Received: 4 July 2019 | Revised: 17 February 2020 | Accepted: 18 February 2020

DOI: 10.1111/psyp.13567

ORIGINAL ARTICLE

PSYCHOPHYSIOLOGY SPR WILEY

The neurophysiological correlates of the triarchic model of psychopathy: An approach to the basic mechanisms of threat conditioning and inhibitory control

Tiago O. Paiva[1,2] | **Pedro R. Almeida**[3] | **Rui C. Coelho**[1] | **Rita Pasion**[1] | **Fernando Barbosa**[1] | **Fernando Ferreira-Santos**[1] | **António J. Bastos-Leite**[2] | **João Marques-Teixeira**[1]

[1]Laboratory of Neuropsychophysiology, Faculty of Psychology and Education Sciences, University of Porto, Porto, Portugal

[2]Faculty of Medicine, Department of Medical Imaging, University of Porto, Porto, Portugal

[3]Faculty of Law, School of Criminology, Interdisciplinary Research Center on Crime, Justice and Security, University of Porto, Porto, Portugal

Correspondence
Tiago O. Paiva, Laboratory of Neuropsychophysiology, Faculty of Psychology and Education Sciences, University of Porto, Rua Alfredo Allen, 4200-135 Porto, Portugal.
Email: tiagopaiva@fpce.up.pt

Funding information
The project was funded by the Portuguese Foundation for Science and Technology under the Horizon 2020 Program (PTDC/PSI-GER/28076/2017). Tiago O. Paiva was supported by a doctoral grant (SFRH/BD/93831/2013) of the Portuguese Foundation for Science and Technology under the POCH/FSE Program. Rita Pasion was supported by a doctoral grant (SFRH/BD/117130/2016) of the Portuguese Foundation for Science and Technology under the POCH/FSE Program

Abstract

The psychopathic traits boldness, meanness, and disinhibition are theorized to be underlined by trait fearlessness and externalizing vulnerability as etiologic neurobiological processes. However, little is known about the neurophysiological correlates of these traits. In this work, we explored how the three traits are associated with event-related potential (ERP) components targeted at the etiological processes in a partial delayed threat conditioning task and in a go/no-go task. Fifty community-dwelling volunteers (25 women), without history of neurological or psychiatric conditions, were recruited and assessed for psychopathic traits using the triarchic psychopathy measure. Participants performed a threat conditioning task, and a go/no-go task while undergoing an electroencephalography recording. Results from the threat conditioning task showed that boldness was significantly associated with reduced late positive potential. Concerning the go/no-go task, disinhibition was significantly associated with reduced error-related negativity ERP component. Overall, distinct psychopathic traits were found to be associated with distinct neurophysiological correlates of threat conditioning and response inhibition. This is consistent with models of psychopathy entailing trait fearlessness and externalizing proneness, and related brain mechanisms, as distinct processes underlying the expression of psychopathic traits.

KEYWORDS

electroencephalography, event-related potential, inhibitory control, psychopathy, threat conditioning, triarchic model of psychopathy

1 | INTRODUCTION

Psychopathy comprises a constellation of personality features, including affective (e.g., lack of empathy or guilt, and lack of deep emotional attachment to others) and interpersonal characteristics (e.g., superficial charm, social potency), as well as impulsive and antisocial behaviors (Hare & Neumann, 2008; Patrick & Drislane, 2015). Advances in research on psychopathy-related constructs have been marked by bewilderment regarding whether or not psychopathy represents a

single nosological entity, and by an attempt to establish the core features of the psychopathic spectrum (Lilienfeld, Watts, Smith, Berg, & Latzman, 2015). In contrast to the classical view of psychopathy as a unitary syndrome (Cleckley, 1941; McCord & McCord, 1964), contemporary views suggest that psychopathy is multifaceted, reflecting the expression of distinct dimensions in the spectrum (Hare, 2003; Patrick & Drislane, 2015; Skeem, Polaschek, Patrick, & Lilienfeld, 2011). The literature also shows that psychopathy scores are normally distributed (Edens, Marcus, Lilienfeld, & Poythress, 2006), and that there are dissociable effects for distinct psychopathic traits both at the behavioral and the brain functioning levels (for a review, see Patrick & Bernat, 2009). As a consequence, psychopathy may be best conceptualized as a distinctive configuration of extreme scores of personality traits reflecting affective, cognitive, and neurobiological processes placed in continuum with normal functioning (Guay, Ruscio, Knight, & Hare, 2007; Vachon et al., 2013). Therefore, research on the etiological mechanisms underlying psychopathic traits, both at the neurobiological and behavioral levels, is paramount to understand their expression (Edens et al., 2006).

In line with dimensional approaches, the triarchic model of psychopathy provides an operationalization of psychopathy traits through personality scales, such as the triarchic psychopathy measure (TriPM; Patrick, 2010). The TriPM assesses three distinct phenotypic constituents of psychopathy: boldness, meanness, and disinhibition.

Boldness refers to the capacity to remain calm in situations involving threat or the ability to swiftly recovering from stressful events, high self-assurance, social efficacy, and increased tolerance for unfamiliarity and danger (Patrick, Fowles, & Krueger, 2009). It is suggested that boldness is the main phenotypical expression of dispositional fearlessness, associated with reduced sensitivity of the so-called defensive system of the brain to cues signaling threat and punishment (Fowles & Dindo, 2009).

Meanness is associated with callousness and coldheartedness. It encompasses poor empathy, disdain for close attachments with others, rebelliousness, excitement-seeking, exploitativeness, and empowerment through cruelty (Patrick et al., 2009). Meanness is highly associated with insecure attachment during development, and shares variance with antisocial and externalizing constituents of psychopathy, being a relevant dimension for criminal behavior (Drislane & Patrick, 2017; Kyranides, Fanti, Sikki, & Patrick, 2017; Patrick et al., 2009).

Disinhibition describes a general phenotypic propensity for poor impulse control and includes lack of planfulness and foresight, impaired regulation of affect and urges, prevalence of immediate gratification, and deficient behavioral restraint. It is assumed that disinhibition is the phenotypical expression of externalizing vulnerability, which is related to frontal brain deficits in inhibitory control (Fowles, 2018; Patrick & Drislane, 2015; Patrick et al., 2009).

The triarchic model of psychopathy reflects the integration of historically relevant concepts conveyed in the literature (Patrick et al., 2009). Boldness, meanness, and disinhibition were described as emerging from two distinct etiological pathways: (a) dispositional fearlessness, theorized to reflect impaired activity of the so-called defensive motivational system of the brain, which includes the amygdalae and affiliated structures and (b) externalizing vulnerability, representing weak inhibitory control as a result of altered activity in the anterior structures of the brain, including the prefrontal cortex and the anterior cingulate cortex (ACC; Patrick & Drislane, 2015; Patrick, Drislane, & Strickland, 2012; Patrick et al., 2009).

The triarchic model of psychopathy frames boldness as an adaptive phenotypic manifestation of a latent predisposition toward fearlessness (Lilienfeld et al., 2016). Its etiological roots derive from an emotional reactivity deficit, as suggested by the low fear hypothesis[1] (Lykken, 1957). According to this hypothesis, psychopathic individuals do not exhibit typical anticipatory responses when facing situations involving punishments that are contingent to a specific behavior (Fowles & Dindo, 2009). Psychophysiological studies reported reduced electrodermal response in psychopaths secondary to the presentation of conditioned stimuli signaling a potential threat,[2] such as an electric shock (Birbaumer et al., 2005), as well as reduced punishment avoidance in avoidance learning tasks (e.g., Lykken, 1957). The latter is associated with deficits in stimulus reinforcement learning, which has been pointed as a basic mechanism implied in the emotional reactivity deficit observed in psychopathy (Blair, 2007; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). Interestingly, several brain regions implicated in threat conditioning, including the central and basolateral nuclei of the amygdala, the insula, the ventromedial prefrontal cortex, the sensory association cortex, the posterior thalamus, and the motor cortex, have been suggested to represent core structures implied in psychopathy (Blair, 2006, 2007; Blair, Meffert, Hwang, & White, 2018). A voxel-based morphometry study showed that boldness was associated with volumetric abnormalities of the right insula, amygdala, striatum, and the lateral

[1]It is worth mentioning that since the integrated emotions systems model was proposed by Blair (2005, 2006), it is widely accepted that emotional deficits observed in psychopathic individuals are associated with automatic mechanisms involved in the detection of potential threats, and not with the conscious experience of fear (for a meta-analysis see Hoppenbrouwers, Bulten, & Brazil, 2016).

[2]Terms as aversive/fear conditioning blur the distinction between unconscious processes involved in threat detection and the conscious expression of fear itself (LeDoux, 2014). As such, the expression "threat conditioning" is used.

orbitofrontal cortex (Vieira et al., 2015). Empirical studies have also demonstrated that psychopathic individuals show deficient acquisition of threat conditioned responses, suggesting impairments in the affective–evaluative mechanisms underlying emotional associations between cues and threatening stimuli (Birbaumer et al., 2005; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Patrick, Bradley, & Lang, 1993). In addition, recent work demonstrated that the fearless dominance dimension of psychopathy (i.e., boldness) is specifically associated with decreased activation of the autonomic nervous system in processing conditioned responses (López, Poy, Patrick, & Moltó, 2013).

Disinhibition, as conceptualized by the triarchic model of psychopathy, reflects general externalizing vulnerability (Patrick et al., 2009), a biobehavioral liability factor for psychopathy-related phenomena, such as antisocial personality and substance abuse (Krueger, 2002; Krueger & Piasecki, 2002). Research has shown a strong relationship between this broad externalizing factor and measures of antisocial behavior included in different psychopathy instruments (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Blonigen et al., 2010; Patrick, Hicks, Krueger, & Lang, 2005). It is thought that externalizing vulnerability is associated with disrupted frontal brain networks relevant for inhibitory control (Fowles, 2018; Patrick & Drislane, 2015). Empirical studies have suggested that certain psychopathic traits, such as impulsive and antisocial dispositional vulnerability reflected by disinhibition, are associated with reduced amplitude of the event-related potential (ERP), error-related negativity (ERN) component (Heritage & Benning, 2013; Pasion, Cruz, & Barbosa, 2016). Thus, externalizing vulnerability may be related to a lack of endogenous feedback arising from response errors (Pasion et al., 2016). Deficits in response inhibition have also been linked to reduced amplitudes of the P3 component in the oddball paradigm (Patrick et al., 2006), and to altered activity of the frontoparietal network (Rodman et al., 2016), whose dysfunction is known to be related to broad impairments of attentional processes in psychopathy (Juárez, Kiehl, & Calhoun, 2013).

Meanness reflects a more complex phenotype within the triarchic model of psychopathy. It was initially described as reflecting a maladaptive manifestation of the fearlessness dimension (Patrick et al., 2009). However, its close relation to antisocial behavior led to the assumption that meanness reflects a distinguishable component of the general externalizing factor, entailing low empathy and stimulation-seeking tendencies, behaviorally associated with patterns of predatory aggression (Patrick et al., 2009, 2012). At the operational level, this is expressed by moderate correlations of meanness with both boldness and disinhibition (e.g., Paiva et al., 2020; Pasion et al., 2016), highlighting the multifaceted associations of this phenotype with both fearlessness and externalizing vulnerability.

The study of the external correlates of psychopathy is relevant to understand its underlying mechanisms and their etiological substrates. As far as we know, there are no studies comprehensively addressing both basic etiological pathways of psychopathy by analyzing the neurophysiological correlates of threat conditioning and response inhibition. In the current study, we aimed to examine the effects of distinct psychopathic traits—conceptualized by the triarchic model of psychopathy—on the behavioral and ERP correlates of threat conditioning and inhibitory control in a community sample.

Regarding threat conditioning, two ERP components have been considered correlates of the acquisition of a conditioned response: the late positive potential (LPP; e.g., Bacigalupo & Luck, 2018) and the contingent negative variation (CNV; e.g., Flor et al., 2002). The LPP indexes emotional salience and perception, and its amplitude increases with the emotional salience of stimuli. Thus, it shows higher amplitudes for cues signaling the presentation of a noxious stimulus (Bacigalupo & Luck, 2018). The CNV reflects an anticipatory response to noxious events and is thought to reflect activity of the defensive motivational system in inhibiting the potentially aversive effects of such events (Flor et al., 2002). The CNV can be decomposed into two distinct components: the early or initial CNV (iCNV) and the late CNV (lCNV). The iCNV represents anticipatory activity primarily elicited by cues signaling potential threat, whereas the lCNV is associated with the anticipation of the noxious stimuli in classical threatening conditioning protocols (Flor et al., 2002).

Both the LPP and CNV components have been studied in relation to psychopathy. Overall, studies have reported reduced LPP amplitudes to salient stimuli in individuals with high scores in the interpersonal facets of psychopathy (Baskin-Sommers, Curtin, & Newman, 2013; Carolan, Jaspers-Fayer, Asmaro, Douglas, & Liotti, 2014), and reduced CNV in the anticipation of threats in individuals with high psychopathy scores (Flor et al., 2002). Although there is evidence of ERP alterations as a function of psychopathy, there are no studies reporting the effects of psychopathy on the behavioral indexes of associative learning. One hypothesis consistently raised in the literature is that a reduced reactivity to emotional salience of stimuli in psychopathy (secondary to emotional reactivity deficit) increases the performance in certain cognitive tasks (e.g., Costello et al., 2019). Nonetheless, this hypothesis was not previously tested in the context of threat conditioning.

Three ERP components have been consistently pointed as correlates of the go/no-go task: the no-go N2, the no-go P3, and the ERN. The no-go N2 shows higher amplitudes when a planned response is successfully inhibited (e.g., Folstein & Van Petten, 2008). It has been suggested that the N2 reflects the frontal brain activity of response conflict monitoring and response inhibition (Donkers & Van Boxtel, 2004). Overall, the P3 is modulated by attentional processes allocated to task relevant or infrequent stimuli,

such as the no-go stimulus of the go/no-go task. Both N2 and P3 amplitudes have been consistently related to externalizing traits, including disinhibitory tendencies as assessed by the TriPM (Nelson, Patrick, & Bernat, 2011; Pasion, Fernandes, Pereira, & Barbosa, 2018; Pasion et al., 2019; Patrick et al., 2006). The ERN is associated with the activity of the ACC (Holroyd & Coles, 2002; Weinberg, Riesel, & Hajcak, 2012) and represents early ERP activity time-locked to the occurrence of incorrect responses. The activity elicited in the same time window by correct responses is commonly referred to as correct-related negativity (CRN). As mentioned, previous studies have shown that externalizing traits are associated with reduced ERN amplitudes (Hall, Bernat, & Patrick, 2007; Nelson et al., 2011; Pasion et al., 2016).

In this study, we aim at disentangling the putative etiological mechanisms of psychopathic traits by analyzing their relation with the ERP correlates of threat conditioning and response inhibition. We hypothesized that: (a) boldness, but not disinhibition, was associated with a reduced amplitude modulation of LPP and CNV responses to aversive cues; (b) disinhibition, but not boldness, was associated with a reduced amplitude modulation of N2 and P3 responses to the no-go signal and with reduced ERN response to the occurrence of errors. We did not postulate a specific hypothesis for meanness, since this phenotypical expression of psychopathy is thought to be shared both with externalizing vulnerability and trait fearlessness (Patrick et al., 2009).

2 | METHOD

2.1 | Participants

Fifty community-dwelling volunteers (25 women) without self-reported history of neurological or psychiatric conditions were recruited. All participants reported to have normal color perception and normal or corrected-to-normal visual acuity. The age of participants ranged from 18 to 46 years ($M = 26.6$; $SD = 6.12$).

For the analysis of threat conditioning and LPP, we excluded data from three participants ($n = 47$), due to a low number of ERP trials (less than 50% of the total number of trials).

For the analysis of CNV, data from seven participants was excluded ($n = 43$), due to excessive noise in the ERP.

For the analysis of the go/no-go paradigm, we excluded data from one participant, due to technical problems during the electroencephalography (EEG) recording. Eight participants were further excluded ($n = 41$), due to lack of sufficient error trials to analyze ERN after denoising (number of good trials < 6; Hajcak, Moser, Yeung, & Simons, 2005).

For the analysis of the N2 and P3 components, data from six participants were excluded ($n = 43$), due to excessive noise in the EEG.

Means and standard deviations for the total number of trials per task and condition are presented in Table S1 (Supporting Information).

2.2 | Experimental setup and self-reported measures

The experiment included two tasks, counterbalanced between participants: the threat conditioning task, and the go/no-go task. During both experimental tasks, participants were seated in a chair, looking at a screen monitor positioned approximately one meter away. All stimuli were presented on a 17-inch Dell P170S monitor.

2.2.1 | Threat conditioning task

The threat conditioning task was adapted from Bacigalupo and Luck (2018) and its structure is shown in Figure 1. It consists of three distinct phases: the habituation phase, the threat conditioning phase, and the extinction phase. Each phase included the passive visualization of a sequence of trials, in which circles (radius of 1.3° of visual angle) were presented in the center of the monitor. The duration of each stimulus was 4 s with a random intertrial interval between 3 and 5 s.

During the habituation phase, the stimulus was a gray (15 cd/cm^2 luminance) circle presented in each trial (eight trials in total). Participants were told to relax and pay attention to the stimuli presented. The habituation phase was intended to prevent novelty-related EEG responses during the threat conditioning phase (Bacigalupo & Luck, 2018).

In the threat conditioning phase, each trial consisted in the presentation of a blue, a green or a yellow circle (15 cd/cm^2). Each color was pseudorandomly selected to represent the conditioned stimulus (CS+) for each participant. The randomization constraint was that each color had to be chosen three times every nine participants. The CS+ was paired with the unconditioned stimulus (US) in 50% of the CS+ presentations. The US consisted of a noxious white noise burst with a duration of 1.5 s presented through Sennheiser HD206 headphones. The maximum intensity was defined to be 100 dB SPL. The modality, duration, and intensity of the US were similar to those used in previous studies (Bacigalupo & Luck, 2018). The remaining color-coded stimuli (CS−) were never paired with the US. CS+ and CS− were presented for 4 s. The US started 2.5 s after the onset of CS+ (in 50% of the CS+ presentations) and co-terminated with the CS+. The threat conditioning phase was divided in three blocks, each containing 32 trials: 16 CS−, 8 CS+ paired with US (CS+ US+),

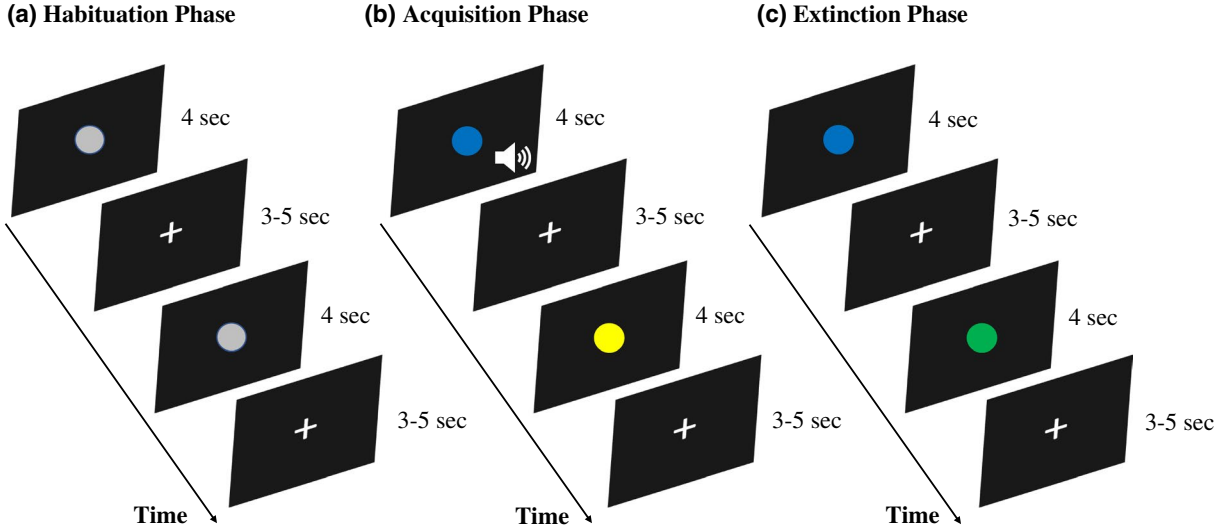

FIGURE 1 Threat conditioning protocol. The protocol consisted of three distinct phases: (a) the habituation phase; (b) the acquisition phase; and (c) the extinction phase. For each participant, one color was chosen as the conditioned stimulus (CS+) to be paired with the US in the acquisition phase, whereas the other two colors (CS−) were never paired with the US. The US was presented on 50% of CS+ trials on the last 1.5 s of the CS+ presentation. During the extinction phase the US was never presented. sec, time in seconds

and 8 CS+ unpaired with the US (CS+ US−). Each trial was presented in pseudorandom order with the constraint that the first three CS+ presentations were always paired with the unconditioned stimulus (i.e., the first three presentations were CS+ US+), and that no more than two successive trials with the same color were presented. Between each block, participants were asked to assess the probability of a loud noise occurring in association with each color using a 9-point Likert scale (1 = not likely at all; 9 = extremely likely). Participants were instructed to relax and pay attention to the stimuli. They were also warned that, at some time points, a loud noise would be generated, but that no response during this sort of stimulation would be required.

The extinction phase consisted in the presentation of the blue, green, and yellow circles in random order, without the presentation of the white noise. Each color was randomly presented 10 times (30 trials in total). Participants were asked to relax and pay attention to the stimuli.

2.2.2 | Go/no-go task

A scheme of the task is shown in Figure 2. During this task, two letters (B and P) were randomly presented in four blocks. In each block, participants were told that they should respond to the target letter, either B or P (go trial), and not to respond to the other letter (no-go trial). Each block contained 125 trials (30 of which were no-go). In each trial, the letter was presented for 100 ms and participants had to respond in the subsequent 450 ms. The intertrial interval was 1,000 ms. The target letter switched at each block (i.e., if participants were responding to the letter P in the first block, they were instructed to respond to the letter B in the second, P in the third, and B again in the fourth). The target letter of the first block was counterbalanced between participants. Participants used a response pad to respond to the target letter and were instructed to do it as fast as possible, whenever the target letter was presented, using the thumb of the dominant hand. No feedback on the response accuracy was given. This experimental procedure was adapted from Maruo, Sommer, and Masaki (2017) and the four experimental blocks were preceded by 10 trials of training.

2.2.3 | Triarchic psychopathy measure

The TriPM (Patrick, 2010) is a 58-item self-report measure designed to assess the three distinct components described in the triarchic model of psychopathy (Patrick et al., 2009). Items are scored using a 4-point Likert-type scale according to the following answers: false, somewhat false, somewhat true, and true. Although it is possible to compute a total psychopathy score, the TriPM is primarily intended for the study of psychopathic dimensions: boldness, as assessed by the TriPM, is scored on a 19 items-based questionnaire of fear and fearlessness addressing optimism, resilience to stress, social dominance, persuasiveness, tolerance for uncertainty, self-confidence, social assurance, and intrepidness; the meanness dimension comprises a 19 items-based index of empathy, relational aggression, destructive aggression, physical aggression, honesty, and excitement-seeking; and disinhibition is scored on the basis of 20 items primarily addressing the general externalizing factor (i.e., irresponsibility, problematic impulsivity, theft, alienation, boredom proneness, impatient urgency, fraudulence, dependability, and planful control). In the present study, the European

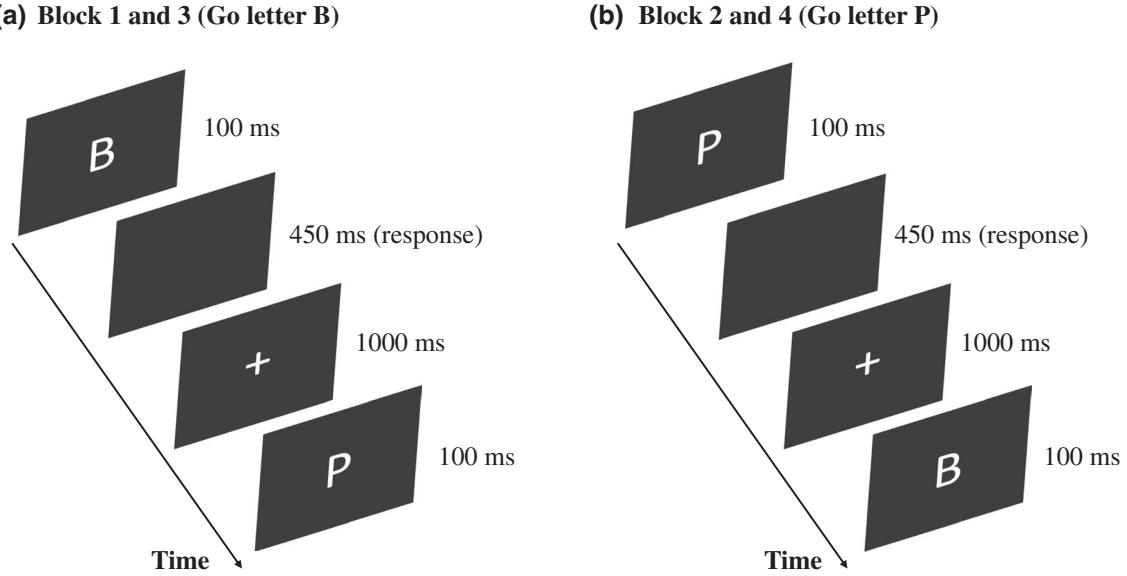

FIGURE 2 Go/no-Go protocol. For each participant, one letter was assigned as the Go letter (e.g., letter B), while the other (e.g., letter P) was assigned as the no-Go letter in (a) blocks 1 and 3. In (b) blocks 2 and 4, the rule switched, and participants had to response to the other letter. Participants were instructed to respond by pressing a button whenever the Go letter appeared on the screen. The first letter to respond was counterbalanced between subjects. ms: time in milliseconds

Portuguese version of the TriPM was used. For a detailed description of the translation and adaptation processes, please see Vieira, Almeida, Ferreira-Santos, Moreira, et al. (2014).

2.3 | Procedure

Upon arrival at the Laboratory facilities, participants were told that the main goal of the study was to explore the relation between individual characteristics and patterns of behavior and brain activity. After signing the written informed consent, participants filled a sociodemographic inquiry, a general health questionnaire, and were asked to fill the TriPM. Participants then performed the threat conditioning and the go/no-go tasks (the order was counterbalanced) while recording the EEG activity. The tasks, measures and procedures where approved by the local ethics committee, and all participants were treated in accordance with the 2013 revision of the declaration of Helsinki. Participants did not receive any sort of compensation to take part in this study.

2.4 | EEG recording and analysis

The EEG was recorded using a NetAmps 300 system from Electrical Geodesics Inc. (Eugene, Oregon, USA) with a 128-channel hydrocel geodesic sensor net from the same company. The signal was digitized at 500 Hz with a vertex reference (Cz). The electrode impedances were kept below 50 KΩ (high-impedance amplifier). During acquisition, an antialiasing filter was automatically applied by the acquisition software (Net Station v4.5). The filter was a Butterworth low pass, designed to have a frequency response as flat as mathematically possible in the passband, rolling off toward zero in the stopband at 250Hz (the Nyquist frequency of the selected sampling rate). Data processing and analysis was conducted using EEGLAB toolbox v13.6.5b (Delorme & Makeig, 2004) and the ERPLAB plugin v6.1.3 (Lopez-Calderon & Luck, 2014), which are open-source MATLAB packages for EEG/ERP analysis.

EEG signals were resampled to 250 Hz and bandpass filtered, using a causal bandpass filter with 0.1 Hz as the low half-amplitude cutoff (transition band width of 0.1 Hz and roll-off at 12 dB/octave) and 30 Hz as the high-amplitude cutoff (transition band width of 7.5 Hz and roll-off at 12 dB/octave). After filtering, we selected recorded signals that included task-related activity and removed channels with excessive noise, never exceeding a number of channels >10% of the total number of electrodes. Descriptive statistics on the number of electrodes, locations of interest, and removed channels are displayed in the Supporting Information section. The EEG was then submitted to a temporal independent component analysis (ICA; Makeig, Bell, Jung, & Sejnowski, 1996). Independent components (ICs) identified as corresponding to eye blinks, saccades, or heart rate were removed, in order to correct for this type of artifacts. The artifact correction procedure was the following: (a) visual inspection of the ICs and preselection of those resembling to correspond to eye blinks, saccades, and heart rate activity; (b) visual inspection of the time course of the preselected ICs; (c) comparison of the original signal with the back-projected signal without the selected

components; (d) if the changes in the corrected signal were circumscribed to the latency of the specific artifacts without changing the EEG morphology at other latencies, the components were removed; if not, no correction was performed at this stage (i.e., remaining artifacts were later removed during visual inspection). The descriptive statistics on the number and type of ICs removed are presented in the Supporting Information section (Table S2). After artifact correction, deleted channels were interpolated by means of spherical spline interpolation, which weights each electrode spatially in a way that is qualitatively consistent with dipole fields (Ferree, 2006). The signal was then re-referenced to the average of all electrodes. Finally, the EEG signal was segmented into epochs around the onset of the stimulus of interest. For the threat conditioning task, two segmentations were performed: (a) 1,000 ms epochs with 200 ms before (baseline) and 800 ms after the onset of CS (CS+ and CS−); (b) 3,200 ms epochs with 200 ms before and 3,000 ms after the onset of CS (CS+ and CS−), in order to extract the initial and late portions of the CNV component. For the go/no-go task, epochs of 1,000 ms with 200 ms before and 800 ms after the onset of each letter and the occurrence of a motor response were created. All segments were subjected to visual inspection, and epochs containing artifacts not corrected using ICA were deleted. The epoch deletion decision was based on visual inspection (i.e., based on EEG morphology) and performed by consensus of two experts. The remaining epochs were baseline corrected and averaged into the conditions of interest.

After visual inspection and on the basis of reported latencies for the components of interest (e.g., Bacigalupo & Luck, 2018; Flor et al., 2002; Folstein & Van Petten, 2008; Pasion et al., 2016; Patrick et al., 2006), we computed the ERP measures. As we used a high-density EEG setup, channels of interest were defined as the regional average of locations defined by the extended international system 10–5: Fz as the average of electrodes E4, E10, E11, E16, E18, and E19; FCz as the average of electrodes E5, E6, E7, E12, E13, E106, and E112; Cz as the average of electrodes E7, E31, E55, E80, E106, and E129; and Pz as the average of electrodes E61, E62, E67, E72, E77, and E78. We conducted a 200-ms baseline correction (before stimulus/motor response onset) for all components.

For the threat conditioning task, the following measures were extracted: (a) LPP calculated as the mean amplitude in the 350–650-ms post-CS onset time window at the Pz-averaged electrode (Bacigalupo & Luck, 2018; Liu, Huang, McGinnis-Deweese, Keil, & Ding, 2012); (b) iCNV calculated as the mean amplitude in the 500–1,000-ms post-CS onset time window at the Fz-averaged electrode (Flor et al., 2002); (c) lCNV calculated as the mean amplitude in the 2,000–2,500-ms post-CS onset time window at the Fz-averaged electrode (Flor et al., 2002). These measures were extracted for each CS type during the acquisition and extinction phases.

Regarding the go/no-go task, we extracted the following ERP measures: (a) ERN and CRN, defined as the mean amplitude in the 50–150-ms time window after the occurrence of a motor response (error or correct response, respectively) at the FCz electrode within the interval defined by Pailing and Segalowitz (2004); (b) N2 calculated as the mean amplitude in the 250–350-ms poststimulus presentation time window at the Cz electrode (Nieuwenhuis, Yeung, & Cohen, 2004); (c) P3 calculated as the mean amplitude in the 300–500-ms poststimulus presentation time window at the Pz electrode (Patrick et al., 2006). All component measures were calculated for the total task. The duration of each measurement window rested under the time constraints defined in the literature and was further confirmed by visual inspection based on the expected ERP morphology.

Finally, the internal consistency of the behavioral (i.e., probability ratings in the threat conditioning task and errors in the go/go-no task) and ERP measures (i.e., LPP, CNV ERN, CRN, N2, and P3) was examined using a split-half approach. In the case of the ERP measures, the correlation between averages of odd and even numbered trials was corrected with the Spearman–Brown prophecy formula (Nunnally, Bernstein, & Berge, 1967). Results indicated satisfactory to excellent internal consistency for the probability ratings (CS+: r = .708; CS−: r = .822), the number of errors (r = .651), the LPP (CS+: r = .764; CS−: r = .710), the CNV (r = .842; CS−: r = .698), the ERN (r = .754), the CRN (r = .976), the N2 (Go: r = .961.; no-Go: r = .954), and for the P3 (Go: r = .972; no-Go: r = .874).

2.5 | Statistical analysis

Statistical analyses were performed using IBM SPSS 24 (IBM Corporation, Armonk, NY, USA). For the TriPM subscales, we computed the Cronbach's alpha value of internal consistency. The Pearson's correlation coefficient was used to test correlations between scores. The effects of the experimental manipulation for each task on the behavioral measures and brain correlates were tested using repeated measures analyses of variance (ANOVA). For the effects of the threat conditioning task on each outcome variable, we implemented a model with CS type as the within-subjects factor (CS+ and CS−). Regarding the go/no-go task, we also implemented a model with one within-subjects factor both for the stimulus (go and no-go) and the response-related ERPs (correct response- and error-related). Data from participants were excluded when at least one outlier was identified from standardized residuals (>3.0 or <−3.0) of the ANOVA model. Finally, in order to test predictive models of psychopathic traits in behavioral and brain-related terms, we performed linear regression analyses

with TriPM scores (boldness, meanness, and disinhibition) as predictors, and behavioral measures and ERP correlates as dependent variables. The alpha threshold for statistical significance was .05. Given the small sample size of this study, we computed a permutation-based analysis of the regression models (1,000 samples with random seed) beyond the standard hypothesis testing. Confidence intervals (CI) at 95% for the standardized beta (β) coefficients were calculated, along with the permutated p values for each predictor. The permutation analysis provides evidence for the systematic nature of the significant bivariate covariation patterns. It is worth mentioning that correlations between self-report measures and ERP or behavioral measures (as those reported here) are expected to be in the .1–.3 range (e.g., Hall et al., 2007) and a sample size of 84 would be necessary to detect a correlation of .3 ($\beta = .80$). Thus, our study is relatively underpowered to detect effects of this magnitude.

3 | RESULTS

3.1 | Triarchic psychopathy measure

The means and standard deviations of the TriPM total, boldness, meanness, and disinhibition scores, and the analyzed ERP measures are displayed in Tables 1 and 2. No sex-related differences were found for TriPM scores.

As expected, boldness was positively correlated with meanness, $r_{(48)} = .448$, $p = .001$, and meanness was positively correlated with disinhibition, $r_{(48)} = .523$, $p < .001$. Boldness and disinhibition were not correlated, $r_{(48)} = -.042$, $p = .771$.

3.2 | Threat conditioning task

3.2.1 | Behavioral measures

Participants reported the white noise as significantly arousing ($M = 6.11$, $SD = 1.91$), $t_{(46)} = 5.76$, $p < .001$, and unpleasant ($M = 3.36$, $SD = 1.93$), $t_{(46)} = -4.05$, $p < .001$. As expected, participants reported that the loud noise was more likely to be presented after the CS+ relative to CS− in all blocks of the acquisition phase ($t = 19.1$, $p < .001$ for block 1, $t = 17.7$, $p < .001$ for block 2, and $t = 20.5$, $p < .001$ for block 3).

TABLE 1 Descriptive statistics for TriPM total score, boldness, meanness, and disinhibition

	Mean	*SD*	Min–Max	Cronbach's α
Total score	54.4	17.2	26–96	.88
Boldness	29.3	7.62	14–48	.80
Meanness	10.1	7.62	1–33	.87
Disinhibition	15.1	8.29	4–38	.82

Linear regression models with boldness, meanness, and disinhibition as predictors revealed no significant associations with ratings of arousal and valence for the white noise. Boldness was the only significant predictor of the probability rating regarding the noise being presented along with CS+ ($\beta = -.311$, $p = .028$). This suggests that boldness was associated with a more accurate estimation of the white noise being presented after the CS+. Actually, estimated probabilities lower than 50% of the CS+ US+ contingencies only occurred in three participants. The permutation-based analysis revealed the following 95% CI for $\beta = [-.600; -.016]$, $p = .045$.

3.2.2 | ERP components

The ERPs at Fz and Pz for each CS type, along with difference waves, are displayed in Figure 3.

Repeated measures ANOVA with CS type as the within-subjects factor for the LPP mean amplitude at Pz revealed a main effect of CS type, $F_{(1,46)} = 10.01$, $p = .003$, $\eta_p^2 = .179$, with higher LPP mean amplitudes for the CS+ ($M = 2.61$, $SD = 2.16$) relative to the CS− condition ($M = 1.90$, $SD = 1.55$). Regarding the extinction phase, the paired samples Student's t test did not reveal significant differences in the LPP at Pz between CS+ and CS−, $t_{(43)} = .95$, $p = .347$.

For the mean amplitude of the iCNV at Fz, the repeated measures ANOVA revealed a main effect of CS type, $F_{(1,42)} = 16.43$, $p < .001$, $\eta_p^2 = .281$, with higher iCNV amplitudes for CS+ ($M = -1.86$, $SD = 1.60$) relative to the CS- condition ($M = -0.99$, $SD = 1.32$). No significant main effect of CS type was found for lCNV at Fz, $F_{(1,42)} = 0.47$, $p = .468$, $\eta_p^2 = .011$. In the extinction phase, the paired samples Student's t test did not show significant differences between CS+ and CS− in both the iCNV, $t_{(42)} = -0.38$, $p = .707$, and lCNV components, $t_{(42)} = -0.79$, $p = .436$.

Linear regression models were tested for the difference values in the amplitude of distinct components (LPP, iCNV, and lCNV) for the total threat conditioning phase according to CS type, using boldness, meanness, and disinhibition as predictors. The statistics of the models and the standardized coefficients for each predictor are displayed in Table 3. Boldness was significantly associated with the LPP amplitude difference for the total threat conditioning phase, with high scores on boldness being associated with lower LPP amplitude differences between CS+ and CS−. No other significant associations were found. The described pattern of results was similar in the permutation-based analysis: 95% CI for $\beta = [-.653; -.020]$, $p = .035$. Significant behavioral and ERP associations are shown in Figure 4. The p values and

TABLE 2 Means and standard deviations (*SD*) for the ERP measures

Threat conditioning	Mean	*SD*	Go/no-go task	Mean	*SD*
LPP at Pz			ERN/CRN at FCz		
CS+	2.61	2.16	Error	−2.37	2.31
CS−	1.90	1.55	Correct	1.85	1.93
iCNV at Fz			N2 at Cz		
CS+	−1.86	1.60	No-Go	−1.07	2.40
CS−	−0.99	1.31	Go	1.11	1.85
lCNV at Fz			P3 at Pz		
CS+	−0.45	2.90	No-Go	2.43	1.87
CS−	−1.00	2.44	Go	2.19	1.72

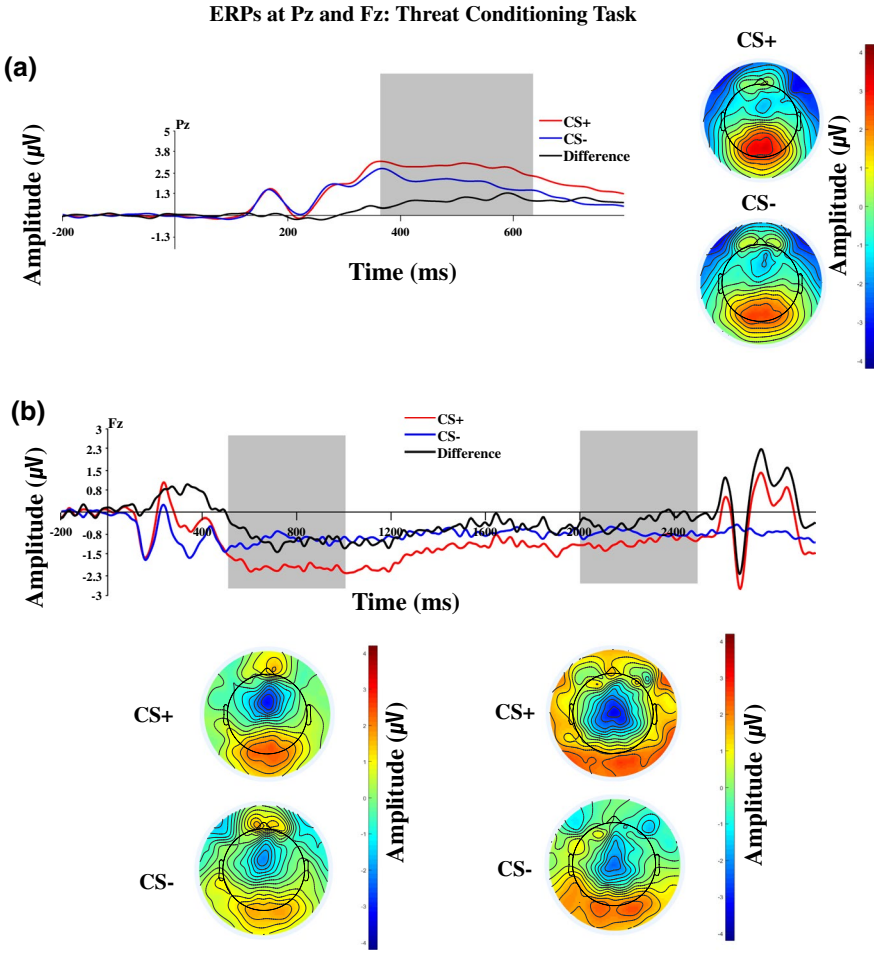

FIGURE 3 ERPs at (a) Pz and (b) Fz for the Threat Conditioning protocol. The time windows in gray shadow represent the (a) LPP and (b) iCNV and lCNV measurement time windows, post CS presentation. Topographic maps represent the mean amplitude for the LPP, iCNV, and lCNV time windows. CS+, Conditioned Stimulus paired with the Unconditioned Stimulus; CS−, Conditioned Stimulus unpaired with the Unconditioned Stimulus; ms, time in milliseconds; μV, amplitude in microvolts

95% CI for β for all permutation analysis are in Supporting Information (Table S3).

3.3 | Go/no-go task

3.3.1 | Behavioral measures

The linear regression model including boldness, meanness, and disinhibition as predictors of number of errors was not a significant, $F_{(3,38)} = 2.37$, $p = .086$. The described pattern of results was similar in the permutation-based analysis (see Supporting Information, Table S3).

3.3.2 | ERP components

ERPs at FCz for each outcome (correct response or error), and for each stimulus type (go or no-go) at Cz and Pz, along with difference waves, are displayed in Figure 5.

TABLE 3 Linear regression models for LPP, iCNV, and lCNV difference scores at the total acquisition phase with boldness, meanness, and disinhibition as predictors

	Model			Boldness		Meanness		Disinhibition	
	F	*p* value	$_{Adj}R^2$	*β*	*p*	*β*	*p*	*β*	*p*
LPP difference value	1.86	.150	.053	**−.350***	**.049**	.055	.785	−.141	.435
iCNV difference value	0.27	.844	−.052	.126	.503	.026	.903	−.012	.952
lCNV difference value	0.47	.703	−.037	.143	.447	.048	.822	−.083	.668

*Significant at $p < .05$.

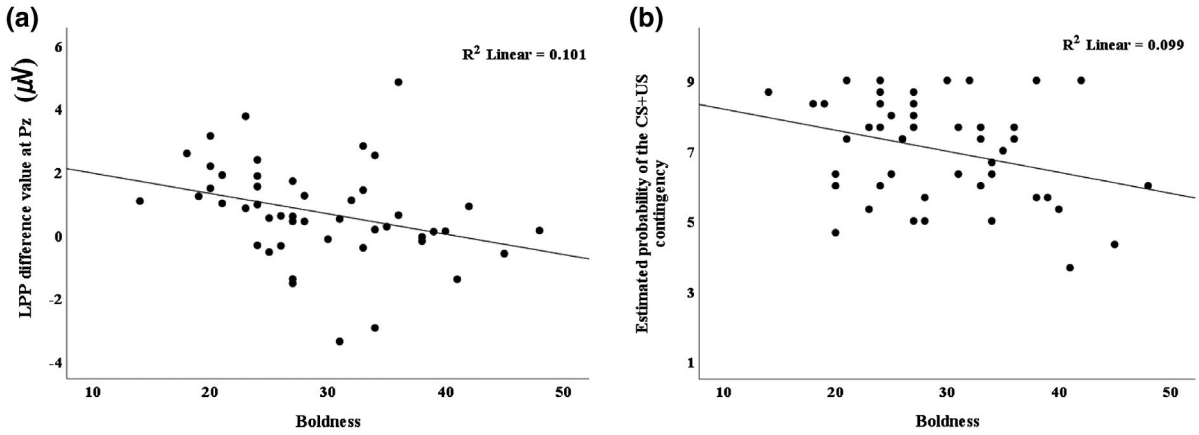

FIGURE 4 Scatter plots for the significant predictors of the ERP and behavioral outcomes of the Threat Conditioning Task: (a) boldness as predictor of LPP difference value at Pz; (b) boldness as predictor of the estimated probability of the CS+-US contingencies. μV, amplitude in microvolts

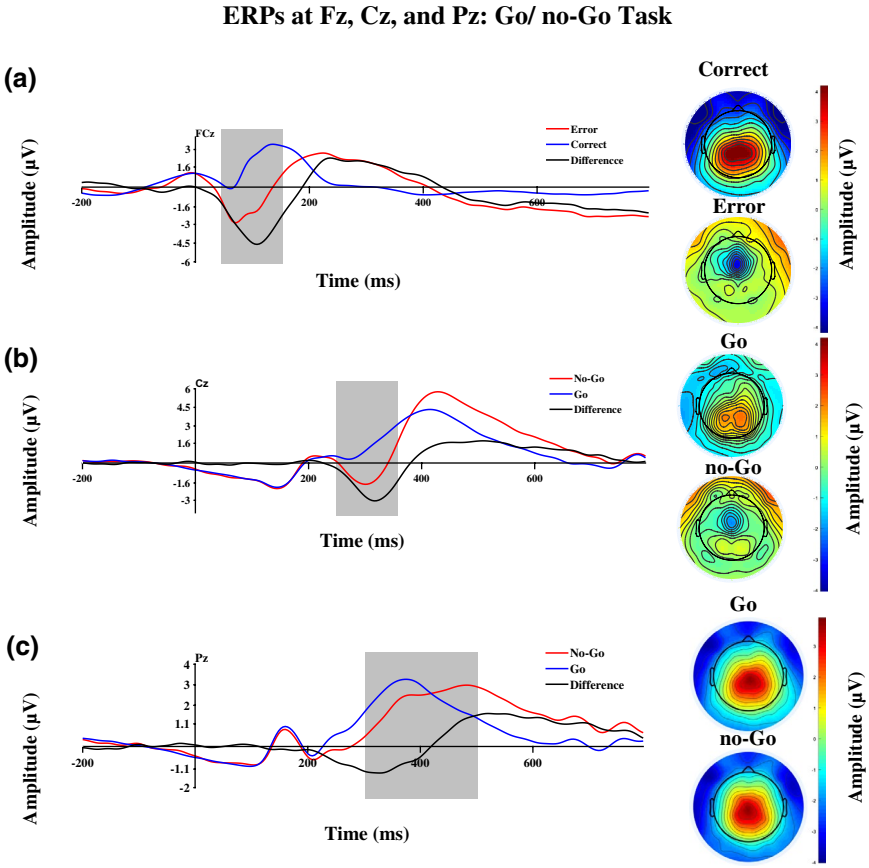

FIGURE 5 ERPs at (a) FCz, (b) Cz, and (c) Pz for the Go/no-Go task. Topographic maps represent the mean amplitude for the (a) ERN/CRN, (b) N2, and (c) P3 time windows. The time windows in gray shadow represent the (a) ERN/CRN, (b) N2, and (c) P3 measurement time windows. ms: time in milliseconds; μV: amplitude in microvolts

The repeated measures ANOVA revealed a significant association of the response type with the mean amplitude of the ERN at FCz, $F_{(1,40)} = 213.6$, $p < .001$, $\eta_p^2 = .842$. In other words, there was increased negative deflection for errors ($M = -2.36$, $SD = 2.42$) in comparison with correct responses ($M = 1.85$, $SD = 1.93$). There was also a significant effect of stimuli type (go or no-go) on the N2 mean amplitude at Cz, $F_{(1,42)} = 75.17$, $p < .001$, $\eta_p^2 = .642$. The N2 mean amplitudes were more negative for no-go ($M = -1.07$, $SD = 2.40$) relative to the go trials ($M = 1.11$, $SD = 1.85$). Finally, no significant effect was found for the event type on P3 mean amplitude at Pz, $F_{(1,42)} = 1.41$, $p = .241$, $\eta_p^2 = .033$.

Linear regression models with boldness, meanness, and disinhibition as predictors were tested for: (a) the ERN to CRN amplitude difference (ERN—CRN) at FCz; (b) the no-go to go difference value (no-go—go) of the N2 mean amplitude at Cz; and (c) the no-go—go of the P3 mean amplitude at Pz. The statistics of the models and the standardized coefficients for each predictor are displayed in Table 4. Disinhibition was significantly associated with the ERN—CRN at FCz (i.e., a reduced amplitude difference was associated with increased disinhibition). The described pattern of results was similar in the permutation-based analysis. The latter revealed a 95% CI for $\beta = [.057; .757]$, $p = .032$. Please note that a more negative difference value is associated with a more negative/less positive mean amplitude for the ERN in comparison with the CRN.

Regarding N2, neither boldness, meanness, nor disinhibition predicted the difference values in the N2 mean amplitude. The same pattern of results was confirmed by the permutation-based analysis (see Supporting Information, Table S3).

Meanness was a significant predictor of the P3 mean amplitude difference value at Pz. Increased meanness scores were associated with increased P3 mean amplitude difference values at Pz. This pattern was confirmed by the permutation-based analysis, which revealed the following 95% CI for $\beta = [.097; .857]$, $p = .026$. The significant associations are shown in Figure 6. The p values and 95% CI for β for all permutation analysis are in Supporting Information (Table S3).

4 | DISCUSSION

Since the introduction of the triarchic model of psychopathy (Patrick et al., 2009), several studies addressed the distinct phenotypical dimensions assessed by the TriPM (e.g., López et al., 2013; Pasion et al., 2016; Sellbom & Phillips, 2013; Vieira, Almeida, Ferreira-Santos, Barbosa, et al., 2014; Vieira et al., 2015). The present study aimed at characterizing

TABLE 4 Linear regression models for ERN/CRN, N2, and P3 difference scores at the total task with boldness, meanness, and disinhibition as predictors

	Model			Boldness		Meanness		Disinhibition	
	F	*p* value	$_{Adj}R^2$	*β*	*p*	β	*p*	*β*	*p*
ERN/CRN	1.78	.169	.055	−.020	.921	−.003	.987	**.356***	**.037**
N2 (No-Go—Go)	1.13	.350	.009	.233	.210	−.291	.144	.230	.175
P3 (No-Go—Go)	2.31	.091	.085	−.274	.127	**.435***	**.025**	−.311	.058$^+$

*Significant at $p < .05$.

$^+$Marginally significant at $p < .06$.

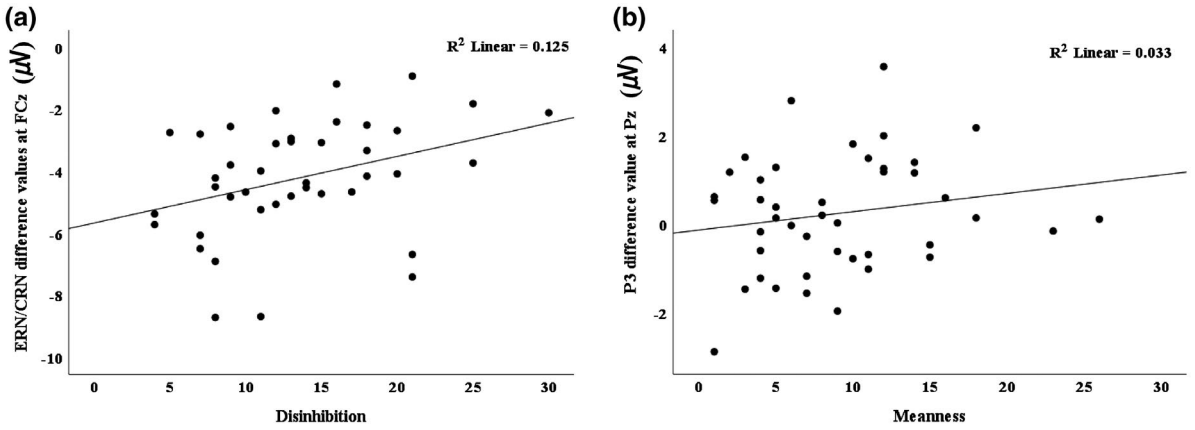

FIGURE 6 Scatter plots for the significant predictors of the ERP outcomes of the go/no-go task: (a) disinhibition as predictor of the ERN/CRN difference value at FCz; (b) meanness as predictor of the P3 difference value at Pz. μV, amplitude in microvolts

the distinct dimensions of the triarchic model of psychopathy by directly assessing the associations of boldness, meanness, and disinhibition with the behavioral and ERP correlates of trait fearlessness and externalizing vulnerability, using two well-established experimental tasks: the threat conditioning task and the go/no-no task.

Our results show that boldness, meanness, and disinhibition are associated with distinct behavioral and brain correlates of threat conditioning and response inhibition, in keeping with dual-process models of psychopathy (Patrick et al., 2009). In the following paragraphs, we separately discuss the results associated with each dimension. It is worth to point out at this point that the phenotypic expressions included in the triarchic model of psychopathy correspond to the integration of historically relevant concepts, which were partly operationalized by distinct psychopathy models (Patrick & Drislane, 2015; Patrick et al., 2009). Therefore, boldness, meanness, and disinhibition—as assessed by the TriPM—show moderate to high correlations with related dimensions assessed by other psychopathy inventories (for a review, see Patrick & Drislane, 2015). This is relevant for the interpretation of the reported results.

4.1 | Boldness

We expected that boldness would be associated with reduced LPP and CNV responses to threat (versus nonthreat) cues, in the threat conditioning protocol. Providing support for trait fearlessness as the underlying mechanism, boldness was found to be significantly associated with a reduced LPP difference value. However, no association was found between boldness and the iCNV or the lCNV. Behaviorally, boldness was related with a more accurate estimation of CS+ US+ contingencies.

As a stable index of emotional stimuli processing, the LPP shows increased amplitude for emotionally salient stimuli, when compared to neutral stimuli. Schupp, Flaisch, Stockburger, and Junghöfer (2006) suggested that the LPP indicates how well the stimuli are maintained in the working memory. In addition, the fact that emotionally charged stimuli induce increased LPP reflects how such stimuli are intrinsically relevant. In threat conditioning protocols, LPP amplitude increases for CS+ relative to CS− (Bacigalupo & Luck, 2018). Furthermore, in a combined EEG-functional magnetic resonance imaging (fMRI) study, the LPP response associated with aversive stimuli processing was found to be coupled with the amygdala complex, insula, and the adjacent temporal and ventrolateral prefrontal brain function (Liu et al., 2012). Interestingly, these regions have also been implicated in threat conditioning processes, and were described as being dysfunctional in psychopathy (Blair, 2007; LeDoux, 2012; Vieira et al., 2015).

Previous studies on the relation between psychopathy and LPP modulation by emotional content revealed an overall effect of psychopathy. Specifically, individuals with high psychopathy scores were found to have reduced LPP modulation secondary to emotionally charged pictures (Carolan et al., 2014; Medina, Kirilko, & Grose-Fifer, 2016). A study by Cheng, Hung, and Decety (2012) also reported reduced LPP differentiation in youth offenders irrespective of their scores in the youth version of the psychopathy checklist (Forth, Kosson, & Hare, 2003). Studies differentiating the dimensions of psychopathy have shown that the affective and interpersonal features of psychopathy (Factor 1 of the psychopathy checklist revised [PCL-R]; Hare, 2003) were associated with reduced LPP to negative pictures (Venables, Hall, Yancey, & Patrick, 2015), and that primary psychopathy—as assessed by the Levenson self-report psychopathy scale (Levenson, Kiehl, & Fitzpatrick, 1995)—was associated with reduced LPP to stimuli showing others in pain, when participants were requested to assess their level of empathic concern (Decety, Lewis, & Cowell, 2015).

Although consistent with the idea of reduced sensitivity to emotional stimuli as a proxy for the affective deficits in psychopathy, these studies did not consider amygdala-mediated associative learning processes, which are believed to be the etiological mechanisms underlying trait fearlessness. The only previous study assessing LPP response to threat conditioning did not report any associations of total psychopathy with LPP amplitudes (Rothemund et al., 2012). Furthermore, no previous study addressed the LPP association with distinct psychopathic traits. In the current study, we analyzed the association of distinct psychopathic phenotypes and ERP correlates of threat conditioning. Therefore, by showing that boldness was the only psychopathic trait to be significantly associated with a reduced LPP amplitude, our study provides added value to understanding how specific psychopathic traits are related with threat conditioning.

The link between trait fearlessness and threat conditioning has also been established using both skin conductance responses, startle reflex potentiation, and the CNV component (Birbaumer et al., 2005; Flor et al., 2002; Patrick et al., 1993; Rothemund et al., 2012). Flor and colleagues (2002) reported the absence of CS+/CS− differentiation in the CNV of psychopaths, suggesting a deficit in association formation possibly related to deficient interaction of subcortical and cortical structures. The CNV component is typically used to measure the anticipation of emotionally salient events. It has been suggested that fearlessness dispositions are associated with reduced anticipation of such type of events; for example, a noxious sound (Flor et al., 2002). Taking together, our results suggest that boldness, reflecting the etiological predisposition toward fearlessness, is significantly associated with reduced LPP amplitude difference values, but not with CNV amplitude difference values. This leads to the interpretation

that boldness expresses a blunted emotional response to a cue signaling a potential threat, but is not associated with the anticipation of the threat itself.

Our results also showed that boldness was associated with a more accurate assessment of CS+ US+ contingencies. This indicates that a reduced response to the emotional salience of a cue might be linked to increased cognitive resources allocated to the task, which is in line with results of previous studies showing that LPP is attenuated by voluntary top-down control (Hajcak, Macnamara, & Olvet, 2010). Furthermore, it supports the hypothesis that boldness embodies positive adjustment characteristics of the psychopathic personality (Patrick & Drislane, 2015). In this context, it is possible that the reduced emotional sensitivity to cues signaling threat is related with a less biased estimation of the CS+ US+ contingencies. More work will be necessary in future studies to confirm this hypothesis.

4.2 | Disinhibition

Given the deficits in inhibitory control expected to occur in the externalizing vulnerability etiological pathway, we hypothesized that disinhibition would be associated with a reduced amplitude of the ERN, N2, and P3 components secondary to the go/no-go task. Disinhibition was found to be significantly associated with a reduced amplitude difference of the ERN subtracted to the CRN at FCz, but no association was found between disinhibition and N2 or P3 amplitudes. Furthermore, disinhibition was not found to be associated with the total number of errors in the go/no-go task.

The ERN is considered to be an index of performance monitoring by measuring the mismatch between the intended (i.e., correct) response and the effective (e.g., error) response (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). As predicted, disinhibition was the only psychopathic trait associated with ERN amplitude differences. This is in line with previous findings relating disinhibition to reduced error detection (Bresin, Finy, Sprague, & Verona, 2014; Hall et al., 2007; Pasion et al., 2016), and is also consistent with the disposition toward disruptive and antisocial behavior in psychopathy. Nonetheless, several previous studies on the relation between psychopathy and ERN modulation did not report any significant associations (Brazil et al., 2009; Brazil et al., 2011; Maurer, Steele, Cope, et al., 2016; Maurer et al., 2018; Maurer, Steele, Edwards, et al., 2016; Munro et al., 2007; Steele, Maurer, Bernat, Calhoun, & Kiehl, 2016). These studies assessed psychopathy in noncommunity samples—offenders (e.g., Maurer, Steele, Edwards, et al., 2016; Munro et al., 2007), youth offenders (e.g., Maurer, Steele, Cope, et al., 2016), and forensic patients (e.g., Brazil et al., 2009)—using the PCL-R (Hare, 2003) or the youth version of the psychopathy checklist (Forth et al., 2003). It is, however, worth to point out that self-report instruments (e.g., TriPM) measure liability factors associated with psychopathy in terms of low-order traits. By contrast, interview-based assessments, such as the PCL-R, usually target discrete symptoms, namely those providing an objective index of social deviance (Drislane, Patrick, & Arsal, 2014). Therefore, it is possible that the TriPM disinhibition subscale might provide a more sensitive measure of tendencies, or low order traits, better related to ERP correlates of inhibitory control. Notably, previous studies using the TriPM and related self-report measures of trait disinhibition have consistently reported reduced ERN amplitudes as a function of disinhibitory tendencies (e.g., Hall et al., 2007; Pasion et al., 2016).

Several studies using the go/no-go task supported the hypothesis that the N2 no-go component mirrors the (dis)inhibition of a planned response in go/no-go tasks (for a review see Folstein & Van Petten, 2008). As previously mentioned, no significant association between disinhibition and N2 amplitude was found in our study. A recent study by Prata and colleagues (2019) described the same pattern of results regarding disinhibition, but reported increased no-go N2 amplitudes for meanness. The N2 is a complex component, possibly reflecting the summation of overlapping ERP activity responsible for several features of response inhibition, such as cognitive control and sequential matching (Folstein & Van Petten, 2008). The study of the associations between distinct psychopathic traits and distinct features of response inhibition reported to modulate the N2 amplitude may constitute a topic for future studies.

We also hypothesized that increased disinhibition would be associated with a reduced P3 amplitude difference in the go/no-go task. Although the linear regression analysis revealed a marginal significant association, the latter did not persist in the permutation-based analysis. A previous study by Patrick and colleagues (2006) found solid evidence for reduced P3 amplitude as a function of externalizing problems. P3 was thought to reflect a diminished capacity of neuronal inhibition, potentiating neuronal hyperexcitability as a common risk factor for general externalizing problems (Begleiter & Porjesz, 1999). In our study, no difference was found in the mean P3 amplitude between go and go-go stimuli. Therefore, we cannot assert whether P3 reflects a meaningful correlate of cognitive processing in the go/no-go task in this context. Possible explanations for our findings include the absence of manipulation of task difficulty (P3 is sensitive to task difficulty), and the temporal overlap between N2 and P3 components.

4.3 | Meanness

Given the multifaceted nature of the meanness phenotype within the triarchic model of psychopathy, no hypothesis

regarding the association of meanness with behavioral and ERP components was formulated. Nonetheless, our results (supported by the permutation-based analysis) suggested that increased meanness is associated with an increase in the mean P3 amplitude difference value, possibly reflecting the allocation of attentional resources to the no-go signal.

The construct of meanness (or callous unemotionality) reflects a biologically based predatory orientation toward aggressive resource seeking, without regard or concern for others (Patrick, 2018). The maladaptive expression of trait fearlessness (Nelson & Foell, 2018), along with descriptions of meanness entailing a dissociable component from externalizing vulnerability—reflecting lack of empathy and the manifestation of instrumental predatory aggression—is present in the description of the phenotypical expression of meanness (Patrick & Drislane, 2015; Patrick et al., 2009, 2012).

A recent study by Van Dongen, Brazil, van Der Veen, and Franken (2018) reported that meanness was not associated with the P3 amplitude secondary to presentation of emotionally charged pictures displaying the perpetration of violent actions, but was associated with reduced LPP amplitudes instead. We did not find any association between meanness and the LPP response in this study. Most probably, the explanation for different associations relies on the content of the used tasks. In our task, the emotional salience of the cue signaling threat had no interpersonal content, whereas Van Dongen and colleagues (2018) used emotional pictures showing violent interpersonal interactions. As the phenotypical expression of meanness highlights the interpersonal nature of its manifestations (e.g., Patrick et al., 2009), it is possible that meanness better relates to stimuli with interpersonal content.

4.4 | Conclusion

Our results can be integrated in both etiological pathways theorized to underlie the phenotypic expressions of the triarchic model of psychopathy.

Boldness was associated with reduced CS type differentiation, which is consistent with the trait fearlessness etiological pathway (Patrick et al., 2009). In addition, the accurate estimation of CS+ US+ contingencies is congruent with descriptions of boldness as reflecting the positive adjustment characteristics of psychopathy (e.g., Patrick & Drislane, 2015).

Disinhibition was found to be the only phenotypical dimension of psychopathy associated with the neurophysiological correlate secondary to the occurrence of errors in the go/no-go task. This speaks to the notion that a lack of inhibitory control underlies a maladaptive externalizing etiological pathway of psychopathy, as previously proposed (e.g., Pasion et al., 2016; Patrick et al., 2012).

Finally, the contribution of meanness for the chosen explanatory framework of psychopathy remains unclear on the basis of our results. Future studies should analyze the etiological mechanisms underlying the expression of this trait. In particular, the use of tasks including contents of interpersonal relevance (e.g., Van Dongen et al., 2018) can provide a very promising lead.

4.5 | Limitations and closing remarks

The small sample size of our study represents the main limitation. We have tried to circumvent the impact of this limitation by analyzing data beyond the standard null hypothesis testing, using permutation-based statistics. Given that permutation-based statistics allows to compute confidence intervals for the distribution of statistical parameters, supporting the likelihood of a true effect being present (Dienes, 2008), we hoped to counteract the well-known reduced statistical power secondary to the small sample size. Nonetheless, permutation analysis does not allow for the assessment of the robustness and replicability of the results in other samples. As small sample sizes may lead to biased estimations of statistical effect sizes, follow-up replication studies with larger sample sizes will be needed to assess for the robustness of the reported findings.

We used an ERP analysis that allows studying the cortical activity with a high temporal precision. Nevertheless, several subcortical structures, such as the amygdalae, are central for the manifestation of threat conditioned responses (LeDoux, 2012). Unfortunately, EEG does not provide direct measures of activity of these structures. Therefore, future studies should embrace other techniques, such as fMRI, to assess subcortical structures during threat conditioning, and their relationship with phenotypical expressions of psychopathy, as assessed by the TriPM.

Finally, our study was designed to map the etiological mechanisms associated with the expression of psychopathic traits. Boldness and disinhibition are thought to reflect the more pure phenotypical expressions of trait fearlessness and externalizing vulnerability (Patrick & Drislane, 2015), but meanness relates both to the emotional and antisocial constituents of psychopathy. In the future, a task directly assessing the core deficit of empathy, expected to be found in meanness (Almeida et al., 2015; von Dongen et al., 2018), should be introduced.

In spite of the abovementioned limitations, our study provided evidence for deficits in threat conditioning and inhibitory control in psychopathy. This should encourage future research on how these broad etiological pathways are linked with distinct brain and behavioral manifestations of psychopathic traits.

ACKNOWLEDGMENTS

We would like to acknowledge the Portuguese Foundation for Science and Technology and the POCH/FSE Program for their financial support.

ORCID

Tiago O. Paiva https://orcid.org/0000-0001-5461-6802

REFERENCES

Almeida, P. R., Seixas, M. J., Ferreira-Santos, F., Vieira, J. B., Paiva, T. O., Moreira, P. S., & Costa, P. (2015). Empathic, moral and anti-social outcomes associated with distinct components of psychopathy in healthy individuals: A Triarchic model approach. *Personality and Individual Differences*, *85*, 205–211. https://doi.org/10.1016/j.paid.2015.05.012

Bacigalupo, F., & Luck, S. J. (2018). Event-related potential components as measures of aversive conditioning in humans. *Psychophysiology*, *55*(4), e13015. https://doi.org/10.1111/psyp.13015

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2013). Emotion-modulated startle in psychopathy: Clarifying familiar effects. *Journal of Abnormal Psychology*, *122*(2), 458. https://doi.org/10.1037/a0030958

Begleiter, H., & Porjesz, B. (1999). What Is inherited in the predisposition toward alcoholism? A proposed model. *Alcoholism: Clinical and Experimental Research*, *23*, 1125–1135. https://doi.org/10.1111/j.1530-0277.1999.tb04269.x

Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., & Flor, H. (2005). Deficient fear conditioning in psychopathy. *Archives of General Psychiatry*, *62*(7), 799. https://doi.org/10.1001/archpsyc.62.7.799

Blair, R. J. (2005). Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology*, *17*(3), 865–891. https://doi.org/10.1017/S0954579405050418

Blair, R. J. (2006). Subcortical brain systems in psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (1st ed., pp. 296–312). New York, NY: Guilford Press.

Blair, R. J. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Sciences*, *11*(9), 387–392. https://doi.org/10.1016/j.tics.2007.07.003

Blair, R. J., Meffert, H., Hwang, S., & White, S. F. (2018). Psychopathy and brain function: Insights from neuroimaging research. In C. J. Patrick (Ed.), *Handbook of Psychopathy* (2nd ed., pp. 401–421). New York, NY: Guilford Press.

Blair, R. J., Peschardt, K. S., Budhani, S., Mitchell, D. G. V., & Pine, D. S. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *47*(3–4), 262–275. https://doi.org/10.1111/j.1469-7610.2006.01596.x

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2005). Psychopathic personality traits: Heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine*, *35*(5), 637–648. https://doi.org/10.1017/S0033291704004180

Blonigen, D. M., Patrick, C. J., Douglas, K. S., Poythress, N. G., Skeem, J. L., Lilienfeld, S. O., … Krueger, R. F. (2010). Multimethod assessment of psychopathy in relation to factors of internalizing and externalizing from the Personality Assessment Inventory: The impact of method variance and suppressor effects. *Psychological Assessment*, *22*(1), 96–107. https://doi.org/10.1037/a0017240

Brazil, I. A., de Bruijn, E. R. A., Bulten, B. H., von Borries, A. K. L., van Lankveld, J. J. D. M., Buitelaar, J. K., & Verkes, R. J. (2009). Early and late components of error monitoring in violent offenders with psychopathy. *Biological Psychiatry*, *65*(2), 137–143. https://doi.org/10.1016/j.biopsych.2008.08.011

Brazil, I. A., Mars, R. B., Bulten, B. H., Buitelaar, J. K., Verkes, R. J., & De Bruijn, E. R. A. (2011). A neurophysiological dissociation between monitoring one's own and others' actions in psychopathy. *Biological Psychiatry*, *69*(7), 693–699. https://doi.org/10.1016/j.biopsych.2010.11.013

Bresin, K., Finy, M. S., Sprague, J., & Verona, E. (2014). Response monitoring and adjustment: Differential relations with psychopathic traits. *Journal of Abnormal Psychology*, *123*(3), 634–649. https://doi.org/10.1037/a0037229

Carolan, P. L., Jaspers-Fayer, F., Asmaro, D. T., Douglas, K. S., & Liotti, M. (2014). Electrophysiology of blunted emotional bias in psychopathic personality. *Psychophysiology*, *51*(1), 36–41. https://doi.org/10.1111/psyp.12145

Cheng, Y., Hung, A. Y., & Decety, J. (2012). Dissociation between affective sharing and emotion understanding in juvenile psychopaths. *Development and Psychopathology*, *24*(2), 623–636. https://doi.org/10.1017/S095457941200020X

Cleckley, H. M. (1941). *The mask of sanity: An attempt to reinterpret the so-called psychopathic personality*. St. Louis: C. V. Mosby Company.

Costello, T. H., Smith, S. F., Bowes, S. M., Riley, S., Berns, G. S., & Lilienfeld, S. O. (2019). Risky business: Psychopathy, framing effects, and financial outcomes. *Journal of Research in Personality*, *78*, 125–132. https://doi.org/10.1016/j.jrp.2018.11.006

Decety, J., Lewis, K. L., & Cowell, J. M. (2015). Specific electrophysiological components disentangle affective sharing and empathic concern in psychopathy. *Journal of Neurophysiology*, *114*(1), 493–504. https://doi.org/10.1152/jn.00253.2015

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Dienes, Z. (2008). *Understanding psychology as a science: An introduction to scientific and statistical inference*. New York: Palgrave Macmillan.

Donkers, F. C. L., & Van Boxtel, G. J. M. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition*, *56*(2), 165–176. https://doi.org/10.1016/j.bandc.2004.04.005

Drislane, L. E., & Patrick, C. (2017). Integrating alternative conceptions of psychopathic personality: A latent variable model of triarchic psychopathy constructs. *Journal of Personality Disorders*, *31*(1), 110–132. https://doi.org/10.1521/pedi_2016_30_240

Drislane, L. E., Patrick, C. J., & Arsal, G. (2014). Clarifying the content coverage of differing psychopathy inventories through reference to the triarchic psychopathy measure. *Psychological Assessment*, *26*(2), 350–362. https://doi.org/10.1037/a0035152

Edens, J. F., Marcus, D. K., Lilienfeld, S. O., & Poythress, N. G. (2006). Psychopathic, not psychopath: Taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology*, *115*(1), 131–144. https://doi.org/10.1037/0021-843X.115.1.131

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, *78*(6), 447–455. https://doi.org/10.1016/0013-4694(91)90062-9

Ferree, T. C. (2006). Spherical splines and average referencing in scalp electroencephalography. *Brain Topography*, *19*(1–2), 43–52. https://doi.org/10.1007/s10548-006-0011-0

Flor, H., Birbaumer, N., Hermann, C., Ziegler, S., & Patrick, C. J. (2002). Aversive pavlovian conditioning in psychopaths: Peripheral and central correlates. *Psychophysiology*, *39*(4), 505–518. https://doi.org/10.1111/1469-8986.3940505

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology*, *45*(1), 152–170. https://doi.org/10.1111/j.1469-8986.2007.00602.x

Forth, A., Kosson, D., & Hare, R. (2003). *The hare psychopathy checklist: Youth version, technical manual*. New York, NY: Multi-Health Systems, Inc.

Fowles, D. C. (2018). Temperament risk factors for psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (2nd ed., pp. 94–126). New York, NY: Guilford Publications.

Fowles, D. C., & Dindo, L. (2009). Temperament and psychopathy: A dual-pathway model. *Current Directions in Psychological Science*, *18*(3), 179–183. https://doi.org/10.1111/j.1467-8721.2009.01632.x

Guay, J., Ruscio, J., Knight, R. A., & Hare, R. D. (2007). A taxometric analysis of the latent structure of psychopathy: Evidence for dimensionality. *Journal of Abnormal Psychology*, *116*(4), 701–716. https://doi.org/10.1037/0021-843X.116.4.701

Hajcak, G., Macnamara, A., & Olvet, D. M. (2010). Event-related potentials, emotion, and emotion regulation: An integrative review. *Developmental Neuropsychology*, *35*(2), 129–155. https://doi.org/10.1080/87565640903526504

Hajcak, G., Moser, J. S., Yeung, N., & Simons, R. F. (2005). On the ERN and the significance of errors. *Psychophysiology*, *42*(2), 151–160. https://doi.org/10.1111/j.1469-8986.2005.00270.x

Hall, J. R., Bernat, E. M., & Patrick, C. J. (2007). Externalizing psychopathology and the error-related negativity. *Psychological Science*, *18*(4), 326–333. https://doi.org/10.1111/j.1467-9280.2007.01899.x

Hare, R. D. (2003). *The psychopathy checklist—Revised manual* (2nd ed.). Toronto, ON: Multi-Health Systems.

Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annual Review of Clinical Psychology*, *4*(1), 217–246. https://doi.org/10.1146/annurev.clinpsy.3.022806.091452

Heritage, A. J., & Benning, S. D. (2013). Impulsivity and response modulation deficits in psychopathy: Evidence from the ERN and N1. *Journal of Abnormal Psychology*, *122*(1), 215. https://doi.org/10.1037/a0030039

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*(4), 679–709. https://doi.org/10.1037/0033-295X.109.4.679

Hoppenbrouwers, S. S., Bulten, B. H., & Brazil, I. A. (2016). Parsing fear: A reassessment of the evidence for fear deficits in psychopathy. *Psychological Bulletin*, *142*(6), 573–600. https://doi.org/10.1037/bul0000040

Juárez, M., Kiehl, K. A., & Calhoun, V. D. (2013). Intrinsic limbic and paralimbic networks are associated with criminal psychopathy. *Human Brain Mapping*, *34*(8), 1921–1930. https://doi.org/10.1002/hbm.22037

Krueger, R. F. (2002). Personality from a realist's perspective: Personality traits, criminal behaviors, and the externalizing spectrum. *Journal of Research in Personality*, *36*(6), 564–572. https://doi.org/10.1016/S0092-6566(02)00506-8

Krueger, R. F., & Piasecki, T. M. (2002). Toward a dimensional and psychometrically-informed approach to conceptualizing psychopathology. *Behaviour Research and Therapy*, *40*(5), 485–499. https://doi.org/10.1016/S0005-7967(02)00016-5

Kyranides, M. N., Fanti, K. A., Sikki, M., & Patrick, C. J. (2017). Triarchic dimensions of psychopathy in young adulthood: Associations with clinical and physiological measures after accounting for adolescent psychopathic traits. *Personality Disorders: Theory, Research, and Treatment*, *8*(2), 140–149. https://doi.org/10.1037/per0000193

LeDoux, J. (2012). Rethinking the emotional brain. *Neuron*, *73*(4), 653–676. https://doi.org/10.1016/j.neuron.2012.02.004

LeDoux, J. (2014). Coming to terms with fear. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(8), 2871–2878. https://doi.org/10.1073/pnas.1400335111

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of Personality and Social Psychology*, *68*(1), 151–158. https://doi.org/10.1037/0022-3514.68.1.151

Lilienfeld, S. O., Smith, S. F., Sauvigné, K. C., Patrick, C. J., Drislane, L. E., Latzman, R. D., & Krueger, R. F. (2016). Is boldness relevant to psychopathic personality? Meta-analytic relations with non-psychopathy checklist-based measures of psychopathy. *Psychological Assessment*, *28*(10), 1172–1185. https://doi.org/10.1037/pas0000244

Lilienfeld, S. O., Watts, A. L., Francis Smith, S., Berg, J. M., & Latzman, R. D. (2015). Psychopathy deconstructed and reconstructed: Identifying and assembling the personality building blocks of Cleckley's Chimera. *Journal of Personality*, *83*(6), 593–610. https://doi.org/10.1111/jopy.12118

Liu, Y., Huang, H., McGinnis-Deweese, M., Keil, A., & Ding, M. (2012). Neural substrate of the late positive potential in emotional processing. *Journal of Neuroscience*, *32*(42), 14563–14572. https://doi.org/10.1523/JNEUROSCI.3109-12.2012

López, R., Poy, R., Patrick, C. J., & Moltó, J. (2013). Deficient fear conditioning and self-reported psychopathy: The role of fearless dominance. *Psychophysiology*, *50*(2), 210–218. https://doi.org/10.1111/j.1469-8986.2012.01493.x

Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of event-related potentials. *Frontiers in Human Neuroscience*, *8*, 213. https://doi.org/10.3389/fnhum.2014.00213

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *The Journal of Abnormal and Social Psychology*, *55*(1), 6–10. https://doi.org/10.1037/h0047232

Makeig, S., Bell, A. J., Jung, T. P., & Sejnowski, T. J. (1996). Independent component analysis of electroencephalographic data. *Advances in Neural Information Processing Systems*, *8*, 145–151. https://doi.org/10.1109/ICOSP.2002.1180091

Maruo, Y., Sommer, W., & Masaki, H. (2017). The effect of monetary punishment on error evaluation in a Go/No-go task. *International Journal of Psychophysiology*, *120*(June), 54–59. https://doi.org/10.1016/j.ijpsycho.2017.07.002

Maurer, J. M., Steele, V. R., Cope, L. M., Vincent, G. M., Stephen, J. M., Calhoun, V. D., & Kiehl, K. A. (2016). Dysfunctional error-related processing in incarcerated youth with elevated psychopathic traits. *Developmental Cognitive Neuroscience*, *19*, 70–77. https://doi.org/10.1016/j.dcn.2016.02.006

Maurer, J. M., Steele, V. R., Fink, B. C., Vincent, G. M., Calhoun, V. D., & Kiehl, K. A. (2018). Investigating error-related processing in incarcerated adolescents with self-report psychopathy measures. *Biological Psychology*, *132*, 96–105. https://doi.org/10.1016/j.biopsycho.2017.11.009

Maurer, M., Steele, V. R., Edwards, B. G., Bernat, E. M., Calhoun, V. D., & Kiehl, K. A. (2016). Dysfunctional error-related processing in female psychopathy. *Social Cognitive and Affective Neuroscience*, *11*(7), 1059–1068. https://doi.org/10.1093/scan/nsv070

McCord, W., & McCord, J. (1964). *The psychopath: An essay on the criminal mind*. Oxford, England: D. Van Nostrand.

Medina, A. L., Kirilko, E., & Grose-Fifer, J. (2016). Emotional processing and psychopathic traits in male college students: An event-related potential study. *International Journal of Psychophysiology*, *106*, 39–49. https://doi.org/10.1016/j.ijpsycho.2016.06.004

Munro, G. E. S., Dywan, J., Harris, G. T., McKee, S., Unsal, A., & Segalowitz, S. J. (2007). ERN varies with degree of psychopathy in an emotion discrimination task. *Biological Psychology*, *76*(1–2), 31–42. https://doi.org/10.1016/j.biopsycho.2007.05.004

Nelson, L. D., & Foell, J. (2018). Externalizing proneness and psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (2nd ed., pp. 127–143). New York, NY: Guilford Press.

Nelson, L. D., Patrick, C. J., & Bernat, E. M. (2011). Operationalizing proneness to externalizing psychopathology as a multivariate psychophysiological phenotype. *Psychophysiology*, *48*(1), 64–73. https://doi.org/10.1111/j.1469-8986.2010.01047.x

Nieuwenhuis, S., Yeung, N., & Cohen, J. D. (2004). Stimulus modality, perceptual overlap, and the go/no-go N2. *Psychophysiology*, *41*(1), 157–160. https://doi.org/10.1046/j.1469-8986.2003.00128.x

Nunnally, J. C., Bernstein, I. H., & Berge, J. M. F. (1967). *Psychometric theory*, Vol. *226*. New York: McGraw-Hill.

Pailing, P. E., & Segalowitz, S. J. (2004). The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. *Psychophysiology*, *41*(1), 84–95. https://doi.org/10.1111/1469-8986.00124

Paiva, T. O., Pasion, R., Patrick, C. J., Moreira, D., Almeida, P. R., & Barbosa, F. (2020). Further evaluation of the triarchic psychopathy measure: Evidence from community adult and prisoner samples from Portugal. *Psychological Assessment*, *32*, e1–e14. https://doi.org/10.1037/pas0000797

Pasion, R., Cruz, A. R., & Barbosa, F. (2016). Dissociation of boldness and disinhibition psychopathic traits in ERN modulation. *Personality and Individual Differences*, *95*, 6–10. https://doi.org/10.1016/j.paid.2016.02.017

Pasion, R., Fernandes, C., Pereira, M. R., & Barbosa, F. (2018). Antisocial behaviour and psychopathy: Uncovering the externalizing link in the P3 modulation. *Neuroscience and Biobehavioral Reviews*, *91*, 170–186. https://doi.org/10.1016/j.neubiorev.2017.03.012

Pasion, R., Prata, C., Fernandes, M., Almeida, R., Garcez, H., Araújo, C., & Barbosa, F. (2019). N2 amplitude modulation across the antisocial spectrum: A meta-analysis. *Reviews in the Neurosciences*, *30*, 781–794 https://doi.org/10.1515/revneuro-2018-0116

Patrick, C. (2010). Operationalizing the Triarchic conceptualisation of psychopathy: Preliminary description of brief scales for assessment of boldness, meanness, and disinhibition. *PhenX Toolkit Online Assessment Catalog*. Retrieved from https://www.phenxtoolkit.org/toolkit_content/supplemental_info/psychiatric/measures/Triarchic_Psychopathy_Measure_Manual.pdf

Patrick, C. (2018). Cognitive and emotional processing in psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (2nd ed., pp. 422–455). New York, NY: Guilford Publications.

Patrick, C. J., & Bernat, E. M. (2009). Neurobiology of psychopathy: A two process theory. In G. G. Berntson & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioral sciences* (Vol. *2*, pp. 1110–1131). New Jersey: John Wiley & Sons Inc. https://doi.org/10.1002/9780470478509.neubb002057

Patrick, C. J., Bernat, E. M., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, *43*(1), 84–92. https://doi.org/10.1111/j.1469-8986.2006.00376.x

Patrick, C., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology*, *102*(1), 82–92. https://doi.org/10.1037/0021-843X.102.1.82

Patrick, C., & Drislane, L. E. (2015). Triarchic model of psychopathy: Origins, operationalizations, and observed linkages with personality and general psychopathology. *Journal of Personality*, *83*(6), 627–643. https://doi.org/10.1111/jopy.12119

Patrick, C., Drislane, L. E., & Strickland, C. (2012). Conceptualizing psychopathy in triarchic terms: Implications for treatment. *International Journal of Forensic Mental Health*, *11*(4), 253–266. https://doi.org/10.1080/14999013.2012.746761

Patrick, C., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Development and Psychopathology*, *21*(3), 913–938. https://doi.org/10.1017/S0954579409000492

Patrick, C., Hicks, B. M., Krueger, R. F., & Lang, A. R. (2005). Relations between psychopathy facets and externalizing in a criminal offender sample. *Journal of Personality Disorders*, *19*(4), 339–356. https://doi.org/10.1521/pedi.2005.19.4.339

Prata, C., Pasion, R., Fernandes, M., Almeida, R., Pereira, M. R., Mazer, P., & Barbosa, F. (2019). Callousness and meanness traits are associated with increased N2 amplitude in a community sample of adolescents and adults. *Neuroscience Letters*, *706*, 1–6. https://doi.org/10.1016/j.neulet.2019.04.056

Rodman, A. M., Kastman, E. K., Dorfman, H. M., Baskin-Sommers, A. R., Kiehl, K. A., Newman, J. P., & Buckholtz, J. W. (2016). Selective mapping of psychopathy and externalizing to dissociable circuits for inhibitory self-control. *Clinical Psychological Science*, *4*(3), 559–571. https://doi.org/10.1177/2167702616631495

Rothemund, Y., Ziegler, S., Hermann, C., Gruesser, S. M., Foell, J., Patrick, C. J., & Flor, H. (2012). Fear conditioning in psychopaths: Event-related potentials and peripheral measures. *Biological Psychology*, *90*(1), 50–59. https://doi.org/10.1016/j.biopsycho.2012.02.011

Schupp, H. T., Flaisch, T., Stockburger, J., & Junghöfer, M. (2006). Emotion and attention: Event-related brain potential studies. *Progress in Brain Research*, *156*, 31–51. https://doi.org/10.1016/S0079-6123(06)56002-9

Sellbom, M., & Phillips, T. R. (2013). An examination of the triarchic conceptualization of psychopathy in incarcerated and nonincarcerated samples. *Journal of Abnormal Psychology*, *122*(1), 208–214. https://doi.org/10.1037/a0029306

Skeem, J. L., Polaschek, D. L. L., Patrick, C. J., & Lilienfeld, S. O. (2011). Psychopathic personality: Bridging the gap between scientific evidence and public policy. *Psychological Science in the Public Interest*, *12*(3), 95–162. https://doi.org/10.1177/1529100611426706

Steele, V. R., Maurer, J. M., Bernat, E. M., Calhoun, V. D., & Kiehl, K. A. (2016). Error-related processing in adult males with elevated psychopathic traits. *Personality Disorders: Theory, Research, and Treatment*, *7*(1), 80–90. https://doi.org/10.1037/per0000143

Vachon, D. D., Lynam, D. R., Widiger, T. A., Miller, J. D., McCrae, R. R., & Costa, P. T. (2013). Basic traits predict the prevalence of personality disorder across the life span: The example of

psychopathy. *Psychological Science*, *24*(5), 698–705. https://doi.org/10.1177/0956797612460249

Van Dongen, J. D. M., Brazil, I. A., Van Der Veen, F. M., & Franken, I. H. A. (2018). Electrophysiological correlates of empathic processing and its relation to psychopathic meanness. *Neuropsychology*, *32*(8), 996–1006. https://doi.org/10.1037/neu0000477

Venables, N. C., Hall, J. R., Yancey, J. R., & Patrick, C. J. (2015). Factors of psychopathy and electrocortical response to emotional pictures: Further evidence for a two-process theory. *Journal of Abnormal Psychology*, *124*(2), 319–328. https://doi.org/10.1037/abn0000032

Vieira, J. B., Almeida, P. R., Ferreira-Santos, F., Barbosa, F., Marques-Teixeira, J., & Marsh, A. A. (2014). Distinct neural activation patterns underlie economic decisions in high and low psychopathy scorers. *Social Cognitive and Affective Neuroscience*, *9*(8), 1099–1107. https://doi.org/10.1093/scan/nst093

Vieira, J. B., Almeida, P. R., Ferreira-Santos, F., Moreira, P. S., Barbosa, F., & Marques-Teixeira, J. (2014). *The Triarchic psychopathy measure (TriPM): Translation and adaptation to European Portuguese (LabReport No. 6)*. Porto, Portugal: Laboratory of Neuropsychophysiology (University of Porto). https://doi.org/10.17605/OSF.IO/5C8EJ

Vieira, J. B., Ferreira-Santos, F., Almeida, P. R., Barbosa, F., Marques-Teixeira, J., & Marsh, A. A. (2015). Psychopathic traits are associated with cortical and subcortical volume alterations in healthy individuals. *Social Cognitive and Affective Neuroscience*, *10*(12), 1693–1704. https://doi.org/10.1093/scan/nsv062

Weinberg, A., Riesel, A., & Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. *Motivation and Emotion*, *36*(1), 84–100. https://doi.org/10.1007/s11031-011-9269-y

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

TABLE S1 Descriptive statistics for the number of noise free trials on each experimental task

TABLE S2 Descriptive statistics for the total number of excluded components, and types of artefacts corrected by means of independent component analysis (ICA) for the threat conditioning and go/no-go tasks

TABLE S3 Permutation analysis: p values and 95% confidence intervals (CI) for standardized β coefficients

How to cite this article: Paiva TO, Almeida PR, Coelho RC, et al. The neurophysiological correlates of the triarchic model of psychopathy: An approach to the basic mechanisms of threat conditioning and inhibitory control. *Psychophysiology*. 2020;57:e13567. https://doi.org/10.1111/psyp.13567