can also evoke dissociation. However, so far analogue studies have lacked control conditions ensuring that dissociation is driven by the aversive-audiovisual or painful stimulation and not by general task, time, or demand effects. Moreover, although many traumatic events (e.g., directly experiencing a car accident or physical violence) incorporate both aversive-audiovisual and painful stimulation, up to now, no study has systematically manipulated more than one event feature at once to examine independent and joint contributions to the rise of dissociation.

1.2. Negative-affect, pain, and dissociation

Theoretical models postulate that during trauma strong negative feelings like negative-affect and pain are linked to the onset of dissociation (Danböck et al., 2021; Schauer & Elbert, 2010). In line with this, reports of trauma-survivors indicate that during real-life trauma higher distress (Bryant et al., 2011; Fikretoglu et al., 2006; Lewis et al., 2014; Otis et al., 2012) and hyperarousal (Sterlini & Bryant, 2002) were linked to higher dissociation-levels. Moreover, trauma survivors reporting more peritraumatic pain also indicated higher peritraumatic dissociation-levels (Beaudoin et al., 2021) and, in a similar vein, mothers reporting more childbirth pain indicated higher perinatal dissociation-levels (Boudou et al., 2007; Zambaldi et al., 2011). Yet, these studies solely rely on retrospective self-reports of negative-affect, pain, and dissociation and thus results might have been influenced by various internal and external factors (Candel & Merckelbach, 2004; de Williams et al., 2000; van der Hart et al., 2008).

To overcome this limitation, analogue studies have started to employ objective negative-affect markers and examine their relationships with dissociation: One study linked dissociation to lower HR during a film displaying car accidents, but only in participants who also responded to separately presented startle trials with HR deceleration (Chou et al., 2014). Another study linked dissociation to higher heart rate (HR) and more corrugator muscle activity during a film displaying a rape scene (Danböck et al., 2021). As negative-affect is reflected by HR deceleration in traffic-related-aversive, but HR acceleration during sexually-aversive films (Arnaudova & Hagenaars, 2017), both studies might implicate that dissociation is linked to higher negative-affect. However, the lack of specificity of HR and other psychophysiological measures for negative-affect (Kreibig et al., 2007) limits the interpretability of these findings fostering the need to investigate the relationship between negative-affect and dissociation using more specific negative-affect markers, e.g. from brain activation patterns. Critically, associations between objective pain markers and dissociation have not been examined yet.

1.3. The current study

The current study aimed to experimentally investigate factors contributing to the rise of peritraumatic dissociation by employing a new trauma-analogue paradigm independently varying aversive-audiovisual and painful-electrical stimulation (Franke et al., 2022). By these means, we wanted to disentangle the relevance of aversive-audiovisual stimulation characterizing almost all traumatic events (e.g., the sounds and images when witnessing a car accident or when being hit by a car) and painful stimulation characterizing only a subset of traumatic events (e.g., the pain when being hit by a car) for the rise of peritraumatic dissociation. Moreover, by employing a multidimensional assessment of negative-affect and pain-responses (as opposed to previous studies mainly relying on retrospective self-report) and exploring their relationship with dissociation, we aimed to provide new insights into the negative-affect-dissociation and pain-dissociation relationships. Specifically, we repeatedly exposed healthy participants to 2(aversive/neutral film) x 2(pain/no pain) experimental conditions while psychophysiological and neural activations were recorded. Afterwards, participants rated the intensity of negative-affect, pain, and dissociation for each condition.

We expected that both aversive-audiovisual and painful-electrical stimulation would elicit dissociation and explored a potential interaction between these factors. Moreover, we hypothesized that stronger negative-affect responses assessed via self-report, psychophysiological (i.e., HR), and neural (i.e., the fMRI-BOLD-based Picture-Induced-Negative-Emotions-Signature; PINES; Chang et al., 2015) markers would be linked to higher dissociation-levels. Last, we expected stronger pain-responses assessed via self-report, psychophysiological (i.e., a pain-specific skin-conductance-response-based signature; PPS; Matthewson et al., 2019), and neural (i.e., the Neurologic-Pain-Signature; NPS; Wager et al., 2013) markers to be associated with higher dissociation-levels.

2. Methods

2.1. Participants

Overall, 74 healthy women without self-reported cardiovascular, neuroendocrinological, pain-related, or mental disorders between the ages of 18 and 35 years recruited via public announcements took part in

the study. Participants reported physical and psychological resilience, did not use medication (except for oral contraceptives), were not pregnant, did not have ferromagnetic implants or other non-removable metal objects, and had a body mass index between 18 and 35 kg/m^2. Furthermore, participants with claustrophobia or high consumption of extremely violent media (more than 2–3 times a week) were excluded. Two participants were excluded due to technical problems and three aborted study participation due to high emotional reactivity, leaving a final sample of 69 participants. Due to technical problems, HR data was only available for 61 participants and electrodermal data for 67 participants. Due to movement artifacts (>3 mm) or abnormalities in brain structure, valid neural data was only available for 65 participants.

Current psychopathology was assessed with the German versions of the long version of the Depression-Anxiety-Stress-Scales (Nilges & Essau, 2015), the State-Trait Anxiety Inventory (Laux et al., 1981), the Questionnaire of Dissociative Symptoms (Freyberger et al., 1998), and the Screening for Somatoform Symptoms 7 (Rief & Hiller, 2003). As detailed in Table 1, distribution of scores was typical for healthy samples.

The study was approved by the local Ethics Committee. All participants provided informed consent and were reimbursed with course credit or money.

2.2. Procedure

After an online pre-assessment of demographic and trait variables, participants were invited to the lab. Sitting on the MRI table, they first underwent a pain calibration procedure. Then, Ag/AgCl electrodes filled with isotonic electrode paste were placed on the lower palm of the non-dominant hand to measure electrodermal responses and a pulse oximeter was clipped on the index finger to assess HR. Next, participants got accustomed to the setting during an 8-min resting state fMRI sequence. Following, participants were repeatedly exposed to neutral and aversive films with and without painful-electrical stimulation while psychophysiological and neural activation was recorded. More precisely, each participant was exposed four times to each of the following conditions: a neutral film without painful stimulation, another neutral film paired with painful stimulation, an aversive film without painful stimulation, and another aversive film paired with painful stimulation. The pairing of films with painful stimulation was counterbalanced across participants and stimuli were presented in pseudorandom order, i.e., with maximally two similar conditions in a row. Intertrial-intervals ranged from 12 to 16s. Afterwards, participants were asked to remember how they felt while viewing each film and retrospectively rate negative-affect and pain while seeing a reminder screenshot of each film. Last, seated in front of a laptop outside of the scanner, participants were asked to rate the dissociation they experienced during each condition. As this study was part of a larger investigation, films were preceded by four images resembling elements of each film-clip. Participants underwent another 8-min resting state fMRI sequence at the end of the session, returned to the lab for another fMRI session 24 h later, and reported pain and audiovisual intrusions during the following days using a smartphone application (Franke et al., 2022).

Table 1
Sample descriptives (N = 69).

	M	*SD*	Range in sample
Age (years)	22.32	3.50	[18; 35]
Education (years)	14.86	2.45	[10; 20]
DASS – D (0-42)	3.58	4.12	[0; 15]
DASS – A (0-42)	2.54	3.23	[0; 15]
DASS – S (0-42)	5.91	5.68	[0; 22]
STAI-T (20–80)	38.72	8.82	[21; 61]
QDS (0–100)	7.46	5.99	[0.45; 30.91]
SOMS-7 ICD-10 somatization index (0-14)	0.97	1.56	[0; 8]

Note. Values indicating non-clinical relevance are ≤10 for DASS - D, ≤6 for DASS – A, ≤10 for DASS – S (Nilges & Essau, 2015), ≤44 for STAI-T (Ercan et al., 2015) and ≤13 for QDS (Rodewald et al., 2006). Average ICD-10 somatization scores within the general population are $M = 1.1$ and $SD = 1.7$ (Rief et al., 2001). Abbreviations: DASS - D/A/S = Depression/Anxiety/Stress-Subscales; STAI-T = State Trait Anxiety Inventory; QDS = Questionnaire of Dissociative Symptoms; SOMS-7 = Screening for Somatoform Symptoms.

2.3. Material and measures

Apparatus and physiological recordings. We used E-Prime 2.0 (Psychology Software Tools, Inc., Pittsburgh, PA, USA) to control stimulus presentation. Moreover, we used Polybench 1.22 (TMSi, Twente Medical Systems International, EJ Oldenzaal, Netherlands), a Porti 32-channels-amplifier (TMSi), and a skin-conductance-amplifier (Becker Meditec, Karlsruhe, Germany) to record skin-conductance data. We recorded the arterial pulse signal using the peripheral finger pulse plethysmography unit of the fMRI scanner (Siemens Magnetom Prisma) and analysed psychophysiological data using ANSLAB 2.6 (Blechert et al., 2016).

Aversive-audiovisual stimulation. We used 16s long film-clips of commercial movies. The two aversive film-clips were extracted from the movie "*Irreversible*" (Noé, 2002) and depicted severe interpersonal violence (i.e., sexual and physical assault). Two neutral control film-clips were extracted from the movies "*Coach Carter*" (Carter, 2005) and "*Mr. Jones*" (Figgis, 1993) and depicted normal human social interactions (i.e., a beach walk and a basketball game). All film clips were previously validated and pairwise matched in content, valence, and arousal (Arnaudova & Hagenaars, 2017).

Painful-electrical stimulation. We used a Digitimer DS7A constant current stimulator (Digitimer Ltd, Hertfordshire, England) with a re-useable concentric 7 mm diameter surface electrode and a platinum-pin WASP-electrode attached to participants' inner side of the left calf (Brainbox Ltd, Specialty Developments, Cardiff, Wales) to deliver electrocutaneous stimulation. Stimulation started with film onset and was applied in seven pulse trains with a duration of 988 ms followed by 400–1300 ms inter-stimulus intervals in order to produce stable stimulus intensities (Mouraux et al., 2014).

An individual stimulation intensity was determined using a stepwise calibration procedure (Rance et al., 2014): Starting at 0.2 mA, stimulation intensity was incremented stepwise by 0.2 mA until participants reported their *perceptual threshold*, i.e. that they noticed a sensation, and their *pain threshold*, i.e. that they noticed a painful sensation. Next, stimulation intensity was incremented stepwise by 5% of the individual pain threshold until participants reported their *maximum pain tolerance threshold*, which was defined as "the moment shortly before one would want to tear the electrode off the calf, similarly to when one wants to drop a coffee cup that is too hot." If the pain tolerance threshold was not reached within three of these 5% trials, stimulation intensity was increased by 15% until it was reached. After having determined these thresholds, the individual stimulation intensity was calculated as following: *Pain threshold* + (30% × (*maximum pain tolerance threshold* - *pain threshold*)). Last, participants were stimulated with this intensity and asked to rate it on a scale from 0 (*no sensation*) to 5 (*painful*) to 10 (*maximum tolerable*). If the rating was not 6 or 7, stimulation intensity was adjusted by 0.1 mA and tested again until the rating was 6 or 7.

FMRI preprocessing and first-level analysis. We pre-processed and analysed fMRI data using SPM12 (Wellcome Department of Cognitive Neurology, London, UK). First, functional images were corrected for geometric distortions by applying the FieldMap toolbox, realigned, unwarped, and slice time corrected to the onset of the first slice. The structural images were segmented and normalized to MNI standard stereotactic space. Resulting parameters were then implemented for normalization of the co-registered functional images, which were resampled to isotropic 3 mm^3 voxels and smoothed with a 6 mm full width at half maximum Gaussian kernel. In the first-level model, each event was convolved by a canonical hemodynamic response function. Regressors for the first-level model included the film clips (16s). We also

added images preceding the film-clips (4s) and the six rigid-body movement parameters determined from realignment as covariates of no interest.

Negative-affect markers.

Self-reported negative-affect. Following prior work (Franke et al., 2022), participants rated the perceived unpleasantness of each condition on a Likert scale from 0 (*very pleasant*) to 10 (*very unpleasant*).

Psychophysiological negative-affect marker. Though not optimal (Kreibig et al., 2007), we used HR as psychophysiological negative-affect marker to provide comparability to previous studies (Chou et al., 2014; Danböck et al., 2021) and averaged across the four trials per condition.

Neural negative-affect marker. The PINES is a machine-learning derived signature predicting negative-affect-level based on fMRI activity in brain regions typically activated (positive weights) and deactivated (negative weights) during negative emotions (Chang et al., 2015). Specifically, it is defined by positive weights for regions associated with negative-affect processing (e.g., amygdala, periaqueductal gray, anterior insula, dorsomedial prefrontal cortex, ventral occipital cortex, presupplementary motor area, ventromedial temporal lobe, and posterior cingulate cortex) and negative weights for regions typically deactivated by negative-affect (e.g., parahippocampal gyrus, right superior temporal gyrus, left temporal parietal junction, right caudate, occipital and somatomotor cortices). The PINES has been shown to outperform classical region-of-interest- and network-based markers regarding their sensitivity and specificity for negative-affect (Chang et al., 2015). We calculated the PINES response for each trial by multiplying the first-level vectorized activation images of film-clip responses with the machine-learning derived PINES pattern weights (Chang et al., 2015) and averaged across the four trials per condition.

Pain markers.

Self-reported pain. As recommended (Dansie & Turk, 2013), participants rated how strongly they experienced pain sensations during each condition on a Likert scale from 0 (*not strong at all*) to 10 (*maximal bearable*).

Psychophysiological pain marker. The PPS is a machine-learning derived signature predicting pain-level based on electrodermal activity (Matthewson et al., 2019). Specifically, it is defined by weights assigned to each time point of the temporal course of the electrodermal response to painful stimulation. It has previously demonstrated good sensitivity and moderate specificity for somatic pain, outperforming conventional approaches like taking the grand average or baseline-to-peak amplitude of the electrodermal response (Matthewson et al., 2019). We calculated the PPS response for each trial by multiplying the electrodermal response at each time point during the trial with the corresponding machine-learning-derived weights indexing pain intensity provided by Matthewson et al. (2019). We averaged across the four trials per condition.

Neural pain marker. The NPS is a machine-learning derived signature predicting pain-level based on fMRI activity in brain regions typically activated and deactivated during somatic pain (Wager et al., 2013). Specifically, the NPS is defined by positive weights for pain-related brain regions like the dorsal anterior cingulate cortex, insula, secondary somatosensory cortex, and thalamus and negative weights for structures typically deactivated by pain such as the ventral medial prefrontal cortex and the visual cortex. The NPS has been shown to be highly specific and sensitive for somatic pain (Krishnan et al., 2016; Wager et al., 2013). We calculated the NPS response for each trial by multiplying the first-level vectorized activation images of film-clip responses with the corresponding machine-learning derived pattern weights. In contrast to the NPS development study (Wager et al., 2013), we combined painful stimulation with visual stimuli which are known to activate occipital areas deactivated during mere painful stimulation (Krishnan et al., 2016) and thus having negative weights in the original NPS. Hence, following previous studies (Duff et al., 2020; Franke et al., 2022; López-Solà et al., 2017), we only used the positive NPS pattern weights. We averaged across the four trials per condition.

Measuring dissociation. We assessed dissociation with the short four-item-version of the German Dissociation-Tension Scale acute ('Dissoziations- Spannungs-Skala'; DSS; Stiglmayr et al., 2003, 2009) which has specifically been developed for use during neuroimaging. Overcoming limitations of previous measures of peritraumatic dissociation (van der Hart et al., 2008), the DSS assesses both, psychological (i.e., depersonalization, derealization) and somatic (i.e., somatoform dissociation, analgesia) facets of dissociation, allowing for a comprehensive assessment of dissociation (Stiglmayr et al., 2009). Participants rated how strongly they experienced depersonalization ("*I had the impression that my body does not belong to me*"), somatoform dissociation ("*I had problems hearing,* e.g. *I heard sounds from nearby as if they came from far away*"), derealization ("*I had the impression other people or things around me were unreal*"), and pain analgesia ("*I had the impression that my body or parts of it are insensitive to pain*") during each condition on a Likert scale from 0 (*not at all*) to 9 (*very strong*). The DSS demonstrated good psychometric properties in validation studies (Stiglmayr et al., 2003, 2009). However, while it previously demonstrated good internal consistency ($\alpha = 0.87$; Stiglmayr et al., 2009), it showed acceptable to questionable internal consistencies (ranging from $\alpha = 0.61$ to $\alpha = 0.73$) within our study. In line with theoretical reasoning (van der Hart et al., 2008), this underlines the importance of taking into account phenomenon-specific deviations from overall effects when studying peritraumatic dissociation.

2.4. Statistical analyses

We computed Bayesian multilevel regression models (BMLMs) in R 4.0.3 (R Core Team, 2019) via the Stan-based package *brms* (Bürkner, 2017; Carpenter et al., 2017) to (1) assess effects of aversive-audiovisual and painful-electrical stimulation on dissociation and (2) examine associations between negative-affect and pain markers and dissociation. An overview of all fitted models is provided as supplemental material (Table S1).

To test whether aversive films and painful stimulation cause higher dissociation, we computed a BMLM. Dissociation measured by the four items of the DSS and fitted with a cumulative model (Bürkner & Vuorre, 2019) served as outcome. As predictors, we entered film- and stimulation-type as dummy coded variables (film-type: neutral = 0, aversive = 1; stimulation-type: no pain = 0, pain = 1), as well as the interaction between film- and stimulation-type. To take into account that responses might differ between the four items of the DSS as these capture different dissociative phenomena, item-type was entered into the model as additional predictor. As item-type was effect-coded (i.e., zero reflecting the grand mean), estimates for effects of the predictors of interest on dissociation are averages across all four items. Hence, these coefficients can be similarly interpreted to effects on an overall mean score of all four dissociation items. To also allow for the possibility that film- and stimulation-type might have different effects on different dissociative phenomena captured by the distinct items, interactions between film- and item-type and stimulation- and item-type were included as well. Thus, coefficients for these interactions indicate whether the effect of the predictor of interest on the respective dissociation item deviates from the effect of the predictor on the mean of all four items. We accounted for the repeated measurement design with 4 (items) x 4(conditions), i.e., 16 observations per subject by including a random intercept and random slopes for film-type, stimulation-type, film-type × stimulation-type, and item-type into the model. As approximate leave-one-out-cross validation (Vehtari et al., 2017) indicated no better fit but more problematic observations for a more complex random effect structure also allowing the film-type × item-type and stimulation-type × item-type effects to randomly differ between participants, we decided against including random slopes for these effects in the final model.

To examine whether higher negative-affect operationalized by the

unpleasantness rating, HR, and PINES is associated with higher dissociation, we calculated three BMLMs. Dissociation operationalized by the four items of the DSS and fitted with a cumulative model served as outcome. As predictors, we entered one z-standardized negative-affect marker per model (i.e., either the unpleasantness rating or HR or PINES), the effect-coded variable item-type, and the interaction between the negative-affect marker and item-type. To account for the repeated measurements, a random intercept and random slopes for the respective negative-affect marker and item-type were included into the model. As approximate leave-one-out-cross validation indicated no better fit but more problematic observations for a more complex random effect structure also allowing the negative-affect × item-type interaction to differ between participants, we decided against including a random slope for this interaction in the final models.

To examine whether pain operationalized by the pain rating, PPS, and NPS is associated with higher dissociation, we calculated three separate BMLMs. Again, dissociation fitted with a cumulative model served as outcome. As predictors, we entered one z-standardized pain marker per model (i.e., either the pain rating or PPS or NPS), the effect-coded variable item-type, and the interaction between the pain marker and item-type. To account for the repeated measurements, a random intercept and random slopes for the respective pain marker and item-type were included into the model. As approximate leave-one-out-cross validation indicated no better fit but more problematic observations for a more complex random effect structure also allowing the pain × item-type interaction to differ between participants, we did not include a random slope for this interaction in the final models.

We report regression coefficients (*b*s), 95% credible intervals (CIs), i.e., Bayesian uncertainty intervals, and posterior probabilities ($PP_{b>0}$) for all predictors of interest (information on item-specific effects are provided as supplemental material). As the probit-link allows for the interpretation of the dependent variable as a normally distributed latent variable with a standard deviation of one, *b*s reflect the increase in the latent dissociation variable expressed in standard deviations when the predictor increases by one predictor-unit. For instance, $b = 0.5$ would reflect an increase in the latent dissociation variable by 0.5 standard deviations for an increase in the predictor variable by one predictor-unit reflecting the difference between conditions for categorical variables and the standard deviation for z-standardized continuous predictors. 95% CIs constitute intervals in which the respective parameter falls with a 95% probability given the observed data, prior, and model assumptions. $PP_{b>0}$ values denote the posterior probability of the respective parameter being greater than zero given the observed data, prior, and model assumptions. In other words, $PP_{b>0}$ values closer to 100% provide evidence that the effect of interest is greater than zero and values closer to 0% convey support for the effect of interest being smaller than zero. 95% CIs and $PP_{b>0}$ values allow for a continuous evaluation of support for our hypotheses. Nevertheless, in line with prior work (Franke et al., 2022), we considered effects significantly different from zero if the estimate's 95% CIs did not include zero which equals a $PP_{b>0}$ of the effect below 2.5% or above 97.5% (this would indicate statistical significance on a 5% level).

We used weakly- or non-informative default priors of brms whose influence on results is negligible (Bürkner, 2017, 2018). All BMLMs converged as indicated by common algorithms-agnostic (Vehtari et al., 2021) and algorithm-specific diagnostics (Betancourt, 2017). There were no divergent transitions, $Rhat < 1.01$ and $ESS > 400$ for all relevant parameters.

3. Results

3.1. Do aversive-audiovisual and painful stimulation elicit dissociation?

Effects of film- and stimulation-type on dissociation are illustrated in Fig. 1. Aversive films elicited more dissociation across items than neutral films ($b = 0.69$, 95% CI = [0.17, 1.27], $PP_{b>0} = 100\%$). This effect was not significantly altered for depersonalization and pain analgesia, but stronger for derealization and weaker for somatoform dissociation (see Table S2).

Moreover, painful stimulation elicited more overall dissociation than no painful stimulation ($b = 0.59$, 95% CI = [0.04, 1.20], $PP_{b>0} = 98\%$). This effect was not significantly altered for any item (see Table S2).

No significant interaction between film- and stimulation-type emerged ($b = -0.37$, 95% CI = [−1.01, 0.23], $PP_{b>0} = 11\%$).

3.2. Are negative-affect markers linked to dissociation?

Associations of negative-affect markers with dissociation are illustrated in Fig. 2.

Self-reported negative-affect. Higher unpleasantness ratings were linked to more dissociation across items ($b = 0.44$, 95% CI = [0.21, 0.72], $PP_{b>0} = 100\%$). This effect was not significantly altered for depersonalization, derealization and pain analgesia, but weaker for somatoform dissociation (see Table S2).

Psychophysiological negative-affect marker. We did not find enough evidence linking dissociation to higher HR across items ($b = 0.16$, 95% CI = [−0.38, 0.67], $PP_{b>0} = 74\%$). This effect was not significantly altered for derealization, pain analgesia and somatoform dissociation, but stronger for depersonalization (see Table S2).

Neural negative-affect marker. We did not find enough evidence linking dissociation to higher PINES activation across items ($b = 0.19$, 95% CI = [−0.10, 0.46], $PP_{b>0} = 91\%$). This effect was not significantly altered for any item (see Table S2).

3.3. Are pain markers linked to dissociation?

Associations of pain markers with dissociation are illustrated in Fig. 3.

Self-reported pain. Higher pain ratings were linked to more dissociation ($b = 0.55$, 95% CI = [0.27, 0.89], $PP_{b>0} = 100\%$). This effect was not significantly altered for depersonalization, derealization and somatoform dissociation, but weaker for analgesia (see Table S2).

Psychophysiological pain marker. Higher PPS responses were linked to more dissociation ($b = 0.24$, 95% CI = [0.03, 0.45], $PP_{b>0} = 99\%$). This effect was not significantly altered for derealization, pain analgesia and somatoform dissociation, but stronger for depersonalization (see Table S2).

Neural pain marker. Higher NPS activation was linked to more dissociation across items ($b = 0.24$, 95% CI = [0.05, 0.46], $PP_{b>0} = 99\%$). This effect was not significantly altered for any item (see Table S2).

4. Discussion

The current study aimed to (1) experimentally examine trauma features contributing to the rise of peritraumatic dissociation by independently varying aversive-audiovisual and painful-electrical stimulation within a 2 × 2 within-subject design and (2) provide new insights into the complex negative-affect-dissociation and pain-dissociation relationships by employing a multidimensional assessment of negative-affect and pain responses. Results indicate that both aversive films (vs. neutral films) and pain-stimulation (vs. no-pain stimulation) elicit dissociation in an additive manner. While self-reported peritraumatic negative-affect was linked to higher dissociation-levels, we did not find enough evidence supporting a link between psychophysiological (HR) and neural (PINES) negative-affect markers and overall dissociation. However, pain was consistently across self-report, psychophysiological (PPS), and neural (NPS) domains linked to higher dissociation. Accordingly, in line with evolutionary accounts (Schauer & Elbert, 2010), our findings suggest a particularly strong and robust connection between pain and dissociation.

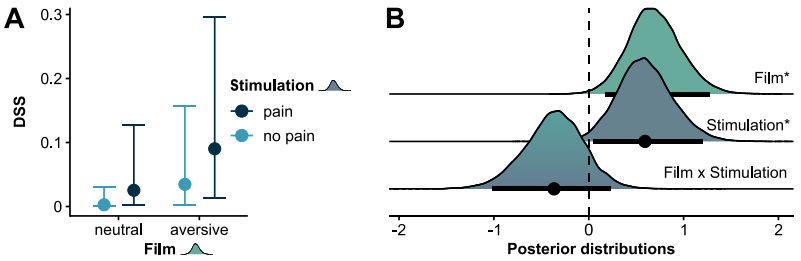

Fig. 1. Effects of film- and stimulation-type on overall dissociation. Panel A displays fitted values of the regression model. Vertical lines represent 95% CIs. Panel B depicts posterior distributions of each effect of interest. The regression coefficient *b*, i.e., the mean of the posterior distribution, is depicted with a bold dot. The 95% CI, i.e., the interval between the 2.5th and 97.5th percentile of the distribution containing 95% of posterior draws, is depicted with a bold line. The $PP_{b>0}$, i.e., the percentage of posterior draws being greater than zero, is represented by the area under the posterior density to the right of zero. Effects whose 95% CIs did not include zero were marked as significant (*). Abbreviations: DSS = Dissociation-Tension Scale, short version.

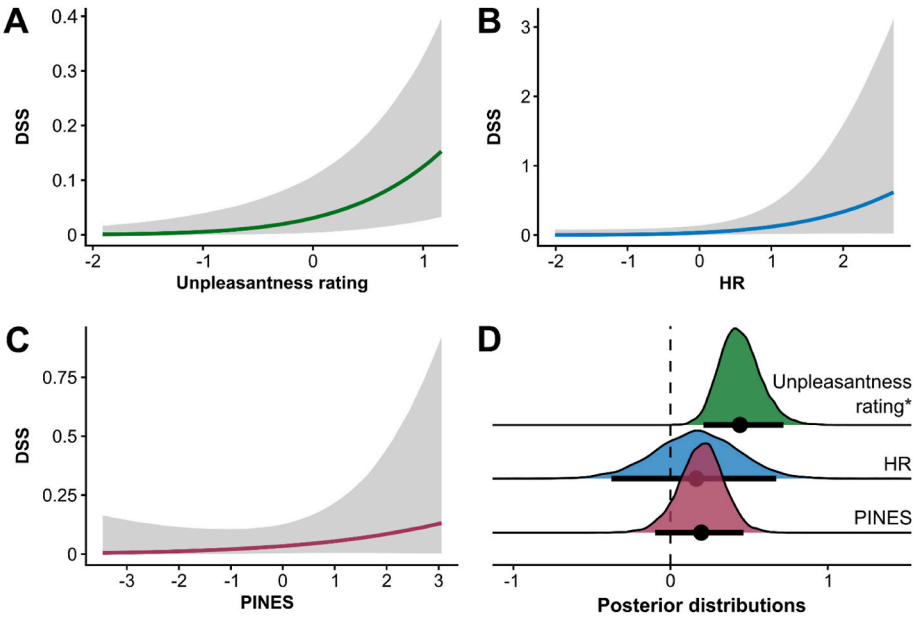

Fig. 2. Effects of negative-affect measures on overall dissociation. Panels A–C display fitted values of regression models. Vertical lines represent 95% CIs. Panel D depicts posterior distributions of each effect of interest. The regression coefficient *b*, i.e., the mean of the posterior distribution, is depicted with a bold dot. The 95% CI, i.e., the interval between the 2.5th and 97.5th percentile of the distribution containing 95% of posterior draws, is depicted with a bold line. The $PP_{b>0}$, i.e., the percentage of posterior draws being greater than zero, is represented by the area under the posterior density to the right of zero. Effects whose 95% CIs did not include zero were marked as significant (*). Abbreviations: DSS = Dissociation-Tension Scale, short version; HR = heart rate; PINES = Picture-Induced-Negative-Emotions-Signature.

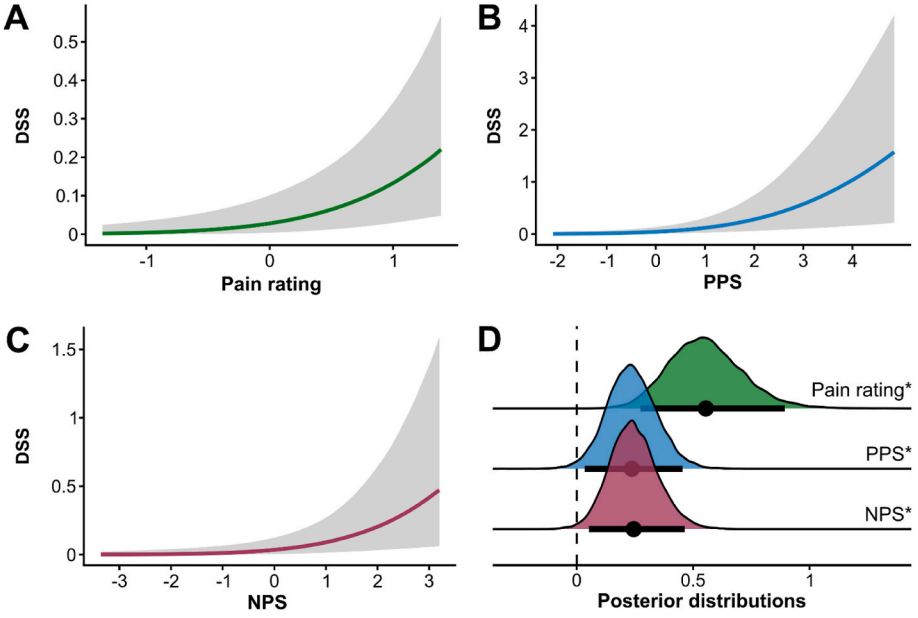

Fig. 3. Effects of pain measures on overall dissociation. Panels A–C display fitted values of regression models. Vertical lines represent 95% CIs. Panel D depicts posterior distributions of each effect of interest. The regression coefficient *b*, i.e., the mean of the posterior distribution, is depicted with a bold dot. The 95% CI, i.e., the interval between the 2.5th and 97.5th percentile of the distribution containing 95% of posterior draws, is depicted with a bold line. The $PP_{b>0}$, i.e., the percentage of posterior draws being greater than zero, is represented by the area under the posterior density to the right of zero. Effects whose 95% CIs did not include zero were marked as significant (*). Abbreviations: DSS = Dissociation-Tension Scale, short version; PPS = physiological skin-conductance-response-based pain-signature; NPS = Neurologic-Pain-Signature.

4.1. Trauma features eliciting dissociation

The current and previous findings (Chou et al., 2014; Holmes et al., 2004, 2006) suggest that aversive-audiovisual stimulation, i.e., mere audiovisual witnessing a traumatic event without being personally involved, might already suffice to evoke dissociation. In line with evolutionary models proposing that encountering a direct physical threat to one's life might be an important factor contributing to the rise of dissociation (Schauer & Elbert, 2010), painful-stimulation also evoked dissociation in our and previous studies (Giesbrecht et al., 2008; Gómez-Pérez et al., 2013; Horowitz & Telch, 2007). This is in line with the idea that pain constitutes a signal of tissue damage which might limit the fight-or-flight capacity and thereby promote dissociation as an adaptive response. When being injured, dissociation might facilitate remaining still which might be helpful to not provoke further injuries either by harming oneself or by attracting the perpetrator's attention (Schauer & Elbert, 2010). Our data did not support an interaction between effects of aversive-audiovisual and painful-stimulation on dissociation suggesting that both factors might independently contribute to the rise of dissociation. In other words, they might influence dissociation in an additive manner with the highest dissociation-levels for combined aversive-audiovisual and painful stimulation and the lowest for the absence of both. Yet, the credibility intervals for the interaction effect were not perfectly centered around zero which may imply that our study was not sufficiently powered to detect a subtle interaction effect.

4.2. Negative-affect, pain, and dissociation

In line with theoretical accounts understanding peritraumatic dissociation as reaction to unbearable distress (Carlson et al., 2012; Dutra & Wolf, 2017; Schauer & Elbert, 2010) and previous studies linking peritraumatic dissociation to higher self-reported peritraumatic distress and pain (Beaudoin et al., 2021; Bryant et al., 2011; Fikretoglu et al., 2006; Lewis et al., 2014; Otis et al., 2012), dissociation was also linked to self-reported negative-affect and pain in our study. Critically, to overcome the limitations of retrospective self-reports and enable a deeper understanding of negative-affect-dissociation and pain-dissociation relationships, we additionally employed psychophysiological and neural markers of negative-affect.

Partly in line with previous findings (Danböck et al., 2021), HR was linked to depersonalization but not to the overall dissociation score. This could indicate that psychophysiological arousal might have a stronger relationship with depersonalization than somatoform dissociation, pain analgesia, and derealisation. However, the absence of evidence for the later relationships might as well be due to the restricted validity of HR acceleration not being consistently linked to negative valence across studies (Kreibig et al., 2007). In particular, the short film duration within our paradigm (16s) might have blurred associations of HR with dissociation, as the HR response to very aversive stimuli in women typically consists of a rapid HR drop (initial orienting response) followed by HR acceleration (Bradley et al., 2001; Lang et al., 2000). We did not find enough evidence for a link between the neural negative-affect marker (PINES) and dissociation. It could be that, even though the PINES has demonstrated high specificity and sensitivity for negative-affect (Chang et al., 2015), it might not be the best marker in our context, as it was trained on and validated for affective pictures. However, exploratory analyses demonstrating sensitivity of PINES responses for the dynamic aversive-audiovisual stimulation in our study contradicts this explanation (for details see Supplements). Alternatively, it could be the case that dissociative experiences are tied to a very specific or extreme negative affect (e.g., hopelessness, intense fear) which the PINES, designed to predict general negative affective states, may not have captured. Hence, although our data does not suggest a link between activation in brain regions involved in general negative-emotion processing with peritraumatic dissociation, we cannot rule out associations between specific emotions or types of negative-affect and dissociation.

Critically, our data support a specific role of pain with subjective, psychophysiological (PPS), and neural (NPS) pain markers being consistently linked to dissociation. The neural pain marker has been shown to be highly specific for somatic pain (Chang et al., 2015). Hence, underpinning evolutionary accounts (Schauer & Elbert, 2010) postulating that dissociation might be especially likely when one is injured, our study provides first evidence for a close coupling between peritraumatic pain assessed on multiple domains, and dissociation.

Altogether, our data link self-reported unpleasant and painful experiences, accelerated HR, a pain-specific electrodermal response pattern (PPS), and a pain-specific neural activation pattern (NPS) but not a negative-affect-specific neural activation pattern (PINES) to peritraumatic dissociation. Interestingly, exploratory analyses revealed several positive correlations between negative-affect and pain markers. Further, except from the PINES pattern (which did not respond to the pain stimulation) all negative-affect and pain markers were influenced by both aversive-audiovisual and painful stimulation (for details see supplements). This raises the question whether negative-affect and pain constitute separable constructs or whether their conceptual and empirical overlap calls for a re-conceptualization of these constructs. As recently discussed by Gilam et al. (2020), negative-affect and pain could be understood as 1) one being a component of the other with the most prominent model defining negative-affect as an intrinsic component of pain or 2) two overlapping constructs sharing some and differing in other conceptual, psychophysiological, and neurophysiological components. While the current state-of-the-art does not yet allow a clear positioning regarding these viewpoints, for the current study it seems critical that both viewpoints imply that negative-affect and pain share some subjective, physiological, and neural patterns. Hence, our finding that pain and negative-affect markers were correlated and probably driven by common factors might suggest that especially these shared patterns could be closely connected with dissociation. To shed light on this, future studies might specifically assess shared (e.g., feelings of helplessness or fear) and unique (e.g., feelings of sadness or guilt) components of negative-affect in relation to pain, examine their association with dissociation, and thus promote further insights into the complex negative-affect-pain-dissociation interplay.

4.3. Future directions

Peritraumatic negative-affect, pain, and dissociation are part of a complex interplay. While negative affect and pain are assumed to initially provoke dissociative responding, dissociation might, once its intensity reaches a certain level, attenuate negative affect and pain experience, resulting in an inverse U-shaped relationship (Danböck et al., 2021; Schauer & Elbert, 2010). In our study, negative affect and pain manipulations elicited dissociation. Moreover, experiential, psychophysiological, and neural markers of pain and in part, negative-affect, were positively associated with dissociation, supporting the conceptualization of dissociation as a response to both emotionally distressing and painful experiences (Schauer & Elbert, 2010). To examine whether dissociation might, particularly at higher-dissociation levels, reduce negative affect and pain, experimentally manipulating dissociation, either behaviorally (Hagenaars et al., 2008; Leonard et al., 1999; Renard, Huntjens, & Pijnenborg, 2018; Shin et al., 2019) or pharmacologically (Feder et al., 2014) in healthy samples and patient samples with a higher propensity to dissociate is indispensable. In a study in healthy participants dissociation induction via mirror-gazing attenuated self-reported negative affect during emotional picture viewing (Shin et al., 2019), supporting the conceptualization of dissociation as a way to numb negative emotions. Future studies might also examine effects of dissociation induction on objective negative affect and pain markers, to account for various internal and external factors influencing self-report measures of negative affect and pain (Candel & Merckelbach, 2004; de Williams et al., 2000). Moreover, it might be

interesting to consider (continuously collectable) behavioural markers of dissociation currently undergoing investigation (i.e., immobility, staring; Abrams et al., 2009, 2012; Cardeña et al., 2017; Danböck et al., 2021; Maia et al., 2015) and examine their instantaneous dynamic associations with (continuously collectable) physiological and neural negative affect and pain markers.

Our data suggest that trauma-related dissociation might initially be caused by aversive-audiovisual or painful events. Individuals regularly witnessing traumatic events (e.g., staff of emergency care units, police officers, firefighters) may react with subtle dissociative symptoms that may be overlooked but might constitute an early warning signal for later PTSD development (Danböck et al., 2021; Lensvelt-Mulders et al., 2008). Thus, routine screening for such symptoms might be a worthwhile strategy.

4.4. Limitations

First, ratings of negative-affect, pain, and dissociation were provided retrospectively at the end of the experimental procedure. However, as the physiological and neural markers for negative-affect and pain obtained directly during each trial complement the results drawn from the self-report measures, we feel confident that reporting biases might be low. Second, due to time and monetary restrictions we only included women. Hence, as biological sex has been shown to influence emotional and stress responses to aversive films (Wilhelm et al., 2017), conclusions do not necessarily generalize to men. Third, our trauma-analogue paradigm only induced mild levels of dissociation. However, as we aimed to specifically examine factors contributing to the initial onset of dissociation, this might be advantageous as higher levels of dissociation could, due to the assumed complex bidirectional relation between dissociation and affective correlates (Danböck et al., 2021; Schauer & Elbert, 2010), limit the interpretability of findings. Moreover, though the overall levels of dissociation were low in our healthy sample, the considerable size of our regression estimates denotes their reliability and substantial value for the prediction of dissociation.

5. Conclusion

Many traumatic incidents involve both, aversive audio-visual input and physical pain experiences. Our findings indicate that both can elicit dissociation. Crucially, self-reported, psychophysiological, and neural pain markers were consistently linked to dissociation-level, suggesting a specific, evolutionarily meaningful, connection between pain and dissociation.

CRediT authorship contribution statement

Sarah K. Danböck: Conceptualization, Methodology, Formal analysis, Investigation, Visualization, Writing – original draft. **Laila K. Franke:** Conceptualization, Methodology, Investigation, Data curation, Project administration, Writing – review & editing. **Stephan F. Miedl:** Conceptualization, Methodology, Software, Formal analysis, Writing – review & editing. **Michael Liedlgruber:** Methodology, Software, Formal analysis, Writing – review & editing. **Paul-Christian Bürkner:** Methodology, Formal analysis, Visualization, Writing – review & editing. **Frank H. Wilhelm:** Conceptualization, Methodology, Supervision, Project administration, Writing – review & editing.

Declaration of competing interest

All authors declare no conflict of interest.

Data availability

Data will be made available on request.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brat.2023.104289.

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