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Influence of acute stress on attentional bias toward threat: How a previous trauma exposure disrupts threat apprehension

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ABSTRACT

While previous trauma exposure is known to be a risk factor for the development and maintenance of many psychological disorders, it remains unclear how it increases individual risk for prospective psychopathology in the aftermath of a new trauma exposure. The aim of this study was to investigate how a prior exposure to trauma affects attentional processing of threat before and after an acute stress task. Specifically, we assessed attentional biases to threat before and after a cold pressor task in 17 individuals who have been exposed to trauma (TE) compared to 18 individuals without trauma exposure (NTE). Behavioral results showed difficulties to disengage from threat in TE but not in the control group prior to stress induction, as well as a switch to an attentional bias toward threat after the cold pressor task in the TE group. For the ERPs, we highlighted (1) decreased N1 negativity in response to threatening stimuli after an acute stress in both groups, and (2) a parallel increase in P1 for such stimuli only in the TE group. Those results suggest a vulnerability presented by previously traumatized individuals when dealing with threats as well as an acute responsitity toward stress. Those results are interpreted in regards with the theorical models of stress and anxiety.

1. Introduction

According to the World Health Organization (WHO), each person will be victim of 1.9 traumatic events during their lifetime (Kessler et al., 2017). At the immediate aftermath of this trauma, a majority will experience symptoms such as flashbacks, sleep disturbances or avoidance of trauma reminders (American Psychiatric Association, 2013). While those symptoms will decrease within a few weeks after the trauma for most individuals, this population represents a particularly at-risk group for the development of mental disorders (post-traumatic stress disorder, depression, substance abuse, gambling disorders, etc.) if they encounter another traumatic episode in the future (Kausch et al., 2006; Kessler et al., 2014; Sarchiapone et al., 2007). One key mechanism involved in both adaptative threat detection and development of anxiety disorders and PTSD are attentional biases toward threat. Therefore, the impact of a first-trauma exposure on one's apprehension of a new emotional situation constitutes an interesting area, even though the processes underlying attentional allocation under stress are poorly understood nowadays. As a first-trauma exposure would establish a bridge between acute stress and anxiety related disorders, results concerning behavioral and neurocognitive correlates of these emotional states may help to build a coherent theoretical framework for predicting risk and resilience factors.

The ability to rapidly detect cues of threat is a highly adaptative behavior, in particular in dangerous or stressful situations, and relies on both automatic and conscious processes in our attentional system. Corbetta and Shulman (2002) described two attentional systems that play distinct but complementary roles in the control of attention: a *top-down* (goal-directed) control system involved in preparing and applying goal-directed selection of stimuli and action responses and a *bottom-up* (stimulus-driven) control system involved in the detection of and orientation toward salient stimuli. The balance of those two systems defines *attentional control*. In their integrative framework of stress, Vine et al. (2016) examined the influence of stress on the two attentional networks proposed by Corbetta and Shulman (2002). These authors suggest that stress might increase the influence of the bottom-up attentional system, leading to heightened distractibility and poorer performance on a given ongoing task. Specifically, an individual submitted to an acute stress and perceiving a threat may not be able to select all the relevant sources needed to accurately perform a task and could have a tendency to focus on irrelevant information. This was corroborated by Rued et al. (2019) who exposed participants to either high or

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low stress conditions during a face in the crowd paradigm displaying threatening and nonthreatening facial targets among distractors. They found that even though both groups were faster to detect threat in comparison with other distractors, highly stressed participants were faster to detect threat and less accurate (Rued et al., 2019). Bishop (2004) further analysed the effect of stress (which she referred to as high *state anxiety*) on neural processing of fear. She observed that participants with high state anxiety displayed an increase amygdala activation to attended and unattended fearful information. She concluded that low-stressed participants were able to modulate their amygdala response to fearful faces by focusing their attentional resources on not-threatening informations. However, high-anxious participants were not able to decrease their amydgala responses by attentional focus (Bishop, 2007). The faster detection of emotional information observed in stressed participants by Rued et al. (2019) might therefore derived from this increased amygdala activation, and the lack of ability to modulate this fear response by focusing on non-threatening information.

Eysenck et al. (2007) described the influence of pathological trait-anxiety on the top-down attentional system (Eysenck et al., 2007; Eysenck and Derakshan, 2011). Through the attentional control theory (ACT), he posits that attentional biases to threat arise from a lack of top-down attentional system, especially during tasks with high cognitive loads. According to the Attentional Control Theory, trait anxiety would facilitate the processes associated with the detection of and orientation toward threat stimuli (bottom-up attentional system) while decreasing the prefrontal control necessary to regulate the allocation of attentional resources to task-relevant stimuli (top-down attentional system). This imbalance would be responsible for the manifestations of attentional biases which are characterized by an initial hypervigilance for threat followed by difficulties to disengage their attention from it (Cisler and Koster, 2010). Furthermore, while trait anxiety might impair attentional control, individuals could maintain their performance if they mobilize additional processing resources (effort). Eysenck et al. (2007) therefore distinguishes processing efficiency (effort needed to reach this performance) from performance effectiveness (i.e. the quality of performance). Theories of Vine et al. (2016) and Eysenck et al. (2007) differentiate the effect of stress and anxiety on attentional control. While stress might only enhance the stimulus-driven attentional system, anxiety might arise from both increased bottom-up system and impaired goal-oriented attentional system. Hence, stress and anxiety seem to have a differential behavioral and neural impact on threat processing. However, even though the confrontation with a traumatic event is known to be the intermediary between those two poles, only a handful of studies have examined the impact of trauma per se (without PTSD) on stress responses toward threat.

In order to assess attentional biases to threat, the dot-probe task has been widely used (Bardeen and Orcutt, 2011; Bullock and Bonanno, 2013; Chan et al., 2013). In this task, the computation of reaction times provides indices of orientation of attention, disengagement of attention and avoidance (Evans and Britton, 2018). To complement these RT analyses, event related potentials (ERPs) provide a high temporal resolution measure of attentional processes during EEG recording as well as a representation of processing efficiency (the mobilization of additional resources needed to accurately perform the task). Therefore, the high temporal resolution of ERPs allows the assessment of the efficiency of both automatic and conscious control processes involved in attentional bias toward threat which extends the knowledge gained by RT alone. Considering that ERPs provided complementary measurement of attentional bias to threat in previous research (Bar-Haim et al., 2005), we can assume that the effects of stress could be better represented by ERPs as well.

Several components have been specifically examined in relation to attentional biases during a dot-probe task (Kappenman et al., 2015; Torrence and Troup, 2017). First, the P1 component peaks at fronto-central sites and reflects early visuospatial orienting of attention. Accordingly, a larger P1 amplitude is known to be found after emotional stimuli – particularly threat stimuli, and represents an increased amount of cognitive resources directed to the processing of those visual stimuli (Zhang et al., 2017). Second, the N1 component is the first negative deflection following the P1 that reflects early attention allocation facilitating further perceptual processing and discrimination of stimuli (Choi et al., 2014; Ernst et al., 2013; Luck, 2014). Third, the P3 component reflects high level cognitive processes and conscious allocation of attentional resource (Torrence and Troup, 2017). It is known to be influenced by the motivational significance of the affective stimuli, and might reflect the allocation of conscious processing resources to motivationally relevant stimuli (Kosonogov et al., 2018).

Some studies have evaluated stress responses toward threatening information with a dot-probe task among healthy participants. In regard to behavioral responses, Carr et al. (2016) highlighted a significant avoidance of threat in healthy women at baseline (presenting low state anxiety), following by a significant change to an attention bias toward threat following an acute induction of stress (therefore presenting a high state anxiety). Referring to electrophysiological components, Jiang et al. (2017) demonstrated that stress suppressed attentional bias toward threatening stimuli, as indexed by a lack of P1 for the threatening information. Those results have been understood as a decreased ability to perceive threat under stress, the distinction between threat and not threat has become more subtle. Stress has also been linked to an increased N1 negativity, representing a higher vigilance toward threat (Qi et al., 2016; Shackman et al., 2011). Finally, to our knowledge only one study investigated this impact of stress on this component. Shackman et al. (2011) showed a decreased P3 amplitude under stressful condition, which they interpreted as attenuated selective attention induced by stress.

Other studies have used the same paradigm among high trait-anxiety individuals. Bar-Haim et al. (2007) depicted an attentional vigilance toward threat, followed by difficulties in disengagement among individuals suffering from chronic anxiety. Helfinstein et al. (2008) demonstrated that stress induced an increased attentional bias toward threat, as revealed by increased P1 and decreased reaction times for such stimuli after the stress exposure. They suggested that a brief exposure to threat increased alertness and vigilance for fearful stimuli among socially anxious patients (Helfinstein et al., 2008). The complex P1/N1 has been investigated by Bar-Haim et al. (2005) who found that highly anxious individuals recorded a larger amplitude on P1 and N1 waveforms when viewing emotional facial expressions. Finally, several modulations of the P3 have been found in anxiety-related pathologies (Bar-Haim et al., 2005; Johnson et al., 2013; Judah et al., 2013).

Finally, a few studies have evaluated the stress response in individuals reporting previous trauma without PTSD. Among these, Zhang et al. (2014) measured RT and ERPs in response to an emotional dot-probe task in a trauma-exposed sample of earthquake survivors compared to a healthy control group. They found larger P1 amplitudes and faster RT to congruent (threat) trials in the trauma-exposed sample (Zhang et al., 2014). Bardeen and Orcutt (2011) have examined attentional bias mechanism of participants with high and low post-traumatic stress symptoms (each group being under the threshold of PTSD) using a dot-probe paradigm with either 150 or 500 ms presentation time of emotional pictures. They described that higher PTSS were associated with greater attention to threat stimuli when stimuli were presented for 500 ms. Specifically, participants with higher PTSS had greater difficulty disengaging from threat stimuli in comparison to participants low in PTSS who seem to have disengaged and shifted their attention from threat stimuli by 500 ms in favor of attending to neutral stimuli (Bardeen and Orcutt, 2011). Recently, Gindt et al. (2017) compared attentional biases measured by a visual search task with a lexical decision component among healthy, anxious and students with post-traumatic stress symptoms (PTSS). While authors reported an expected attentional bias toward threat in both the control and anxiety group, in contrast, the PTSS group presented disengagement difficulties (Gindt et al., 2017).

This study aimed to investigate the role of previous exposure to a

traumatic event on AB in response to threatening pictures before and after an acute stress induction. Acute stress was induced using a Cold Pressor Task (CPT, Bryant et al., 2013; Nicholson et al., 2014) and salivary alpha amylase (sAA) levels, which correlate robustly with sympathetic arousal, were used to evaluate response to the stress task. Attentional biases were measured with an emotional dot probe task before (at baseline) and after the stress induction via behavioral and ERP component. According to previous studies, we expected different attentional biases and different response to stress between TE and healthy controls. Previously traumatized individuals might present an attentional pattern closer to those observed in anxiety-related disorders while healthy controls should respond to stress according to Vine et al. (2016) conceptual framework of stress. Specifically, we predicted NTE would present a facilitated engagement toward threat while TE should show disengagement difficulties at baseline (Gindt et al., 2017; Kappenman et al., 2015). Those effect are expected in terms of RT, as shown by (1) a higher orientation and disengagement index for the NTE group and ERPs, as shown by (2) a smaller P3 amplitude following threatening information for the TE group. After the stress induction, we expect a switch toward facilitated engagement in the TE group (Carr et al., 2016), as indexed by (3) an increased orientation index, (4) an increased P1 amplitude (Zhang et al., 2014) and (5) an increased P3 amplitude following threat (Johnson et al., 2013) compared with baseline. We also expect a general impact of stress in both group, as indexed by (1) a decrease of RT to identify threat (Rued et al., 2019), and an increase of N1 negativity after stress (Qi et al., 2016; Shackman et al., 2011).

2. Method

2.1. Participants

37 participants took part in the study (18 females and 19 males, mean age = 25.27, *SD* = 6.11). They were recruited through an existing pool of participants listed by the University of Tasmania, including eligible first-year psychology students who received 2 h of course credit in return for participation. We firstly accessed to pre-encoded demographic information, including trauma-exposure which allowed us to create our groups and specifically contact eligible participants. We secondly excluded participants who had reported a psychiatric history apart from PTSD, diagnosed attentional deficits, neurological disease, substance abuse, and traumatic brain injury. Participants were then classified into two groups according to their responses to the Traumatic Events Questionnaire (TEQ: Vrana and Lauterbach, 1994). The first group was comprised of 20 trauma-exposed (TE) participants reporting at least one Criterion A trauma on the TEQ who were allocated to the TE group and the second group comprised 19 non-trauma-exposed (NTE) individuals. In order to avoid the inclusion of potential acute stress disorder, TE participants were screened for exposure to a traumatic event occurring more than 6 months before the completion of the TEQ. When those individuals were contacted, they completed the Post-Traumatic Stress Disorder Checklist (PCL-5: Weathers et al., 2013) to assess the presence of PTSD symptoms. We excluded participants who reported a clinical PTSD symptoms (corresponding to a total score above 33). Two participants were excluded due the presence of high PTSD score, two to poor behavioral performance (below 3 standard deviations from the mean), which resulted in 18 participants in the TE group and 17 participants in the NTE group. The two groups were matched for sex.

2.2. General procedure

Written informed consent was obtained from participants after they read an information sheet and asked any questions they may have had. Participants then provided an initial saliva sample to assess baseline salivary alpha-amylase (sAA). The medical history, TEQ and Depression, Anxiety, and Stress Scale were already encoded in the database prior to testing, and PCL-5 was completed before the EEG cap was prepared and securely fitted to the scalp. Participants were seated 50 cm from the NeuroSCAN computer screen and given full instructions about how to complete the dot-probe task. All participants undertook five practice trials using *International Affective Picture System* (IAPS) images which had similar valence and arousal to the experimental set but were not included in the actual task. Each participant then completed two counterbalanced blocks of 57 trials, for a total of 114 trials. Block A and B were constructed of different images with an equal distribution of congruent, incongruent, and neutral trials, and all participants completed them in the same order. Following the 57 trials of block A, participants undertook the cold pressor task, immediately after which they provided a second saliva sample to assess the effect of stress on sAA following the CPT, before beginning block B of the Dot-Probe Task. Following the completion of the 57 trials of block B, the EEG cap was removed. Testing time averaged 90 min including placement and removal of the EEG cap, dot-probe task, saliva sampling and cold pressor task. Participants had the opportunity to ask any follow-up questions before departing. The study was approved by the University of Tasmania Social Sciences Human Research Ethics Committee.

2.3. Attentional bias and stress induction

2.3.1. Dot-probe task (DPT)

The dot-probe task was created in line with tasks used by Carr et al. (2016) and displayed on a computer screen using a custom NeuroSCAN STIM computer. Each dot-probe trial involved an initial black screen for 400 ms, followed by two paired IAPS images (either a neutral/neutral pair or a negative/neutral pair) being displayed on screen for 1000 ms. A total of 152 neutral (e.g. boat, painting) and 75 negative (e.g. vicious dog, car accident) pictures were selected for the IAPS. The neutral images had a mean valence of 5.62 (*SD* = 0.98) and arousal of 3.67 (*SD* = 0.89) respectively, while the negative images had a mean valence of 2.92 (*SD* = 0.92) and arousal of 5.86 (*SD* = 0.82) respectively. Immediately following offset of the images, a white dot appeared on the left or right of the screen, depending on condition. The congruent condition involved the dot-probe appearing on the same side of the screen as the negative image in the previous pair, while the incongruent condition was the opposite. The neutral condition had the dot-probe following a pair of neutral images. In consequence, the location of the probe did not matter and was randomly distributed within the neutral pairs. Participants then had 2000 ms to select the A (left screen) or L (right screen) key on the keyboard to indicate probe location before the trial cycle begins again. Participants performed a first block of 57 trials before the cold pressor task and a second block of 57 trials after this stress induction.

2.3.2. The Cold Pressor Task (CPT)

The CPT is a widely-used and standardized stress induction task that is employed to induce physiological arousal (Mitchell et al., 2004). This task requires participants to submerge their hand past the wrist into a tub of water maintained at 4 degrees Celsius, which has been found to reliably invoke a sympathetic nervous system response which includes an increase in the release of NE in the body (Victor et al., 1987). A time limit of 3 min was placed on the task due to the minimum-risk ethics approval. Participants were told to remove their hand from the water at the point where they could no longer tolerate the discomfort, where they felt pain, or once the 3 min upper-limit had been reached.

2.3.3. Salivary Alpha-Amylase (sAA)

sAA has been validated as a biomarker of endogenous noradrenaline that is secreted by the salivary glands and reflects sympathetic nervous system activation when individuals are under stress (Rohleder and Nater, 2009; Thoma et al., 2012). We collected saliva samples from participants using the passive drool method before and after the acute stress induction.

2.4. Self-reported questionnaires

2.4.1. Traumatic Events Questionnaire (TEQ: Vrana and Lauterbach, 1994)

The TEQ is a 11 items self-report questionnaire assessing nine events which may have occurred in an individual's life, such as a serious accident, being a victim of physical or sexual abuse, or witnessing someone dying in a violent manner. The TEQ has been found to be a valid and reliable measure of traumatic exposure with high test-retest reliability in primary care and non-clinical samples (Crawford et al., 2008). It was administered to all participants before testing began, to identify whether participants had been exposed to a Criterion A traumatic event, meaning an event in which they were at risk of injury or death, or witnessed someone being in such a situation, through assault, disaster, or war (APA, 2013). Results from the TEQ were therefore considered an appropriate measure for TE group allocation.

2.4.2. Post-Traumatic Stress Disorder Checklist for DSM-5 (PCL-5: Weathers et al., 2013)

The PCL-5 is a 20 item self-report scale used to assess an individual's experience of the symptoms of PTSD, as described in the Diagnostic and Statistical Manual of Mental Disorders – 5th edition (DSM-5: American Psychiatric Association, 2013). Possible total scores range from 0 to 80 using a 5-point Likert scale (0 = not at all, 4 = extremely). The PCL-5 was used in this study to ensure appropriate allocation of participants into the TE group. Classification of possible PTSD begins with individuals having a score of 33 or higher, no participants in the current study reached this cut-off. The PCL-5 has excellent reliability, with a Cronbach's Alpha level of 0.93 found in a study by Lowe et al. (2015), similar to reliability of earlier iterations of the PCL.

2.4.3. Depression, Anxiety, and Stress Scale (DASS-21: Lovibond and Lovibond, 1995)

The DASS-21 is a 21 item scale which is used to determine an individual's recent mood through a 4-point Likert scale (0 = did not apply to me at all, 3 = applied to me very much or all of the time) which assesses three subscales of depression, anxiety, and stress. DASS scores were summed and used to assess individuals' variation on level of depressed mood (DASS-D), anxiety (DASS-A), and stress (DASS-S) leading up to time of testing, and each subscale was summed separately and individually analysed as a covariate of reaction time measures to ensure that depression, anxiety, and stress levels did not have a significant effect on the data or group allocation. The DASS-21 has been validated as a reliable indicator of separate depression, anxiety, and stress (Cronbach's Alpha = 0.93) and has been suggested to be a more effective measure than the full-scale DASS (Henry and Crawford, 2005).

2.5. Physiological apparatus and recording, data reduction

EEG data was recorded for the dot-probe task using Neuroscan SCAN 4.5 software (Compumedics Neuroscan, 2003) and a SymAmps2 system which was connected to a 32-channel EEG Quick-cap with silver and silver chloride electrodes. EEG recordings were taken from 32 sites with eight midline, parietal, and occipital sites being utilised for analysis based upon inspection of the grand mean average data. Placement of electrodes on the scalp was completed in accordance with the International 10–20 system of electrode placement (Jasper, 1958), with all electrodes referenced by linked mastoids and grounded by an AFz ground electrode. Electro-oculogram electrodes were placed above and below the left eye and at the outer corner of both eyes to allow for control of horizontal eye movement and eye blinks. Electrode impedance was maintained at or below 10 KΩ. The continuous sampling rate was 1000 Hz, amplified at 200 Hz, and data was rejected based on horizontal and vertical electro-oculogram activity as well as artefact exceeding ±125 μV. The data was filtered at 30 Hz using a low-pass filter, with epoching completed from 100 ms pre-stimulus onset to 900 ms at stimulus offset. ERP components were selected in relation to a 100 ms baseline window before each stimulus onset. We selected a window surrounding the maximal peak of each ERP component for amplitude analysis, resulting in a positive wave between 80 and 150 ms after image onset (i.e. P1, averaged across CPZ and CZ,), a negative wave between 100 and 220 ms after image onset (i.e. N1, averaged across P3 and P4,), and a positive wave between 200 and 330 ms after image onset (i.e. P3, averaged across CPZ and CZ). Those electrode sites and windows were chose based a review of previous research conducted using an emotional dot-probe task (Bar-Haim et al., 2005; Torrence and Troup, 2017), as well as an observation of the location of maximal grand-average waveform effects. Analyses were conducted on mean amplitudes of each component. The mean amplitude for each component was examined for outliers.

Analysis of NE was undertaken by collecting saliva samples from participants using the passive drool method. Salivary NE levels were analysed by standard assays of sAA at Macquarie University Pathology Lab with samples stored frozen at −20 °C until assay. On the day of experiment, the samples were thawed and analysed using commercially available kits according to the manufacturer's instructions (Salimetrics, USA). Thawed samples were centrifuged at 1500 ×*g* for 15 min to collect clear saliva and this saliva was used without further processing for all assays. All samples were brought to room temperature before adding to assay wells and all samples were analysed in duplicate.

Concerning behavioral data, analyses were conducted on reaction times (RT) latencies for correct trials only. RTs longer than 2000 milliseconds or shorter than 150 milliseconds were excluded of further analyses. Data beyond 2.5 standard deviation below or above each participant's mean were discarded as outliers (1.57% of the data at baseline, 3.8% after the stress induction). The Response-Based Computation (RBC) is a recently proposed calculation method which captures intra-individual variability of threat-related attention by comparing individual RT in each valid (or invalid) trial to the individual mean RT in invalid (or valid) trials. This method minimizes data loss and allows to measure vigilance and avoidance. Using RBC, individual reaction times on congruent trials can be *separately* indexed against their individual average reaction time on neutral trials (i.e., Trial 1: $RT_{NeutralMean}$ - $RT_{Congruent\ 1}$, Trial 2: $RT_{NeutralMean}$ - $RT_{Congruent\ 2}$, …, Trial $_{n:\ RTNeutralMean}$ - $RT_{Congruent\ n}$). The different scores across trials are subsequently aggregated to generate separate measures of orientation (positive scores indicate vigilance while negative score indicate avoidance processes) and disengagement. For a detailed description of this computation method, see (Evans et al., 2020; Evans and Britton, 2018).

2.6. Statistical analyses

Statistical significance was accepted at p value of 0.05. To minimize type I errors, the Greenhouse-Geisser correction was applied in all repeated-measures analysis of variance (ANOVA). Data were distributed normally (Kolmogorov-Smirnov $p > 0.05$ for all variables). Analyses have been completed using the software IBM SPSS Statistics for Windows (Version 21, Armonk, NY, IBM Corp.)

2.6.1. Demographic data

Between-groups comparisons were performed on demographic (age, gender, history of trauma) using Pearson chi-square tests and on psychopathological characteristics (post-traumatic symptoms, depression, anxiety) using one-way analysis of variance (ANOVA).

2.6.2. Mean reaction time data

A repeated-measures analysis of variance (ANOVA) was performed on mean RT with Time (T1, pre-stress and T2, post-stress) and Congruence (Congruent and Incongruent) as within-subject factor and Group (TE and NTE) as between-subject factor.

2.6.3. Response-Based Computation (RBC)

As RBC scores are computed from different RT trials, those variables do not represent different values of a same factor. Therefore, RBC orientation and disengagement bias were analysed distinctively through two repeated-measures analysis of variance (ANOVA). Time (T1, pre-stress and T2, post-stress) was entered as within-subject factor and Group (TE and NTE) as between-subject factor.

2.6.4. Salivary alpha amylase (sAA)

A repeated-measures analysis of variance (ANOVA) was performed on sAA levels with Time (T1, pre-stress and T2, post-stress) as within-subject factor and Group (TE and NTE) as between-subject factor.

2.6.5. ERP Components

First, repeated-measures analysis of variance (ANOVA) were performed on N1, P1 and P3 ERP components with Time (T1, pre-stress and T2, post-stress) Congruence (Congruent and Incongruent) as within-subject factor and Group (TE and NTE) as between-subject factor. Only one interaction effect was highlighted (time*condition*group), which was analysed accordingly to prior hypothesis through (1) paired *t*-tests for a within group interaction between groups and (2) one-way analysis of variance (ANOVA) for a between group interaction.

3. Results

3.1. Demographics

Data are presented in Table 1. Groups differ on every measure except from sex, symptoms of intrusion (PCL-B), negative mood and cognitions (PCL-C) and anxiety (DASS-A).

3.2. Behavioral data

RT and RBC measures can be found in Table 2.

3.3. Reaction Time

A significant main effect of Time was found ($F(1,33) = 21.64$, $p < 0.001$, $\eta^2_p = 0.396$) with significantly faster RT at T2 (M = 287.10, SD = 5.96) than at T1 (M = 302.69, SD = 6.42). Neither congruency ($F(1,33) = 0.851$, $p = 0.353$, $\eta^2_p = 0.025$) or Group ($F(1,33) = 3.163$, $p = 0.085$, $\eta^2_p = 0.087$) had significant an effect.

Table 1
Demographic data's of TE and NTE groups.

	TE	NTE	Statistic	*p* value
Sex	7F, 11M	10F, 7M	$\chi^2 = 1.391$	0.238
Age	27.44 (5.94)	23.11(6.28)	$F = 4.383$	**0.044***
LEC	1.78 (1.06)	0 (0)	$F = 47.713$	**0.000****
PCL-B	2.89 (4.29)	1.29 (1.68)	$F = 2.042$	0.162
PCL-C	1.56 (1.58)	0.76 (0.9)	$F = 3.251$	0.081
PCL-D	4.72 (3.86)	1.94 (2.10)	$F = 6.875$	**0.013***
PCL-E	3.78 (2.16)	2.11 (2.088)	$F = 5.341$	**0.027***
PCL-5 total	12 (741)	5.82 (5.37)	$F = 7.882$	**0.008***
DASS-D	3.22 (2.89)	0.71 (0.77)	$F = 12.127$	**0.001****
DASS-A	2.28 (2.84)	1.35 (1.49)	$F = 1.422$	0.242
DASS-S	4.72 (3.10)	2.59 (2.89)	$F = 4.413$	**0.043***
DASS-total	9.44 (7.22)	4.47 (5.09)	$F = 5.488$	**0.025***

Note. LEC = Life-Event Checklist, PCL-B = symptoms of intrusions on the *Post-Traumatic Stress Disorder Checklist for DSM-5* (PCL-5), PCL-C = symptoms of avoidance on the PCL-5, PCL-E = symptoms of negative alterations in cognitions and mood in the PCL-5, PCL-E = symptoms of Alterations in arousal and reactivity on the PCL-5.

* *p*-Values < 0.01.

** *p*-Values < 0.05.

Table 2
Behavioral components from the dot-probe task.

	T1		T2	
	TE M (SD)	NTE M (SD)	TE M (SD)	NTE M (SD)
RT for congruent trials	312.09 (36.54)	291.06 (41.86)	292.30 (35.45)	277.04 (33.35)
RT for incongruent trials	316.93 (39.44)	290.59 (35.76)	298.33 (40.11)	276.74 (38.71)
Orientation index	−1,18 (15)	7.27 (11.19)	10.02 (25.04)	15.04 (16.7)
Disengagement index	−3.93 (16.44)	8.97 (18.07)	6.35 (26.00)	15.76 (14.91)

T1 = Pre-stress induction (baseline), T2 = Post-stress induction, TE = Trauma-exposed ($N = 18$), NTE = Not Trauma Exposed ($N = 17$).

3.4. Response-Based Computation (RBC) indexes

On RBC orientation bias, only a main effect of Time was highlighted ($F(1,33) = 10.399$, $p = 0.003$, $\eta^2_p = 0.240$), both groups increased their vigilance toward threat after the acute stress task, indicating that the arousal has been successfully increased with the cold pressor task. On RCB disengagement bias, both Time ($F(1,33) = 4.482$, $p = 0.042$, $\eta^2_p = 0.120$) and Group ($F(1,33) = 4.632$, $p = 0.039$, $\eta^2_p = 0.123$) had a significant effect. The main effect of Time is to be understood as an increased difficulty to disengage from threat after the acute stress task (both mean score increasing from T1 to T2). Furthermore, the NTE group presented slower disengagement at both T1 and T2.

3.5. Noradrenaline (sAA)

Repeated measures ANOVA revealed a significant main effect of Time ($F(1,33) = 4.160$, $p = 0.036$, $\eta^2_p = 0.119$), with an increase in sAA at T2 (M = 111.96, SD = 13.76) compared with T1 (M = 96.93, SD = 11.43). No group difference were observed, at baseline or after the stress induction.

3.6. Event-Related Potentials (ERPs)

The original grand average waveforms for each condition and groups are presented in Fig. 1.

3.7. P1

Grand average ERPs averaged across CPZ and CZ recording site are shown in Fig. 2. Neither Group ($F(1,32) = 0.386$, $p = 0.539$, $\eta^2_p = 0.013$), Time ($F(1,32) = 0.326$, $p = 0.572$, $\eta^2_p = 0.011$) or Condition ($F(1,32) = 0.003$, $p = 0.958$, $\eta^2_p < 0.000$) had a significant main effect. However, repeated measures ANOVA revealed an interaction effect of Time*Condition*Group ($F(1,30) = 5.977$, $p = 0.021$, $\eta^2_p = 0.116$).[1] Line-plots were computed in order to have a representation of this interaction effect. As depicted in Fig. 2 and according with our research questions, we decided to investigate the potential effect of group for threat-related P1 amplitude at T2. Considering that we hypothesized that the TE group might react stronger to stress than the NTE group, we first conducted a paired *t*-test to compare the increase of P1 amplitude from T1 to T2 only in congruent trials. As expected, the TE group showed higher P1 amplitude for those trials at T2 ($M = 3.07$, $SD = 2.16$) in comparison with T1 ($M = 4.67$, $SD = 2.16$; $t(16) = -3.11$; $p = 0.004$). This result was not found for incongruent trials (T1: $M = 3.33$, $SD = 2.58$; T2: $M = 3.65$, $SD = 1.93$; $t(16) = -0.419$; $p = 0.680$), or within the NTE group (in all comparisons; $p > 0.144$). Secondly, accordingly with

[1] The effect remained when age was considered as a covariable: ($F(1,30) = 4.709$, $p = 0.038$).

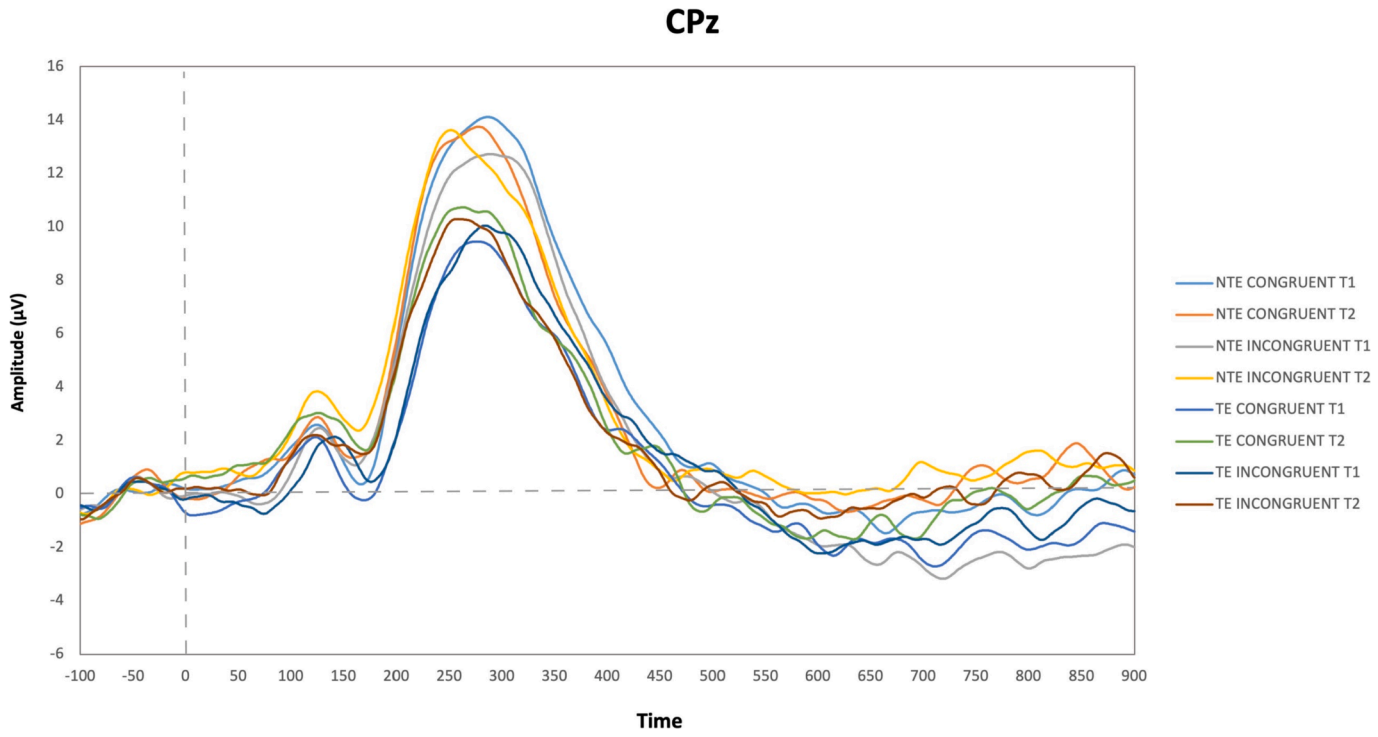

Fig. 1. Original grand average waveform for each condition and group at the CPz recording site. T1 = Pre-stress induction (baseline), T2 = Post-stress induction.

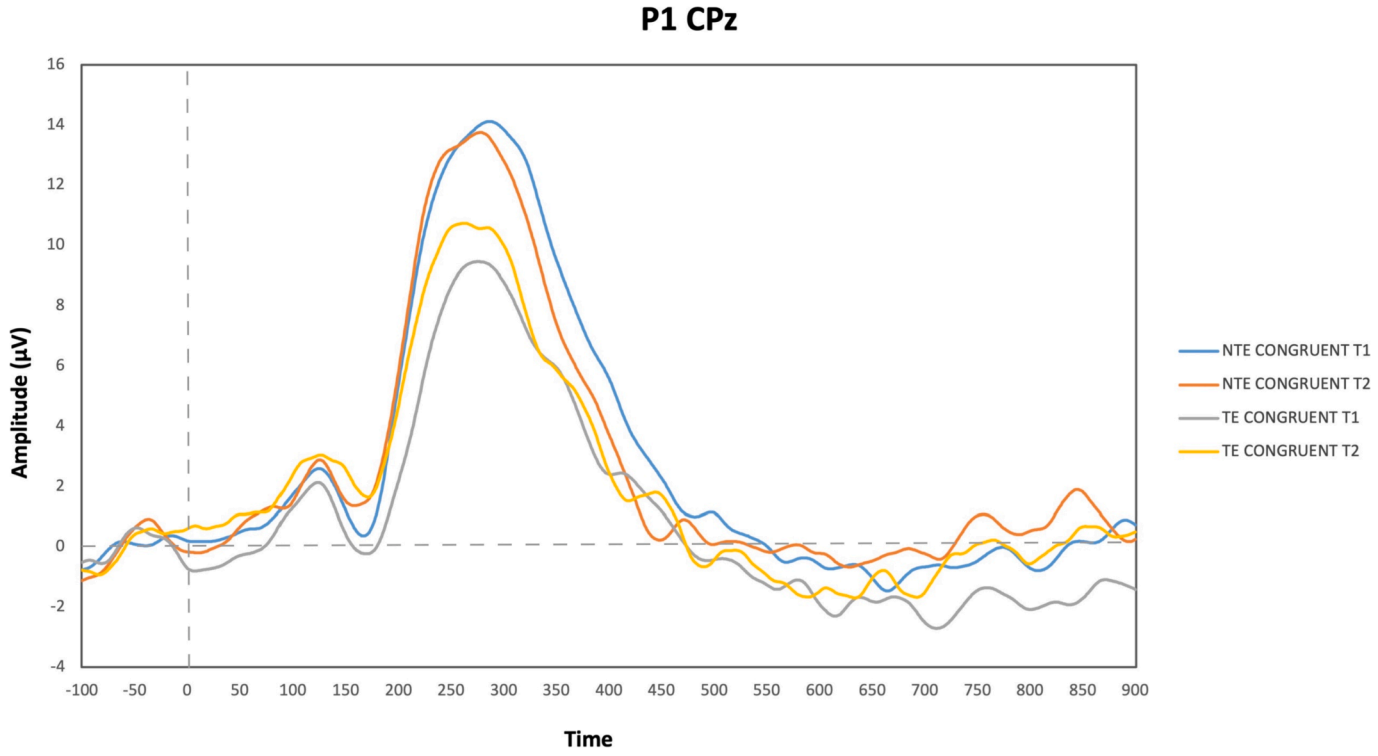

Fig. 2. Grand average ERPs following the presentation of congruent pictures averaged across CPZ and CZ recording site. T1 = Pre-stress induction (baseline), T2 = Post-stress induction.

the effect depicted in Fig. 2 and our hypothesis of a differential threat processing between groups, we performed a single ANOVA on the amplitude of P1 for congruent trials after the stress induction. We found that the TE group displayed significantly higher P1 amplitude (M = 4.88, SD = 2.03) for the congruent trials at T2 compared with the NTE group (M = 2.61, SD = 2.78; $F(1,31) = 7.100$; $p = 0.012$).

3.8. N1

Grand average ERPs at N1 recording site are shown in Fig. 3. Repeated measures ANOVA revealed a significant main effect of Time ($F(1,33) = 7.871$, $p = 0.003$, $\eta^2_p = 0.193$), with decreased amplitudes at T2 (M = -1.577, SD = 0.49) as compared to T1 (M = -2.42, SD = 0.49)

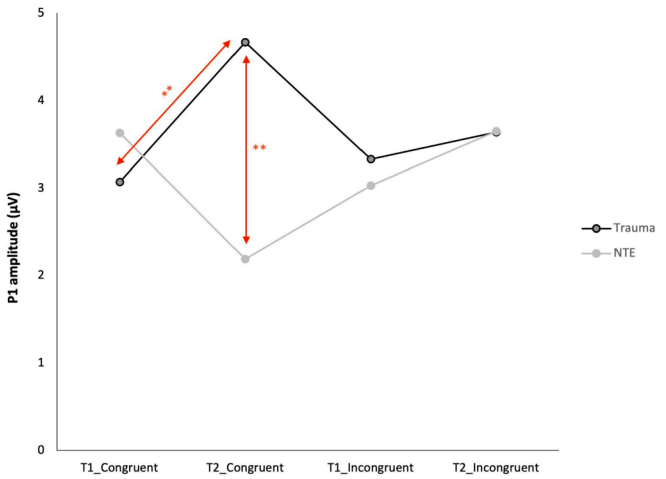

Fig. 3. Line-plot representation of the Interaction effect (Time*-Condition*Group) highlighted on the P1 Amplitude evoked by the presentation of congruent and incongruent pictures. Mean P1 amplitude. T1 = Pre-stress induction (baseline), T2 = Post-stress induction. Post-test were performed (1) within the Trauma group on mean P1 amplitude evoked by the presentation of congruent trials (between T1 and T2), and (2) between groups on mean P1 amplitude evoked by the presentation of congruent trials at T2.

for every condition, in both groups. This effect is shown on Fig. 4. Neither Group ($F(1,33) = 0.005$, $p = 0.945$, $\eta^2_p < 0.000$) or Condition ($F(1,32) = 3.040$, $p = 0.091$, $\eta^2_p = 0.007$) had a significant main effect.

3.9. P3

Repeated measures ANOVA revealed no significant effect regarding the P3, neither Group ($F(1,32) = 2.371$, $p = 0.133$, $\eta^2_p = 0.069$), Time ($F(1,32) = 0.354$, $p = 0.556$, $\eta^2_p = 0.026$) or Condition ($F(1,32) = 3.56$, $p = 0.068$, $\eta^2_p = 0.1$) had a significant main effect.

4. Discussion

The aim of this study was to investigate how a previous exposure to a traumatic event affects attentional processing of threat before and after an acute stress task. Specifically, we assessed attentional biases to threat before and after a cold pressor task in individuals who have been exposed to trauma (TE) compared to individuals without trauma exposure (NTE). In line with our hypothesis, we were able to highlight both behavioral and neural evidences of differential threat processing at baseline as well as after being stressed.

The first main finding of this study is an increase in P1 amplitude for threatening stimuli only for the trauma exposed individuals after the stress induction. As P1 reflects allocation of attention to stimuli, our results suggest that stressed TE individuals present an enhanced attentional orienting toward threat. This might thus indicate a stress-induced hypervigilance to threat, which is mirrored by the reaction time data. Indeed, we observed a switch toward facilitated engagement in the TE group as indexed by shortened reaction times at locations cued by a negative picture. This increase in P1 aplitude has been previously observed in anxiety-related disorders (Bar-Haim et al., 2005). Considering that (a) an hypervigilance pattern similar to previous finding of anxiety-related disorders is found within the TE group after the stress induction and (b) the increased P1 to threat-related cues is larger for TE group compared to healthy controls individuals, we can suggest that TE individuals who are under stress show an attentional pattern closer to anxious individuals than healthy controls.

To further explore the impact of stress on attentional allocation, we noted a smaller N1 amplitude in response to threatening stimuli after an acute stress in both groups. This is contrary to our hypothesis, based on previous studies that have found larger N1 amplitude during a stressful task (Qi et al., 2016). As the N1 component in known to be sensitive to the level of vigilance and that an acute stress elicits a state of heightened vigilance and arousal, a larger N1 amplitude under stress was not surprising. However, in the study of Qi et al. (2016), the task itself constituted a stress induction. They performed a modified version of a mental arithmetic task in which the stressful situation was induced by a time pressure and a social-evaluative threat (evoked by a judgement on the task performance). We can argue that their task induced a

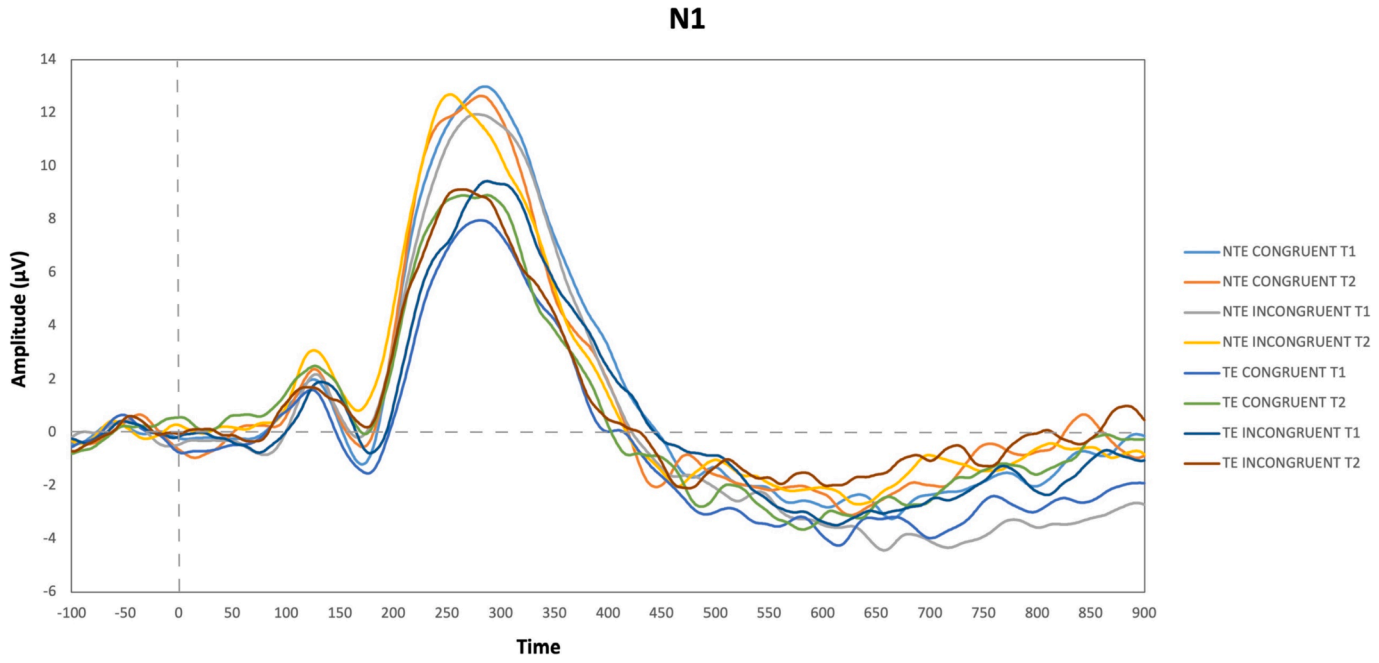

Fig. 4. Grand-average ERPs evoked by the presentation of congruent and incongruent pictures at the N1 recording site. T1 = Pre-stress induction (baseline), T2 = Post-stress induction.

performance anxiety rather than a stress response to threat. Furthermore, as components of the task (such as answers provided) were socially evaluated, it seems normal that higher attentional resources were allocated to such stimuli. Our study employed a cold-water immersion task that has been used in several studies to test sympathetic nervous reactivity (Santa Ana et al., 2006). This classic physical stress test was independent from the main task (the emotional dot-probe) and required participants to calm themselves in order to correctly perform the task, instead of being gradually stressed by the task as it has been the case in Qi et al. (2016) study. This protocol aimed to be more ecological, given that when a threat has been encountered (i.e.: a robbery), individuals needs to perform actions that are not in direct relation with the stressor (i.e.: calling emergency services). As the N1 component has also been linked with early discrimination processes of stimuli (Vogel and Luck, 2000), the smaller N1 amplitude found for every stimulus type in this study suggests a harder discrimination between threat and not threat under stress.

Taken together, the combination of both larger P1 and the smaller N1 presented by the previously trauma-exposed individuals after stress might be linked to increased amygdala activation in this population. The amygdala is a central perceptual node where information from olfactory, visual, auditory, and tactile modalities converges (Jacobs et al., 2012). The amygdala exerts effects on fear-related behaviours, including on spatial attention and eye movements. Previous studies have highlighted that stress shifted the amygdala toward higher sensitivity as well as lower specificity (van Marle et al., 2009). That is, a stress induction augmented amygdala response for both threat-related and positively valenced stimuli, thereby diminishing a threat-selective response pattern. The cold pressor task might therefore have significantly increased the amygdala activity, resulting in a combination of smaller N1 amplitude and larger P1 amplitude. Those two phenomena respectively representing a harder discrimination of threat versus not-threat information and a higher sensitive toward threat once it has been identified. This could mean that after a stress induction, while both controls and previously traumatized individuals seems to have difficulty to discriminate threat and neutral information (as indexed by a smaller N1 amplitude in both groups), only previously traumatized individuals present a significative increased sensitivity toward threat. Trauma survivors might therefore be more sensitive to acute stress than healthy controls, and present an increased amygdala activity under stress.

The second main result of this study is the presence of initial disengagement difficulties in previously traumatized individuals in comparison with healthy controls. Before undergoing an acute stress induction, trauma-exposed participants showed longer reaction times after incongruent trials, indicating disengagement difficulties (Aupperle et al., 2012). Their attentional resources seemed to have been captured by the threatening information while the probe appeared on the opposite site of the screen (Mogg et al., 2008). This pattern is comparable to the one found in several pathological populations, disengagement difficulties having been found in anxiety-related disorders, depression, and individuals suffering from PTSD (Cisler and Koster, 2010; Evans et al., 2016; Yaroslavsky et al., 2019). Experiencing difficulties in moving one's attentional focus from threatening information toward goal-relevant information could therefore represent a cognitive vulnerability that might lead to clinical pathologies. Based on previous work, such impairments are known to specifically contribute to the clinical profile of PTSD and lead to the use of alternative coping styles such as avoidance.

Our results allow to clarify the processes underlying threat apprehension at baseline as well as following a stress induction of previously traumatized individuals. First, we found that TE participants presented difficulties moving their attentional focus from threatening information toward goal-relevant information once they process negative information (as indexed by RT data's). This vulnerability when dealing with threat which could contribute to the development of PTSD after another traumatic event (Aupperle et al., 2012). Second, trauma exposed individuals partially responded to stress in the way predicted by Vine et al. (2016) conceptual framework of stress. These authors suggested that stress led to heightened distractibility with a tendency to focus on irrelevant information, which is corroborated by the N1 amplitude increased positivity found in our study after the stress induction (in both TE and HC, as it is supposed to represent a normal reaction to stress). However, TE participants also presented a stress-induced hypervigilance toward threatening information, which was not predicted by Vine et al. (2016), but rather falls in line with the attentional control theory of Eysenck et al. (2007). Hence, the attentional patterns presented by trauma exposed individuals does not completely fit either one or the other theory, suggesting that this population constitutes a vulnerable class between the normal and pathological pole. This gives evidence to the idea that a previous trauma constitutes a risk factor to negative mental health outcomes.

Our findings should be considered cautiously regarding a number of limitations. The first main limitation is the lack of consideration of trauma types in the TE group. As we did not have access to the detailed history of each participant, we are not aware of the kind of trauma, listed in the Trauma Events Questionnaire, that mainly raised in our sample. However, this limitation needs to be considered in a context of almost null PTSD symptoms. Indeed, our research question was centered on the impact of a past traumatic event, as defined by the criteria A of the DSM-5, without current symptomatology on threat apprehension. The impact of the trauma type has specifically been highlighted regarding the levels of PTSD symptomatology it originally induces (see Kessler et al., 2017 present in our manuscript), which is irrelevant in our study considering the level PTSD symptoms reported. Instead, we were only interested in the life-threatening aspect of the trauma, which has been carefully considered through the TEQ. However, it would be informative for future research conducted on larger sample size to assess if, in the absence of PTSD symptomatology, threat apprehension varies between trauma-type. Secondly, the current study used one stimulus duration of 1000 ms in an attempt to replicate and extend Carr and colleagues' paradigm (Carr et al., 2016), which may promote avoidance or disengagement difficulties. Future research should include several stimulus durations windows, for example, 100 ms, 500 ms, and 1250 ms presentations, similar to Koster et al. (2005). The current study may also have been limited by the omission of control group who did not complete the cold pressor task which would have allowed the separation of stressor effects from practice effects that may have affected the post-stressor block B. Such a group would complete a warm water control, where the process is identical to the cold pressor task but the water is maintained at 37 degrees-Celsius instead (Deuter et al., 2012). Research by McHugh et al. (2010) highlighted that the use of a control group who do not complete a stress induction would give a better understanding of the effect of stress compared to the effect of learning. Finally, the findings of this study need to be considered as preliminary considering the small sample size and therefore, replications on larger samples are warranted. It is to be noted that, although psychopathology was assessed thought widely validated questionnaires, we were not able to compute Cronbach's alpha for the current sample considering that we had access to pre-encoded total score of such questionnaires.

5. Conclusion

In summary, this study provides empirical support of the risk factor that constitutes a previous exposition to trauma. Under baseline conditions, when confronted with a threat, trauma-exposed individuals present a slow disengagement pattern which could represent a vulnerability when dealing with threats. This is similar to the subclinical low trait anxious individuals pattern, which supports the idea that a previous exposition to a traumatic experience place to individual between healthy and pathological poles of stress regulation. Once those individuals are stressed, their ability to discriminate threat among distractors decreases (as expected) and they allocate more attentional resources toward threatening information once recognized, which is in

line with evidence of enhanced attentional biases toward threat in anxiety related disorders. In conclusion, our results are in line with the growing literature highlighting that trauma, even without clinical post-traumatic symptoms can induce long lasting neural changes. This study helps understanding how a previous experience of trauma negatively affects threat processing.

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