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Low defensive cardiac reactivity as a physiological correlate of psychopathic fearlessness: Gender differences

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ABSTRACT

Affective/interpersonal features of psychopathy have been consistently associated with diverse psychophysiological indicators of low threat sensitivity, suggesting an underlying deficit in the reactivity of the brain's defensive motivational system. This study examined the Cardiac Defense Response (CDR) —a complex pattern of heart rate changes in response to an aversive, intense, and unexpected stimulus— and its second accelerative component (A2), as a new physiological indicator of the fearlessness trait component of psychopathy. The differential contribution of dispositional fearlessness, externalizing proneness, and coldheartedness to the CDR pattern elicited during a defense psychophysiological test was examined in a mixed-gender sample of 156 undergraduates (62% women) assessed by the Psychopathic Personality Inventory-Revised (PPI-R). Higher PPI-R Fearless Dominance scores were related to lower heart rate changes throughout the CDR in women, but not in men. Further analyses on scales conforming the fearless dominance factor revealed that the hypothesized reduced A2 was specifically related to higher PPI-R Fearlessness scores only in women. Our findings provide initial evidence for the utility of the A2 to better understand the physiological aspects of fearlessness tendencies and its potential distinct manifestations across genders.

1. Introduction

Psychopathy is considered a multifaceted personality disorder which involves prominent behavioral deviance in a context of distinctive emotional and interpersonal traits (Cleckley, 1976; Hare & Neumann, 2008; Patrick et al., 2009). Dual-process models of psychopathy (Fowles & Dindo, 2009; Patrick & Bernat, 2009) postulate that two separable constructs of individual differences, with a distinctive neurobiological foundation, contribute to the impulsive/antisocial and the affective/interpersonal symptom components of psychopathy: *externalizing vulnerability* —reflecting impairments in frontocortical systems that mediate functions such as planning, anticipation, and behavioral control— and *trait fearlessness* —reflecting an under-reactivity of the brain's defensive motivational system to threat cues—, respectively. The most widely used measures to assess psychopathy in incarcerated (Psychopathy Checklist-Revised; PCL-R; Hare, 2003) and community samples (Psychopathic Personality Inventory-Revised; PPI-R; Lilienfeld & Widows, 2005) reflect these two symptom components in their bifactorial structures, with a first factor assessing the affective/interpersonal features of psychopathy (albeit through different configurations of these traits; see Marcus et al., 2013; Patrick et al., 2009), and a second factor assessing its externalizing tendencies.

In support of the view of psychopathy as a multifaceted disorder with different etiological substrates, empirical studies have demonstrated that the affective/interpersonal traits of psychopathy are particularly related to reduced aversive startle potentiation (ASP), which is considered one of the most reliable and well-validated psychophysiological correlates of the hypothesized deficits in threat responsivity believed to underlie the affective/interpersonal features of psychopathy (see Oskarsson et al., 2021, for a review). In fact, Kramer et al. (2012) conducted a quantitative-structural analysis of scale measures of fear/-fearlessness —including the scales loading on the PPI-R Fearless Dominance factor (i.e., Social Influence, Stress Immunity, and Fearlessness) as indicators of low fear— and found evidence of a general factor which could be interpreted as a bipolar dimension of dispositional threat sensitivity (THT+), that was appreciably heritable (~.5) and accounted for individual differences in (threat-neutral) ASP (see also Vaidyanathan et al., 2009). In this way, these differing self-report measures would act as indicators of a common underlying dimension of threat sensitivity, with the low end marked by social dominance,

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affective imperturbability, and thrill-seeking —intersecting with the affective/interpersonal traits of psychopathy—, and the high end characterized by intense responsiveness to cue-elicited fear and threatening situations and avoidance of risky activities —intersecting with specific phobic disorders (see Nelson et al., 2016). Consequently, while ASP is diminished in high psychopathic fearless individuals (Benning et al., 2005; Esteller et al., 2016), patients with phobic disorders show an enhanced ASP (Cuthbert et al., 2003; Lang & McTeague, 2009).

In addition to ASP, research has also shown that psychopathy, or specifically its affective/interpersonal traits, is reliably associated with other psychophysiological measures, such as reduced corrugator muscle tension (Flor et al., 2002) and electrodermal reactivity (López et al., 2013) in fear conditioning procedures, diminished late positive potential (LPP) brain response amplitudes to aversive versus neutral pictures (Venables et al., 2015), or reduced heart rate (HR) acceleration while experiencing negative pictures (Casey et al., 2013). In this line, Yancey et al. (2016) demonstrated that some of these physiological indicators —ASP, corrugator electromyography reactivity, HR acceleration— can be combined with scores on a report-based measure of dispositional threat sensitivity (cf. Kramer et al., 2012; Vaidyanathan et al., 2009) to delineate a cross-domain index of THT+. The resulting factor showed positive robust associations with other physiological criterion measures (e.g., general muscle tension, noise-probe P3) and with symptoms of phobic disorders —which would be negatively related to features in the low pole of the fear/fearlessness dimension, e.g., the affective/interpersonal traits of psychopathy. The THT+ factor found by Yancey et al. (2016) can be considered a neurobehavioral construct that serves as a reference for the future research on neurobiology of individual differences in threat sensitivity.

In this study, the Cardiac Defense Response (CDR; Vila et al., 1992) is examined as a potential new physiological measure of the fearlessness trait component of psychopathy. The CDR is characterized by a complex pattern of heart rate changes which are produced in response to an aversive, discrete, intense, and unexpected stimulation —preferentially acoustic or electrocutaneous. The response lasts approximately 80 s after stimulus onset and, in unselected participants under resting conditions, consists of two alternating accelerative and decelerative components: acceleration-deceleration-acceleration-deceleration. Results of studies using physiological measures that indirectly index sympathetic or parasympathetic control (such as pre-ejection period, pulse transit time and beta-adrenergic blockade vs. respiratory sinus arrhythmia and baroreceptor reflex) suggest that during the first accelerative/decelerative components (A1/D1) there is parasympathetic dominance —inhibition and activation, respectively—, while the second acceleration/deceleration (A2/D2) is controlled by both sympathetic and parasympathetic influences that work reciprocally —sympathetic activation accompanied by parasympathetic inhibition, and sympathetic inhibition accompanied by parasympathetic activation, respectively— mediated primarily by the sympathetic nervous system (Fernández & Vila, 1989; Garrido et al., 2020; Reyes del Paso et al., 1993; 1994). This cardiac pattern seems to reflect the succession of two defensive phases, showing the transition from attention to action: an attentional protective phase reflected in the first acceleration/deceleration —cessation of ongoing activity and heightened attention to external cues—, and a motivational protective phase reflected in the second acceleration/deceleration —metabolic mobilization for active defense, and retrieval if danger disappears (Vila et al., 2007). Of note, this complex pattern of heart rate changes (acceleration-deceleration-acceleration-deceleration) is observed in unselected samples of participants in laboratory settings when no other task is imposed. In natural settings, the imminence of a predator (or, in other words, the severity and type of danger and its spatial and temporal proximity) involves a fast shift from cardiac deceleration to cardiac acceleration (i.e., from D1 to A2) and to overt defensive actions (fight, flight), whose metabolic requirements will be supported by the major physiological changes in cardiac, electrodermal and somatic systems (see Lang et al., 1997). Paralleling responses to imminent threat in real-world situations, the cardiac defense response becomes a single, pronounced acceleration (without subsequent deceleration) when the unexpected noise occurs in the context of viewing unpleasant or phobic pictures. This pattern suggests that the motivational phase (readiness for defensive actions) has been temporarily advanced to better respond to the threat (Ruiz-Padial et al., 2005; Sánchez et al., 2002).

The CDR may have some advantages over other cardiac measures in laboratory studies on cue-specific defensive reactivity in psychopathy. On the one hand, HR measured during aversive-picture viewing prototypically shows a large deceleration, representing only the first part of the defense response (Bradley et al., 2001): in this context, in which aversive stimuli do not pose a real and imminent danger, the second acceleration —i.e., the readiness for active defense— does not occur, and only the previous phase of attentional orienting is present. In contrast, the CDR tracks the entire defense cascade sequence, from heightened attention in its earlier accelerative/decelerative components to readiness for action (second acceleration) and recovery (second deceleration) in its later components (for the defense cascade model, see Bradley & Lang, 2000 or Lang et al., 1997). On the other hand, the reduced HR found in individuals with psychopathy (in resting and task conditions) seems not to be specific to any cluster of psychopathic traits (de Looff et al., 2022), whereas the CDR, as a psychophysiological defensive response to imminent danger, could be related to the affective/interpersonal features of psychopathy, but not to its externalizing traits.

Thus, converging lines of evidence suggest that the CDR, and more specifically its second accelerative component (A2), could be a relevant psychophysiological indicator of the fearlessness trait component of psychopathy. CDR patterns characterized by a reduced/absent first deceleration and/or higher A2 amplitudes have been associated with different internalizing problems and traits —including post-traumatic stress disorder (Norte et al., 2019; Schalinski et al., 2013), chronic worry (Delgado et al., 2009), and trait anxiety (López et al., 2016)— and with focal fear disorders more particularly —i.e., specific phobias (Ruiz-Padial et al., 2002, 2005; Sánchez et al., 2009; Wannemueller et al., 2017). Individuals without the second accelerative component of the CDR have also been found to show deficient fear learning (López et al., 2009) and to be characterized by high extraversion and low neuroticism in terms of personality (Richards & Eves, 1991), which represent the personality trait configuration most characteristic of psychopathic fearless dominance (Miller & Lynam, 2012). In this regard, Vila et al. (2007) suggested that different CDR patterns would reflect differences in the way individuals face danger and that preexisting clinical states could contribute to modify the activation threshold of the defensive motivational system and, subsequently, the coping response to a dangerous stimulus. Thus, a lowered threshold in the case of fear disorders would carry out an earlier and oversized defensive response, whereas the affective/interpersonal traits of psychopathy could increase that threshold, leading to a poor defensive response.

Therefore, based on the above-revised evidence, this study aims to examine for the first time whether a reduced CDR, and particularly its second accelerative component (A2), which would be indexing a blunted reactivity of the defensive motivational system, could be an additional physiological indicator of the affective/interpersonal traits of psychopathy. To this end, we examined the differential contribution of psychopathic traits, at both the factor and scale levels of the PPI-R, to the CDR pattern elicited by a defense psychophysiological test in a mixed-gender sample of undergraduates. In light of the foregoing evidence, we expected that a lower defensive reactivity —as indexed by the second accelerative component of the CDR— would be specifically associated with higher scores on the PPI-R Fearless Dominance factor, but no with scores on the PPI-R Impulsive Antisociality factor or the Coldheartedness scale. As a secondary objective, if appropriate in light of the results, we were also interested in examining the differential contribution of the constituent scales within the significant factor(s) in predicting reduced

cardiac reactivity. Considering previous research on individual differences in the CDR, which has relied almost exclusively on female samples (e.g., Delgado et al., 2009; Ruiz-Padial et al., 2002, 2005; Sánchez et al., 2009; Schalinski et al., 2013; Vila & Beech, 1978), another relevant feature of this study was to test for gender effects on psychopathy-related differences in CDR patterns.

2. Method

2.1. Participants

Participants were 168 undergraduates (65 men) from the Universitat Jaume I of Castellón (Spain). None presented visual, auditory, or cardiovascular deficits. Ten participants were excluded due to equipment failure, and two because they were undergoing psychiatric and/or pharmacological treatment. The final sample comprised a total of 156 participants (60 men) who were aged between 18 and 25 years (M = 20.2, SD = 2.0).

The Spanish adaptation (López et al., 2013) of the PPI-R (Lilienfeld & Widows, 2005) was used to assess psychopathic traits. The PPI-R is a self-report measure that consists of 154 items presented in a 4-point Likert-type format (1 = *false*, 2 = *somewhat false*, 3 = *somewhat true*, 4 = *true*). This inventory provides a total index score of psychopathy, two factor scores (Fearless Dominance and Impulsive Antisociality; Benning et al., 2003), and eight content scale scores: *Social Influence* (18 items; When people are mad at me, I usually win them over with my charm), *Stress Immunity* (13 items; I can remain calm in situations that would make many other people panic), and *Fearlessness* (14 items; I would find the job of a movie stunt person exciting) — scores on the Fearless Dominance factor are obtained by summing scores on these three scales—; *Machiavellian Egocentricity* (20 items; I get mad if I don't receive special favors I deserve), *Rebellious Nonconformity* (16 items; I have always seen myself as something of a rebel), *Blame Externalization* (15 items; Some people have gone out of their way to make my life difficult), and *Carefree Nonplanfulness* (19 items; A lot of times, I repeat the same bad decisions) —scores on the Impulsive Antisociality factor are obtained by summing scores on these four scales—; and *Coldheartedness* (16 items; A lot of times, I worry when a friend is having personal problems, reversed) —a subscale that does not load distinctively on either higher-order factor, thus tapping a distinct third dimension or factor (see Benning et al., 2003).

Table 1 reports the PPI-R scale scores' reliabilities, means, standard deviations, and ranges for the overall sample and for women and men separately. Independent t-tests revealed that men scored significantly higher than women in both factors and all PPI-R scales ($ts > |2.61|$; $ps < .01$), except for Social Influence and Blame Externalization ($ts < |.56|$; $ps > .579$).

2.2. Instruments

2.2.1. Defense psychophysiological test

The defense psychophysiological test to obtain the CDR (cf. Vila et al., 2007) consisted of the unexpected presentation of an intense white noise of 105 dB, 500 ms, and instantaneous risetime, delivered binaurally through 3a Insert Earphone (Eartone), after a resting period of 8 min. Participants were seated in a comfortable armchair and were instructed to breathe spontaneously and to remain with their eyes open throughout the recording time. They were informed that the purpose of the experiment was to record their electrocardiogram during a period of resting conditions for several minutes, without mentioning the upcoming noise presentation. Electrocardiogram recording lasted from 15 s prior to stimulus onset (baseline) to 80 s after its presentation. A single trial per participant was conducted, as previous evidence has demonstrated rapid habituation of the CDR with repeated presentations of the noise (Eves & Gruzelier, 1984; Mata et al., 2009; Ramírez et al., 2005; Turpin, 1986; Vila & Beech, 1978; Vila et al., 1992), and that individual differences in the CDR have been found only for the first presentation of the stimulus (e.g., Schalinski et al., 2013).

2.2.2. Physiological data recording and reduction

Stimuli control, data acquisition and reduction were accomplished using VPM software (Cook, 2002). Ag/AgCl surface electrodes (Standard Lead II) filled with hypertonic electrolyte paste provided 1000 samples/second electro-cardiograph analogical signals to a Coulbourn V75–04 High Gain Bioamplifier, and then to a Coulbourn S81–02 generator and gated through a Coulbourn S82–24 audio-mixer amplifier. Interbeat intervals were recorded to the nearest millisecond and reduced offline into heart rate in beats per minute, in half-second bins. Data for the 80-s recording period were transformed to averages for every second, and HR change scores were computed by subtracting the pretrial 15 s baseline average.

To facilitate statistical analysis without altering CDR topography, the 80 second-by-second HR change scores were reduced to ten values corresponding to the medians of 10 progressively longer intervals (cf. Vila et al., 2007): 2 of 3 s, 2 of 5 s, 3 of 7 s, and 3 of 13 s (from this point on, M1 to M10). In this simplified representation of the CDR, M1 reflects the first acceleration (A1), M2 to M4 the first deceleration (D1), M5 to M8 the second acceleration (A2), and M9 to M10 the second deceleration (D2; Vila et al., 2007). See Fig. 1a to illustrate the CDR pattern and the corresponding medians. Additionally, we undertook a temporal Principal Component Analysis (PCA; Dien, 2012) on the 80 second-by-second HR change scores to verify the pattern conforming the CDR and to derive its four components in a data-driven manner. This approach has been widely applied to other psychophysiological measures (e.g., event-related potentials; Dien, 2012). In brief, temporal PCA

Table 1
PPI-R Factor and Scale Scores Reliability, Means, Standard Deviations, and Ranges in the Overall Sample, and for Women and Men separately.

	α	*Overall (N = 156)*		*Women (N = 96)*		*Men (N = 60)*		*Gender Comparison*	
		M (SD)	*Min.-Max.*	*M (SD)*	*Min.-Max.*	*M (SD)*	*Min.-Max.*	*t*	*p*
PPI-R Factors									
Fearless Dominance	.85	113.42 (16.05)	61–160	109.22 (15.51)	61–148	120.15 (14.66)	91–160	**-4.37**	< .0001
Impulsive Antisociality	.90	144.90 (23.13)	72–206	139.50 (22.59)	72–206	153.55 (21.59)	107–201	**-3.85**	.0002
PPI-R Scales									
Fearlessness	.82	33.95 (8.49)	14–53	31.49 (8.28)	14–53	37.88 (7.31)	19–53	**-4.90**	< .0001
Social Influence	.84	47.44 (8.61)	24–65	47.74 (9.21)	24–65	46.95 (7.59)	32–64	0.56	.579
Stress Immunity	.84	32.04 (7.24)	15–51	29.99 (6.96)	15–46	35.32 (6.47)	21–51	**-4.77**	< .0001
Machiavellian Egocentricity	.86	40.82 (9.69)	20–64	38.05 (9.66)	20–64	45.25 (7.99)	32–61	**-4.83**	< .0001
Rebellious Nonconformity	.79	35.66 (7.81)	15–59	34.20 (7.60)	15–50	38 (7.63)	25–59	**-3.04**	.002
Blame Externalization	.89	31.71(8.83)	15–58	31.77 (8.61)	15–58	31.60 (9.24)	18–56	0.12	.907
Carefree Nonplanfulness	.81	36.72 (7.64)	20–60	35.48 (7.82)	20–57	38.7 (6.97)	27–60	**-2.61**	.010
Coldheartedness	.80	29.39 (6.73)	17–51	27.5 (5.61)	17–46	32.42 (7.29)	19–51	**-4.74**	< .0001

Note. PPI-R = Psychopathic Personality Inventory Revised (Lilienfeld & Widows, 2005); α = Cronbach's alpha
Significant comparisons are highlighted in bold.

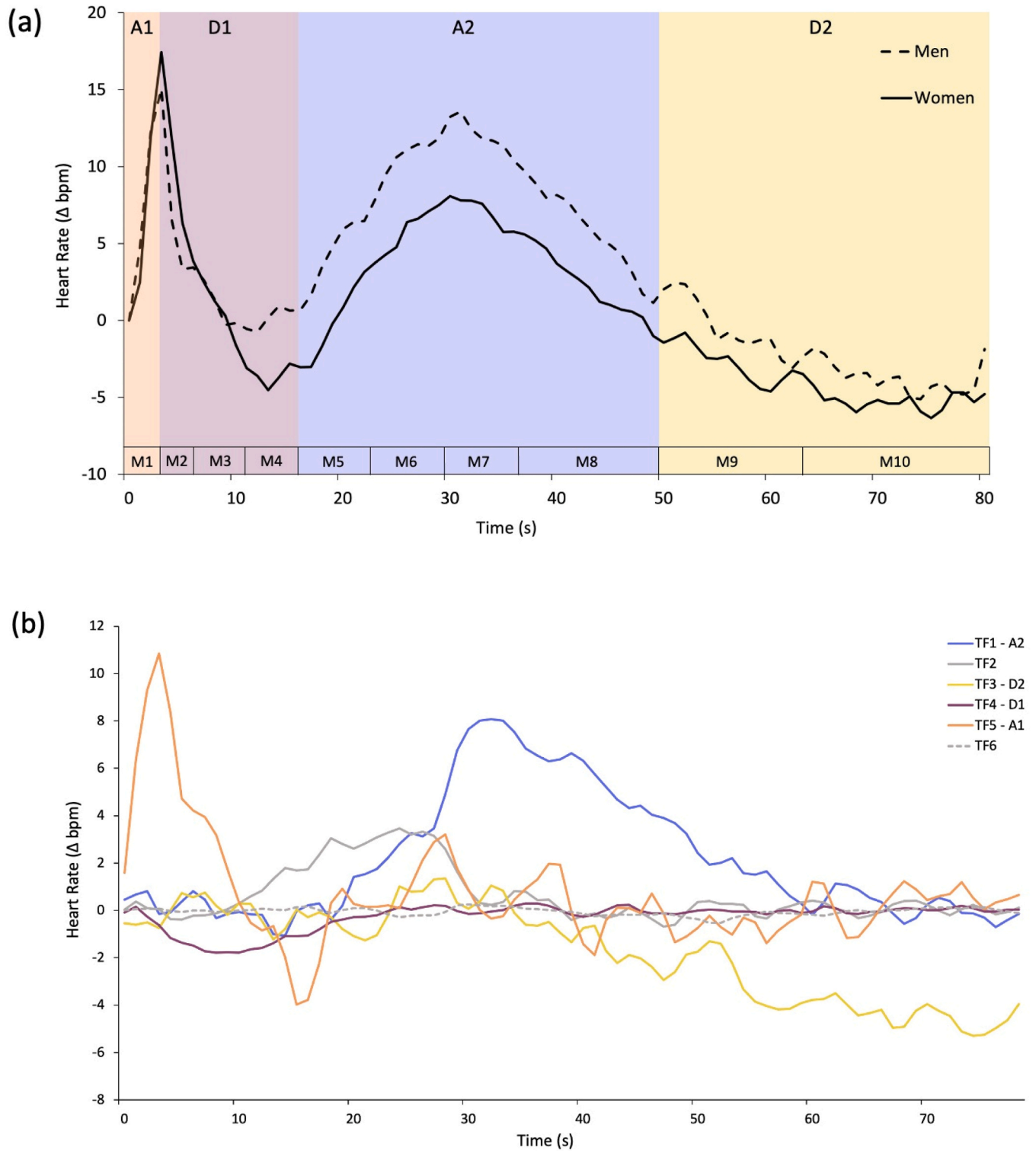

Fig. 1. Cardiac Defense Response Pattern showing the First Accelerative Component (Orange Color), the First Decelerative Component (Purple Color), the Second Accelerative Component (Blue Color), and the Second Decelerative Component (Yellow Color). (a) Heart Rate Changes Scores for Women (Solid Lines; N = 96) and Men (Dotted Lines; N = 60) separately. (b) Temporal Factors of the Principal Component Analysis corresponding to the CDR Components. Note. CDR = Cardiac Defense Response; Δ bpm = beats per minute change scores; M = Median; TF = Temporal Factor; A1 = First acceleration; D1 = First deceleration; A2 = Second acceleration; D2 = Second deceleration.

computes the covariance between time points, which tends to be higher between time points involved in the same component than the other time points. Thus, this method allows to extract and quantify each component in a more independent way from the influence of the other components. Following this, a temporal PCA was conducted to compute the covariance between the 80 second-by-second HR change scores following the presentation of the white noise, with Promax rotation and Kaiser normalization using the ERP PCA Toolkit version 2.93 (Dien, 2010). Based on Scree plot, 6 temporal factors were retained and extracted for rotation. Fig. 1b represents the six extracted components,

rescaled to HR change scores, which is achieved by multiplying the factor loadings by the factor scores for each component. The A2 was evident on the first temporal factor, D2 on the third, D1 on the fourth, and A1 on the fifth. The output of the temporal PCA —temporal factor scores— can be used as an estimate of each underlying component, and are linearly related to the original scale (i.e., second-by-second HR changes). These scores were therefore extracted for subsequent statistical analyses to examine the consistency between the two procedures to reduce the 80 second-by-second HR change scores. The CDR components obtained by PCA fitted highly with its corresponding medians (mean *r* between PCA components and its corresponding medians vs. other medians: .72 vs. .26 for A1, .84 vs. .27 for D1, .82 vs. .39 for A2, and .93 vs. .39 for D2). The temporal factors that were not selected (i.e., TF2 and TF6) showed mean *r*s $< .59$. Additionally, when appropriate, the PCA derived A2 scores were also included in correlational analyses with psychopathy traits to further explore significant effects on medians composing the second acceleration of the CDR.

2.3. Procedure

The experimental session was conducted individually in a soundproofed and dimly lit room. Before the session, participants were informed about the nature of the study and provided their written informed consent. The PPI-R was completed anonymously in different sessions of a maximum of 50 participants during the first semester of the academic year, whereas the experimental session was conducted during the second semester. This study was approved by the Ethical Committee of the University and complied with ethical principles for human research set in the Declaration of Helsinki.

2.4. Data analysis

Statistical analyses were performed using IBM SPSS Statistics 28 software. First, the CDR pattern was examined by conducting a 2 (Gender) × 10 (Median) repeated measures ANOVA. Significant effects of gender on CDR were followed up by conducting independent *t*-tests between men and women for each median.

Second, the effects of psychopathic traits on the CDR pattern were examined by including concurrently PPI-R Fearless Dominance, PPI-R Impulsive Antisociality, and PPI-R Coldheartedness scores as continuous between-subjects factors in a repeated measures general linear model (GLM) along with the discrete variables (Gender, Median) and their interactions. Secondarily, when appropriate as indicated by significant effects of Fearless Dominance or Impulsive Antisociality, we further explored the contribution of its constituent scales by conducting a 2 (Gender) × 10 (Median) repeated measures GLM in which corresponding scale scores were included as continuous between-subjects factors. To decompose significant gender interactions, analyses were conducted for men and women separately. Finally, significant PPI-R scores × Median interactions were explored in depth using Pearson's *r* correlations. Analyses yielding hypothesized significant effects of psychopathic traits on medians composing the second acceleration were further corroborated by correlational analyses with scores on the A2 component obtained by PCA. Corresponding depictions of the top versus bottom quartiles of the score distribution for significant psychopathic traits were presented to illustrate the nature of the effects. For repeated measures analyses, Greenhouse-Geisser correction was applied where appropriate.

3. Results

3.1. Cardiac defense response and gender

Table 2 presents the descriptive statistics (means and standard deviations) for each CDR median. The 2 (Gender) × 10 (Median) repeated measures ANOVA revealed a significant cubic effect of Median, $F(1154) = 158.97$, $p < .001$, $\eta_p^2 = .508$, which confirmed the presence of a typical CDR pattern with a first acceleration at M1 followed by a first deceleration reaching its minimum value at M4, and then a second acceleration with maximum peak at M7 followed by a last deceleration with a peak amplitude in M10. This pattern was consistent with previous research (e.g., Delgado et al., 2009; López et al., 2016; Ruiz-Padial et al., 2005; Sánchez et al., 2009). Analyses also revealed a Gender × Median interaction, $F(9, 1386) = 3.90$, $p = .004$, $\eta_p^2 = .025$, $\varepsilon = .43$, reflecting higher values in men than in women from M4 to M8, *t*s$(154) > 2.02$, *p*s $< .045$, *d*s > 10.63. Fig. 1a illustrates these findings.

3.2. Cardiac defense response and psychopathic traits

Table 2 presents the descriptive statistics (means and standard deviations) for each CDR median. The GLM including concurrently PPI-R Fearless Dominance, PPI-R Impulsive Antisociality, and PPI-R Coldheartedness scores revealed significant main effects of Median, $F(9, 1332) = 2.49$, $p = .008$, $\eta_p^2 = .017$, $\varepsilon = .43$, and Gender, $F(1, 148) = 5.31$, $p = .023$, $\eta_p^2 = .035$ —with men showing higher heart rate changes on average than women (3.43 vs. 1.33)—, and a significant interaction Gender × PPI-R Fearless Dominance, $F(1, 148) = 6.32$, $p = .013$, $\eta_p^2 = .041$. There were no other significant main (*F*s $< .11$; *p*s $> .747$, $\eta_p^2 < .001$) nor interaction effects (*F*s < 1.65; *p*s $> .164$, $\eta_p^2 < .011$). The effect of Gender on the PPI-R Fearless Dominance-CDR association was pursued by conducting correlational analyses for men and women separately. Higher fearless dominance scores were significantly associated with lower CDR averages across medians in women, $r(96) = -.21$, $p = .034$, with a trend in the opposite direction in men, $r(60) = .24$; $p = .062$.

In order to further explore the role of scales conforming the PPI-R

Table 2
Means and Standard Deviations for CDR Medians and the PCA Derived A2 in the Overall Sample, and for Women and Men separately.

	Overall (N = 156)		*Women (N = 96)*		*Men (N = 60)*		*Gender Comparison*	
CDR Medians	*M*	*SD*	*M*	*SD*	*M*	*SD*	*t*	*p*
M1	11.81	8.25	11.88	8.69	11.70	7.56	0.13	.897
M2	5.57	10.92	6.65	11.36	3.85	10.03	1.57	.119
M3	-.09	11.43	-.35	11.46	.34	11.48	-.37	.713
M4	-2.41	10.88	-4.04	9.95	.20	11.85	**-2.41**	.017
M5	2.17	10.87	.27	10.81	5.21	10.35	**-2.82**	.005
M6	8.41	13.61	6.68	13.95	11.17	12.68	**-2.02**	.045
M7	8.63	13.13	6.75	12.92	11.65	13.01	**-2.30**	.023
M8	2.83	11.09	1.41	10.77	5.10	11.30	**-2.04**	.043
M9	-2.47	8.01	-3.07	8.25	-1.52	7.59	-1.18	.242
M10	-5.18	7.04	-5.90	7.22	-4.04	6.66	-1.62	.108
PCA derived A2	.59	.99	.46	.96	.81	1.03	**-2.17**	.031

Note.
Significant comparisons are highlighted in bold.

Fearless Dominance factor on the CDR, a 2 (Gender) × 10 (Medians) GLM including PPI-R Fearlessness, Social Influence, and Stress Immunity scores as continuous between-subjects factors was performed. In addition to significant main effects of Median, $F(9, 1332) = 3.10$, $p = .001$, $\eta_p^2 = .021$, $\varepsilon = .44$, and Gender, $F(1, 148) = 5.47$, $p = .021$, $\eta_p^2 = .036$, analyses revealed a significant Gender × Median × PPI-R Fearlessness interaction, $F(9, 1332) = 2.94$, $p = .002$, $\eta_p^2 = .019$, $\varepsilon = .44$, with no other significant main effects or interactions, $Fs < 2.28$; $ps > .061$, $\eta_p^2 < .015$.

Effects of Gender on the Median × PPI-R Fearlessness interaction were pursued by conducting analyses for men and women separately. Both analyses revealed significant main effects of Median, $Fs > 2.83$, $ps < .027$, $\eta_p^2 = .046$; for women, a significant Median × PPI-R Fearlessness interaction was also found, $F(9, 846) = 3.10$; $p = .019$, $\eta_p^2 = .032$, $\varepsilon = .40$ ($p = .094$ in men). Follow-up correlational analyses (see Table 3) revealed significant bivariate associations between PPI-R Fearlessness scores and CDR medians from M5 to M9 only in women. To confirm that this result was not due to shared variance between scales within the fearless dominance factor, partial correlational analyses were conducted. After controlling for Stress Immunity and Social Influence scores, the association between Fearlessness scores and medians corresponding to the second accelerative component —i.e., M5 to M8— remained significant, *partial rs* (96) > −.21, *ps* < .05. Fig. 2a illustrates the nature of this finding, depicting the CDR pattern for women scoring in the upper and lower quartiles on PPI-R Fearlessness scores. Factor scores in the A2 component obtained in the PCA also correlated significantly with PPI-R Fearlessness scores in women, $r(96) = -.28$, $p < .01$ (see Fig. 2b), even after controlling for Stress Immunity and Social Influence scores (see Table 3).

4. Discussion

This is the first study to examine individual differences in the affective/interpersonal traits of psychopathy, operationalized by the PPI-R, in relation to the CDR (and particularly its second accelerative component), a measure of cue-specific defensive reactivity, in a mixed-gender undergraduate sample. The typical CDR pattern was obtained in the overall sample, with men showing greater cardiac reactivity than women in the second accelerative component (A2) of the CDR, consistent with the only study examining gender differences in this cardiac pattern (Vila et al., 1992). Regarding the association between components of psychopathy and cardiovascular reactivity, PPI-R Impulsive Antisociality and Coldheartedness scores were unrelated to CDR measures. Importantly, women scoring higher in PPI-R Fearless Dominance showed a significantly lower CDR. Analyses at the scale level revealed, more interestingly, that the hypothesized reduced A2 amplitude was related exclusively to PPI-R Fearlessness scores in women; lower order traits of psychopathy were unrelated to the CDR pattern in men. These gender-specific findings suggest a differential pattern of cardiac reactivity to dangerous physical cues as a function of trait fearlessness in women, likely mediated by the sympathetic branch of the autonomic nervous system (cf. Fernández & Vila, 1989; Garrido et al., 2020; Reyes del Paso et al., 1993, 1994; see also Vila et al., 2007), which could result in a lessened readiness for a defensive fight or flight response.

Our results seem to support dual process models of psychopathy (cf. Fowles & Dindo, 2009; Patrick & Bernat, 2009), positing that the low reactivity of the neurobiological system that modulate responses to threat is exclusively associated with the affective/interpersonal features of psychopathy (Anderson et al., 2011; Benning et al., 2005; Esteller et al., 2016; López et al., 2013; Vanman et al., 2003), and not with its externalizing traits, coming together well with prior studies which have found diminished fear learning in participants without the second accelerative component of the CDR (López et al., 2009). A novel finding of our study is that, at least in women, the fearless dominance component of psychopathy seems to be associated with a general reduction in defensive cardiac reactivity, while a diminished metabolic mobilization for active defense —as indexed by a lower A2 component— was specifically related to PPI-R Fearlessness scores, and not to scores on the other two PPI-R scales in the fearless dominance dimension, namely, Stress Immunity and Social Influence. These scales index the capacity to remain calm in pressure or anxiety-provoking situations and the ability to be engaging and skillful in influencing others, respectively, whereas PPI-R Fearlessness assesses the absence of fear when faced with physical threats and the enjoyment of engaging in risky activities (Lilienfeld & Widows, 2005). Therefore, the reduced mobilization of the organism's resources to give a defensive response to an unexpected aversive stimulus appears to be better captured by a narrower assessment of the fear/fearlessness dimension, rather than by the lack of distress in relation to threatening situations or social potency skills, which may not be as central to understanding the diminished responsivity to initial threat observed here.

In contrast to other psychophysiological measures, such as ASP, which appear to function as indicators of a broad dimension of fear/fearlessness but not of any facet in particular (see Kramer et al., 2012), the second accelerative component of the CDR appears to be more closely related to aspects of low fear to physical threats and preference to engage in risky behaviors. These results highlight the need to consider the complex interlinkages between personality dimensions and different psychophysiological measures to gain insights into their underlying mechanisms. For example, a study by Dindo & Fowles (2011) found that reduced anticipatory skin conductance (SC) responses to loud noise during the first trial of a countdown procedure were specifically related to the fearlessness dimension —but not to the social influence or stress immunity dimensions— of the PPI-R. In this regard, finding psychophysiological measures such as the A2 which appear to index a psychological attribute more specifically (e.g., fearlessness) could contribute to multi-method measurement models targeting narrower symptom facets (Patrick et al., 2019), potentially leading to more precise operationalizations of homogeneous dimensions linked to psychopathic personality, which could, in turn, help in designing more effective treatments for such problems.

In line with this, it may also be important to further consider the

Table 3
Bivariate/Partial Correlations between PPI-R Scale Scores and CDR Medians and the PCA Derived A2 in Women (N = 96) and Men (N = 60).

	Fearlessness		**Social Influence**		**Stress Immunity**	
CDR Medians	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*
M1	-.02/.01	-.24/-.24	-.00/-.01	.01/.03	-.17/-.17	.03/.05
M2	-.04/.02	.20/.18	**-.21***/**.21***	.14/.06	-.10/-.11	.21/.16
M3	-.01/.05	.20/.16	-.13/-.14	.25/.19	-.07/-.08	.17/.07
M4	-.07/-.03	.13/.09	-.16/-.16	**.26***/.23	-.09/-.10	.13/.03
M5	**-.21***/**.21***	.20/.16	-.02/.02	**.26***/.17	-.04/-.02	.25/.17
M6	**-.25***/**.25***	.11/.09	.04/.09	.02/-.04	-.08/-.05	.15/.14
M7	**-.26****/**.28****	.15/.16	.08/.13	-.01/-.03	-.08/-.04	-.01/-.01
M8	**-.31****/**.29****	-.00/-.03	-.05/.00	.15/.14	-.12/-.08	.06/.01
M9	**-.23***/-.20	-.11/-.15	-.13/-.10	.15/.11	-.11/-.09	.20/.17
M10	-.13/-.12	-.09/-.13	-.08/-.06	.21/.17	-.03/-.02	.19/.14
PCA derived A2	**-.28****/**.29****	.09/.09	.04/.09	.06/.05	-.09/-.06	.00/-.03

Note.
Significant correlations are highlighted in bold.
* $p < .05$, ** $p < .01$

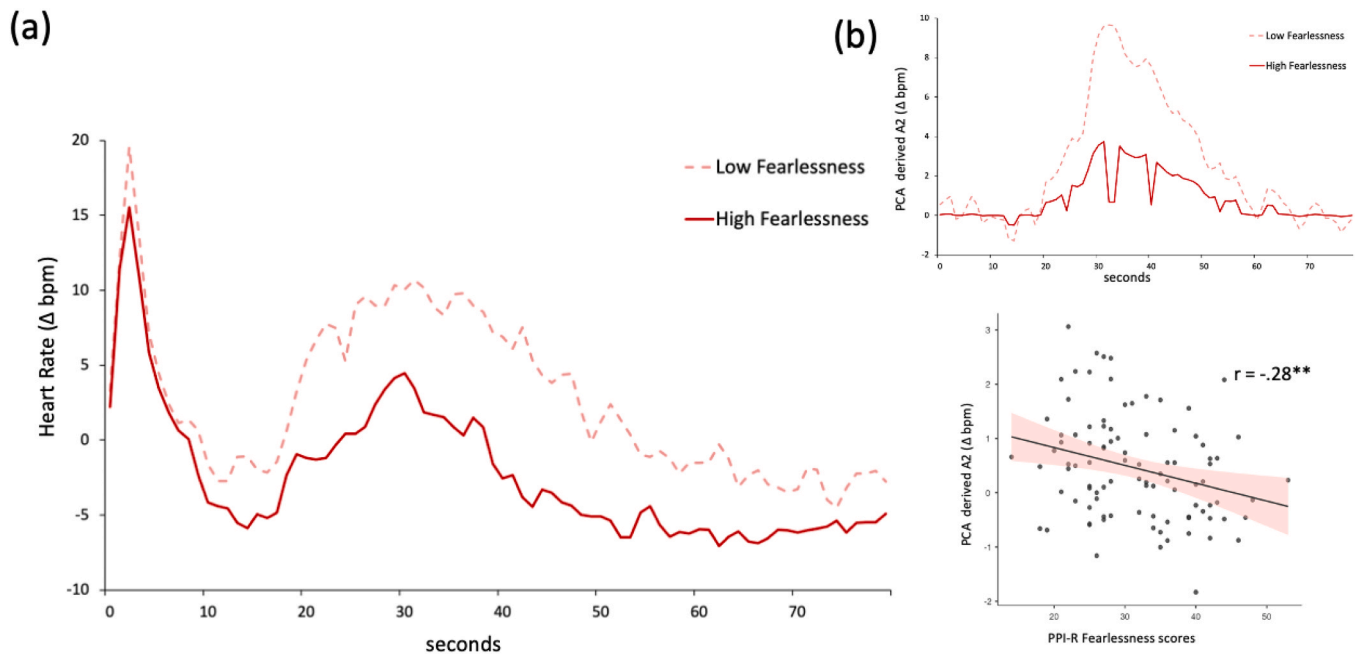

Fig. 2. Relationship between PPI-R Fearlessness and the Second Acceleration of the CDR in Women (N = 96). (a) Cardiac Defense Response Pattern in Women Classified as a Function of PPI-R Fearlessness Scores (highest and lowest quartile values). (b) Top: PCA A2 Component in Women Classified as a Function of PPI-R Fearlessness Scores (highest and lowest quartile values). Bottom: Scatterplot depicting the Correlation Between PPI-R Fearlessness scores and the PCA A2 Component in Women. Note. Δ bpm = beats per minute change scores; ** $p < .01$.

possible implications of the CDR within the context of dimensional models of psychopathology, such as the Hierarchical Taxononomy of Psychopathology (HiTOP; Kotov et al., 2017). Given that alterations in CDR patterns have been linked to problems and traits subsumed in the internalizing spectrum of HiTOP (Watson et al., 2022) —such as anxiety (López et al., 2016), chronic worry (Delgado et al., 2009), or post-traumatic stress disorders (Norte et al., 2019)— as well as to disorders more specifically included in the fear subfactor—such as specific phobias (Ruiz-Padial et al., 2002, 2005; Wannemueller et al., 2017)—, studies assessing a broader range of symptoms and traits within this spectrum —along with fearlessness tendencies— may prove useful to better disentangle the potential relevance of this physiological correlate to psychopathic traits compared to other internalizing problems and traits.

Another question that remains to be explained is why the association between fearless dominance/fearlessness and low defensive cardiac reactivity is absent in men. The positive trend-level association found between CDR and PPI-R Fearless Dominance scores would suggest elevated reactivity for men scoring higher in fearless dominance —perhaps driven by slight, non-significant positive correlations between scores on the scales conforming this factor and medians from M2 to M6 (see Table 3). This unexpected trend should be considered with caution, as it is in need of further confirmation in larger samples of men. Further, given that the evidence on individual differences in the CDR pattern has been obtained in samples composed exclusively, or mostly, of women, generalization of the results to men is uncertain. Indeed, the only study on the CDR that used a balanced mixed-gender sample (López et al., 2016) found a relationship between A2 amplitude and trait anxiety only in women. Our findings offer similar results insofar as individual differences in this defensive cardiac reflex are differentially related to gender, unlike other psychophysiological measures of threat sensitivity (e.g., Esteller et al., 2016; Kramer et al., 2012; López et al., 2013), which could be speculatively attributed to baseline differences in the neural circuitry that modulates fear responses (see Davis, 1992; Davis et al., 2010; LeDoux, 2000; Tovote et al., 2015). The central nucleus of the amygdala is the main structure that receives the inputs, but the outputs are projected to different subcortical areas to mediate specific defensive reactions. For example, while the nucleus reticularis pontis caudalis mediate startle responses, autonomic responses, such as the CDR, are mediated by the lateral hypothalamus. Although more research is needed to elucidate whether there are gender differences in the functioning of these areas that could affect the way of responding defensively, some evidence regarding connectivity have already been found. The brain regions with which the amygdala communicates under resting conditions are different in men and women, with women showing connections between a more active left amygdala and hypothalamus (for a review of gender differences in the human brain, see Zaidi, 2010). Furthermore, it is known that the cardiovascular system does not function identically in men and women, and from a clinical standpoint, these gender differences could be affecting the prevention and treatment of cardiovascular diseases. In fact, women present higher mortality related to cardiovascular disease, largely because prevention, diagnosis and treatment are based on basic research and clinical trials in male samples (Humphries et al., 2017). This fact, and also our results on gender-effects on fearlessness-related differences in A2 amplitudes, highlight the relevance of incorporating gender in empirical studies on cardiac reactivity to better understand human functioning and avoid biased conclusions.

The present study has some limitations that might constrain the generalizability of our findings and highlight directions for future research. First, we used a homogeneous undergraduate sample, so future research on larger samples of different types (clinical, criminal) and more heterogeneous in age and educational level would be necessary to examine the generalizability and robustness of our findings. Second, it would be very enlightening to complement the psychophysiological measure of defensive reactivity here employed with behavioral tests (e.g., body motor reactions; Bastos et al., 2016; Volchan et al., 2017) and/or self-report descriptions of defensive behaviors (Harrison et al., 2015) in response to various threatening scenarios. This would allow testing whether changes in the CDR pattern are accompanied by the expected behavioral changes (i.e., the way of facing danger through fight or flight responses) —which could provide further support for interpreting the A2 as an indicator of readiness for active defense—, and whether those defensive behaviors are related to individual differences

in the fearlessness trait in the same way as the A2 component of the CDR. To address this, it would be necessary to assess the convergence between the A2 component —as a potential physiological correlate of responsivity to initial threat— and other psychophysiological indicators of threat reactivity —such as ASP or anticipatory SC in countdown tasks— to work towards a more comprehensive multimodal measurement model (Patrick, Iacono, et al., 2019) of individual differences in threat sensitivity (Kramer et al., 2012; Vaidyanathan et al., 2009; Yancey et al., 2016) that can also include indicators which are relevant to narrower facets (e.g., behavioral fearlessness), and to understand their relevance to psychopathic personality. Third, in order to avoid single measure biases, it would also be useful to assess fearlessness with other available operationalizations of these tendencies, such as total and facet scales scores of the new Boldness Inventory (Patrick, Kramer et al., 2019) —the dispositional trait from the triarchic model of psychopathy (Patrick et al., 2009) most conceptually aligned with the fearless dominance component of the PPI-R— or the Thrill-Adventure Seeking subscale of the Sensation Seeking Scale (Zuckerman, 1979), to further confirm that the A2 could be considered a suitable non-report indicator of the stimulation seeking tendencies identified within structural models of dispositional threat sensitivity (Kramer et al., 2012).

Despite these limitations, our study provides preliminary evidence that the CDR acts as a general measure of reactivity to threat related to the fearless dominance dimension of psychopathy, whereas the second CDR acceleration functions as a more specific correlate of low defensive reactivity specifically associated with psychopathic fearlessness in women, highlighting a gender-specific differential defense cardiac reactivity involving fear/fearlessness traits. This result underscores the potential use of the A2 to better understand the physiological aspects of psychopathic fearlessness tendencies and its differing manifestations across genders.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Declaration of generative AI and AI-assisted technologies in the writing process

The authors did not use generative AI technologies for preparation of this work.

References

Anderson, N. E., Stanford, M. S., Wan, L., & Young, K. A. (2011). High psychopathic trait females exhibit reduced startle potentiation and increased P3 amplitude. *Behavioral Sciences & the Law, 29*(5), 649–666. https://doi.org/10.1002/bsl.998

Bastos, A. F., Vieira, A. S., Oliveira, J. M., Oliveira, L., Pereira, M. G., Figueira, I., Erthal, F. S., & Volchan, E. (2016). Stop or move: Defensive strategies in humans. *Behavioural Brain Research, 302*, 252–262. https://doi.org/10.1016/j.bbr.2016.01.043

Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D. M., & Krueger, R. F. (2003). Factor structure of the Psychopathic Personality Inventory: Validity and implications for clinical assessment. *Psychological Assessment, 15*, 340–350. https://doi.org/10.1037/1040-3590.15.3.340

Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology, 42*(6), 753–762. https://doi.org/10.1111/j.1469-8986.2005.00353.x

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion, 1*(3), 276. https://doi.org/10.1037/1528-3542.1.3.276

Bradley, M. M., & Lang, P. J. (2000). Measuring emotion: Behavior, feeling, and physiology. In R. Lane, & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 242–276). New York: Oxford University Press.

Casey, H., Rogers, R. D., Burns, T., & Yiend, J. (2013). Emotion regulation in psychopathy. *Biological Psychology, 92*(3), 541–548. https://doi.org/10.1016/j.biopsycho.2012.06.011

Cleckley, H. (1976). The mask of sanity. St. Louis, MO: Mosby.

Cook, E.W., III (2002). VPM reference manual. Birmingham, AL: Author.

Cuthbert, B. N., Lang, P. J., Strauss, C., Drobes, D., Patrick, C. J., & Bradley, M. M. (2003). The psychophysiology of anxiety disorder: Fear memory imagery. *Psychophysiology, 40*(3), 407–422. https://doi.org/10.1111/1469-8986.00043

Davis, M. (1992). The role of the amygdala in fear-potentiated startle: implications for animal models of anxiety. *Trends in Pharmacological Sciences, 13*, 35–41. https://doi.org/10.1016/0165-6147(92)90014-W

Davis, M., Walker, D. L., Miles, L., & Grillon, C. (2010). Phasic vs sustained fear in rats and humans: Role of the extended amygdala in fear vs anxiety. *Neuropsychopharmacology, 35*(1), 105–135. https://doi.org/10.1038/npp.2009.109

Delgado, L. C., Guerra, P., Perakakis, P., Mata, J. L., Pérez, M. N., & Vila, J. (2009). Psychophysiological correlates of chronic worry: Cued versus non-cued fear reaction. *International Journal of Psychophysiology, 74*(3), 280–287. https://doi.org/10.1016/j.ijpsycho.2009.10.007

Dien, J. (2010). The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. *Journal of Neuroscience Methods, 187*(1), 138–145. https://doi.org/10.1016/j.jneumeth.2009.12.009

Dien, J. (2012). Applying principal components analysis to event-related potentials: A tutorial. *Developmental Neuropsychology, 37*(6), 497–517. https://doi.org/10.1080/87565641.2012.697503

Dindo, L., & Fowles, D. (2011). Dual temperamental risk factors for psychopathic personality: evidence from self-report and skin conductance. *Journal of personality and Social Psychology, 100*(3), 557. https://doi.org/10.1037/a0021848

Esteller, À., Poy, R., & Moltó, J. (2016). Deficient aversive-potentiated startle and the triarchic model of psychopathy: The role of boldness. *Biological Psychology, 117*, 131–140. https://doi.org/10.1016/j.biopsycho.2016.03.012

Eves, F. F., & Gruzelier, J. H. (1984). Individual differences in the cardiac response to high intensity auditory stimulation. *Psychophysiology, 21*(3), 342–352. https://doi.org/10.1111/j.1469-8986.1984.tb02946.x

Fernández, M. C., & Vila, J. (1989). Sympathetic-parasympathetic mediation of the cardiac defense response in humans. *Biological Psychology, 28*(2), 123–133. https://doi.org/10.1016/0301-0511(89)90094-X

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/10.1017/S0048577202394046

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

Garrido, A., Duschek, S., Árbol, J. R., Usera, I. G., Vila, J., & Mata, J. L. (2020). Sympathetic contributions to habituation and recovery of the cardiac defense response. *Biological Psychology, 151*, Article 107846. https://doi.org/10.1016/j.biopsycho.2020.107846

Hare, R.D. (2003). Hare Psychopathy Checklist Revised (2nd edition). Toronto: Multi-Health Systems.

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

Harrison, L. A., Ahn, C., & Adolphs, R. (2015). Exploring the structure of human defensive responses from judgments of threat scenarios. *PLoS One, 10*(8), Article e0133682. https://doi.org/10.1371/journal.pone.0133682

Humphries, K. H., Izadnegahdar, M., Sedlak, T., Saw, J., Johnston, N., Schenck-Gustafsson, K., Shah, R. U., Regitz-Zagrosek, V., Grewal, J., Vaccarino, V., Wei, J., & Merz, C. B. (2017). Sex differences in cardiovascular disease–impact on care and outcomes. *Frontiers in Neuroendocrinology, 46*, 46–70. https://doi.org/10.1016/j.yfrne.2017.04.001

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., & Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology, 126*(4), 454. https://doi.org/10.1037/abn0000258

Kramer, M. D., Patrick, C. J., Krueger, R. F., & Gasperi, M. (2012). Delineating physiologic defensive reactivity in the domain of self-report: Phenotypic and etiologic structure of dispositional fear. *Psychological Medicine, 42*(6), 1305–1320. https://doi.org/10.1017/S0033291711002194

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. *Attention and Orienting: Sensory and Motivational Processes, 97*, 135.

Lang, P. J., & McTeague, L. M. (2009). The anxiety disorder spectrum: Fear imagery, physiological reactivity, and differential diagnosis. *Anxiety, Stress, & Coping, 22*(1), 5–25. https://doi.org/10.1080/10615800802478247

Lilienfeld, S.O., & Widows, M.R. (2005). Psychopathic Personality Inventory-Revised (PPI-R) professional manual. Psychological Assessment Resources.

de Looff, P. C., Cornet, L. J., De Kogel, C. H., Fernández-Castilla, B., Embregts, P. J., Didden, R., & Nijman, H. L. (2022). Heart rate and skin conductance associations with physical aggression, psychopathy, antisocial personality disorder and conduct disorder: An updated meta-analysis. *Neuroscience & Biobehavioral Reviews*. https://doi.org/10.1016/j.neubiorev.2021.11.003

LeDoux, J. (2000). Listen to the brain. In R. D. Lane, & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 129–155). Oxford University Press.

López, R., Poy, R., Pastor, M. C., Segarra, P., & Moltó, J. (2009). Cardiac defense response as a predictor of fear learning. *International Journal of Psychophysiology, 74*(3), 229–235. https://doi.org/10.1016/j.ijpsycho.2009.09.006

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

López, R., Poy, R., Segarra, P., Esteller, À., Fonfría, A., Ribes, P., Ventura, C., & Moltó, J. (2016). Gender-specific effects of trait anxiety on the cardiac defense response. *Personality and Individual Differences, 96*, 243–247. https://doi.org/10.1016/j.paid.2016.03.014

Marcus, D. K., Fulton, J. J., & Edens, J. F. (2013). The two-factor model of psychopathic personality: Evidence from the Psychopathic Personality Inventory. *Personality Disorders: Theory, Research, and Treatment, 4*(1), 67–76. https://doi.org/10.1037/a0025282

Mata, J. L., Rodríguez-Ruiz, S., Ruiz-Padial, E., Turpin, G., & Vila, J. (2009). Habituation and sensitization of protective reflexes: Dissociation between cardiac defense and eye-blink startle. *Biological Psychology, 81*(3), 192–199. https://doi.org/10.1016/j.biopsycho.2009.04.006

Miller, J. D., & Lynam, D. R. (2012). An examination of the Psychopathic Personality Inventory's nomological network: A meta-analytic review. *Personality Disorders: Theory, Research, and Treatment, 3*(3), 305. https://doi.org/10.1037/a0024567

Nelson, L. D., Strickland, C., Krueger, R. F., Arbisi, P. A., & Patrick, C. J. (2016). Neurobehavioral traits as transdiagnostic predictors of clinical problems. *Assessment, 23*(1), 75–85. https://doi.org/10.1177/1073191115570110

Norte, C. E., Volchan, E., Vila, J., Mata, J. L., Arbol, J. R., Mendlowicz, M., Berger, W., Pires, M., Rocha-Rego, V., Figueira, I., & Guerra, G. (2019). Tonic immobility in PTSD: exacerbation of emotional cardiac defense response. *Frontiers in Psychology, 10*, 1213. https://doi.org/10.3389/fpsyg.2019.01213

Oskarsson, S., Patrick, C. J., Siponen, R., Bertoldi, B. M., Evans, B., & Tuvblad, C. (2021). The startle reflex as an indicator of psychopathic personality from childhood to adulthood: A systematic review. *Acta Psychologica, 220*, Article 103427. https://doi.org/10.1016/j.actpsy.2021.103427

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* (pp. *1110–1131*). John Wiley & Sons. https://doi.org/10.1002/9780470478509.neubb002057.

Patrick, C. J., 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. J., Iacono, W. G., & Venables, N. C. (2019). Incorporating neurophysiological measures into clinical assessments: Fundamental challenges and a strategy for addressing them. *Psychological Assessment, 31*(12), 1512. https://doi.org/10.1037/pas0000713

Patrick, C. J., Kramer, M. D., Vaidyanathan, U., Benning, S. D., Hicks, B. M., & Lilienfeld, S. O. (2019). Formulation of a measurement model for the boldness construct of psychopathy. *Psychological Assessment, 31*(5), 643. https://doi.org/10.1037/pas0000690

Ramírez, I., Sánchez, M. B., Fernández, M. C., Lipp, O. V., & Vila, J. (2005). Differentiation between protective reflexes: Cardiac defense and startle. *Psychophysiology, 42*(6), 732–739. https://doi.org/10.1111/j.1469-8986.2005.00362.x

Reyes del Paso, G. A. R., Godoy, J., & Vila, J. (1993). Respiratory sinus arrhythmia as an index of parasympathetic cardiac control during the cardiac defense response. *Biological Psychology, 35*(1), 17–35. https://doi.org/10.1016/0301-0511(93)90089-Q

Reyes del Paso, G. A. R., Vila, J., & García, A. (1994). Physiological significance of the defense response to intense auditory stimulation: A pharmacological blockade study. *International Journal of Psychophysiology, 17*(2), 181–187. https://doi.org/10.1016/0167-8760(94)90034-5

Richards, M., & Eves, F. F. (1991). Personality, temperament and the cardiac defense response. *Personality and Individual Differences, 12*(10), 999–1007. https://doi.org/10.1016/0191-8869(91)90030-F

Ruiz-Padial, E., Mata, J. L., Rodríguez, S., Fernández, M. C., & Vila, J. (2005). Non-conscious modulation of cardiac defense by masked phobic pictures. *International Journal of Psychophysiology, 56*(3), 271–281. https://doi.org/10.1016/j.ijpsycho.2004.12.010

Ruiz-Padial, E. R., Sánchez, M. B., Thayer, J. F., & Vila, J. (2002). Modulación no consciente de la respuesta cardiaca de defensa por imágenes fóbicas [Non-conscious modulation of the cardiac defense response by phobic pictures]. *Psicothema, 14*(4), 739–745.

Sánchez, M. B., Guerra, P., Muñoz, M. A., Mata, J. L., Bradley, M. M., Lang, P. J., & Vila, J. (2009). Communalities and differences in fear potentiation between cardiac defense and eyeblink startle. *Psychophysiology, 46*(6), 1137–1140. https://doi.org/10.1111/j.1469-8986.2009.00861.x

Sánchez, M. B., Ruiz-Padial, E., Pérez, N., Fernández, M. C., Cobos, P., & Vila, J. (2002). Modulación emocional de los reflejos defensivos mediante visualización de imágenes afectivas [Emotional modulation of defensive reflexes by viewing affective pictures]. *Psicothema, 14*(4), 702–707.

Schalinski, I., Elbert, T. R., & Schauer, M. (2013). Cardiac defense in response to imminent threat in women with multiple trauma and severe PTSD. *Psychophysiology, 50*(7), 691–700. https://doi.org/10.1111/psyp.12051

Tovote, P., Fadok, J. P., & Lüthi, A. (2015). Neuronal circuits for fear and anxiety. *Nature Reviews Neuroscience, 16*(6), 317–331. https://doi.org/10.1038/nrn3945

Turpin, G. (1986). Effects of stimulus intensity on autonomic responding: The problem of differentiating orienting and defense reflexes. *Psychophysiology, 23*(1), 1–14. https://doi.org/10.1111/j.1469-8986.1986.tb00583.x

Vaidyanathan, U., Patrick, C. J., & Bernat, E. M. (2009). Startle reflex potentiation during aversive picture viewing as an indicator of trait fear. *Psychophysiology, 46*(1), 75–85. https://doi.org/10.1111/j.1469-8986.2008.00751.x

Vanman, E. J., Mejia, V. Y., Dawson, M. E., Schell, A. M., & Raine, A. (2003). Modification of the startle reflex in a community sample: Do one or two dimensions of psychopathy underlie emotional processing. *Personality and Individual Differences, 35*(8), 2007–2021. https://doi.org/10.1016/S0191-8869(03)00052-7

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. https://doi.org/10.1037/abn0000032

Vila, J., & Beech, H. R. (1978). Vulnerability and defensive reactions in relation to the human menstrual cycle. *British Journal of Social and Clinical Psychology, 17*, 93–100.

Vila, J., Fernández, M. C., & Godoy, J. (1992). The cardiac defense response in humans: Effects of stimulus modality and gender differences. *Journal of Psychophysiology, 6*, 140–154.

Vila, J., Guerra, P., Muñoz, M.Á., Vico, C., Viedma-del Jesús, M. I., Delgado, L. C., Perakakis, P., Kley, E., Mata, J. L., & Rodríguez, S. (2007). Cardiac defense: From attention to action. *International Journal of Psychophysiology, 66*(3), 169–182. https://doi.org/10.1016/j.ijpsycho.2007.07.004

Volchan, E., Rocha-Rego, V., Bastos, A. F., Oliveira, J. M., Franklin, C., Gleiser, S., Berger, W., Souza, G. C. L., Oliveira, L., David, I. A., Erthal, F. S., Pereira, M. G., & Figueira, I. (2017). Immobility reactions under threat: a contribution to human defensive cascade and PTSD. *Neuroscience & Biobehavioral Reviews, 76*, 29–38. https://doi.org/10.1016/j.neubiorev.2017.01.025

Wannemueller, A., Adolph, D., Joehren, H. P., Blackwell, S. E., & Margraf, J. (2017). Psychophysiological reactivity of currently dental phobic-, remitted dental phobic- and never-dental phobic individuals during exposure to dental-related and other affect-inducing materials. *Behaviour Research and Therapy, 90*, 76–86. https://doi.org/10.1016/j.brat.2016.12.009

Watson, D., Levin-Aspenson, H. F., Waszczuk, M. A., Conway, C. C., Dalgleish, T., Dretsch, M. N., Eaton, N. R., Forbes, M. K., Forbush, K. T., Hobbs, K. A., Michelini, G., Nelson, B. D., Sellbom, M., Slade, T., South, S., Sunderland, M., Waldman, I., Witthöft, M., Wright, A. G. C., Kotow, R., & Krueger, R. F. (2022). Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): III. Emotional dysfunction superspectrum. *World Psychiatry, 21*(1), 26–54. https://doi.org/10.1002/wps.20943

Yancey, J. R., Venables, N. C., & Patrick, C. J. (2016). Psychoneurometric operationalization of threat sensitivity: Relations with clinical symptom and physiological response criteria. *Psychophysiology, 53*(3), 393–405. https://doi.org/10.1111/psyp.12512

Zaidi, Z. F. (2010). Gender differences in human brain: A review. *The Open Anatomy Journal, 2*(1). https://doi.org/0.2174/1877609401002010037.

Zuckerman, M. (1979). Sensation seeking: Beyond the optimal level of arousal. Erlbaum.