DOCTORAL THESIS

Validation of the triarchic model of psychopathy:

Physiological indicators for threat sensitivity, affiliative capacity, and inhibitory control

Victoria Branchadell Capdevila

Supervisors: Francisco Javier Moltó Brotons and Rosario Poy Gil

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Validación del modelo triárquico de la psicopatía: indicadores fisiológicos para la sensibilidad a la amenaza, capacidad afiliativa y control inhibitorio

Memoria presentada por Victoria Branchadell Capdevila para optar al grado de doctor/a por la Universitat Jaume I

VICTORIA| BRANCHADELL| CAPDEVILA
Firmado digitalmente por VICTORIA| BRANCHADELL| CAPDEVILA
Fecha: 2023.09.13 20:45:15 +02'00'

Victoria Branchadell Capdevila

FRANCISCO JAVIER| MOLTO| BROTONS
Firmado digitalmente por FRANCISCO JAVIER| MOLTO|BROTONS
Fecha: 2023.09.14 09:09:45 +02'00'

Francisco Javier Moltó Brotons

ROSARIO |POY|GIL
Firmado digitalmente por ROSARIO|POY|GIL
Fecha: 2023.09.14 09:31:18 +02'00'

Rosario Poy Gil

Castelló de la Plana, julio de 2023

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List of abbreviations

A1: First Accelerative Component of the Cardiac Defense Response

A2: Second Accelerative Component of the Cardiac Defense Response

ADHD: Attention-Deficit/Hyperactivity Disorder

ADS: Alcohol Dependence Scale

AMP: Alternative Model for Personality Disorders

APA: American Psychological Association

APSD: Antisocial Process Screening Device

ASP: Aversive Startle Potentiation

ATI: Adult Temperament Inventory

AUDIT: Alcohol Use Disorders Identification Test

BHR: Behavior Report on Rule-Breaking

BMI: Body Mass Index

CDR: Cardiac Defense Response

CID-SAM: Composite International Diagnostic Instrument–Substance Abuse Module

CS+/CS-: Excitatory Conditioned Stimulus/Inhibitory Conditioned Stimulus

CU: Callous-Unemotional

D1: First Decelerative Component of the Cardiac Defense Response

D2: Second Decelerative Component of the Cardiac Defense Response

DAST-A: Drug Abuse Screening Test

DSM: Diagnostic and Statistical Manual of Mental Disorders

ECG: Electrocardiogram

EDR: ECG-Derived Respiration

EEG: Electroencephalogram/Electroencephalography

EFA: Exploratory Factor Analysis

ERN: Error-Related Negativity

ERP: Event-Related Potential

ESI: Externalizing Spectrum Inventory

ESI-100: Externalizing Spectrum Inventory 100-item version

ESI-BF: Externalizing Spectrum Inventory–Brief Form

ESI-CA: Externalizing Spectrum Inventory–Callous Aggression

ESI-DIS: Externalizing Spectrum Inventory–Disinhibition Scale

FD: Fearless Dominance Factor of the Psychopathic Personality Inventory

FFM: Five Factor Model

FFMRF: Five-Factor Model Rating Form

fMRI: Functional Magnetic Resonance Imaging

HF-HRV: High frequency band of HRV

HiTOP: Hierarchical Taxonomy of Psychopathology

HR: Heart Rate

HRV: Heart Rate Variability

ICU: Inventory of Callous-Unemotional Traits

IES: Integrated Emotion System

IPI-NEO-120: International Personality Item Pool–Neuroticism, Extraversion and Openness

ITI: Inter-trial Interval

LPP: Late Positive Potential

LSRP: Levenson Self-Report Psychopathy Scale

M1-M10: Values corresponding to the medians of 10 intervals in the CDR pattern

MDOs: Mentally Disordered Offenders

Mini-IPIP: International Personality Item Pool—Short Form

MMPI-Tri: Minnesota Multiphasic Personality Inventory Triarchic Scales

MPQ: Multidimensional Personality Questionnaire

MPQ-BF: Brief Form of the Multidimensional Personality Questionnaire

MPQ-DIS: Multidimensional Personality Questionnaire Disinhibition Scale

MPQ-Tri: Multidimensional Personality Questionnaire Triarchic Scales

N170: N170 ERP Component

N2: N2 ERP Component

NEO-FFI: NEO Five-Factor Inventory

NEO PI-R: NEO Personality Inventory Revised

NEO-Tri: NEO Personality Inventory Triarchic Scales

P200: P200 ERP Component

P3: P3 ERP Component

PAI: Personality Assessment Inventory

PCA: Principal Component Analysis

PCL: Hare Psychopathy Checklist

PCL-R: Hare Psychopathy Checklist–Revised

PCL:SV: Psychopathy Checklist: Screening Version

PCL:YV: Psychopathic Checklist: Youth Version

Pe: Error Positivity

PID-5-Tri: Personality Inventory for DSM-5 Triarchic Scales

PPI: Psychopathic Personality Inventory

PPI-DIS: Psychopathic Personality Inventory Disinhibition

PPI-R: Psychopathic Personality Inventory–Revised

PPI-SF: Psychopathic Personality Inventory–Short Form

PPI-Tri: Psychopathic Personality Inventory Triarchic Scales

RT: Reaction Time

SART: Sustained Attention to Response Task

SC: Skin Conductance

SCI: Self-Centered Impulsivity Factor of the Psychopathic Personality Inventory

SCR: Skin Conductance Reactivity

SDAST: Short Drug Abuse Screening Test

SDQ: Strengths and Difficulties Questionnaire

SNAP-F-Tri: Schedule for Nonadaptive and Adaptive Personality Forensic Version Triarchic Scales

SO: Socialization Scale

SSS-V: Sensation-Seeking Scale V

SSRT: Stop Signal Reaction Time

SRP-III: Self-Report Psychopathy Scale

SRP-4: Self-Report Psychopathy Scale 4

SRP-SF: Self-Report Psychopathy Scale, Short Form

TF: Trait Fear Inventory

TF#SF#: Temporal Factor (number) Spatial Factor (number)

THT: Cross-Domain Index of Threat Sensitivity

TPQ: Cloninger's Tridimensional Personality Questionnaire–Short Form

TriPM: Triarchic Psychopathy Measure

tsPCA: Temporospatial Principal Component Analysis

VIM: Violence Inhibition Model

vmHRV: Resting Vagally-Mediated Heart Rate Variability

YPI-Tri: Youth Psychopathic Traits Inventory Triarchic Scales

Extended summary

Psychopathy is a multifaceted personality disorder that is characterized by distinctive affective and interpersonal traits manifested in a context of behavioral deviance. The triarchic model of psychopathy has been proposed with the purpose of integrating different theoretical perspectives and serving as a framework for neurobiological research on this disorder.

According to this model, psychopathy is defined in terms of three distinct but interrelated trait constructs: *boldness*, which encompasses characteristics such as high social dominance, high self-esteem, or the ability to remain calm under threatening situations; *meanness*, which entails attributes such as lack of empathy, disdain toward others, absence of close personal attachments, or aggressive manipulation of others; and *disinhibition*, which includes impulsivity, irritability, irresponsibility, and difficulty in controlling impulses. These trait constructs are formulated in biobehavioral terms, using self-report measures as provisional referents to identify distinct indicators from other measurement domains (behavioral, physiological) and thus conform comprehensive nomological networks that contribute to a better understanding of each triarchic disposition.

Hence, the boldness trait construct is related to the biobehavioral dimension of *threat sensitivity*, which is presumed to reflect individual differences in the brain's defensive system reactivity. It has been associated with several physiological indicators of low fear (e.g., reduced aversive startle potentiation) but also with indicators of good adjustment (e.g., better task performance under threatening conditions), which suggests a more adaptive aspect of this dimension. The meanness trait construct corresponds to the biobehavioral dimension of *affiliative capacity*. Previous research has documented the association between

meanness/callous-unemotional traits and deficits in the recognition of emotional facial expressions (especially fear), as well as a reduced brain reactivity to them. It has recently been proposed that the brain's pain network could play a key role in the empathic deficits that characterize this dimension, reflected in a reduced brain reactivity to pictures of others in situations of pain or distress. Finally, *inhibitory control*, the biobehavioral disposition corresponding to the disinhibition trait construct, has well-known behavioral and neurophysiological correlates. One of the most studied of these correlates is the reduced amplitude of the P3 component of event-related potentials (ERPs), which is presumed to reflect deficits in the elaborative processing of meaningful stimuli. In addition, recent research on response-related components of ERPs also suggests an association between trait disinhibition and deficits in error-processing.

The general objective of this thesis is to provide evidence for the biobehavioral dispositions underlying the trait constructs of the triarchic model of psychopathy by identifying new physiological correlates for each one. Thus, for threat sensitivity, the aim was twofold: first, to explore the association of boldness with resting vagally-mediated heart rate variability (vmHRV), as a physiological indicator of emotional self-regulation (*Study 1*); second, to investigate the relationship between boldness and the Cardiac Defense Response (CDR), and specifically its second accelerative component (A2), indexing metabolic mobilization of the defensive motivational system (*Study 2*). For affiliative capacity, we examined the relationship between meanness/callousness traits and electrocortical processing of pain, depending on the adopted perspective (self vs. other), through the Late Positive Potential (LPP) of ERPs, as an indicator of sustained attention to relevant stimuli (*Study 3*). Finally, for inhibitory control (*Study 4*), the aim was to explore the relationship between the triarchic disposition of disinhibition and electrocortical

indicators from a modified flanker-stop-signal task indexing inhibitory processing —Stop P3— and error monitoring —Flanker and Stop ERNs (Error-Related Negativity).

A positive relationship between boldness and vmHRV was found in *Study 1*, suggesting that suitable emotional self-regulation is one of the adaptive traits conforming boldness, and supporting the potential use of the vmHRV as a new physiological marker of this trait construct. In *Study 2*, boldness —measured by the Fearless Dominance factor (FD) from the Psychopathic Personality Inventory-Revised (PPI-R)— was inversely related to the average change in heart rate throughout the CDR in women, whereas the specific traits of low fear (assessed by the Fearlessness scale, one of the three scales conforming the FD) showed the same pattern in relation to the A2 amplitude. These results suggest that boldness is associated with an overall reduced cardiac reactivity to unexpected threat, whereas the specific reduced metabolic mobilization for an active defense (indexed by the A2) would be linked to a more specific assessment of the fear/fearlessness dimension, at least in women. *Study 3* demonstrated that callousness is associated with reduced amplitudes of the LPP —a well-validated electrophysiological indicator of sustained attention to emotionally relevant stimuli— while viewing pictures of pain under the instruction to imagine that the person in the picture was someone unknown, but not under the instruction to imagine that the person in the picture was oneself. These results suggest reduced brain reactivity to others' distress in higher callous individuals, encouraging the usefulness of electrocortical studies on pain processing to better characterize the empathic deficits associated with psychopathic meanness from a neurobiological perspective. Finally, *Study 4* found that disinhibition was related to reduced amplitudes of both ERN variants (i.e., stop and flanker), but not of the Stop P3 component. These results suggest that deficits in inhibitory control associated with disinhibition are located in early stages of error monitoring, rather than in later stages of elaborative processing of relevant stimuli.

In conclusion, this thesis has made possible the identification of different physiological correlates that contribute to advance knowledge about the distinct biobehavioral dispositions underlying the trait constructs of the triarchic model of psychopathy. The relationship of boldness with higher vmHRV and lower CDR (and A2) demonstrates, on the one hand, and in a novel way, that low threat sensitivity is associated with adequate emotional self-regulation, and, on the other hand, with lower metabolic mobilization of the defensive motivational system, thus providing a new physiological indicator to those already established on responses to threat. The relationship between meanness and lower LPP amplitudes to pain in others suggests that low affiliative capacity is associated with blunted reactivity to others' distress (but not to one's distress), probably pointing to the lower relevance of these stimuli for higher callous individuals, which is a new contribution to the study of empathic deficits in psychopathy. Finally, the relationship between disinhibition and lower flanker and stop ERN amplitudes suggests that low inhibitory control is associated with deficits in early error monitoring (both interference and inhibition) in a complex task, adding to previous results new evidence of individual differences in error processing (regardless of type) in relation to disinhibition. Thus, this research work aims to contribute to conform more comprehensive nomological networks for each triarchic disposition and to improve our understanding of this multifaceted personality disorder from a biobehavioral perspective.

Resumen ampliado

La psicopatía es un trastorno multifacético de la personalidad caracterizado por distintos rasgos afectivos e interpersonales que se manifiestan en un contexto de desviación conductual. El modelo triárquico de la psicopatía ha sido propuesto con el propósito de integrar distintas perspectivas teóricas y servir como marco de referencia para la investigación neurobiológica de este trastorno.

Según este modelo, la psicopatía se define en términos de tres constructos de rasgo distintos pero interrelacionados: audacia (*boldness*), que engloba características como la alta dominancia social, la elevada autoestima o la capacidad para mantener la calma en situaciones amenazantes; maldad (*meanness*), que comprende atributos como la falta de empatía, el desprecio hacia los demás, la ausencia de vínculos estrechos con otras personas, o la manipulación agresiva de otros; y desinhibición (*disinhibition*), que incluye la impulsividad, la irritabilidad, la irresponsabilidad, y la dificultad para controlar los impulsos. Estos constructos de rasgo se formulan en términos bioconductuales, utilizando medidas de autoinforme como referencia provisional para identificar indicadores en otros dominios de medida (conductual, fisiológico) y así establecer redes nomológicas completas que contribuyan a mejorar la comprensión de cada disposición triárquica.

Así, el rasgo disposicional de audacia se relaciona con la dimensión bioconductual de *sensibilidad a la amenaza*, que se supone refleja diferencias individuales en la reactividad del sistema defensivo del cerebro. Ha sido asociada con varios indicadores fisiológicos de bajo miedo (por ejemplo, una reducida potenciación aversiva del reflejo de sobresalto) pero también con indicadores de buen ajuste (por ejemplo, un mejor rendimiento conductual bajo condiciones amenazantes), lo que sugiere una parte más adaptativa de esta dimensión. Al rasgo disposicional de maldad le corresponde la dimensión bioconductual de *capacidad*

afiliativa. Investigaciones previas han documentado la relación entre los rasgos de maldad y crueldad/falta de emocionalidad y los déficits en el reconocimiento de expresiones faciales emocionales (especialmente de miedo), así como una reducida reactividad cerebral ante las mismas. Recientemente se ha propuesto que la red cerebral del dolor podría desempeñar un papel fundamental en los déficits empáticos que caracterizan esta dimensión, manifestándose en una reducida reactividad cerebral ante imágenes de otros en situaciones de dolor o malestar. Por último, el *control inhibitorio*, la disposición bioconductual correspondiente al rasgo disposicional de desinhibición, presenta correlatos conductuales y neurofisiológicos bien conocidos. Uno de los más estudiados es la reducida amplitud del componente P3 de los potenciales evocados (ERPs), que se supone refleja déficits en el procesamiento elaborativo de estímulos significativos. Además, investigaciones recientes sobre componentes de los ERPs relacionados con la respuesta también sugieren la existencia de una asociación entre el rasgo de desinhibición y alteraciones en la monitorización de errores.

El objetivo general de esta tesis es aportar evidencia sobre las disposiciones bioconductuales que subyacen a los constructos de rasgo del modelo triárquico de la psicopatía mediante la identificación de nuevos correlatos fisiológicos para cada uno de ellos. Así, para la *sensibilidad a la amenaza* se planteó un doble objetivo: primero, explorar la relación entre la audacia y la variabilidad de la tasa cardíaca en reposo mediada vagalmente (vmHRV), como un indicador fisiológico de la autorregulación emocional (*Estudio 1*); segundo, investigar la relación entre la audacia y la Respuesta Cardíaca de Defensa (RCD), y especialmente su segundo componente acelerativo (A2), que refleja la movilización metabólica del sistema motivacional defensivo (*Estudio 2*). Para la *capacidad afiliativa*, se planteó examinar la relación entre los rasgos de maldad/crueldad y el procesamiento electrocortical del dolor, en función de la perspectiva adoptada (uno mismo

vs. otro), a través del Potencial Positivo Tardío (LPP) de los ERPs, como indicador de la atención sostenida hacia estímulos relevantes (*Estudio 3*). Por último, para el *control inhibitorio*, el objetivo fue explorar la relación entre la disposición triárquica de desinhibición y los indicadores electrocorticales en una tarea modificada de flancos con señal de *stop* que indexan el procesamiento inhibitorio —*Stop* P3— y la monitorización de errores —*Flanker* y *Stop* ERNs (Negatividad Relacionada con el Error).

En el *Estudio 1* se encontró una relación positiva entre la audacia y la vmHRV, lo que sugiere que la adecuada autorregulación emocional es uno de los rasgos adaptativos que conforman la audacia, y respalda el uso potencial de la vmHRV como un nuevo marcador fisiológico de este rasgo disposicional. En el *Estudio 2*, la audacia —medida por el factor de *Fearless Dominance* (FD) del *Psychopathic Personality Inventory-Revised* (PPI-R)— se relacionó inversamente con el cambio promedio en la tasa cardiaca a lo largo de toda la CDR en las mujeres, mientras que sus características específicas de bajo miedo (evaluadas mediante la escala *Fearlessness*, una de las tres que conforman el factor FD) hicieron lo propio con la amplitud del componente A2. Estos resultados sugieren que la audacia está asociada con una reactividad cardíaca atenuada en general ante la amenaza inesperada, mientras que la reducción específica en la movilización metabólica para una defensa activa (reflejada en el A2) se vincularía con una evaluación más concreta de la dimensión de miedo/ausencia de miedo, al menos en mujeres. En el *Estudio 3* se demostró que la crueldad se asocia con una reducida amplitud del componente LPP —un indicador electrofisiológico bien validado de atención sostenida hacia estímulos emocionalmente relevantes— durante la visión de imágenes de dolor bajo la instrucción de imaginar que la persona de la imagen era alguien desconocido, pero no bajo la instrucción de imaginar que se trataba de uno mismo. Estos resultados sugieren una reactividad cerebral reducida hacia el malestar en otros en las personas con mayores rasgos de crueldad, y respaldan la utilidad de los estudios

electrocorticales del procesamiento del dolor para caracterizar mejor, desde una perspectiva neurobiológica, los déficits empáticos asociados a la maldad psicopática. Finalmente, en el *Estudio 4* se encontró que la desinhibición se relacionaba con amplitudes reducidas en ambas variantes del ERN (*Stop* y *Flanker*), pero no con la amplitud del componente *Stop* P3. Estos resultados sugieren que los déficits en el control inhibitorio asociados a la desinhibición se localizan en etapas tempranas de la monitorización de los errores, más que en las etapas tardías del procesamiento elaborativo de estímulos relevantes.

En conclusión, esta tesis ha posibilitado la identificación de distintos correlatos fisiológicos que contribuyen al avance del conocimiento sobre las distintas disposiciones bioconductuales que subyacen a los constructos de rasgo del modelo triárquico de la psicopatía. La relación de la audacia con una mayor vmHRV y una menor CDR (y A2) demuestra, por un lado, y de manera novedosa, que la baja sensibilidad a la amenaza está asociada con una adecuada regulación emocional, y por otro lado, con una menor movilización metabólica del sistema motivacional defensivo, aportando así un nuevo indicador fisiológico a los ya establecidos sobre la respuesta a la amenaza. La relación entre la maldad y una menor amplitud del LPP ante el dolor en otros sugiere que la baja capacidad afiliativa está asociada con una reactividad atenuada ante la angustia ajena (pero no ante la propia), señalando probablemente la menor relevancia que tienen estos estímulos para los individuos con rasgos de crueldad, lo que supone una nueva aportación para el estudio de los déficits empáticos en la psicopatía. Finalmente, la relación entre la desinhibición y una menor amplitud de los componentes *Flanker* ERN y *Stop* ERN sugiere que el bajo control inhibitorio está asociado con déficits en la monitorización temprana de los errores (tanto de interferencia como de inhibición) en una tarea compleja, sumando a los resultados previos nueva evidencia sobre las diferencias individuales en el procesamiento del error (con independencia del tipo) en relación a la desinhibición. De este modo, este trabajo de

investigación pretende contribuir a conformar redes nomológicas más completas para cada disposición triárquica y mejorar nuestra comprensión de este trastorno de la personalidad multifacético desde una perspectiva bioconductual.

BACKGROUND

CHAPTER 1. Psychopathy from the triarchic model: Background and theoretical accounts

Psychopathy is currently considered a multifaceted personality disorder characterized by distinctive affective and interpersonal traits, as well as prominent behavioral deviance. However, there are ongoing debates about the unitary versus multifaceted nature of the disorder, the differences between psychopathy and antisocial personality disorder, and the pathognomonic components of psychopathy.

To address these issues, the triarchic model of psychopathy (Patrick et al., 2009) has been proposed as a conceptual framework that integrates different existing theoretical perspectives, both historical and contemporary, and serves as a reference for coordinating research on the neurobiological mechanisms underlying psychopathy. The model defines psychopathy in terms of three trait constructs that correspond to distinct symptom features of psychopathy and appear to relate more clearly to biobehavioral systems and processes (Patrick, 2022; Patrick et al., 2019):

- **Boldness**, which involves social dominance, high self-esteem, venturesomeness, the ability to remain calm and focused under pressure or threat, and the capacity to recover quickly from stressful events, corresponds to the biobehavioral process of *threat sensitivity***.**
- **Meanness**, which encompasses a constellation of attributes including a lack of empathy, disdain for and absence of close attachments with others, rebelliousness, excitement seeking, empowerment through cruelty, and aggressive manipulation of others, relates to the biobehavioral system of *affiliative capacity.*
- **Disinhibition**, which involves boredom proneness, irritability, irresponsibility, lack of planning and foresight, deficient behavioral restraint,

propensity toward impulse control problems, and insistence on immediate gratification, is related to the biobehavioral process of *inhibitory control.*

1.1 Triarchic trait constructs in alternative historical accounts of psychopathy

The triarchic trait constructs can be observed to varying degrees in different classic and contemporary conceptualizations of psychopathy, beginning with Hervey Cleckley and his classic and renowned book "The Mask of Sanity" (1941/1976). In this book, Cleckley provided a detailed description of the personality and behavior of a group of patients who were institutionalized in a psychiatric hospital. As the title of his book suggests, Cleckley viewed psychopathy as a severe pathological condition that is concealed, or "masked", by an appearance of good mental health, or "sanity", which contrasts with other patients who exhibited a disturbed and confused state, such as those with schizophrenia.

Against the background of decades following Pinel's work [1962 (1806)], which named the syndrome '*manie sans délire'*, and encompassed aspects of a lack of behavioral restraint but without "deliriums", Cleckley attempted to stablish a more precise usage of the term "psychopathy" by proposing 16 criteria for diagnosis. These criteria have been classified into three categories: mask features (e.g., social charm, absence of irrationality, and low anxiety, which differentiate psychopathy from other psychiatric conditions), behavioral deviance features (antisocial behaviors, irresponsibility, or lack of life planning), and shallow-deceptive features (absence of remorse, inability to love, absence of loyalty, affective shallowness, and insincerity; Patrick, 2006, 2018a; see Table 1).

Table 1. *Cleckley's (1941/1976) 16 Diagnostic Criteria for Psychopathy Grouped into Categories According to Patrick (2018a)*

Category	Item and description
Mask features	1. Superficial charm and good "intelligence"
	2. Absence of delusions and other signs of irrational thinking
	3. Absence of "nervousness" or psychoneurotic manifestations
	14. Suicide rarely carried out
Behavioral deviance features	7. Inadequately motivated antisocial behavior
	8. Poor judgment and failure to learn by experience
	4. Unreliability
	13. Fantastic and uninviting behavior with drink and sometimes without
	15. Sex life impersonal, trivial, and poorly integrated
	16. Failure to follow any life plan
Shallow-deceptive features	5. Untruthfulness and insincerity
	6. Lack of remorse or shame
	10. General poverty in major affective reactions
	9. Pathological egocentricity and incapacity for love
	11. Specific loss of insight
	12. Unresponsiveness in general interpersonal relations

Given that high externalizing proneness (i.e., behavioral deviance features and lack of control of impulses) is often accompanied by internalizing problems (Achenbach & Edelbrock, 1978; Krueger, 1999; Vaidyanathan et al., 2011), the patients described by Cleckley could be considered markedly uncommon. From the standpoint of the triarchic model, the psychopathy described by Cleckley reflects the concurrence of two triarchic dispositions: the behavioral deviance features would be well represented by **disinhibition**, whereas the lack of internalizing symptomatology would be related to **boldness**. Interestingly, Patrick (2022) studied how varied both dispositions along personality disorders. Notably, it was demonstrated that the presence of boldness blunted the higher

internalizing symptomatology (i.e., avoidant, dependent, obsessive-compulsive, or depressive personality disorders) related to disinhibition, whereas the confluence of both triarchic dispositions showed the highest antisocial personality disorder symptomatology.

In parallel, Cleckley's contemporary authors (Karpman, 1941/1948; Lykken 1957) proposed two variants of psychopathy. Karpman (1951/1948) proposed primary psychopathy (also named 'idiopathic' or 'essential' psychopathy) that arises from a high genetic predisposition occurring in the minority of cases, whereas the secondary psychopathy (also named 'symptomatic' psychopathy) is caused by adverse experiences and environmental conditions, without the need for genetic vulnerability, being the most prevalent. In a related line, Lykken (1957), attending to the criteria proposed by Cleckley, demonstrated empirically two primary and secondary psychopathy subtypes differing in their anxiety and fear features: lower for primary psychopathy, higher for secondary (also named 'neurotic').

In contrast to Cleckley's portrayal of psychopathic psychiatric patients, historical writers focused on psychopathy in incarcerated criminal populations (Lindner, 1944; McCord & McCord, 1964) painted a more antagonistic picture, emphasizing features such as emotional detachment, hostility, defiance, selfish disregard, and callous exploitativeness, along with a lack of behavioral control. To address these characteristics, the triarchic model includes the dimension of **meanness**. It is important to note that when studying these features in children or adolescent samples, the term callous-unemotional (CU) traits (Frick et al., 2014) is used instead of meanness.

In summary, as discussed in this review, the triarchic dispositions have been represented, to varying degrees, in different historical conceptualizations of psychopathy. All theories agree on the presence of unrestrained behavior, which is most strongly connected with the **disinhibition** trait construct but it is not sufficient by itself for a diagnosis

of psychopathy. Cleckley (1941/1976), in his view on psychiatric patients, empathized the characteristics of low anxiety and high social ability, which are preferentially represented by the **boldness** dimension. McCord & McCord (1964), in their studies on incarcerated samples, highlighted the hostility and cruelty components reflected by **meanness** traits. How the different possible configurations of these three dispositions can account for the diverse phenotypic expressions of psychopathy is a question that remains to be defined.

1.2 The assessment of psychopathy

The main goal of obtaining a reliable measure for psychopathy led professor Robert Hare to develop what is now considered the most widely validated and influential instrument for the clinical and forensic assessment of psychopathy*: The Hare Psychopathy Checklist* (PCL; Hare, 1980). Based on the traditional clinical descriptions of psychopathy, the revised version (PCL-R; Hare, 1991, 2003) is a clinical scale composed of 20 items that are scored on a 3-point scale (0-2), depending on whether the raters consider it to be applicable to a given individual. The standard administration procedure involves a semi-structured interview and the review of the individual's file and supporting information, which requires a long evaluation time (approximately 90 minutes) and extensive training for raters.

Although the PCL-R arose from a unitary perspective of psychopathy, research studying its internal item structure has shown that it can fall statistically and conceptually into distinguishable groups or factors. Firstly, a two-factor structure highly correlated with each other ($r \approx .50$; Hare et al., 1991; Harpur et al., 1988, 1989) was found: PCL-R Factor 1 (or F1), which includes affective/interpersonal traits, and PCL-R Factor 2 (or F2), which encompasses lifestyle/antisocial features. Later, a four-facets model (using 18 items) posited that both factors could be divided into two separable facets each (see Table 2; Hare, 2003).

Table 2. *Hare Psychopathy Checklist-Revised (PCL-R; Hare, 2003) Factors, Facets, and Items*

Factor	Facets and items
Factor 1 **Affective/Interpersonal**	*Facet 1 Interpersonal*
	1. Glibness/superficial charm
	2. Grandiose sense of self-worth
	4. Pathological lying
	5. Conning/manipulative
	Facet 2 Affective
	6. Lack of remorse of guilt
	7. Shallow affect
	8. Callous/Lack of empathy
	16. Failure to accept responsibility
Factor 2 **Lifestyle/Antisocial**	*Facet 3 Lifestyle*
	3. Need for stimulation
	9. Parasitic lifestyle
	13. No realistic, long-term goals
	14. Impulsivity
	15. Irresponsibility
	Facet 4 Antisocial
	10. Poor behavioral controls
	12. Early behavioral problems
	18. Juvenile delinquency
	19. Revoke conditional release
	20. Criminal versatility

As a result of its widespread usage and the high reliability and validity demonstrated by the PCL-R, clinical scales directly derived from it were developed, such as the *Psychopathy Checklist: Screening Version* (PCL:SV ; Hart et al., 1995) —a 12-item version of PCL-R that serves as a screen for psychopathy, but is commonly used as a standalone instrument for research with forensic psychiatric and non-criminal populations— and the *Psychopathic Checklist: Youth Version* (PCL:YV; Forth et al., 2003) —designed to assess

psychopathy in adolescents between 12 and 18 years, demonstrating similar psychometric properties and factorial structures as the PCL-R.

Although assessing psychopathy using self-report measures may seem nonsensical due to the tendency of individuals with higher psychopathy traits to lie or show lack of insight, the use of these measures represents other relevant advantages, such as brevity, ease to completion, and affordability, in addition to allowing the detection of response styles that may be especially problematic in psychopaths (i.e., over- and underreporting of maladaptive symptoms), as well as yielding useful information about the absence of affective traits (Sellbom et al., 2018). Self-report measures patterned after the PCL-R have been developed to assess psychopathy in non-institutionalized samples. Worth noting is the *Levenson Self-Report Psychopathy Scale* (LSRP; Levenson et al., 1995), a self-report consisting of 26 items answered on a 4-point Likert scale that are divided into two scales: LSRP Primary (e.g., "people who are stupid enough to get ripped off usually deserve it") and LSRP Secondary (e.g., "I don't plan anything very far in advance"). These differing scales were intentionality constructed as counterparts of PCL-R F1 and F2 and supported by initial exploratory factor analyses revealing two factors that appeared to parallel those of the PCL-R (Brinkley et al., 2001; Lynam et al., 1999; but see Brinkley et al., 2008; Garofalo et al., 2019; Sellbom, 2011). Another scale is the *Self-Report Psychopathy Scale: Version III* (Williams et al., 2007), now formally named SRP-4 (Paulhus et al., 2017), consisting of 64 items that show a four-factor structure: Interpersonal Manipulation, Callous Affect, Erratic Lifestyle, and Criminal Tendencies.

In addition to measures designed following the PCL-R approach, the most widely used measure in community samples has been the *Psychopathic Personality Inventory* (PPI; Lilienfeld & Andrews, 1996) and its revised version (PPI-R; Lilienfeld & Widows, 2005). The PPI was developed through an iterative exploratory process, with item-level factor

analyses used to delineate content scales. It comprises 154 items presented in a 4-point Likert-type format and provides a total score as a global index of psychopathy, along with scores on eight content scales indexing specific facets of psychopathy (see Table 3 for the definition of each content scale and item examples). Initial exploratory factor analyses of the eight content scale scores revealed a two-factor structure (Benning et al., 2003; Benning, Patrick, Bloningen et al., 2005; but see Neumann et al., 2008), with PPI-R Social Influence, Fearlessness, and Stress Immunity scales loading on a first factor, named Fearless Dominance (FD), and PPI-R Machiavellian Egocentricity, Rebellious Nonconformity, Blame Externalization, and Carefree Nonplanfulness scales loading on a second factor, initially termed Impulsive Antisociality (IA), but changed to Self-Centered Impulsivity (SCI; Lilienfeld & Widows, 2005). The Coldheartedness scale does not load distinctively on either higher-other factor and is often used as a separate dimension. In contrast to the two factors of PCL-R, which are moderately correlated, the two PPI-R factors seem to be orthogonal ($r \approx .05$-$.12$; Marcus et al., 2013; Miller & Lynam, 2012).

Table 3. *Psychopathic Personality Inventory–Revised (PPI-R; Lilienfeld & Widows, 2005) Factors and Content Scales*

Factor	Content scales
PPI-R Fearless Dominance / PPI-R Factor 1	**Social Influence:** Perceived ability to influence and manipulate others (18 items; "When I meet people, I can often make them interested in me with just one smile")
	Fearlessness: Absence of anticipatory anxiety concerning harm and willingness to participate in risky activities (14 items; "Sometimes I do dangerous things on a dare")
	Stress Immunity: Absence of marked reactions to anxiety-provoking events (13 items; "I don't get nervous under pressure")

Factor	Content scales
PPI-R Self-Centered Impulsivity / PPI-R Factor 2	**Machiavellian Egocentricity:** Narcissistic and ruthless attitudes in interpersonal functioning (20 items; "I tell people only the part of the truth they want to hear")
	Rebellious Nonconformity: Reckless lack of concern regarding social norms (16 items; "I pride myself on being offbeat and different from others")
	Blame Externalization: Tendency to blame others for one's problems and to rationalize one's misbehavior (15 items; "I often been betrayed by people I trusted")
	Carefree Nonplanfulness: Attitude of indifference in planning one's actions (19 items: "I like to act first and think later")
PPI-R Coldheartedness: Propensity toward callousness, guiltlessness, and lack of sentimentality (16 items; "I look out for myself before I look out for anyone else")	

Finally, due to its special relevance to this research, the *Inventory of Callous-Unemotional Traits* (ICU; Frick, 2004; Kimonis et al., 2008) deserves mention, although it does not measure all the traits that characterize psychopathy. Rather, it focuses on the assessment of callous-unemotional (CU) traits, using a 24-item scale rated on a 4-point Likert scale. The ICU is considered one of the most comprehensive measures of these traits (Viding & Kimonis, 2018).

1.2.1 The Triarchic Psychopathy Measure

The *Triarchic Psychopathy Measure* (TriPM; Patrick, 2010) is a self-report questionnaire specifically designed to assess the three trait constructs proposed by the triarchic model of psychopathy (Patrick et al., 2009). The measure consists of 58 items answered using 4-point Likert scale (0 = false, 1 = somewhat false, 2 = somewhat true, 3 = true). The items of the TriPM Disinhibition and Meanness scales come from the

Externalizing Spectrum Inventory (ESI; Krueger et al., 2007), which is an inventory comprising 23 scales that assess behavior problems associated with externalizing psychopathology. The ESI has a hierarchical structure where all scales load into a general Disinhibitory factor, with residual variances for certain subscales delineating distinct subfactors of Callous-Aggression and Substance Abuse. The 20 items that compose the TriPM Disinhibition scale were specifically selected from the subscales with lower saturations in the residual subfactors and strongly predict the general Disinhibitory factor (r > .90), reflecting impulsivity, irresponsibility, or rule-breaking tendencies. The TriPM Meanness scale consists of 19 items selected to strongly predict (r > .80) the Callous-Aggression residual subfactor, after controlling for moderate overlap with the scores on the TriPM Disinhibition scale, and indexing an uncaring and exploitative propensity. The TriPM Boldness scale comprises 19 items derived from a separate inventory that indexes a fear/fearlessness bipolar dimension (Kramer et al., 2012; Vaidyanathan et al., 2009) or threat sensitivity dimension (Yancey et al., 2016) that includes scales specific to fear and social dominance (e.g., *Fear Survey Schedule-III*; Arrindell et al., 1984; the Thrill and Adventure Seeking subscale from the *Sensation Seeking scale*; Zuckerman, 1994; and the three content scales comprising the PPI-R Fearless Dominance Factor; Lilienfeld & Widows, 2005).

The TriPM has shown good reliability, with Cronbach's alpha between .79-.89, 83-.91, and .77-.89 for the TriPM Disinhibition, Meanness and Boldness scales, respectively (Poy et al., 2014; Sellbom & Philips, 2013; Smith et al., 2013; Stanley et al., 2013; Venables et al., 2014). Regarding the relationship between the three scales, TriPM Disinhibition and Meanness tend to be moderately correlated (rs = .36 to .54), TriPM Meanness and Boldness show lower correlations (rs = .17 to .20), whereas TriPM Disinhibition and Boldness are marginally correlated (rs = -.10 to -.03; Drislane et al., 2014; Stanley et al., 2013; Strickland et al., 2013).

Another important issue to note here is the extent to which the three dimensions of the triarchic model are represented in other psychopathy measures that have been reviewed above. The most influential and widely used measures to assess psychopathy have certainly been the PCL-R (Hare, 2003) and the PPI-R (Lilienfeld and Widows, 2005). Both present a two-factor structure, with a first factor indexing the affective/interpersonal features of psychopathy (although through different configurations of these traits), and a second factor assessing its externalizing tendencies.

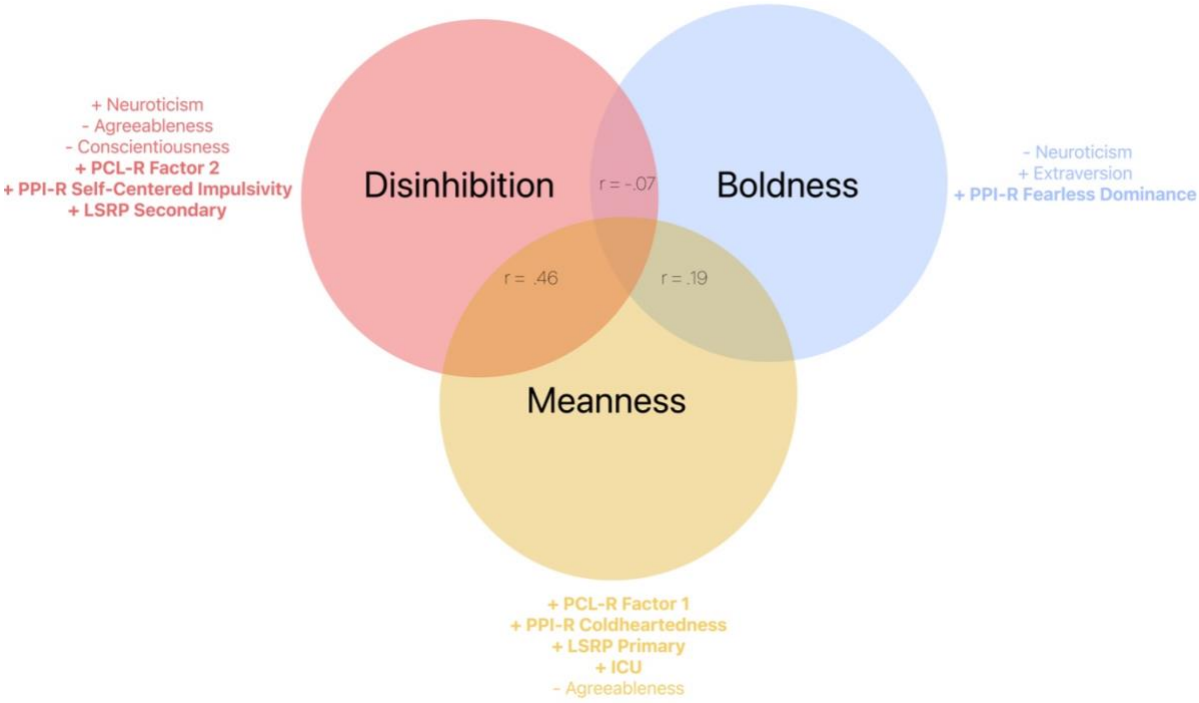
The triarchic model of psychopathy (Patrick et al., 2009) incorporates this latter component in the trait construct of **disinhibition** and separates the affective/interpersonal traits into two distinct phenotypic manifestations (depending on differential developmental factors): the trait construct of **meanness**, encompassing deficient empathy, disdain for and lack of close attachments with others, rebelliousness, excitement seeking and empowerment trough cruelty (best captured by PCL-R Factor 1 and the PPI-R Coldheartedness scale), and the trait construct of **boldness**, entailing a capacity to remain calm and focused on situations involving pressure or threat, high self-assurance, and social efficacy (aligned with the PPI-R Fearless Dominance factor).

The LSRP (Levenson et al., 1995) structure would perform in a similar way to the PCL-R, with its Primary scale representing the trait construct of **meanness** to a greater extent and the Secondary scale representing **disinhibition**. As discussed above, the ICU (Frick, 2004; Kimonis et al., 2008) assesses specifically the CU traits, i.e., the trait construct of **meanness**.

Notably, the trait constructs of the triarchic model also interface easily with well-established models of general personality. Hence, following the five-factor model (FFM) of normal personality (Costa & McCrae, 1992), the trait construct of **disinhibition** seems to be characterized by extremely low Conscientiousness, along with low Agreeableness and high

Neuroticism. The trait construct of **meanness** is defined mainly by low Agreeableness and, to a lesser extent, low Conscientiousness, whereas the trait construct of **boldness** is marked mainly by high Extraversion and low Neuroticism and low Agreeableness (Miller et al., 2016; Poy et al., 2014). Figure 1 illustrates the relationship between the TriPM trait constructs and psychopathic and normal personality measures, and Table 4 summarizes the reported correlations between the TriPM scales and the other psychopathic and normal personality measures (see also Table 5 for more information about sample and psychopathy/personality measure versions in the reviewed studies).

Figure 1. *Relationship between TriPM Trait Constructs and Psychopathic and Normal Personality Measures*



Note. ICU = *Inventory of Callous-Unemotional Traits* (Frick, 2004; Kimonis et al., 2008); LSRP = *Levenson Self-Report Psychopathy Scale* (Levenson et al., 1995); PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); PPI-R = *Psychopathic Personality Inventory–Revised* (Lilienfeld & Widows, 2005)

Table 4. *Correlations between Psychopathy and Normal Personality Measures with Scale Measures of the Triarchic Model Trait Constructs (Adapted from Patrick, 2022; Supplemental Table 2)*

Measures	Average *r*s with triarchic scale scores			Published Source
	Boldness	**Meanness**	**Disinhibition**	
Psychopathy Measures				
PCL-R				
Total	.18	**.29**	**.26**	Brislin et al. (2015, 2017); Gerbrandij et al. (2019); Hall et al. (2014); Sellbom et al. (2015); Venables et al. (2014); Wall et al. (2015); Yoon et al. (2022)
Factor 1	**.20**	.19	.10	
Factor 2	.10	**.32**	**.36**	
LSRP				
Total	.16	**.58**	**.56**	Drislane et al. (2014, 2015, 2017, 2019); Sellbom & Phillips (2013); Sellbom et al. (2015, 2016); Shou et al. (2016)
Primary	.18	**.60**	**.44**	
Secondary	-.19	**.38**	**.60**	
PPI				
Total	**.52**	**.59**	**.54**	Anderson et al. (2014); Drislane et al. (2014, 2015, 2019); Hall et al. (2014); Sellbom & Philips (2013); Sica et al. (2015); Stanley et al. (2013); van Dongen et al. (2017)
Fearless Dominance	**.78**	**.27**	.05	
Self-Centered Impulsivity	.12	**.52**	**.71**	
Coldheartedness	**.23**	**.49**	.07	
ICU				
Total	.13	**.55**	**.30**	Drislane et al. (2014, 2015, 2017); Hall et al. (2014); Kyranides et al. (2017); Sellbom & Philips (2013); Sellbom et al. (2016); Sica et al. (2020)
Personality Measures				
Five Factor Model				
Neuroticism	**-.50**	.08	**.37**	Blagow et al. (2016); Drislane et al. (2014, 2017, 2018, 2022); Hall et al. (2014); Miller et al. (2016); Poy et al. (2014); Shou et al., (2016); Sica et al. (2015); Somma et al. (2016)
Extraversion	**.49**	-.07	-.08	
Openness	.19	-.06	.03	
Agreeableness	-.18	**-.56**	**-.35**	
Conscientiousness	.16	**-.39**	**-.50**	

Note. PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); ICU = *Inventory of Callous-Unemotional Traits* (Frick, 2004; Kimonis et al., 2008); LSRP = *Levenson Self-Report Psychopathy Scale* (Levenson et al., 1995); PPI = *Psychopathic Personality Inventory* (Lilienfeld & Andrews, 1996).
Correlations in table are the weighted average *r*s across samples from cited sources. Coefficients with values > .20 are in bold. Information about sample and psychopathy/personality measure versions is presented in Table 5.

Table 5. *Summary of Reviewed Studies to Obtain the Correlations between Psychopathy and Normal Personality Measures with Scale Measures of the Triarchic Model Trait Constructs*

Published source	Sample				Interview/Self-Report measures					Triarchic scales	
	Criminal/ Psychiatric		Community		Psychopathy				Personality		
	M	W	M	W	PCL-R	LSRP	PPI	ICU	FFM	TriPM	Other
Anderson et al. (2014)			335	276			×[c,d]			×	
Blagov et al. (2016)			28	92					NEO-FFI	×	
Brislin et al. (2015)	242[a]				×						MPQ-Tri
Brislin et al. (2017)	190[a]	216[a]	98	248	×						MPQ-Tri
Drislane et al. (2014)			347	271		×[i]	×	×	NEO PI–R[j]	×	
Drislane et al. (2015)			289	361		×	×	×			YPI-Tri
Drislane et al. (2017)			263	304		×		×	NEO PI–R[j]	×	
Drislane et al. (2018)			353	498					NEO PI–R	×	
Drislane et al. (2019)			140	100		×	×[c]				PID-5-Tri
Drislane et al. (2022)[k]	273[a]	83[a]							NEO PI–R	×	PPI-Tri, MMPI-Tri, NEO-Tri
Gerbrandij et al. (2019)	100[b]				×						SNAP-F-Tri
Hall et al. (2014)	1341[a]		365	285	×[f]		×[h, g]	×[g]	NEO PI–R[j]	×[i]	PPI Tri
Kyranides et al. (2017)			49	50				×		×	

Published source	Sample				Interview/Self-Report measures					Triarchic scales	
	Criminal/ Psychiatric		Community		Psychopathy				Personality		
	M	W	M	W	PCL-R	LSRP	PPI	ICU	FFM	TriPM	Other
Miller et al. (2016)			147	188					IPIP-NEO-120	×	
Poy et al. (2014)			96	253					NEO PI–R	×	
Sellbom and Phillips (2013)	209[a]		204	423		×[i]	×[e]	×[g]		×	
Sellbom et al. (2015)	160[a]		140	100	×[f]	×[g,i]					PPI-Tri
Sellbom et al. (2016)		209[a]	112	166		×	×[e]	×			MMPI-Tri
Shou et al. (2016)	83[b]	110[b]				×				×	
Shou et al. (2017)			44	182					Mini-IPIP[g]	×	
Sica et al. (2020)			278	249				×		×	
Sica et al. (2015)			114	172			×[c]		NEO-FFI	×	
Somma et al. (2016)			455	687					FFMRF	×	
Stanley et al. (2013)	93[a]	48[a]					×[d]		BFI	×	
van Dongen et al. (2017)	195[a]	22[a]	243	253			×[c]			×	
Venables (2014)	326[a]				×					×	
Wall et al. (2015)	152[a]				×					×	
Yoon et al. (2022)	152[a]				×[f]					×	

Note. M = Men; W = Women; BFI = *Big Five Inventory* (John et al., 1991); FFM = Five Factor Model; FFMRF = *Five-Factor Model Rating Form* (Mullins-Sweatt et al., 2006); ICU = *Inventory of Callous-Unemotional Traits* (Frick, 2004; Kimonis et al., 2008); IPI-NEO-120 = *International Personality Item Pool–Neuroticism, Extraversion and Openness* (Maples et al., 2014); LSRP = *Levenson Self-Report Psychopathy Scale* (Levenson et al., 1995); Mini-IPIP = *International Personality Item Pool—Short Form* (Donnellan et al., 2006); MMPI-Tri = *Minnesota Multiphasic Personality Inventory Triarchic Scales* (Sellbom et al., 2016); MPQ-Tri = *Multidimensional Personality Questionnaire Triarchic Scales* (Brislin et al., 2015); NEO-FFI = *NEO Five-Factor Inventory* (Costa & McCrae, 1992); NEO PI-R = *NEO Personality Inventory Revised* (Costa & McCrae, 1992); NEO-Tri = NEO Personality Inventory Triarchic Scales (Drislane et al., 2018); PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); PID-5-Tri = *Personality Inventory for DSM-5 Triarchic Scales* (Drislane et al., 2019); PPI = *Psychopathic Personality Inventory* (Lilienfeld & Andrews, 1996); PPI-R = *Psychopathic Personality Inventory–Revised* (Lilienfeld & Widows, 2005); PPI-SF = *Psychopathic Personality Inventory–Short Form* (Lilienfeld & Hess, 2001); PPI-Tri = *Psychopathic Personality Inventory Triarchic Scales* (Hall et al., 2014); SNAP-F-Tri = *Schedule for Nonadaptive and Adaptive Personality Forensic Version Triarchic Scales* (Gerbrandij et al., 2019); TriPM = *Triarchic Psychopathy Measure* (Patrick, 2010); YPI-Tri = *Youth Psychopathic Traits Inventory Triarchic Scales* (Drislane et al., 2015)

[a] This study was carried out on a criminal sample

[b] This study was carried out on a psychiatric sample

[c] This study used the PPI-R

[d] This study used the PPI-SF

[e] This study used the PPI in the criminal sample and PPI-R in the community sample

[f] This study reported scores only on the criminal sample

[g] This study reported scores only in the community sample

[h] This study reported only scale scores

[i] This study reported only total scores

[j] This study reported only the Antagonism scale (i.e., Agreeableness reverse scored)

[k] This study reported correlations between the TriPM and PPI, as well as correlations between the other psychopathy/personality scales and the PPI triarchic scale

1.2.2 The Antisocial Personality Disorder

The relationship between psychopathy and the antisocial personality disorder, which was proposed as the counterpart to psychopathy in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychological Association, APA, 1952-2013) can be considered controversial. Although earlier versions of the DSM were based to some extent on descriptions proposed by Cleckley, it was not until the DSM-III (APA, 1980) that the diagnostic reliability was improved. Feighner et al. (1972) created a diagnostic system of the antisocial personality disorder (based on Robin's 19-item criteria set, 1966) that, representing the psychopathy view of Cleckley, proposed more specific and explicit criteria.

Concurrently with the emergence of DSM-III, the *Psychopathy Checklist* (PCL) was developed by Hare (1980), and the main criticism was directed at the DSM diagnostic for focusing almost exclusively on assessing behaviors, emphasizing criminality, whereas the PCL also considered personality traits. This criticism led to the inclusion of an item pertaining to lack of remorse in the revised version of the DSM-III (DSM-III-R; APA, 1987) (Widiger et al., 1988). By the time of the subsequent edition of the DSM (DSM-IV; APA, 1994), the PCL had already been replaced by its revised version, the PCL-R (Hare, 1991), and the two-factor structure of the PCL had been proposed. It was shown that the DSM-III and DSM-III-R criteria correlated more strongly with the second factor of the PCL-R, which confirmed that the DSM-III-R assessed antisocial or criminal behaviors more than personality traits. However, the PCL-R showed greater validity in predicting criminal recidivism than the DSM-III-R (e.g., Hart et al., 1988; Serin et al., 1990). Therefore, in the DSM-IV, an attempt was made to move closer to the PCL-R conceptualization, also removing several of the specific behavioral requirements that Feighner et al. (1972) included from the work of Lee Robins (1966).

In recent years, empirical research has focused more on psychopathy than the antisocial personality disorder. Although the diagnostic criteria for antisocial personality disorder in the latest edition of the DSM (DSM-5; APA, 2013) have remained virtually unchanged from the fourth edition and its revision (DSM-IV-TR; APA, 2000), an alternative dimensional model has been proposed in the section III of the DSM-5 to supplement the current categorical perspective. According to this model, the personality disorders are defined by deficits in personality functioning in areas such as identity, self-direction, empathy, and privacy, as well as pathological personality traits. For antisocial personality disorder, the proposed traits include manipulation, insensitivity, deception, hostility (indicatives of **meanness**), and assumption of risk, impulsivity, and recklessness (indicatives of **disinhibition**) as measured by the *Personality Inventory for DSM-5* (PID-5; Krueger et al., 2012). The diagnostic criteria also require that these traits are generalizable to various situations and stable over time. Additionally, a specifier for "with psychopathic features" was added, which is defined by low anxiety, low social withdrawal, and high attention-seeking, representing **boldness** traits. Research has demonstrated a stronger association between the current proposal in the section III of the DSM-5 with other psychopathy measures, than the DSM-4 —particularly with **boldness** (Anderson et al., 2014)— and with the section II of the same version of the manual (Wygant et al., 2016). For a comprehensive review on this topic, see Crego and Widiger (2015).

1.3 Biobehavioral research in psychopathy

The triarchic model trait constructs are formulated in biobehavioral terms, using the self-report measures as provisional referents to identify potential indicators from other domains, such as neurophysiological or behavioral, to construct their own nomological network. The aim is to improve the understanding of each disposition and stablish

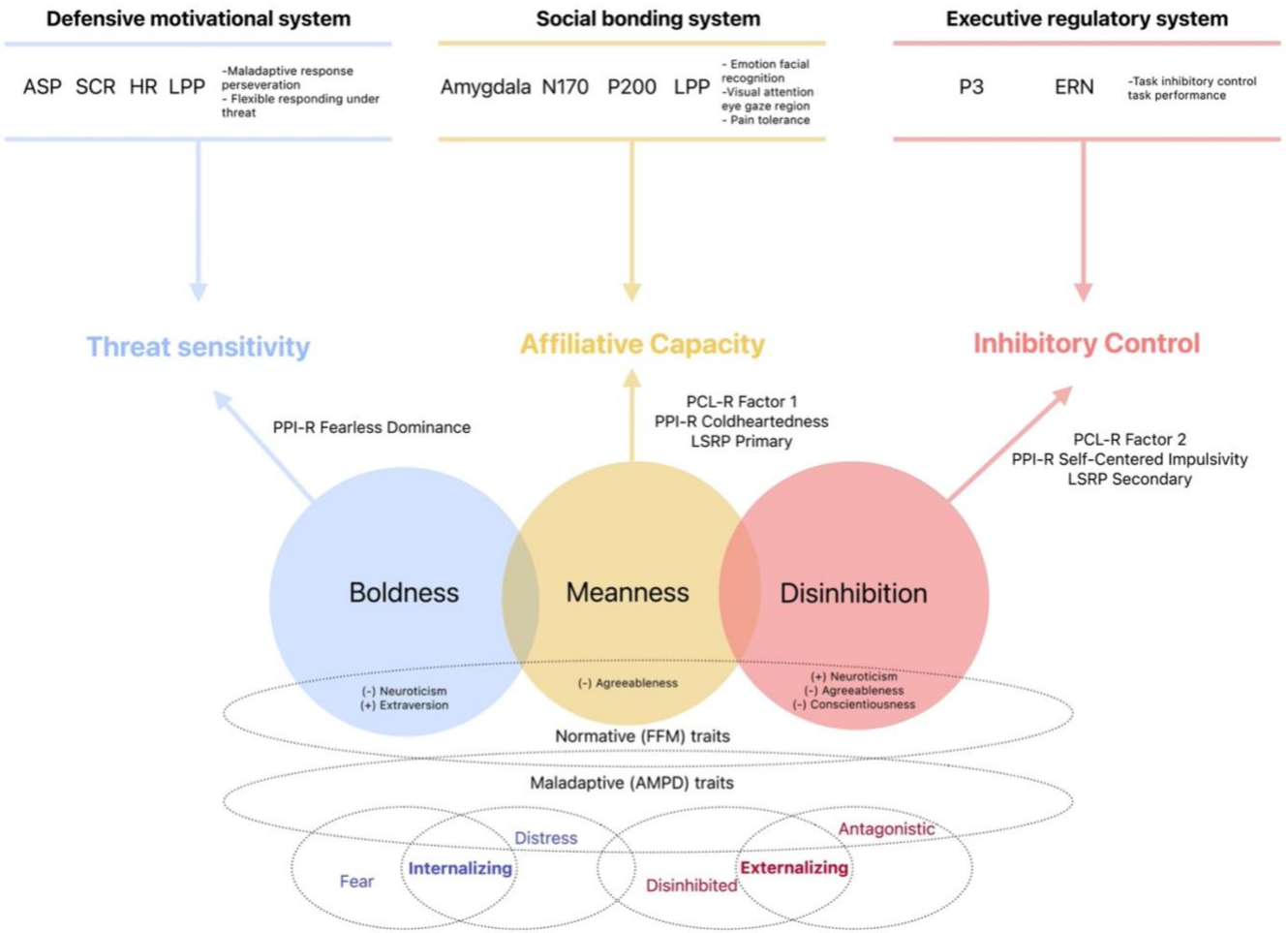
multimodal measurement models (Patrick et al., 2019). Therefore, the triarchic dispositions should be viewed as open-ended constructs that are subjected to revision based on accumulating knowledge of their nomological networks (Patrick, 2022). To understand current neurobehavioral research on triarchic dimensions, it is important to know its origins.

Early etiological models viewed psychopathy as a unitary syndrome that arose from a single underlying deficit. Some authors proposed a deficient affective reactivity (*Low fear hypothesis*; Lykken, 1957, 1995), while others suggested deficits in cognitive and attentional processing affecting inhibitory control and punishment learning (*Response modulation hypothesis*; Newman, 1998; Patterson & Newman, 1993) as the primary underlying cause. However, following the identification of distinct PCL-R factors, empirical research provided evidence that impaired affective response, especially to aversive cues, was preferentially related to the affective/interpersonal traits (PCL-R Factor 1: Patrick, 1994; Patrick et al., 1993; PPI-R Fearless Dominance: Benning, Patrick & Iacono, 2005), whereas the deficits in cognitive-attentional processing were more related to the impulsive/antisocial features (PCL-R Factor 2: Moltó et al., 2007; Morgan & Lilienfeld, 2000; PPI-R Impulsive Antisociality: Carlson et al., 2009). This challenged the unitary conceptualization of psychopathy, suggesting instead that the PCL-R/PPI-R factors partly reflect different deficits. As a result, *dual-process* (Fowles & Dindo, 2009) or *two-process* (Patrick & Bernat, 2009) models of psychopathy emerged, positing that psychopathy can be understood in terms of two separable individual differences constructs with distinctive neurobiological underpinnings contributing to the affective/interpersonal (i.e., **boldness** and **meanness** in triarchic terms) and the impulsive/antisocial (i.e., **disinhibition**) symptom components, respectively: *trait fearlessness* —reflecting an under-reactivity of the brain's defensive motivational system— and *externalizing vulnerability* —supposed to reflect impairments in frontocortical systems that mediate anticipation, planfulness, and behavioral control.

Nonetheless, empirical evidence has demonstrated a prominent role of weak threat sensitivity in the **boldness** facet of psychopathy (see Patrick, 2018b), which has recently led to the proposal that low affiliative capacity could contribute to a greater extent to **meanness** (or callous-unemotional) traits (Blair et al., 2018).

Hence, the **boldness** trait construct corresponds to a biobehavioral dimension of *threat sensitivity*, presumed to reflect individual differences in the reactivity of the brain's defensive system. The **meanness** disposition corresponds to the biobehavioral disposition of *affiliative capacity*, theorized to reflect a biologically based predatory tendency involving the aggressive pursuit of one's goals regardless of others. The **disinhibition** disposition corresponds to the biobehavioral dimension of *inhibitory control*, assumed to reflect differences in frontal brain activity affecting emotional regulation and behavioral control. These neurobehavioral dimensions represent hypothetic biobehavioral trait dimensions for which provisional indicators from differing modalities have been identified, serving to interface the neural system constructs (i.e., defensive motivational system, social bonding system, and executive regulatory system) with each corresponding trait and symptom dimensions (i.e., boldness, meanness, and disinhibition, respectively). The following sections will review the biobehavioral indicators identified so far for each neurobehavioral dimension (see Figure 2 for a schematic depiction of this biobehavioral framework in the study of psychopathy).

Figure 2. *Biobehavioral Framework of Psychopathy (Adapted from Patrick, 2022; Figure 3)*



Note. AMP = Alternative Model for Personality Disorders*;* ASP = Aversive Startle Potentiation; ERN = Error Related Negativity; FFM = Five Factor Model; HR = Heart Rate; LPP = Late Positive Potential; LSRP = *Levenson Self-Report Psychopathy Scale* (Levenson et al., 1995); N170 = N170 ERP Component; P200 = P200 ERP Component; P3 = P3 ERP Component; PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); PPI-R = *Psychopathic Personality Inventory–Revised* (Lilienfeld & Widows, 2005); SCR = Skin Conductance Reactivity

1.3.1 Threat sensitivity

The biobehavioral dimension of threat sensitivity is associated with the construct of boldness and reflects individual differences in the brain's defensive system reactivity. There is a long-standing idea in the literature that psychopathy involves a deficit in fear reactivity. The first studies investigating this issue found a reduced electrodermal conditioning to cues signaling aversive results in offenders diagnosed with psychopathy (Hare, 1965; Lykken, 1957), which was interpreted as a difficulty in inhibiting punished responses in these individuals. Research has continued to show deficient fear conditioning, as indicated by reduced responses in various physiological indicators, including facial corrugator activity, heart rate (HR), startle blink, ERP components, and skin conductance, to a cue signaling the emergence of aversive outcomes. These results have been found in male community samples scoring high in PCL-R, either Factor 1 or Factor 2 (Flor et al., 2002), male offenders scoring high in PCL:SV Factor 1, regardless of their scores on PCL:SV Factor 2 (Rothemund et al., 2012), and undergraduates high in PPI-R Fearless Dominance (Dindo & Fowles, 2011; López et al., 2013) and, importantly, in TriPM Boldness (Paiva et al., 2020).

The study of the affective modulation of the startle reflex, an index of defensive reactivity that is shown modulated by the affective valence of the stimulus context in which it is evoked, has typically shown an enhanced startle blink response (named "aversive startle potentiation"; ASP) during the viewing of aversive pictures compared to neutral pictures (Lang, 1995; Lang et al., 1990). Research has consistently shown a reduced ASP related to PCL-R Factor 1 in male offenders (Patrick, 1994; Patrick et al., 1993; Levenston et al., 2000; Vaidyanathan et al., 2011), in female offenders (Verona et al., 2013), and in mixed-gender community samples (Vanman et al., 2003). This response has also been related to PPI-R Fearless Dominance in community men (Benning, Patrick & Iacono, 2005), women (Anderson et al., 2011), and mixed-gender youth/undergraduate samples (although in a

cueing-with-distraction condition; Dvorak-Bertsch et al., 2009). Moreover, TriPM Boldness has been shown to be related to a reduced ASP in a mixed-gender undergraduate sample (Esteller et al., 2016). In this regard, Kramer et al. (2012) extended prior work of Vaidyanathan et al. (2009) and conducted a structural modeling analyses of several fear/fearlessness self-report measures, including the content scales conforming the PPI-R Fearless Dominance factor, and found a general factor that accounts for individual differences in ASP. Going beyond, Yancey et al. (2016) demonstrated that ASP, along with other physiological indices of defensive activation during aversive-picture viewing (i.e., corrugator electromyography and HR acceleration), can be combined with the self-report measure of fear/fearlessness (cf. Kramer et al., 2012; Vaidyanathan et al., 2009) to delineate a cross-domain index of threat sensitivity (THT). This index is presumed to reflect individual differences in the tendency to exhibit defensive action mobilization to explicit aversive cues. For a recent and more extensive review about ASP and psychopathy, see Oskarsson et al. (2021).

Other studies examining brain and physiological reactivity to aversive or unpleasant stimuli have demonstrated a reduced Late Positive Potential (LPP), which is presumed to reflect the sustained allocation of attention to emotionally relevant stimuli, to aversive pictures in male undergraduates in relation to TriPM Boldness (Ellis et al., 2017) and PPI-R Fearless Dominance (Medina et al., 2016). Furthermore, there is evidence of reduced skin conductance responses to aversive images in relation to PPI-R Fearless Dominance in a community male sample (Benning, Patrick & Iacono, 2005), as well as of reduced HR reactivity to violent stimuli and resting heart rate in relation to TriPM Boldness in a mixed-gender community sample (Kyranides et al., 2017).

Interestingly, Bertoldi et al. (2023) found, in a large sample of twins (N = 710) from a longitudinal study of antisocial behavior risk, that low childhood resting HR predicted

adult antisocial behavior, both violent and non-violent, and that this association was mediated by scores on TriPM Disinhibition and Boldness. Additionally, they found that disinhibition mediated the relation between low childhood HR and more externalizing problems in adulthood, but boldness mediated the relation between low childhood HR and fewer internalizing problems in adulthood, suggesting a protective effect of boldness regarding internalizing psychopathology (see also Latzman et al., 2020).

Finally, in behavioral terms, TriPM Boldness has been related to maladaptive response perseveration (more cards played, and less money earned) in the face of punishment (Ribes-Guardiola, Poy, Segarra et al., 2020), as well as greater accuracy and quicker reaction times during threat blocks compared to safe blocks on a task-switching procedure (Yancey et al., 2019, 2022), in mixed-gender community samples. Taking together, these results suggest that the underlying low fear, or the insensitivity to punishment cues in high-boldness individuals may lead to difficulties in inhibiting reward-approach behavior despite increasing punishment contingencies, while also allowing for flexible responding and good task performance under threat conditions.

To summarize, empirical findings suggest that a deficient defensive reactivity underlies boldness traits. Its corresponding biobehavioral dimension of *threat sensitivity* is represented by different physiological and task performance indicators, including impaired fear conditioning, reduced aversive startle potentiation, reduced physiological reactivity (e.g., skin conductance, heart rate, or LPP amplitude) to aversive cues/stimuli, as well as maladaptive response perseveration, indicative of the low fear deficit exhibited by individuals higher in boldness who tend to engage in risk taking behaviors and show venturesomeness or social-dominance/high self-esteem traits. However, it is important to highlight the potential protective effect of boldness traits, mediating the positive relationship between childhood resting heart rate and adulthood internalizing problems, as well as its

capacity to maintain a good task performance even under stressful conditions, which may indicate a more adaptive component of this dimension. For a summary of the reviewed studies, please refer to Table 6.

Table 6. *Summary of Reviewed Empirical Studies for the Biobehavioral Disposition of Threat Sensitivity*

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal		Community				
	M	W	M	W			
Anderson et al. (2011)				57	PPI-R	ASP	PPI-R Fearless Dominance (-) ASP
Benning, Patrick and Iacono (2005)			355[a]		MPQ	Startle and SC in a passive picture-viewing task	MPQ Fearless Dominance (-) ASP and SC to aversive pictures
Bertoldi et al. (2023)			310[a]	400[a]	TriPM	resting HR	TriPM Boldness and Disinhibition mediate the relationship between resting HR and adult antisocial behavior
Dindo and Fowles (2011)			131		PPI	Fear conditioning	PPI Fearless Dominance (-) fear conditioning
Dvorak-Bertsch et al. (2009)			35	20	MPQ-BF	ASP	MPQ Fearless Dominance (-) ASP
Ellis et al. (2017)			65		TriPM	Passive picture viewing task - EEG	TriPM Boldness (-) LPP to negative pictures
Esteller et al. (2016)			72	108	TriPM	ASP	TriPM Boldness (-) ASP
Flor et al. (2002)			21		PCL-R	Fear conditioning	Psychopathic group (-) aversive conditioning (reduced SC, startle, and corrugator reactivity)
Kramer et al. (2012)			544[a]	1426[a]	TF	ASP	THT (+) ASP
Kyranides et al. (2017)			44	44	TriPM	resting HR and HR in a picture-viewing task	TriPM Boldness (-) resting and reactivity HR to violent stimuli
Levenston et al. (2000)	36				PCLR	ASP	Psychopathic group (-) ASP
López et al. (2013)			32	42	PPI-R	Fear conditioning	PPI-R Fearless Dominance (-) fear conditioning (reduced SC)
Lykken (1957)	35	19			Clecley's criteria	Fear conditioning	Primary Psychopathic group (-) fear conditioning (reduced GSR reactivity)

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal		Community				
	M	W	M	W			
Medina et al. (2016)			33		PPI-R	Passive picture-viewing task - EEG	High psychopathic group (-) LPP to emotional pictures
Paiva et al. (2020)			30	25	TriPM	Fear conditioning	Boldness (-) fear conditioning
Patrick et al. (1993)	54				PCL-R	ASP	Psychopathic group (-) ASP
Ribes-Guardiola, Poy, Segarra et al. (2020)			80	142	TriPM	Card perseveration task	TriPM Boldness (+) maladaptive response perseveration
Rothemund et al. (2012)	11		11		PCL-SV	Fear conditioning	Psychopathic group (-) fear conditioning (reduced startle and SC)
Vaidyanathan et al. (2009)			41	59	TF	ASP	THT (+) ASP
Vaidyanathan et al. (2011)	108				PCL-R	ASP	PCL-R F1 (-) ASP
Vanman et al. (2003)			70	10	PCL-R	ASP	PCL-R F1 (-) ASP
Verona et al. (2013)		48			PCL-R	ASP	PCL-R F1 (-) ASP
Yancey et al. (2016)			221	233	TF	ASP	THT (+) ASP
Yancey et al. (2019)			44	46	TriPM Boldness	Task-switching paradigm	TriPM Boldness (+) task performance
Yancey et al. (2022)			43	49	TriPM Boldness	Task-switching paradigm with acute shock cueing	TriPM Boldness (+) task performance

Note. M = Men; W = Women; ASP = Aversive Startle Potentiation; HR = Heart Rate; MPQ = *Multidimensional Personality Questionnaire* (MPQ; Tellegen, 1982); MPQ-BF = brief form of the *Multidimensional Personality Questionnaire* (Patrick et al., 2002); PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); PCL:SV = *Psychopathy Checklist–Revised Short Version* (Hart et al., 1995); PPI = *Psychopathic Personality Inventory* (Lilienfeld & Andrews, 1996); PPI-R = *Psychopathic Personality Inventory–Revised* (Lilienfeld & Widows, 2005); SC = Skin Conductance; TF = *Trait Fear Inventory* (Kramer et al., 2012; Vaidyanathan et al., 2009); THT = cross-domain index of threat sensitivity; TriPM = *Triarchic Psychopathy Measure* (Patrick, 2010)

[a] This study was carried out on a twin sample

1.3.2 Affiliative capacity

Affiliative capacity is a relatively recent biobehavioral dimension proposed for the study of meanness. Initially, the low empathy traits characterizing meanness were explained by a reduced capacity to feel fear (the same as for the boldness disposition): it was suggested that a weakness in the capacity to experiment fear would limit the learning of conditioned affective responses to aversive/punishment cues, which is considered a key procedure to develop a good socialization (Lykken, 1995). However, the focus on empathic processing instead of low fear or threat sensitivity emerged from studies that found male offenders, diagnosed with psychopathy using the PCL-R, exhibited reduced electrodermal reactivity while viewing images of individuals in distress (e.g., crying or screaming; Blair et al., 1997) and judged textual descriptions of transgressions as more moral, regardless of the authority jurisdiction (Blair et al., 1995). Similarly, reduced electrodermal reactivity to pictures of other people in distressing situations or threatening scenes was related to high Factor 1 scores of the *Antisocial Process Screening Device* (APSD; a screening measure patterned after the PCL-R; Frick & Hare, 2001) in an adolescent sample (Blair, 1999). Based on these findings, Blair (1995, 2001) proposed the "violence inhibition model" (VIM), which suggests that the lack of empathy in psychopathy is due to a deficit in a neurocognitive process that automatically inhibits aggressive responses when others show signs of distress.

In line with this, empirical research on psychopathy has focused on the study of emotional facial expressions recognition. Individuals scoring higher on psychopathy assessed by APSD have shown reduced accuracy in recognizing negative emotions, especially fear, in mixed-gender nonclinical children (Blair & Coles, 2000), as well as in male youth samples with emotional and behavioral problems (Blair et al., 2001; Stevens et al., 2001). Muñoz (2009) further supported these findings, showing that reduced accuracy in recognizing fear facial expressions and fear body poses was specifically associated with CU

traits, as indexed by the ICU, in boys. However, when the accuracy in recognizing affective vocalizations was studied, the results were mixed. In youth male samples, one study found that individuals scoring higher on APSD showed reduced recognition accuracy of sad but not fear vocalizations (Stevens et al., 2001), while another study found a reduced accuracy in the recognition of fear but not sad vocalizations (Blair et al., 2005), which was replicated in a sample of male offenders scoring high in PCL-R (Blair et al., 2002). Based on these findings, Blair (2007) proposed the "integrated emotion system" (IES), suggesting that the reduced capacity for emotion facial recognition associated with psychopathy would be related to reduced amygdala reactivity to affective, especially negative/fearful, facial expressions.

The hypothesis that a reduced amygdala reactivity is associated with a lower capacity for emotion facial recognition in psychopathy is supported by fMRI evidence. Marsh et al. (2008) found a reduced activity of the right amygdala to fearful versus neutral facial expressions in male adolescents/children groups diagnosed with oppositional defiant or conduct disorder, compared to nonclinical control and attention deficit and hyperactivity disorder (ADHD) groups. Jones et al. (2009) also found reduced amygdala activity to fearful faces in a group characterized by high CU traits (assessed by APSD) and impulsive control problems compared to the low-callous control group. A great contribution comes from Viding et al. (2012), who compared the brain reactivity to fearful versus neutral faces in three adolescent male groups: two with high conduct problems, one showing high CU traits and the other low CU traits (assessed by the *Inventory of Callous-Unemotional Traits*; ICU; Frick, 2004; Kimonis et al., 2008), and a third control group (i.e., low CU and low conduct problems). The main finding was a reduced right amygdala reactivity in response to fearful faces exhibited only in the high conduct problems and high CU traits group, providing evidence that the lower amygdala reactivity to fearful expressions is a deficit specific to CU

traits. White et al. (2012) replicated and extended this result, demonstrating a reduced amygdala reactivity to fearful expressions in a high callous (assessed by APSD) and conduct problems group (vs. a healthy control group), particularly under low attentional load conditions, suggesting that this deficit is not attributable to enhanced attentional control.

Interestingly, studies in male samples of children/adolescents have found an association between CU traits (assessed using a combined APSD/SDQ[1] system, as outlined in Dadds et al., 2005) and reduced levels of eye contact with parents (Dadds et al., 2011), a fewer and shorter eye fixations on fearful expressions (Dadds et al., 2008), and deficits in recognizing fearful facial expressions, except when participants are asked to look at the eyes gaze region (Dadds et al., 2006). These findings suggest that individuals with higher levels of psychopathic/CU traits may have deficits in recognizing fear facial expressions, which could be associated with reduced attention toward the eye gaze region. These results have been replicated in adult men samples of offenders, showing fewer fixations on the eyes regions of fear facial expressions related to PCL-R Factor 1 (Dargis et al., 2018), and undergraduate samples, showing reduced number and duration of fixations on the eyes regions, regardless of emotion, related to LSPR Primary (Gillespie et al., 2015).

More recent studies in mixed-gender undergraduate/adult community samples have also found reduced accuracy in recognizing fearful expressions associated with TriPM Meanness traits (Brislin et al., 2018; only for medium intensity of fearful expressions, Brislin & Patrick, 2019). Additionally, these results were extended with the inclusion of electrocortical measurements, demonstrating reduced amplitude of both N170 and P200 ERP components to fear expressions, specifically predicted by meanness traits (Brislin et al., 2018; Brislin & Patrick, 2019). Of particular interest, Palumbo et al. (2020) attempted to integrate measurements from different modalities using data from a large sample of twins

[1] SDQ: Strengths and Difficulties Questionnaire by Goodman (1997).

(N = 508) as a first step toward a multimodal measurement model for the affiliative capacity biobehavioral dimension. Among the neurophysiological measures of particular relevance here, N170 to fearful facial expressions (vs. happy) in a context of an emotional Stroop task and P200 to fearful facial expressions (vs. neutral) in a binocular rivalry task showed reduced amplitudes related specifically to ESI Callous-Aggression, a scale representing meanness/callous-unemotionality traits from the ESI (Krueger et al., 2007). Considering the extensive evidence, and as genetic research seems to point (see Petitclerc et al., 2019), the reduced recognition and responsivity toward fearful facial expressions could be considered as a potential endophenotype of meanness/callous-unemotional traits.

However, research on this topic has not stopped here. Recently, it has been proposed that the brain's pain network may play an important role in empathic capacity. Given the partial overlap between the areas implicated in the vicarious and personal pain processing (Decety et al., 2009), as well as the capacity of pain as a salient stimulus that attracts the attention of others and promote care and protective social functions —contrary to the aggressive behaviors and low empathic traits exhibited by high-meanness individuals—, research on pain processing (particularly pain in others) promises to be a useful and ecological means of studying the empathic deficits underlying meanness/callousness psychopathic traits. In that sense, assuming that a lower perception of ones's own pain (or a higher pain threshold) could lead to an underestimation of the pain experienced by others, higher pain tolerance has been demonstrated in individuals exhibiting higher aggressive behaviors (in a male community sample, Niel et al., 2007), and importantly, this is related to meanness/callousness traits of psychopathy (assessed by TriPM Meanness; Brislin et al., 2016; assessed by SPR-III Callous Affect; Miller et al., 2014, both in mixed-gender community samples).

Other studies have directly investigated brain reactivity to signals of pain in others, mostly using a picture-viewing paradigm where fMRI/EEG reactivity is recorded while participants view pictures of hands and feet in painful and non-painful situations (as a benchmark). Notably, some fMRI studies have incorporated a perspective instruction, asking their participants to adopt either a first-person perspective, imagining that the hand or foot appearing in the image is their own, or a third-person perspective, imagining that the hand or foot belongs to someone else. The main objective of this instruction is to separate the empathic distress reactivity elicited by viewing of distress in others from that evoked by the perception of one's own pain. Consistently, psychopathic meanness/callousness traits have been found to be related with reduced activation of relevant areas conforming the pain network (such as the anterior insula, anterior cingulate cortex, and/or the amygdala; Decety, 2011) when participants adopt a third-person perspective. This has been demonstrated in different samples and psychopathic measures: in children/adolescent samples related to ICU and PCL:YV Factor 1 (Lockwood et al., 2013; Marsh et al., 2013, respectively), and in male incarcerated and community samples related to PCL-R Factor 1 (Decety et al., 2013; but see Yoder et al., 2022 in a female incarcerated sample), and SRP affective/interpersonal factor (Seara-Cardoso et al., 2015), respectively.

In addition to neuroimaging studies, limited evidence from EEG research has found that meanness/callousness traits, as indexed by TriPM Meanness and LSRP Primary, predict a reduced LPP to pain pictures (compared to non-painful images) in a passive-picture viewing task (Brislin et al., 2022), and under the instruction of 'empathic concern' which focuses on the amount of concern felt for the person depicted in the picture (Decety et al., 2015), both in mixed-gender community samples. However, to date, there are no studies that have incorporated perspective taking to the study of EEG pain processing in relation to psychopathic traits.

Consistent with this evidence, recent studies have shown reduced affective modulation of the LPP component to aggressive interaction images in a passive-viewing task (van Dongen et al., 2018) and to affective pictures (both pleasant and unpleasant) only when the picture content was relevant for the task (Ribes-Guardiola et al., 2023) in relation to TriPM Meanness, both in mixed-gender samples. Although the results of this latest study seem to suggest an overall blunted elaborative processing of affective stimuli, regardless of their valence, more research is needed to elucidate whether this deficit linked to meanness traits is generally present towards all type of affective stimuli or specifically towards those with negative valence, as the literature have indicated so far.

In short, the lack of empathy underlying meanness traits of psychopathy seems to be accounted by reduced reactivity, and impaired ability to recognize or pay attention to signals of distress in others. Empirical studies have supported this by relating meanness/CU traits with an impaired ability to recognize negative facial expressions, especially fear, accompanied by reduced attention toward eyes gaze region and a reduced brain reactivity, evidenced by reduced N170 and P200 amplitudes and diminished right amygdala reactivity towards these facial expressions. Recent studies on pain processing seem to support these findings, demonstrating reduced reactivity to pictures of others in distress/painful situations evidenced by reduced LPP amplitudes and decreased reactivity of amygdala, anterior cingulate cortex, or insula, in relation to meanness traits. This could represent a new line of study that helps to better delineate the biobehavioral disposition of affiliative capacity. For a summary of the reviewed studies, see Table 7.

Table 7. *Summary of Reviewed Empirical Studies for the Biobehavioral Disposition of Affiliative Capacity*

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal/ Psychiatric		Community				
	M	W	M	W			
Blair (1999)	32[c]		16		APSD	SC in a passive-viewing task	Psychopathic group (-) SC to pictures of others in distress and threat
Blair et al. (1995)	40[a,b]				PCL-R	Judgement of moral transgressions	Psychopathic group (+) moral ratings of transgressions
Blair et al. (1997)	36[a,b]				PCL-R	SC in a passive-viewing task	Psychopathic group (-) SC to pictures of others in distress
Blair et al. (2001)	51[c]				APSD	Facial emotion recognition task	Psychopathic group (-) sad and fear recognition
Blair et al. (2002)	49[a]				PCL-R	Vocal emotion recognition task	Psychopathic group (-) fear vocal recognition
Blair et al. (2005)	43[c]				APSD	Vocal emotion recognition task	Psychopathic group (-) fear vocal recognition
Blair and Coles (2000)			54		APSD	Facial emotion recognition task	Psychopathic traits (-) sad and fear recognition
Brislin et al. (2016)			42	58	TriPM	Pain Tolerance	TriPM Meanness (+) pain tolerance
Brislin et al. (2018)			23/164[d]	43/90[d]	TriPM and MPQ-BF	Facial emotion recognition task – EEG	TriPM Meanness (-) fear recognition and N170, P200 amplitudes to fear expressions
Brislin et al. (2022)			60	58	TriPM	Pain picture viewing – EEG	TriPM Meanness (-) LPP to pain pictures
Brislin and Patrick et al. (2019)			62	65	TriPM	Facial emotion recognition task – EEG	TriPM Meanness (-) fear recognition (only in the medium intensity), and N170, P200, LPP amplitudes to fear expressions
Dadds et al. (2006)			33/65[d]		APSD/SDQ	UNSW Facial Emotion Task	CU traits (-) fear recognition, except when participants are instructed to look at the eyes

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal/ Psychiatric		Community				
	M	W	M	W			
Dadds et al. (2008)			100		APSD/SDQ	UNSW Facial Emotion Task – Eye tracker	CU traits (-) number and duration of fixations on eye gaze to fear expressions
Dadds et al. (2011)	92[c]				APSD/SDQ	Eye contact during free play with parents	CU traits (-) eye contact with parents
Dargis et al. (2018)	108[a]				PCL-R	Facial emotion recognition task – Eye tracker	PCL-R F1 (-) number of fixations on eye gaze to fear expressions and fear recognition
Decety et al. (2013)	121[a]				PCL-R	Pain picture viewing – fMRI	PCL-R F1 (-) anterior insula and amygdala activation to others distress
Decety et al. (2015)			39	20	LSRP	Pain picture viewing – EEG	LSRP Primary (-) LPP to pain under "empathic concern" conditions
Gillespie et al. (2015)			38		LSRP	Facial emotion recognition task – Eye tracker	LSRP Primary (-) number and duration of fixations on eye gaze to all emotional expressions
Jones et al. (2009)	17[c]		13		APSD	Facial emotion expressions viewing task – fMRI	CU group (-) right amygdala reactivity to fear expressions
Lockwood et al. (2013)	37[c]				ICU	Pain picture viewing – fMRI	ICU (-) anterior cingulate cortex and anterior insula activation to others distress
Marsh et al. (2008)	36[c]				APSD	Facial emotion expressions viewing task – fMRI	CU group (-) right amygdala reactivity to fear expressions
Marsh et al. (2013)	14[c]				PCL:YV	Pain picture viewing – fMRI	PCL-YV F1 (-) amygdala and anterior cingulate cortex activation to others distress
Miller et al. (2014)			30	74	SRP-III	Pain tolerance	SPR-III Callous Affect (+) pain tolerance
Muñoz (2009)			55		ICU	Body postures and facial emotion recognition tasks	ICU (-) fear body postures and facial expressions recognition

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal/ Psychiatric		Community				
	M	W	M	W			
Niel et al. (2007)			72		Response Choice Aggression Paradigm	Pain tolerance	Aggression (+) pain tolerance
Palumbo et al. (2020)			251[e]	257[e]	ESI-CA	Emotional Stroop task and Binocular Rivalry task (among others)	ESI-CA (-) N170 amplitude in the Stroop task and P200 amplitude in the Binocular Rivalry task, both to fear expressions
Petitclerc et al. (2019)			1005[e,f]		Similar items of ICU and ASPD	Facial emotion recognition task	CU (-) fear recognition, mediated by genetic load
Ribes-Guardiola et al. (2023)			30	114	TriPM	Picture viewing task	TriPM Meanness (-) LPP to affective pictures only when picture content was relevant for the task
Seara-Cardoso et al. (2015)			46		SRP-SF	Pain picture viewing – fMRI	Affective/Interpersonal traits (-) anterior insula, and midcingulate cortex activation to others distress
Stevens et al. (2001)	18[c]				APSD	Vocal and facial emotion recognition tasks	Psychopathic group (-) sad and fear facial recognition and sad vocalization recognition
Van Dongen et al. (2018)			36	34	TriPM	Passive picture viewing task	TriPM Meanness (-) LPP to violent pictures
Viding et al. (2012)	30[c]		16		ICU	Facial emotion expressions viewing task – fMRI	CU group (-) right amygdala reactivity to fear expressions
White et al. (2012)	15[c]		17		APSD	Facial emotion expressions viewing task – fMRi under high and low attentional load	CU group (-) right amygdala reactivity to fear expressions under the low attentional load

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal/ Psychiatric		Community				
	M	W	M	W			
Yoder et al. (2022)	109[a]				PCL-R	Pain picture viewing - fMRi	PCL-R F1 (n.s.) amygdala, anterior cingulate cortex, anterior insula reactivity PCL-R F1 (+) connectivity to sensoriomotor cortex and temporal pole to others distress

Note. M = Men; W = Women; APSD = *Antisocial Process Screening Device* (Frick & Hare, 2001); CU = Callous-Unemotional; ESI-CA = *Externalizing Spectrum Inventory – Callous Aggression* (Krueger et al., 2007); ICU = *Inventory of Callous-Unemotional Traits* (Frick, 2004; Kimonis et al., 2008); LPP = Late Positive Potential; LSRP = *Levenson Self-Report Psychopathy Scale* (Levenson et al., 1995); n.s. = no significant; PCL-R = *Hare Psychopathy Checklist–Revised* (Hare, 2003); PCL:YV = *Psychopathy Checklist: Screening Version* (Hart et al., 1995); SC = Skin conductance; SDQ = *Strengths and Difficulties Questionnaire* (Goodman, 1997); SRP-III = *Self-Report Psychopathy Scale* (Paulhus et al., 2012); SRP-SF = *Self-Report Psychopathy Scale, Short Form* (Paulhus et al., 2015); TriPM = *Triarchic Psychopathy Measure* (Patrick, 2010)

[a] This study was carried out on a criminal sample

[b] This study was carried out on a psychiatric sample

[c] This study was carried out on children with emotional and behavioral difficulties

[d] Sample size in the Study 1/Sample size in Study 2

[e] This study was carried out on a twin sample

[f] This study does not report information about gender sample

1.3.3 Inhibitory control

Inhibitory Control is a well-defined biobehavioral disposition that corresponds to the disinhibition trait construct, with established behavioral and neurophysiological correlates. It is important to note that, in the psychopathological research literature, the disinhibition construct of the triarchic model has been studied as an externalizing liability factor. The ESI externalizing proneness factor (Krueger et al., 2007) matches the triarchic disinhibition disposition, as opposed to the ESI callous-aggression factor, which is equivalent to the triarchic meanness disposition (for a review about the linkages between externalizing spectrum model and psychopathy, see Nelson & Foell, 2018).

Studies on mixed-gender youth community samples have found that externalizing tendencies, assessed by a brief factor-scale of disinhibition from ESI (ESI-DIS; Patrick et al., 2013; Yancey et al., 2013) or by several externalizing indicators, are related to poorer task performance in antisaccade (Venables et al., 2018; Young et al., 2009), and stop signal-go/no go tasks (Venables et al., 2018; modified oddball similar to a go/no go task: Brennan & Baskin-Sommers, 2018), and slower reaction times in incongruent trials in Stroop tasks (Venables et al., 2018; Young et al., 2009), supporting the deficits in behavioral control presumed to reflect disinhibitory tendencies. However, the most extensively studied indicator of externalizing proneness is the visual P3 ERP (for an extensive review, see Pasion et al., 2018). The relationship between externalizing/disinhibition and reduced P3 amplitudes has been demonstrated in various types of tasks, including oddball (Bowyer et al., 2020; Nelson et al., 2011; Venables et al., 2018; Yancey et al., 2013), flanker (Nelson et al., 2011; Venables et al., 2018; Ribes-Guardiola, Poy, Patrick et al., 2020), gambling feedback (Nelson et al., 2011; Venables et al., 2018), and go/no go tasks (Delfin et al., 2020; Brennan & Baskin-Sommers, 2018; Ribes-Guardiola, Poy, Patrick, et al., 2020). Because of the diversity of tasks used to elicit P3 and the significant impact of the task parameters on

P3 amplitude, it has been challenging for empirical studies to determine the neurocognitive process that P3 reflects in its association with trait disinhibition.

Venables et al. (2018) recently shed light on this issue by attempting to disentangle the relationship between P3 amplitude and task performance indicators. To achieve this, they collected data from a mixed-gender sample of undergraduates on several self-report measures, including brief scale versions specifically selected to index disinhibitory tendencies from well-known inventories (i.e., ESI-DIS, Patrick et al., 2013; MPQ-DIS, *Multidimensional Personality Questionnaire-Disinhibition scale*, Brislin et al., 2015, 2017; PPI-DIS, Hall et al., 2014) and the *Socialization Scale* (SO, Gough, 1960), along with P3 amplitude to visual-motor tasks (novelty oddball, flanker, and pseudo-gambling tasks) and behavioral performance indicators from inhibitory control tasks (accuracy on stop signal and antisaccade tasks, and reaction time on a Stroop and sustained attention response tasks). Through the use of structural equation modeling, domain factors were formed by covarying the measures of each response domain (scale, behavioral, and neurophysiological), and each factor loaded in turn onto a higher-order, cross-domain inhibition-disinhibition factor, with loadings of .40, -.60, and -.77, respectively. The strong load of both the behavioral and P3 factors to the cross-domain inhibition-disinhibition factor accounted for their mutual associations with the self-report assessment of disinhibition, indicating that the variance relating P3 with disinhibition could reflect a common process leading to impaired performance on inhibitory control tasks.

In addition, it has been posited that the association between reduced P3 amplitude and high disinhibition traits could entail an elevated genetic load. Thus, Young et al. (2009) collected data on behavioral disinhibition, including conduct disorder and ADHD symptoms and rates of substance abuse (tobacco, alcohol, and other illicit drugs), from a larger sample of mixed-gender twins (n = 584) at ages 12 and 17. They also collected data on laboratory

inhibitory control tasks performance, such as the Stroop, stop-signal and antisaccade tasks, only at the age of 17. From this data, a trait disinhibition factor and a task-based inhibition factor were constructed, which were robustly and negatively related, with this association largely attributable to shared genetic influences. In another twin study that also incorporated brain measures, Yancey et al. (2013) demonstrated that disinhibition traits, as measured by a factor extracted from a 30-item subset of the 100-items version of ESI (Krueger et al., 2007), mediated the relationship between antisocial/addictive symptom/disorders and P3 amplitude (in an oddball task), and that this relationship was largely explained by the genetic liability shared between the disinhibition traits and externalizing symptom/disorders. Similarly, Venables et al. (2017) demonstrated that a factor composed of scale measures (the same as Yancey et al., 2013) along with the Aggression scale of the brief-form Multidimensional Personality Questionnaire; MPQ-BF, Patrick et al., 2002) and brain indicators (oddball P3) of disinhibition, resulted in a purer index of genetic liability for substance abuse problems.

Although there is limited evidence linking disinhibitory tendencies with error processing ERPs, studies have found a reduction in the Error Related Negativity (ERN) in mixed-gender/male adult samples in a lexical decision stop signal task related to impulsive-antisocial traits (ESI; Heritage & Benning), in a Simon task related to TriPM Disinhibition (Pasion et al., 2016), and in go/no go tasks related to externalizing proneness (100-items version of ESI along with other externalizing measures; Hall et al., 2007) and TriPM Disinhibition (Paiva et al., 2020; but not in a flanker task: Ribes-Guardiola, Poy, Patrick et al., 2020). It is worth noting that, in the latest study, the covariance among ERP extracted from the go/no-go and a flanker task was examined. The study found two factors, each consisting of one error-locked and one stimulus-locked ERP. One factor was related to parietal reactivity (P3/Pe), and the other was related to frontal reactivity (N2/ERN). TriPM

Disinhibition showed a significant association with the P3/Pe factor, but not with the N2/ERN factor. This suggests that the shared variance among P3 and Pe components reflects a common process accounting for the individual differences in disinhibition traits. However, the no-go ERN contained distinct disinhibition-related variance that differed from that shared with the N2 component. Interestingly, a recent study by Pasion et al. (2023) has demonstrated that P3 and ERN factors, derived from a flanker task, a flanker-threat task, and a go/no go task, independently predict externalizing factor scores, but not the specific problem dimensions (i.e., antisocial, alcohol, and drug use symptoms, and effortful control) loading on it. These results suggest, first, that reduced P3 and ERN amplitudes represent distinct neural processes contributing to externalizing proneness, and, second, that both index a general liability for general (but not specific) externalizing problems.

In summary, the evidence suggests that deficits in the elaborative processing of significant stimuli account for the lack of behavioral and affective control in high-disinhibition individuals, as indicated by a robust negative relationship between P3 amplitude and trait disinhibition. This association appears to reflect a common process that leads to impaired inhibitory control, which involves a high genetic load. Further research on error-related potentials could help to clarify potential deficits in error monitoring processing related to disinhibition, and thus potentially provide additional physiological indicators for the inhibitory control biobehavioral disposition. Please refer to Table 8 for a summary of the reviewed studies.

Table 8. *Summary of Reviewed Empirical Studies for the Biobehavioral Disposition of Inhibitory Control*

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal		Community				
	M	W	M	W			
Bowyer et al. (2020)			178	156	PID-5 Disinhibition factor	EEG: Flanker and oddball tasks	PID-5 Disinhibition factor (-) P3 factor
Brennan and Baskin-Sommers (2018)			59	30	DAST-A, AUDIT, SSS-V Disinhibition subscale	Behavioral performance and EEG: Modified oddball task	Externalizing/Disinhibition factor (-) oddball task accuracy and P3 amplitude
Delfin et al. (2020)	27[a]		20		ESI-BF Disinhibition scale	EEG: Go/no go task	MDOs group and ESI-BF Disinhibition (+) No-go P3 latency/ MDOs group (-) No-go P3 amplitudes
Hall et al. (2007)			34	58	ESI-100	EEG: Flanker task	High externalizing group (-) ERN amplitude
Heritage and Benning (2013)			39	50	MPQ-BF	Behavioral performance and EEG: Stop signal task	MPQ-BF Impulsive Antisociality (+) SSRT and (-) ERN amplitude
Nelson et al. (2011)			33	55	ESI-100, ADS, SDAST, BHR, SO	EEG: Flanker, gambling feedback and oddball tasks	ESI-100/Disinhibition factor (-) EEG factor/flanker response-ERN, flanker-target P3, and gambling-feedback P3 amplitudes
Paiva et al. (2020)			30	25	TriPM	EEG: Go/no-go task	TriPM Disinhibition (+) ERN – CRN difference
Pasion et al. (2016)			32		TriPM	EEG: Simon task	TriPM Disinhibition (-) ERN amplitude
Pasion et al. (2023)			84	98	PAI, ATI	EEG: Flanker, flanker-threat and go/no go tasks	Externalizing factor (-) ERN factor and P3 factor (independently)
Ribes-Guardiola, Poy, Patrick et al. (2020)			41	101	TriPM	EEG: Go/no-go and flanker tasks	TriPM Disinhibition (-) No-go P3, flanker-incongruent P3, and no-go ERN amplitudes

Published source	Sample				Psychopathic Measures	Paradigm	Main findings
	Criminal		Community				
	M	W	M	W			
Venables et al. (2018)			85	64	ESI-DIS, MPQ-DIS, PPI-DIS and SO	Behavioral performance: Stop signal, antisaccade, Stroop, SART tasks EEG: oddball, flanker, pseudo-gambling (choice-feedback) tasks	Disinhibition (-) antisaccade accuracy, target P3 (oddball task), and flanker response P3e amplitudes (flanker task) Disinhibition (+) SART RT variability
Yancey et al. (2013)			199[b]	220[b]	ESI-100	EEG: Oddball task	ESI Trait Disinhibition (-) target-P3 amplitude
Young et al. (2009)			315[b]	269[b]	Conduct disorder and ADHD symptoms, CID-SAM, TPQ novelty seeking	Behavioral performance: Stop signal, antisaccade and Stroop tasks	Trait/Behavioral disinhibition factor (-) task-based-inhibition factor

Note. M = Men; W = Women; ADHD = Attention-deficit/hyperactivity disorder; ADS = *Alcohol Dependence Scale* (Skinner & Allen, 1982); ATI = *Adult Temperament Inventory* (Evans & Rothbart, 2007); AUDIT = *Alcohol Use Disorders Identification Test* (Reinert & Allen, 2007); BHR = *Behavior Report on Rule-Breaking* (items from several measures: Clark & Tifft, 1966; Hindelang et al., 1981; Nye & Short, 1957); CID-SAM = *Composite International Diagnostic Instrument–Substance Abuse Module* (Cottler et al., 1989); DAST-A = *Drug Abuse Screening Test* (Skinner, 1982); ESI-100 = *Externalizing Spectrum Inventory 100-item version* (Krueger et al., 2007); ESI-BF = *Externalizing Spectrum Inventory–Brief Form* (Patrick et al., 2013); ESI-DIS = *Externalizing Spectrum Inventory–Brief Form Disinhibition Scale* (Patrick et al., 2013); MPQ-BF = brief form of *Multidimensional Personality Questionnaire* (Patrick et al., 2002); MPQ-DIS = *Multidimensional Personality Questionnaire Disinhibition Scale* (Brislin et al., 2015, 2017); PAI = *Personality Assessment Inventory* (Morey, 2004); PID-5 = *Personality Inventory for DSM-5* (Krueger et al., 2012); PPI-DIS = *Psychopathic Personality Inventory Disinhibition Scale* (Hall et al., 2014); SART = Sustained Attention to Response task; SDAST = *Short Drug Abuse Screening Test* (Skinner, 1982); SO = *Socialization Scale* (Gough, 1960); SSS-V = *Sensation-Seeking Scale V* (Zuckerman et al., 1978); TPQ = *Cloninger's Tridimensional Personality Questionnaire–Short Form* (Heath et al., 1994); TriPM = *Psychopathy Triarchic Measure* (Patrick, 2010)

[a] This study was carried out on mentally disordered offenders (MDOs)

[b] This study was carried out on a twin sample

EXPERIMENTAL STUDIES

CHAPTER 2. Aims and hypothesis

The **general objective** of this research is to provide empirical evidence on the biobehavioral dispositions that underlie the trait constructs of the triarchic model of psychopathy by identifying physiological indicators.

Threat Sensitivity – Boldness:

Study 1. Psychopathy and heart rate variability: A new physiological marker for the adaptive features of boldness

Specific objective 1: To investigate the relationship between resting vagally-mediated heart rate variability (vmHRV) —a physiological index of emotional self-regulation— and boldness, controlling for the meanness and disinhibition dispositions of the triarchic model of psychopathy, in order to examine the potential of vmHRV as a positive physiological correlate of boldness.

> **Hypothesis 1:** High vmHRV will be positively associated with self-reported boldness scores, after controlling for meanness and disinhibition scores.

Study 2. Low defensive cardiac reactivity as a physiological correlate of psychopathic fearlessness: Gender differences

Specific Objective 2: To investigate whether a reduced Cardiac Defense Response (CDR), especially its second accelerative component (A2), which would be indexing a deficient reactivity of the defensive motivational system, could serve as an additional physiological indicator of the affective/interpersonal traits of psychopathy.

Hypothesis 2: Lower defensive reactivity, as indexed by A2, will be specifically related to the fearless dominance factor scores of the PPI-R, and not to the self-centered impulsivity factor or the coldheartedness scale scores.

Affiliative Capacity – Meanness:

Study 3. Psychopathic callousness and perspective taking in pain processing: An ERP study

Specific Objective 3: To investigate whether perspective taking affects electrocortical responses to pain processing in relation to the callousness traits of psychopathy.

Hypothesis 3: Callousness factor scores will be associated with reduced Late Positive Potential (LPP) amplitudes to pain pictures in the imagine-other perspective condition, but not in the imagine-self condition.

Inhibitory Control – Disinhibition:

Study 4. Disinhibition and electrocortical correlates of inhibitory control

Specific Objective 4: To investigate the relationship between disinhibition traits of psychopathy and the amplitude of ERPs components indexing inhibitory processing and error monitoring.

Hypothesis 4: Disinhibition factor scores will be inversely related to Stop P3, Stop ERN, and Flanker ERN amplitudes in a modified flanker-stop-signal task.

CHAPTER 3

STUDY 1 Psychopathy and heart rate variability: A new physiological marker for the adaptive features of boldness

Study 1

Psychopathy and Heart Rate Variability: A New Physiological Marker for the Adaptive Features of Boldness

Pilar Segarra, Rosario Poy, Victoria Branchadell, Pablo Ribes-Guardiola, & Javier Moltó

Affective Neuroscience Lab, Department of Basic and Clinical Psychology, and Psychobiology. Universitat Jaume I, Castelló, Spain

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Abstract

The boldness disposition of the triarchic model of psychopathy is theorized to entail, aside from maladaptive proclivities (narcissism, fearless risk-taking), some adaptive features (e.g., immunity to stressful events, high self-esteem, and emotional resilience) that seem to predispose high boldness individuals to an effective emotional regulation in response to environmental demands. The high frequency band of heart rate variability —an index of parasympathetic cardiac vagal activity— is a well-validated physiological index of emotional self-regulation and mental health resilience. The aim of this study was to examine the unique predictive contributions of triarchic dispositions of boldness, meanness, and disinhibition on resting vagally-mediated heart rate variability (vmHRV) in a sample of 241 undergraduates (60 men) assessed via the Triarchic Psychopathy Measure (TriPM; Patrick, 2010). A multiple regression analysis was conducted on vmHRV in which TriPM Boldness, Meanness, and Disinhibition scores were entered as predictors, along with gender, age, body mass index, mean resting heart rate, and respiratory activity. Results showed that only TriPM Boldness —but not Meanness or Disinhibition— scores significantly predicted vmHRV (positively), thus evidencing that adequate emotional self-regulation is one of the adaptive features encompassed by the boldness disposition. These findings encourage further use of vmHRV as a physiological marker of boldness and contribute to shedding light on the nomological network surrounding the construct of boldness in psychopathy.

Keywords: Triarchic Model of Psychopathy, Heart Rate Variability, Boldness

Introduction

Nowadays, the triarchic model (Patrick et al., 2009) is one of the most influential conceptualizations of psychopathy because, among other reasons, it links symptomatic features of psychopathy to neurobiological systems and processes. Specifically, the triarchic model conceptualizes psychopathy in terms of three distinct but interrelated biobehavioral dispositions or psychological constructs related to normal-range personality traits that have direct neural and behavioral referents (Patrick & Drislane, 2015).

The disinhibition disposition entails a phenotypic style characterized by impulsiveness, lack of behavioral restraint, and difficulties in affective control. Disinhibition is viewed as a liability factor for externalizing psychopathology and corresponds to the neurobehavioral dimension of inhibitory control that putatively reflects frontal-brain-based differences in the capacity for behavioral restraint. The meanness disposition encompasses a constellation of phenotypic features linked to a lack of ability to understand other people's feelings and welfare that are manifested behaviorally by strategic exploitation of others, callousness, lack of close attachments and predatory aggression, among others. Meanness is thought to reflect a biologically based predatory orientation involving deficient empathic sensitivity and weak affiliation/attachment capacity that is expressed in antagonistic externalizing psychopathology. The boldness disposition follows Cleckley's (1941/1976) concept of psychopathy as a personality disorder that entails an outward appearance of psychological (adaptive) normality that masks a severe pathological maladjustment. With this in mind, boldness is theorized to represent the paradox underlying psychopathy, thus comprising a phenotypic style characterized by maladaptive proclivities (e.g., narcissism, fearless risk-taking, and failures to learn from punishment experiences) in conjunction with certain adaptive features (e.g., low levels of anxiety or fear, immunity to stress, a socially potent interpersonal style). Boldness is hypothesized to correspond to the neurobehavioral

dimension of threat sensitivity, reflecting individual differences in the reactivity of the brain's defensive motivational system —based on the amygdala and affiliated structures. Several studies support this by showing diminished startle potentiation during exposure to threat stimuli (e.g., Esteller et al., 2016), reduced cortical late positive potential (LPP) to negative versus neutral pictures during passive viewing (Ellis et al., 2017), and deficient CS+/CS- electrodermal (López et al., 2013) and electrocortical differentiation (Paiva et al., 2020) in fear conditioning in high fearless dominance/boldness individuals. In addition, boldness has shown consistent negative associations with self-report measures of fearfulness/anxiety and internalizing symptomatology (Latzman et al., 2020; Poy et al., 2014). It is worth nothing that boldness has been related to maladaptive response perseveration in the face of increasing punishment contingencies (Ribes-Guardiola et al., 2020), but also to an enhanced task switching performance under threat conditions (Yancey et al., 2019), suggesting that resistance to the impact of danger or punishment cues might involve adverse outcomes or an adaptive resilient style depending on the situation. Boldness has recently been shown to predict greater emotional well-being during COVID-19 outbreak (Sica et al., 2021) and reduced frequency of protective behaviors (Paiva et al., 2021), supporting the idea that boldness' protection from emotional distress may in turn lead to behaviors that increase the risk of getting the disease. From this standpoint, it seems likely that their low threat sensitivity might be somehow responsible for the fact that high boldness individuals show both good emotional regulation (resilient functioning) and behavioral dysregulation (reckless and unrestrained responses) under stressful or threatening situations. Although the neurophysiological correlates of threat processing deficits linked to triarchic boldness (e.g., reduced fear-potentiated startle, deficient fear conditioning, diminished LPP to aversive stimuli) are relatively well-studied, empirical evidence about physiological

correlates for the potentially adaptive features of boldness (as adequate emotional regulation) is still lacking.

Resting heart rate variability (HRV) —the variation in the time interval between consecutive heartbeats in milliseconds— is currently widely considered as a well-validated physiological index of emotional regulation capacity (see Appelhans & Luecken, 2006, and Balzarotti et al., 2017, for reviews). The high frequency band of HRV (HF-HRV) —which reflects the parasympathetic influence (via the vagus nerve) on the sinoatrial node of the heart (Berntson et al., 1997)— has been specifically proposed to be a transdiagnostic biomarker of self-regulation (the ability to regulate behavioral, emotional, and cognitive processes) and mental health resilience (e.g., Beauchaine & Thayer, 2015; Thayer et al., 2012).

In this line, the neurovisceral integration model (Thayer & Lane, 2000, 2009) emphasizes the interplay between this vagally-mediated cardiac activity and the brain structures related to emotional processing, and suggests that several prefrontal cortical areas modulate cardiovascular activity via the inhibition (in safe contexts) and disinhibition (under threat conditions) of the amygdala (see Thayer & Lane, 2000, for a review of the three routes by which the amygdala would lead to cardiac vagal control). Given that HF-HRV is positively related to amygdala-medial prefrontal cortex functional connectivity and shows similar associations with criterion measures of the nomological network of boldness (i.e., positive correlations with stress immunity, emotional resilience, subjective well-being, and negative relationships with internalizing psychopathologies and self-report measures of fearfulness; see Thayer et al., 2012), we hypothesized that HF-HRV and triarchic boldness would be positively related. The only study that has examined the relationship between vagally-mediated heart rate variability and psychopathy features supports this hypothesis to some extent. Thus, in a sample of male prisoners, Hansen et al. (2007) found that the

interpersonal facet of psychopathy —which partially captures the measurement domain of boldness (Venables et al., 2014)— explained most of the HF-HRV. However, the predictive contribution of boldness features to HF-HRV is yet to be directly tested.

The main goal of this study was to explore the association between resting vagally-mediated heart rate variability (vmHRV) and boldness above and beyond the meanness and disinhibition dispositions of the triarchic model of psychopathy, in order to examine the usefulness of vmHRV as a positive, physiological correlate of boldness. On the basis of the close link between vagally-mediated cardiac activity and emotional regulation ability via amygdala inhibition/disinhibition (see Thayer et al., 2009), and the fact that certain features of the boldness disposition are theoretically related to emotional adjustment such as immunity to stressful events or emotional resilience (see Patrick et al., 2009), it was expected that high vmHRV would be exclusively associated with higher self-reported boldness scores.

Method

Participants

Study participants were 245 volunteer undergraduate psychology students between the ages of 18 and 25 years. No participant reported diagnosis of mental disorder or pharmacological treatment that could alter cardiac activity at the time of testing. Four male undergraduates were excluded from the analysis after participation due to alcohol use 24 h prior to the ECG recording. The final sample comprised a total of 241 Caucasian participants (181 women, 60 men), with a mean age of 20.01 years (*SD* = 1.95). All participants signed a written informed consent form and received academic credit for their participation.

Measures

Triarchic Psychopathy Measure (TriPM; Patrick, 2010). The TriPM is a 58-item self-report inventory specifically designed to measure the three phenotypic domains proposed in the triarchic model of psychopathy (Patrick et al., 2009). The items were

answered using a 4-point Likert scale (3 = *true*, 2 = *somewhat true*, 1 = *somewhat false*, 0 = *false*). The Spanish translation of the TriPM had previously shown good criterial validity (Esteller et al., 2016; Poy et al., 2014). Internal consistencies (alpha coefficients) for Boldness, Meanness, and Disinhibition scores in the current sample were .78, .81, and .79, respectively.

Heart Rate Variability (HRV). The electrocardiogram (ECG) was recorded using 8 mm In Vivo Metric Ag/AgCl surface electrodes (Standard Lead II placement) at a 1000 Hz sampling rate using a Coulbourn V75-04 High Gain Isolated Bioamplifier (with high and low cutoffs set at 40 Hz and 8 Hz, respectively). Electrodes were placed on the right wrist and the left ankle; the ground electrode was placed on the right ankle. Analog ECG signals provided by VPM software (Cook, 2002) were transferred to Kubios HRV analysis Package 2.2 (Tarvainen et al., 2014). Artifacts detected within the R-to-R series were removed by applying an artifact correction level that differentiated abnormal inter-beat-intervals (IBIs), in milliseconds, from median IBIs using a piecewise cubic spline interpolation method. Following established guidelines (Laborde et al., 2017), the R-to-R intervals were subjected to an autoregressive power spectrum density method (AR model order = 16) to obtain absolute powers of high frequency (HF; 0.15-0.4 Hz) band, an index of vagal cardiac activity mediated by the parasympathetic system (Berntson et al., 1997). An ECG-derived respiration (EDR) measure was also computed using the Kubios' algorithm to estimate sinus respiratory arrhythmia from peak HF-HRV values.

Procedure

During the first semester, participants filled out the TriPM at sessions with a maximum of 45 subjects. ECG recording was conducted individually in an isolated, dimly lit room during the second semester. All participants were asked not to smoke, engage in vigorous physical activity, or drink caffeine 2 h before the experiment.

After arriving at the laboratory, participants were weighed, measured, and informed about the experimental session protocol, which had been approved by the ethics committee of the university and was carried out in accordance with the Declaration of Helsinki. The electrodes were then attached to the participants and, after a period of acclimatization, the 5-min baseline-resting ECG recording started. As recommended by guidelines reports (e.g., Laborde et al., 2017; Quintana et al., 2016), participants were seated in a comfortable armchair (with knees at a 90º angle, both feet resting on the footrest and hands on the armrests) and were asked to breathe spontaneously and to remain with their eyes open throughout the recording time. After a short break, students who voluntarily kept on participating in the experimental session underwent a passive picture viewing task that included measurement of startle reflex responses (results reported in Esteller et al., 2016). Participants then completed a brief survey asking for their age, alcohol consumption in the 24 h prior to the experiment, and time elapsed since the last tea or coffee consumption.

Statistical analysis

Prior to all analyses, HF-HRV power values in m^2 were transformed into natural logarithms (lnHF) to fit to the assumptions of the linear analysis. A three-stage regression analysis was conducted to test our main hypothesis about the unique predictive contribution of boldness disposition to vmHRV. Step 1 included gender (0 = men, 1 = women), age (in years), body mass index (BMI; Kg/m^2), mean resting heart rate (HR; in beats per minute), and ECG derived respiration (EDR) as predictors, given their demonstrated influence on inter-individual differences in HRV (e.g., Laborde et al., 2017; Quintana et al., 2016). TriPM Meanness and Disinhibition scores were entered at Step 2, and TriPM Boldness scores were added at Step 3 to test for the predictive contribution of this disposition alone to vmHRV after controlling for its overlap with the physiological variables and the other triarchic dispositions.

Table 1. *Descriptive Statistics and Pearson's zero-orden Correlations for Study Variables (left side), and Regression Coefficients Predicting Vagally-Mediated Resting Heart Rate Variability (lnHF) at Step 3 of the Hierarchical Regression Model (right side)*

Variable	M	SD	1	2	3	4	5	6	7	Predictors	B	SE	*β*	t	*p*	95% CI [LL, UL]
										Intercept	8.648	0.823		10.510	**.000**	
1.ln (HF)	6.61	0.87	___							Gender	0.340	0.143	**.169**	2.377	**.018**	[0.029, 0.308]
2.Age	20.01	1.95	. 09	___						Age	0.003	0.026	.007	0.116	.907	[-0.108, 0.122]
3. BMI (kg/m^2)	22.88	3.92	.02	.05	___					BMI	0.024	0.013	.107	1.795	.074	[-0.010, 0.225]
4.EDR	0.25	0.05	**-.22**	**-.17**	**.14**	___				EDR	-3.145	1.114	**-.168**	-2.825	**.005**	[-0.286, -0.051]
5.HR (bpm)	78.02	12.23	**-.45**	-.13	.04	.10	___			HR	-0.033	0.004	**-.468**	-7.519	**.000**	[-0.591, -0.346]
6.Boldness	27.83	8.13	**.18**	.09	**-.13**	**-.18**	**-.13**	___		Boldness	0.014	0.006	**.127**	2.114	**.036**	[0.009, 0.246]
7.Meanness	11.52	7.01	.12	.02	.02	**-.19**	**-.13**	**.22**	___	Meanness	0.008	0.009	.067	0.907	.366	[-0.078, 0.211]
8.Disinhibition	17.66	7.98	**.13**	-.04	.03	-.11	**-.18**	.08	**.51**	Disinhibition	0.001	0.007	.012	0.182	.856	[-0.119, 0.143]

Note. Significant associations are highlighted in bold (for values greater than |.16|, $p < .01$).
CI = confidence interval for *β*; LL = lower limit, UL= upper limit

Results

Descriptive statistics and bivariate correlations between variables of interest are shown in Table 1 (left). Regression coefficients predicting lnHF at Step 3 of the hierarchical regression model are shown in Table 1 (right)[1]. As expected, HR (β = -.47) and EDR (β = -.19) were significant predictors of vmHRV at Step 1, $F(5, 235) = 14.94$, $p < .001$, $R^2 = .241$. Neither TriPM Meanness nor TriPM Disinhibition scores contributed to the prediction of lnHF at Step 2 ($\Delta R^2 = .006$, $p = .41$), in which HR (β = -.47), EDR (β = -.18), and Gender (β = .14) were significant predictors, $F(7, 233) = 10.92$, $p < .001$, $R^2 = .247$. TriPM Boldness scores significantly increased the explained variance at Step 3 ($\Delta R^2 = .014$, $p = .036$), overall model $F(8, 232) = 10.26$, $p < .001$, $R^2 = .261$. Thus, consistent with our hypothesis, inter-individual differences in boldness accounted for physiological differences in vmHRV above and beyond the triarchic dispositions of meanness and disinhibition, even after adjusting for the contribution of known relevant covariates.

Discussion

This was the first study to focus explicitly on the relationship between cardiac vagal activity —measured via resting vagally-mediated heart rate variability (vmHRV)— and the behavioral dispositions of boldness, meanness, and disinhibition of the triarchic model of psychopathy (Patrick et al., 2009). Our results demonstrated, as hypothesized, that vmHRV was positively related to TriPM Boldness scores. This finding is consistent with numerous studies showing that both HF-HRV and boldness can be adequate indicators —coming from different assessment domains (physiological and self-report, respectively)— of an adaptive and healthy psychological functioning. Thus, it is important to highlight that heart rate

[1] The sensitivity power analyses conducted in G*Power (see Faul et al., 2007) revealed that the linear multiple regression (fixed model, R^2 deviation from zero) with 241 participants and eight predictors would be sensitive to an effect size of $f^2 = 0.06$ (critical $F = 1.98$).

variability has traditionally been considered as a protective factor against internalizing symptomatology (e.g., Beauchaine & Thayer, 2015), just as the boldness disposition of the triarchic model has recently been demonstrated to be (e.g., Latzman et al., 2020). Furthermore, these two measures are linked with psychologically well-adjusted personality variables such as high emotional resilience, good executive functioning, high-self-esteem, and subjective well-being —for example, see Sleep et al. (2019) for correlations of boldness, and Holzman & Bridget (2017) and Thayer et al. (2012) for relationships of heart rate variability with aforementioned variables.

Although the relevance of the triarchic boldness disposition for psychopathy has recently been the subject of intense scientific debates (for a review, see Lilienfeld et al., 2012; Miller & Lyman, 2012)—, the fact that this disposition is assumed to reflect individual differences in the reactivity of the brain's defensive motivational system to threat signals makes it especially relevant to the field of psychopathy. Boldness is the only triarchic disposition consistently related to low neuroticism and high extraversion (e.g., Miller et al., 2016; Poy et al., 2014), personality traits theoretically associated with a weak behavioral inhibition system (BIS; Gray, 1987, and Gray & McNaughton, 2000), responsible for inhibiting or regulating approach behavior that might lead to adverse outcomes in response to threats of punishment. Indeed, research is beginning to learn about the specific role of boldness in some well-documented threat processing psychopathic deficits including maladaptive response perseveration in the face of punishment (Ribes-Guardiola et al., 2020), diminished startle responses to threat pictures (Esteller et al., 2016), and reduced amplitudes of late positive potentials to aversive signals (Ellis et al., 2017; Paiva et al., 2020).

Together with the aforementioned markers, the findings of the present study suggest that vmHRV could also be incorporated into psychopathy research as a potential new

physiological indicator of boldness and, in addition, it could be extremely useful to explore the role of threat sensitivity and parasympathetic cardiac activation in certain phenotypic outcomes of psychopathy. The vmHRV indexes the capacity of the prefrontal cortex to modulate subcortical circuits of fear responses, and the association between vmHRV and individual differences in boldness could also contribute to shedding light on the top-down regulatory control processes of negative emotions that have recently been hypothesized to underlie this disposition of psychopathy (see Yancey et al., 2019). This potential etiological hypothesis would be consistent both with evidence of impaired prefrontal-amygdala connectivity in psychopathy (e.g., Motzkin et al., 2011), and with proposed links between the prefrontal cortex and the septo-hippocampal system (Gray & McNaughton, 2000), the core neurobiological substrate of the BIS. At this point, two key aspects of the triarchic model of psychopathy need to be emphasized (see Patrick, 2018): (1) this model does not assume that the dispositions of boldness, meanness, and disinhibition should correspond directly with neurobiological systems of threat reactivity, affiliative capacity, and inhibitory control, respectively, but rather proposes that these dispositions, as dimensions of variation in biobehavioral functioning across individuals, can be operationalized using indicators from different measurement domains and serve to establish bridges between clinical problems and neurobiological processes, and (2) from this standpoint, psychopathological symptoms reflect the interplay of biobehavioral systems with environmental influences over time and developmental stages. Consequently, the first step in understanding the etiology of psychopathy would be to identify multiple correlates of these systems and to explore how individual differences in their functioning are related to distinct configurations of psychopathic expressions. The relevance of the finding of the present study lies in the identification of resting heart rate variability as a marker of boldness and, therefore, stands

as the first step toward identifying variables from the physiological and behavioral response domains that correlate with the biobehavioral system of threat reactivity.

However, these results should be considered in light of some limitations. On the one hand, the use of a sample of undergraduates with a small age range makes it difficult to generalize our results to other populations and age ranges. In addition, the use of a sample with unequal gender ratio may influence our results, although participants' gender was included as a variable in the analyses. Future research in gender-balanced samples of different types (community, clinical, criminal) and ages (child, adolescent, adult) is needed to investigate these issues. On the other hand, the use of self-report instruments to assess dimensions in undergraduates may narrow the range of scores on a particular triarchic disposition of psychopathy. Moreover, it would be desirable to incorporate different operationalizations of the triarchic dispositions other than the TriPM scales. Future work should also try to operationalize constructs of psychopathy using indicators from multiple domains of measurement (physiological, behavioral, self-report), in order to establish a psychoneurometric quantification of biobehavioral dispositions.

Despite these limitations, the present study provides evidence supporting vmHRV as a potential physiological correlate of the boldness disposition that could help advance our understanding of the different etiological processes and pathways underlying psychopathic dispositions as specified in the triarchic model. Once the patterns of associations (convergent and discriminant) of vmHRV with other reliable multiple indicators have been established, researchers should be able to determine whether this indicator contributes to the emergence, expression, and temporal course of a given psychopathic phenotype. This research strategy will not only provide a comprehensive picture of the correlates associated with (maladaptive and adaptive) features of the boldness disposition, but it will undoubtedly also have a major impact on the

understanding of psychopathy in neurophysiological terms, thus contributing to the National Institute of Mental Health's Research Domain Criteria (NIMH RDoC; Insel et al., 2010) framework, which promotes a multidomain, biobehavioral approach to examine the nature of mental health and clinical psychopathologies.

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CHAPTER 4

STUDY 2 Low defensive cardiac reactivity as a physiological correlate of psychopathic fearlessness: Gender differences

Study 2

Low Defensive Cardiac Reactivity as a Physiological Correlate of Psychopathic Fearlessness: Gender Differences

Victoria Branchadell, Rosario Poy, Pilar Segarra, Pablo Ribes-Guardiola, & Javier Moltó

Affective Neuroscience Lab, Department of Basic and Clinical Psychology, and Psychobiology. Universitat Jaume I, Castelló, Spain

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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.

Keywords: Psychopathic Personality Inventory-Revised (PPI-R), Fearless Dominance, Fearlessness, Cardiac Defense Response (CDR)

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, 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.

Method

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).

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.

Instruments

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).

Physiological data recording and reduction

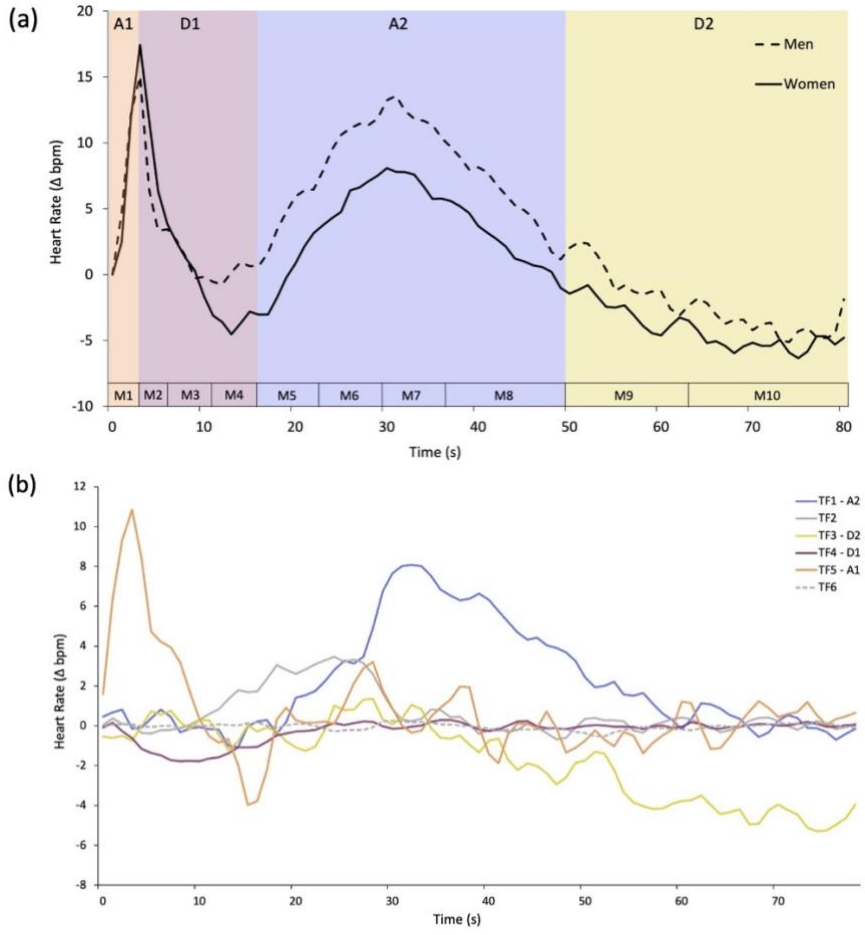
Stimuli control, data acquisition and reduction were accomplished using VPM software (Cook, 2002). Ag/AgCl surface electrodes (Standard Lead II) filled with hypertonic electrolyte paste provided 1000 samples/second electro-cardiograph analogical signals to a Coulbourn V75-04 High Gain Bioamplifier, and 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 13s (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 Figure 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 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. Figure 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.

Figure 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

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.

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.

Results

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, $F(1,154) = 158.97$, $p < .001$, $\eta_p^2 = .508$, 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, ts(154) > 2.02, ps < .045, ds > 10.63. Figure 1a illustrates these findings.

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, η_p^2 = .041. There were no other significant main (Fs < .11; ps > .747, η_p^2 < .001) nor interaction effects (Fs < 1.65; ps > .164, η_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.

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

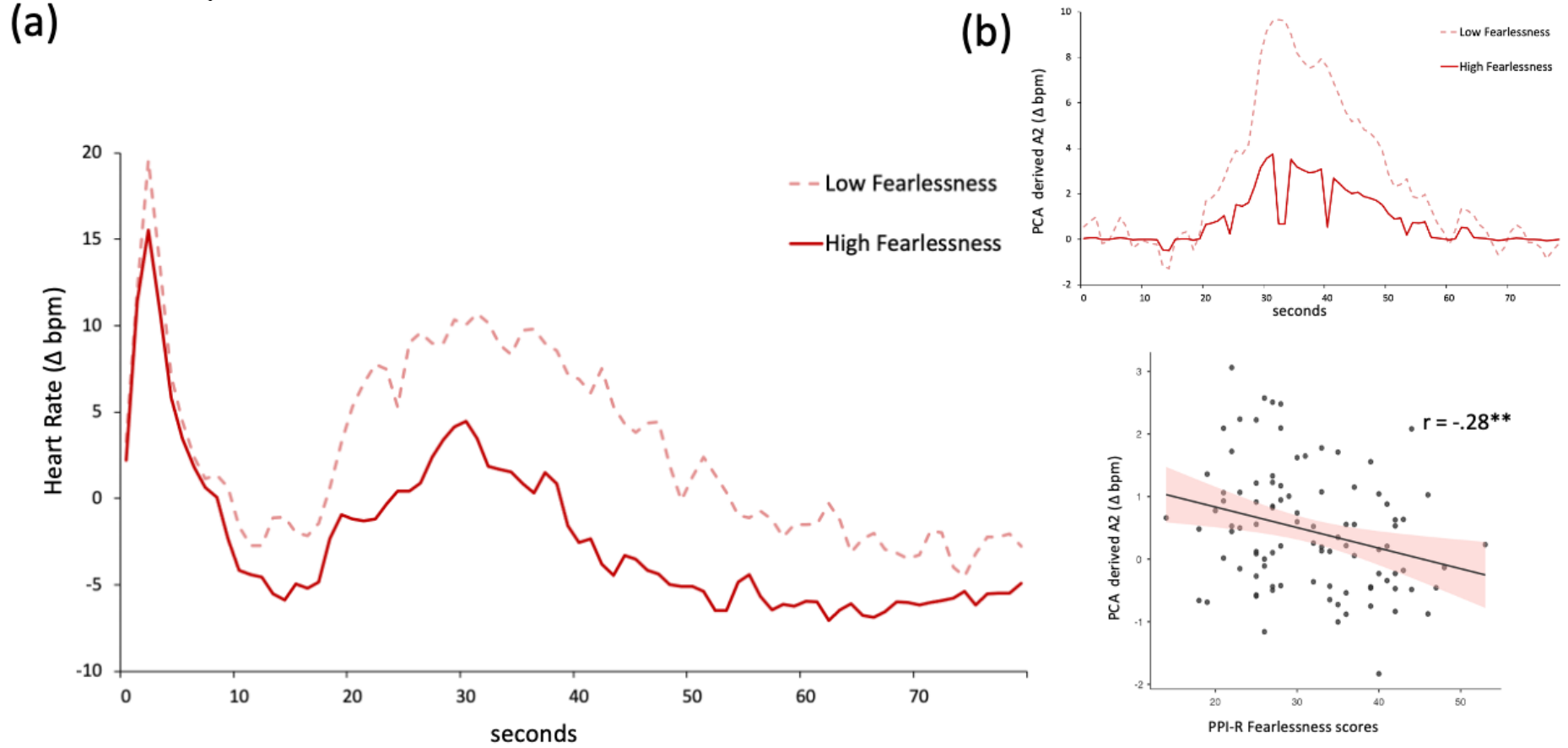
Note.
Significant comparisons are highlighted in bold.

In order to further explore the role of scales conforming the PPI-R 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 r*s (96) > -.21, ps < .05. Figure 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 Figure 2b), even after controlling for Stress Immunity and Social Influence scores (see Table 3).

Figure 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; $^{**}p < .01$

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.
*$p < .05$, ** $p < .01$

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 high 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, Iacono, 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 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.

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CHAPTER 5

STUDY 3 Psychopathic callousness and perspective taking in pain processing: An ERP study

Study 3

Psychopathic Callousness and Perspective Taking in Pain Processing: An ERP Study

Victoria Branchadell, Rosario Poy, Pablo Ribes-Guardiola, Pilar Segarra, & Javier Moltó

Affective Neuroscience Lab, Department of Basic and Clinical Psychology, and Psychobiology. Universitat Jaume I, Castelló, Spain

Abstract

Psychopathy is a multifaceted personality disorder characterized by distinct affective/interpersonal traits, including callousness-unemotionality/meanness, which are often considered the hallmarks of empathic deficits. It has been posited that the processing of others' pain could play an important role in empathy capabilities. This study aimed to investigate the influence of perspective taking on electrocortical responses during pain processing in relation to psychopathic callousness. The Late Positive Potential (LPP) —a well-established electrophysiological indicator of sustained attention to motivationally significant stimuli— was measured while 100 female undergraduates viewed images depicting bodily injuries while adopting an imagine-self or an imagine-other perspective. Callousness factor scores —computed as regression-based component scores from EFA on three relevant self-report measures of this dimension— predicted reduced LPP amplitudes to pain pictures under the imagine-other (but not imagine-self) perspective, even after controlling for other LPP conditions. This result suggests that high-callous individuals exhibit diminished brain responsiveness to others' distress, potentially contributing to the empathic deficits observed in psychopathy. This finding highlights the usefulness of electrocortical studies on pain processing to refine our understanding of the selfish and remorseless characteristics of psychopathy in biobehavioral terms.

Keywords: Psychopathy, Callousness, Pain Processing, Perspective Taking, Late Positive Potential (LPP)

Introduction

Empathy is defined as the capability to understand and share the affective states of others, and plays a fundamental role in social interactions. It facilitates prosocial behaviors and inhibits antisocial or aggressive actions (Decety & Cowell, 2018; Decety & Svetlova, 2012). Impaired empathy can result in significant social disfunctions, which characterizes various forms of psychopathology. Psychopathy, a multifaceted personality disorder involving distinctive emotional, interpersonal, and behavioral deviations, that is marked by symptom features such as callousness, lack of guilt, and shallow affect (Cleckley, 1976; Hare & Neumann, 2008; Patrick et al., 2009), can be regarded as the archetypal empathy disorder (Lockwood, 2016). Lack of empathy would explain the tendency of psychopathic individuals to harm and violate the rights of others and their lack of insight and remorse for their actions.

Consistent with the multifaceted perspective of psychopathy (Fowles & Dindo, 2009; Patrick & Bernat, 2009), impairments in empathic-emotional processing within psychopathy are particularly associated with its callousness-unemotionality/meanness traits (Campos et al., 2022, for a recent meta-analysis), which encompass phenotypic attributes such as lack of close attachments with others, emotional coldness and insensitivity, absence of guilt, and empowerment through cruelty (see, for example, Patrick et al., 2009). Empirical studies have supported this relationship, demonstrating that psychopathy callousness traits —mainly assessed by the Meanness scale of the *Triarchic Psychopathy Measure* (TriPM; Patrick, 2010) and by the *Inventory of Callous-Unemotional traits* (ICU; Frick, 2004; Kimonis et al., 2008)— predict decreased recognition accuracy and blunted electrocortical responses to fearful faces (Brislin et al., 2018; Brislin & Patrick, 2019), reduced reactivity of the right amygdala to fear expressions (Viding et al., 2012), diminished potentiation of the noise-elicited startle reflex in response to violent films (Fanti et al., 2016), and reduced elaborative

processing —as indexed by diminished amplitudes of the Late Positive Potential (LPP)— of pictures depicting aggressive interactions (van Dongen et al., 2018), and task-relevant affective pictures (Ribes-Guardiola et al., 2023).

Building on this, research on empathy deficits in psychopathy has also focused on pain empathy, hypothesizing that the brain's pain network could play a crucial role in empathic capabilities (Decety, 2011). Others experiencing pain is a particularly significant signal, which can capture attention and promote caring and protective social functions. Therefore, responsiveness to others' pain could serve as a valuable and ecologically valid indicator for empathic processing (Lamm et al., 2011). Paradigms involving pain experience have revealed higher pain tolerance in individuals exhibiting aggressive behavior (Niel et al., 2007) and psychopathic callousness traits (Brislin et al., 2016, 2022; Miller et al., 2014). These findings suggest that elevated pain thresholds may act as an underlying mechanism which contributes to the underestimation of others' pain experience and, consequently, insensitivity towards others' distress. Regarding the concern for others' pain, research in psychopathy has primarily used pain-viewing paradigms in which participants view images of hands and feet in painful or nonpainful situations while their brain activity is recorded. Neuroimaging studies have consistently reported a specific association between the selfish, callous, and remorseless use of others component of psychopathy and reduced activation in key regions conforming the 'pain matrix' (such as the anterior insula, anterior cingulate cortex and/or amygdala; Decety, 2011) when participants adopt an other-perspective in which they imagine that the hand or foot in the picture belongs to someone else (Decety et al., 2013; Lockwood et al., 2013; Marsh et al., 2013; Seara-Cardoso et al., 2015; but see Yoder et al., 2022, which reports no differences in a female offender sample). No callousness-related differences in the activation of these areas have been found under self-perspective conditions, when participants imagine that the hand or foot in the picture is their

own (Decety et al., 2013; Marsh et al., 2013; Yoder et al., 2022). These results suggest that individuals with higher psychopathic callousness traits exhibit reduced brain activity in response to signals of distress in others, while maintaining typical activity levels when referring to themselves.

In addition to fMRI studies, research has also employed event-related potentials (ERPs) to characterize the temporal dynamics of pain empathy. A recent meta-analysis revealed that early and mid-latency components (< 300ms) do not consistently show modulation in response to pain conditions, but reliable enhancements were observed in later components (P3/LPP) when comparing pain and no pain stimuli (Coll, 2018). The LPP (Late Positive Potential) is a sustained positive deflection in the ERP waveform, typically measured over centroparietal scalp regions, occurring between 400 to 1000 ms after stimulus presentation. It is a well-established ERP component associated with affective processing and has been theorized to reflect the sustained engagement of attention towards motivationally significant cues that activate the brain's appetitive or aversive motivational systems (Hajcak & Foti, 2020). Considering this, the LPP appears to be a suitable electrocortical measure to investigate the effects of perspective taking on pain processing. However, limited research has explored this possibility thus far, with only one study reporting a greater differentiation between pain and no pain pictures in self-perspective conditions, but not in other-perspective conditions, when focusing on the early portion of this brain response (Li & Han, 2010).

Relevant to the current study, only two previous studies have examined pain processing in relation to psychopathic traits using ERPs. These studies have demonstrated callousness-related reductions in LPP amplitudes when participants viewed visual depictions of others in pain (Brislin et al., 2022; Decety et al., 2015). However, neither of these studies examined the potential moderating role of perspective taking. In one of these studies, Decety

et al. (2015) presented participants with pictures of others' hands and feet in painful situations and instructed them to either focus on the amount of concern they felt for the individuals, or the intensity of the pain the individuals in the pictures would experience. The authors found that psychopathic and callousness traits were associated with reduced LPP responses to pain stimuli only when participants were instructed to focus on their level of concern for others. This provides evidence for specific impairments in the capacity for empathic concern when processing distress signals in others at the electrophysiological level. Furthermore, psychopathic and callousness traits were associated with lower ratings of both empathic concern and pain intensity in this study.

In the second study, Brislin et al. (2022) found that meanness/callousness traits of psychopathy predicted reduced LPP amplitudes in response to pictures of others in pain during a passive viewing task without specific perspective taking instructions. Additionally, these traits were associated with lower ratings of pain intensity in both self- and other-perspective conditions. Unfortunately, this study did not investigate whether the blunted electrocortical processing of pain in individuals with higher callousness may be differentially modulated by the adopted perspective.

The current study

To obtain a more comprehensive understanding of callousness-related differences in pain processing at the electrophysiological level, this study aimed to investigate, for the first time, the influence of perspective taking on LPP amplitudes elicited by pain pictures in relation to psychopathic callousness traits. To achieve this objective, EEG data were recorded while a sample of female undergraduates viewed pictures depicting bodily injuries while imagining that the person in the picture was either themself (self-perspective) or an unknown other (other-perspective). Callousness traits were assessed using a multi-measurement approach, by extracting scores on a factor index of this trait dimension using

three self-report scales that have been demonstrated to be suitable indicators of the callousness traits of psychopathy (see Drislane et al., 2014), and that have also been used in prior research in pain empathy (Brislin et al., 2022; Decety et al., 2015; Lockwood et al., 2013): the TriPM Meanness scale, the Primary Psychopathy scale of the *Levenson Self-Report Psychopathy Scale* (LSRP; Levenson et al., 1995) and the ICU.

Building upon prior evidence demonstrating associations between the callousness traits of psychopathy and reduced brain reactivity —both with fMRI and EEG measures— to pain in others (e.g., Marsh et al., 2013; Decety et al., 2013, 2015), it was hypothesized that callousness factor scores would be specifically correlated with reduced LPP amplitudes to pain stimuli under the other-perspective but not under the self-perspective viewing instructions.

Method

Participants

The initial sample consisted of 105 female undergraduates recruited from the Universitat Jaume I of Castellón (Spain). Before the experimental session, five participants were excluded from the study because they were undergoing psychiatric or pharmacological treatment at the time of the experiment. The final sample comprised a total of 100 participants, ranging in age from 18 to 35 years (M = 19.44, SD = 2.6). The experimental research procedures were approved by the Ethical Committee of the Universitat Jaume I and adhered to the ethical principles for human research outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants, and they received academic credit as compensation for their participation.

Self-report Measures

The *Triarchic Psychopathy Measure* (TriPM; Patrick, 2010; Spanish version, Poy et al., 2014) is a questionnaire specifically developed to assess the three trait dimensions

proposed in the triarchic model of psychopathy (Patrick et al., 2009). The TriPM Meanness scale measures lack of empathy and remorse, scorn for and absence of close attachments with other people, defiance of rules, and cruelty (e.g., "I've injured people to see them in pain"). It consists of 19 items that are answered using a 4-point Likert scale (0 = false, 1 = somewhat false, 2 = somewhat true, 3 = true).

The *Levenson Self-Report Psychopathy Scale* (LSRP; Levenson et al., 1995; Spanish version, Andreu-Rodríguez et al., 2018) was developed to assess both factors of *Hare's Psychopathy Checklist-Revised* (PCL-R; Hare, 2003) in non-institutionalized young adult samples. The 16-item LSRP Primary scale measures tendencies towards deception and manipulation, lack of guilt, and emotional coldness or insensitivity (e.g., ''Success is based on survival of the fittest; I am not concerned about the losers"). Each item is ranked on a 4-point Likert scale (1 = disagree strongly, 2 = disagree somewhat, 3 = agree somewhat, and 4 = agree strongly).

The *Inventory of Callous-Unemotional Traits* (ICU; Frick, 2004; Kimonis et al., 2008; Spanish version, Ezpeleta et al., 2013) is a 24-item questionnaire specifically developed to assess the construct of callous unemotionality, encompassing traits such as carelessness and lack of emotional responsiveness, in individuals across various age groups, including children, adolescents and adults (e.g., "I do not feel remorseful when I do something wrong"). Items are rated on a 4-point Likert scale, from 0 (not at all true) to 3 (definitely true).

All three scales demonstrated good internal consistency reliability in the current sample, with Cronbach's α coefficients being .72 for TriPM Meanness, .78 for LSRP Primary, and .83 for ICU. Descriptive statistics (mean, standard deviation, range) for the scores of these scales are presented in Table 1. An exploratory factor analysis (EFA) on scores from these scales was conducted to obtain a general Callousness factor representing

the shared variance between the different measures. Results of the principal-axis factor analysis (Barlett's $\chi^2 = 88.5$, $p < .001$; KMO = .693) and the parallel analysis revealed one single factor (eigenvalue = 1.69), accounting for 56.3% of the total variance. The factor loadings were .82, .77, and .66 for the TriPM Meanness, ICU, and LSRP Primary scales, respectively. The regression-based estimation method was used to compute a factor score for each participant, reflecting the sum of beta-weighted scores on the three callousness measures.

Procedure and experimental task

Before the experimental session, participants completed the self-report measures in anonymous group sessions. The experimental session was conducted individually in an isolated and dimly lit room. Participants were seated 110 cm away from a monitor screen where stimuli were displayed. Presentation® v.20.1 software (Neurobehavioral Systems, Inc. Albany, CA, USA) was used to control the order, sequence, and timing of stimulus presentations on a PC Pentium Core 2 Duo (Intel) computer. During EEG recording, participants viewed a total of 128 pictures depicting hands and feet of individuals in both painful and non-painful everyday situations (Jackson et al., 2005) —for example, cutting a cucumber with a finger under the knife (pain), or without the finger under the knife (no pain). Each picture was presented twice, with two different perspectives. Participants were instructed to adopt either a self-perspective ('imagine the person in the picture is you') or an other-perspective ('imagine the person in the picture is someone unknown') while viewing the pictures within each block of stimuli.

Each trial began with a fixation cross displayed on the screen for a duration of 2000, 2500 or 3000 ms, followed by the presentation of a pain or no pain (neutral) picture for 1500 ms. The task consisted of eight blocks, each containing 32 trials, resulting in a total of 256 trials. The pictures were randomly presented within each block, and the perspective

instruction changed between consecutive blocks, with a 30-second rest period between blocks. The overall duration of the task, including 8 practice trials and breaks, was approximately 22 min.

Table 1. *Descriptive Statistics for Self-report and ERP Data for the Overall Sample (N = 100)*

Variable	*M (SD)*	*Min.*	*Max.*
Self-report Data			
TriPM Meanness	9.28 (5.44)	1	28
LSRP Primary	27.52(6.03)	18	40
ICU	18.42 (8.17)	4	40
ERP Data			
LPP Pain Self	1.21 (1.05)	-.86	3.44
LPP Pain Other	1.20 (1.02)	-1.22	3.81
LPP No Pain Self	1.12 (.96)	-.63	3.75
LPP No Pain Other	.83 (.80)	-.81	2.80

Note. TriPM = Triarchic Psychopathy Measure (Patrick, 2010); LSRP = Levenson Self-Report Psychopathy Scale (Levenson et al., 1995); ICU = Inventory of Callous-Unemotional Traits (Frick, 2004); LPP = Late Positive Potential

Psychophysiological recording and data reduction

EEG activity was recorded from 257 electrodes using an Electrical Geodesic (EGI; OR, USA) high-density EEG system. The signals were amplified and filtered (analog filters: 0.10–100 Hz bandpass) with a NetAmps 400 amplifier system with NetStation v5.4.1.2 installed on a MacBook Pro (Apple) computer. The EEG data were continuously digitized at a sampling rate of 250 Hz using a 24-bit analog-to-digital converter. The reference electrode was placed on the vertex scalp site (Cz), and scalp impedances were kept below 50 kΩ, following the manufacturer's guidelines.

Offline preprocessing of the raw EEG data was performed using Brain Electrical Source Analysis software (BESA v7.1.2.1; MEGIS software GmbH, Germany). Visual inspection of the raw recordings was performed to identify and interpolate data for bad

electrodes. Eyeblink (EOG) and electrocardiogram (EKG) artifacts in the continuous EEG data were manually corrected using a principal component analysis-based adaptive artifact-correction method in BESA. The artifact corrected data were then subjected to a low-pass filter with a cutoff frequency of 30 Hz. Stimulus-synchronized epochs were extracted from −200 to +1000 ms after the picture onset, and baseline correction was applied using the 200 ms period preceding the stimulus onset. A semi-automated procedure was then used to detect and reject epochs containing amplitude deflections exceeding 75 μV between successive sampling points or surpassing an amplitude threshold of 120 μV. Additionally, epochs with a low signal threshold of 0.01 μV were discarded. The accepted epochs were subsequently converted to the average reference.

ERP measurement

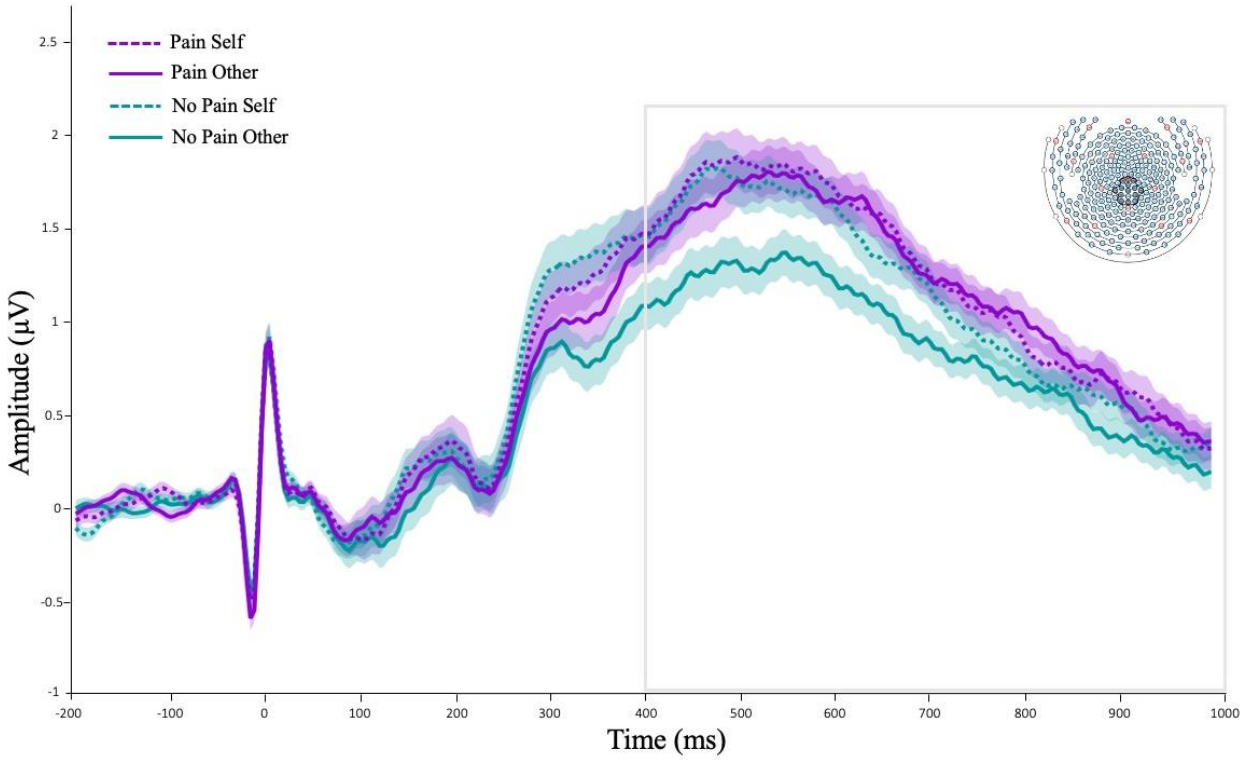
For each participant, separate ERP averages were computed for each sensor and condition. The LPP was scored as the mean amplitude of a 14-sensor centroparietal cluster (EGI sensors: 45, 79, 80, 81, 89, 90, 100, 101, 129, 130, 131, 132, 143, and 257; see Ribes-Guardiola et al., 2023, for the same electrode configuration) during a 400–1000 ms time window following stimulus onset. This time window was selected based on prior research investigating LPP amplitudes for pain pictures in relation to callousness (Brislin et al., 2022; Decety et al., 2015).

The reliability of LPP amplitudes, assessed using split-half (odd-even method) correlations adjusted for attenuation using the Spearman-Brown prophecy formula, was found to be moderate-to-high for all conditions: .77 for LPP Pain Self, .68 for LPP Pain Other, .78 for LPP No Pain Self, and .70 for LPP No Pain Other.

Upon visual inspection of the grand averaged waveforms, a trigger-related artifact was observed around the EEG ground electrode affecting electrodes at central parietal locations (see Figure 1). This artifact coincided with the timing of the trigger codes for visual

stimuli sent by the Presentation software to the acquisition software. The magnitude of the artifact did not vary across conditions, and thus did not affect the results or their interpretation. To further investigate whether the presence of this artifact-related activity affected the results reported using time-windowed analyses, we conducted a temporal Principal Component Analysis (PCA; Dien, 2012) on the averaged epochs (-200 to 1000 ms) at the centroparietal cluster. This allowed us to separate the LPP component from the influence of other components. The results of these analyses are presented in the Supplemental Material, showing the same pattern of results reported in the main text.

Figure 1. *Grand Average Event-Related Potentials Waveforms for Pain (Purple) and No Pain (Green) under Self-Perspective (Dotted Lines) and Other-Perspective Instructions (Solid Lines) at the Centroparietal Sensor Cluster (EGI Sensors: 45, 79, 80, 81, 89, 90, 100, 101, 129, 130, 131, 132, 143, and 257)*



Statistical analyses

The data analyses were conducted using Jamovi 2.3.21.0 software (The Jamovi Project, Sydney, Australia). To address outliers and prevent their disproportionate influence on the relationships with callousness scores, a winsorization procedure (Wilcox, 2012) was

applied. Scores exceeding the 75th percentile plus 1.5 times the interquartile range, or falling below the 25th percentile minus 1.5 the interquartile range, were replaced with the maximum or minimum value within these ranges. This resulted in a correction of 2.25% of all scores (LPP Pain Self: 4%; LPP Pain Other: 1%; LPP No Pain Self: 1%; LPP No Pain Other: 3%). The pattern of results for analyses with non-winsorized data were virtually the same as reported below.

First, to validate the selected task procedure, the effects of pain and perspective on LPP amplitudes were tested by conducting a 2 × 2 repeated measures ANOVA with *Pain* (pain, no pain) and *Perspective* (self, other) as within-subjects factors. Significant interaction effects were further explored using post-hoc comparisons.

Second, to investigate the relationships between LPP responses and callousness, bivariate Pearson's *r* correlations were calculated between LPP amplitudes and callousness scores (omnibus component and individual scale scores). In addition, a hierarchical regression analysis was conducted to test for the unique predictive contribution of callousness factor scores on LPP reactivity to pain in others. In this analysis, LPP amplitudes to pain under the other-perspective condition served as the criterion, and the LPP amplitudes to the other conditions were included as predictors in Step 1 (to account for the overlap between LPP conditions), followed by Callousness factor scores in Step 2.

Results

Task effects

Descriptive statistics for LPP amplitudes are shown in Table 1. The ANOVA revealed significant main effects of *Pain*, $F(1, 99) = 14.21$, $p < .001$; $\eta_p^2 = .13$, and *Perspective*, $F(1, 99) = 6.98$, $p = .010$, $\eta_p^2 = .07$, indicating that LPP amplitudes were larger for pain than for no pain pictures (1.20 vs. 0.98 μV, respectively), and in the self- than in the

other-perspective condition (1.17 vs. 1.02 μV, respectively). Furthermore, there was a significant *Pain* × *Perspective* interaction, F (1, 99) = 5.91, p = .017, η_p^2 = .06. Post-hoc comparisons revealed that LPP amplitudes were significantly larger for pain than for no pain pictures only in the other-perspective condition, $t(99) = 3.53$, $p < .001$ ($p = .702$ for the self-perspective condition; see Figure 1).

Callousness effects

Bivariate Pearson's correlations between callousness measures and LPP amplitudes can be found in Table 2. Callousness factor scores (as well as ICU scores) showed a significant negative correlation with LPP amplitudes for pain pictures in the other-perspective condition ($rs > -.23$, $ps < .02$). This negative association remained significant in the subsequent hierarchical regression analysis, where LPP amplitudes for the other three conditions (LPP Pain Self, LPP No Pain Self, LPP No Pain Other) were entered as predictors in Step 1, followed by Callousness factor scores in Step 2 (see coefficients in Table 3). After controlling for the other LPP conditions, the Callousness factor scores contributed significantly to the prediction of LPP amplitudes for other-perspective pain pictures, ΔF (1, 95) = 4.23, p = .043, R^2 = .019. This finding reflects smaller LPP responses during depictions of pain in others for participants with higher levels of callousness. Figure 2 visually illustrates this result, showing the grand averaged waveforms for median-split groups on Callousness factor scores (Figure 2a), the LPP scalp distribution (Figure 2b), and the scatterplot depicting the association between Callousness factor scores and LPP amplitudes for pain pictures under the other-perspective viewing instruction (Figure 2c).

Table 2. *Pearson Correlations between Self-report and ERP Data for the Overall Sample (N = 100)*

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*
Self-Report Data								
1. ICU	-							
2. LSRP Primary	.50***	-						
3. TriPM Meanness	.63***	.54***	-					
4. Callousness Factor	.85***	.73***	.91***	-				
ERP Data								
5. LPP Pain Self	-.19	-.05	-.09	-.14	-			
6. LPP Pain Other	-.25*	-.14	-.18	-.23*	.70***	-		
7. LPP No Pain Self	-.13	.06	-.06	-.07	.70***	.66***	-	
8. LPP No Pain Other	-.18	-.07	-.08	-.13	.62***	.56***	.58***	-

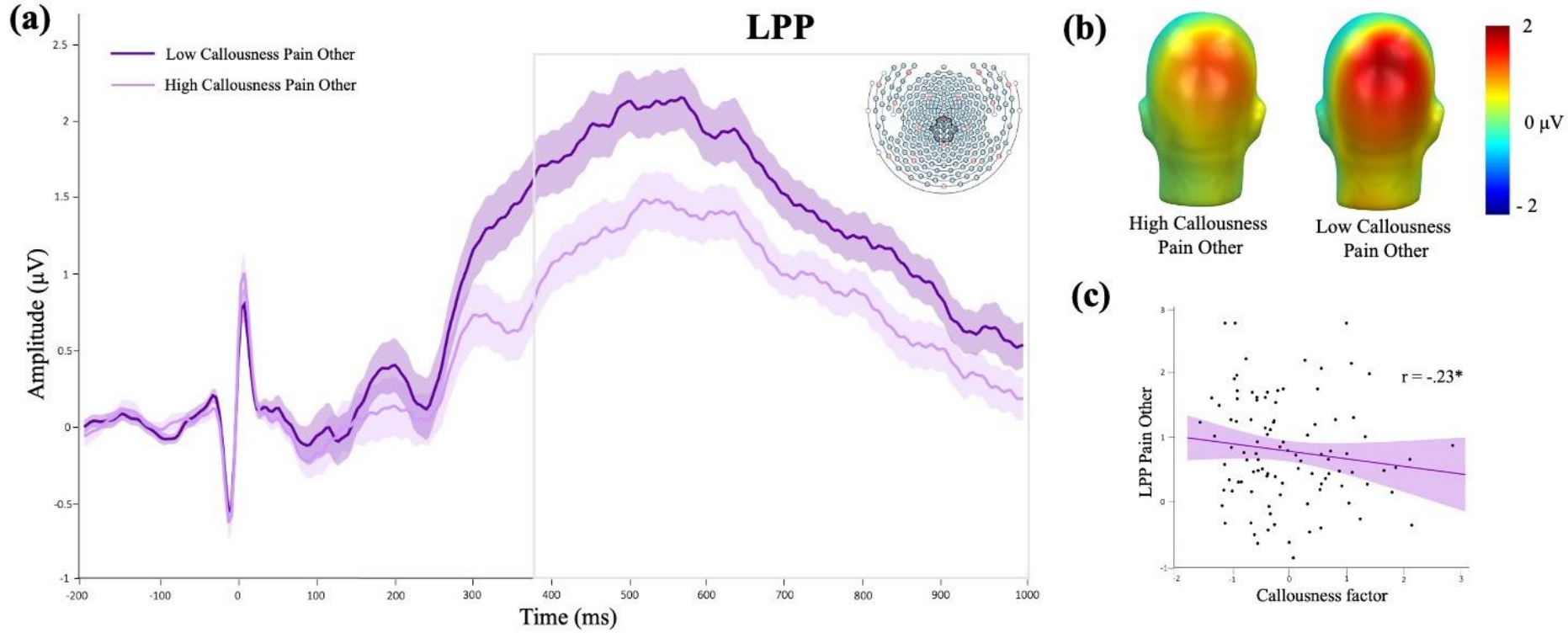
Note. TriPM = Triarchic Psychopathy Measure (Patrick, 2010); LSRP = Levenson Self-Report Psychopathy Scale (Levenson et al., 2995); ICU = Inventory of Callous-Unemotional Traits (Frick, 2004); LPP = Late Positive Potential
*$p < .05$, ** $p < .01$, *** $p < .001$

Table 3. *Standardized β Weights from Hierarchical Regression Predicting for Other-Perspective Pain Pictures*

Step 1	β_1	$R^2 = .563$
LPP Pain Self	**.42*****	
LPP No Pain Self	**.30****	
LPP No Pain Other	.12	
Step 2	β_2	ΔR^2 **= .019***
LPP Pain Self	**.40*****	
LPP No Pain Self	**.32****	
LPP No Pain Other	.23	
Callousness Factor	**-.14***	

Note. β_x = standardized β for each predictor in the model at Step x; ΔR^2 = increase in explained variance at Step 2. Significant associations are highlighted in bold.
*$p < .05$, ** $p < .01$, *** $p < .001$

Figure 2. *Relationship between LPP Amplitudes for Other-Perspective Pain Pictures and Callousness Factor Scores. (a) Grand Average Event-Related Potentials Waveforms for the Other-Perspective Pain Condition at the Centro-Parietal Cluster for Participants on the Bottom (Dark Purple) and Top (Light Purple) Median-Split Groups on the Callousness Factor. (b) LPP Scalp Distribution for the 400-1000 ms Time Window. (c) Scatterplot Depicting the Correlation between Callousness Factor Scores and LPP Amplitudes for Other-Perspective Pain Pictures*



Note. LPP = Late Positive Potential
*p = .022

Discussion

The present study aimed to examine, for the first time, the influence of perspective taking on electrocortical processing of pain in relation to the callousness traits of psychopathy. Consistent with our hypothesis, individuals with higher levels of callousness exhibited reduced LPP amplitudes for pain pictures when imagining someone else (and not themself) in the painful situation. This finding suggests that callousness traits of psychopathy are related to a blunted elaborative processing of distress cues in others, indicative of impaired empathic responding.

Prior research on fMRI has widely investigated the differences in brain reactivity between pain and no pain stimuli (Gu and Han, 2007; Jackson et al., 2005), revealing a more extensive activation of areas conforming the pain network during self- compared to other-perspective conditions (Jackson, Brunet et al., 2006; Jackson, Rainville et al., 2006; Lamm et al., 2007). Meta-analysis evidence on pain empathy ERPs has also demonstrated higher brain reactivity to pain compared to neutral stimuli in late components (Coll, 2018), fitting with our results which show higher LPP amplitudes for pain-related pictures. However, there is limited research examining how ERP components related to pain processing are modulated by perspective taking. Thus, our study makes a significant contribution to electrocortical research on pain processing by incorporating perspective taking, enabling the differentiation of the empathic distress response elicited by others' pain from the perception of one's own pain experience. Furthermore, regardless of the picture type (i.e., pain or no pain), self-perspective consistently elicited greater LPP amplitudes than other-perspective instructions, indicating an enhanced sustained allocation of attention towards self-referenced stimuli. Interestingly, the LPP amplitude differentiation between pain and no pain pictures was observed only when participants imagined someone else in the painful situation, but not when they imagined themselves in the same situation. This finding contrasts with the only

other study to date that has examined the perspective taking modulation of electrocortical responses to perceived pain (Li & Han, 2010). Our results can be interpreted from the theoretical perspective of the LPP as an index of stimulus significance (Bradley et al., 2009; Hajcak & Foti, 2020). In our sample, the self-relevant condition enhanced brain reactivity regardless of the stimulus content while the condition involving someone else allowed to differentiate pain vs. no pain responses, with another person in a non-painful situation being the less motivationally relevant condition in electrocortical terms. Although more EEG studies examining the influence of perspective taking are needed, our results seem to suggest that the LPP can be considered a valuable electrocortical measure to study neural deficits in pain empathy capacity as a function of perspective.

Indeed, by explicitly instructing participants to adopt either a self- or other-oriented perspective towards pain pictures it was possible to confirm the blunted neural responsiveness to others' distress that is theoretically linked to the callousness traits of psychopathy. This result represents the main contribution of the current study. As hypothesized, we found that callousness scores were associated with reduced LPP amplitudes to pain pictures when adopting an imagine-other perspective, even after controlling for the remaining conditions. These findings are consistent with prior evidence on callousness traits which has demonstrated reduced amplitudes of the LPP in conditions involving empathic concern (Decety et al., 2015) and reduced activation of brain regions associated with empathy for pain (Decety et al., 2013; Lockwood et al., 2013; Marsh et al., 2013; Seara-Cardoso et al., 2015). Taken together, these results suggest that diminished neural responses to others' distress may be linked to the lack of concern and disdain for others that characterizes psychopathic meanness/callousness. Given that neural reactivity at this fundamental level can facilitate affiliative behaviors (Decety & Svetlova, 2012), the

absence of such reactivity may contribute to the callousness-unemotionality traits of psychopathy.

Our results must also be considered in light of prior work reporting significant associations between callousness traits and lower ratings of pain intensity under self- and other-oriented perspectives (Brislin et al., 2022). In contrast, our results show that the LPP seems to be more sensitive to the influence of perspective taking on pain processing in relation to callousness traits. Considered together, it seems that, for high-callous individuals, pain stimuli are perceived as less intense in general, regardless of their own involvement (i.e., self or other; see Brislin et al., 2022) —being this latter result consistent with evidence indicative of the higher pain tolerance of higher callous individuals (Brislin et al., 2016, 2022; Miller et al., 2014)—, but its motivational relevance is diminished only when affecting someone else. The lack of convergence of measures from different modalities (self-report, electrophysiological) could be explained by the aspect of pain processing that each of them captures: subjective quantification of perceived pain (ratings) versus stimulus significance (LPP modulation). Brislin et al. (2022) did not find significant correlations between pain intensity ratings under self- and other-perspectives and LPP amplitudes to pain stimuli during passive picture viewing, suggesting that these two types of measures might assess different aspects of pain processing. Unfortunately, we did not collect ratings of pain intensity and arousal in the overall sample, but only in a small subsample in order to characterize relevant subjective dimensions (pain intensity, arousal) on which the stimuli employed to elicit electrocortical responses were expected to vary (see Supplemental Material). In this regard, it would be highly informative for future studies on electrocortical processing of pain to obtain ratings assessing the stimulus significance/relevance from self- and other-perspectives, in addition to arousal and intensity, to complement evidence about electrocortical responsiveness to others' distress.

Another notable contribution of our work is that the reduced reactivity of the LPP to others' pain was found to be related to callousness traits, as indexed through a multi-measurement approach, which offers some advantages. Alternative measures developed to operationalize callousness-unemotionality/meanness show moderate correlations and, though converging in the assessment of the core traits subsumed in this construct, they also diverge by assessing other less central traits (Viding & Kimonis, 2018). For example, some measures assess lack of interest in one's own performance (ICU: 'I do not care about doing things well'), concern for material gains (e.g., LSRP Primary scale: 'My main purpose in life is getting as many goodies as I can'), or sensation seeking (e.g., TriPM Meanness: 'Things are more fun if a little danger is involved'). In fact, the scale scores used in our EFA did not equally contribute to the general callousness factor. By using a single omnibus factor that encompassed the common variance across alternative operationalizations of the callousness-unemotionality/meanness dimension, we obtained a theoretically valid measure that de-emphasized unique and error variance associated with each instrument. This approach allowed us to confirm that it is the shared variance among these alternative scales, rather than the unique variance of each of them, that relates to reduced neural responsiveness to others' pain (see Supplemental Material for exploratory analyses showing that none of the individual scale scores of callousness significantly related to LPP reactivity to others' pain when controlling for the general callousness factor).

In future work, it would be valuable to extend this approach to examine how alternative self-report indicators of callousness relate to other established physiological and behavioral indicators of affective processing of distress cues in others, such as reduced amygdala reactivity to fearful faces (Viding et al., 2012), diminished early ERP amplitudes and recognition accuracy to fear expressions (Brislin et al., 2018; Brislin & Patrick, 2019), or reduced elaborative processing of aggressive interactions (van Dongen et al., 2018).

Systematically studying patterns of covariance among these established physiological and behavioral indicators would be needed to refine our understanding of the biobehavioral processes linked to callousness traits (cf. affiliative capacity; Patrick, 2022; Patrick et al., 2019; see also Palumbo et al., 2020).

Some limitations of the current study should be acknowledged. First, our sample consisted exclusively of women, which may limit the generalizability of our findings. Although previous research has not found gender effects on reductions in LPP for pain pictures related to meanness/callousness (Brislin et al., 2022; Decety et al., 2015), follow-up studies using mixed-gender samples are needed to confirm that the association between electrocortical responsiveness to others' pain and callousness is not gender-dependent. Furthermore, our undergraduate unselected sample showed a restricted range of callousness scores, which might have potentially attenuated the effects found. Therefore, in future studies, it would be valuable to preselect the sample based on callousness scores to ensure a better representation of high scores and allow for more robust effects to be observed. Additionally, considering the lack of convergence between self-report and electrophysiological measures of pain reactivity (as discussed earlier), it would be beneficial to complement the EEG measurements with subjective ratings of pain intensity, arousal, and stimuli relevance.

Despite these limitations, our study provides further evidence regarding the association between callousness-unemotionality/meanness traits and the processing of pain in others. These findings contribute to better understanding empathy deficits, which have been long linked to the selfish and remorseless use of others, a symptom of psychopathy. Finally, our results highlight the utility of perspective-taking in electrocortical research on pain responsiveness, providing support for the use of the LPP as an indicator that has the potential

to elucidate the biobehavioral processes associated with the meanness/callousness traits of psychopathy.

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Psychopathic Callousness and Perspective Taking in Pain Processing: an ERP study

Supplemental Material

Contents

1. Pain ratings

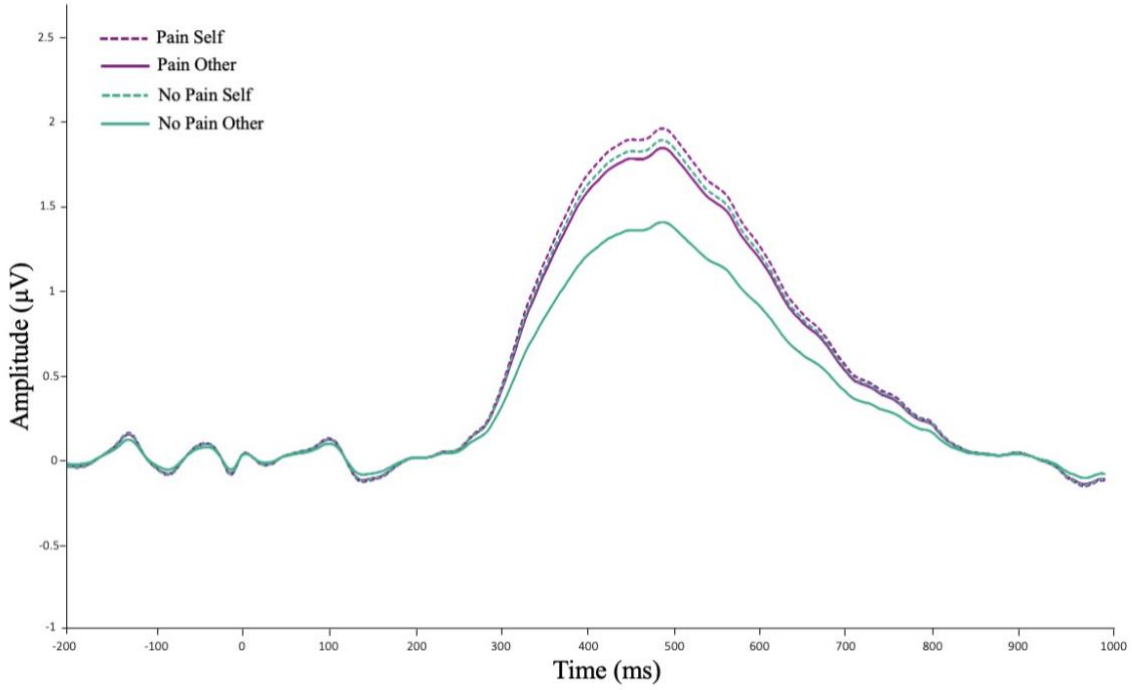
Two subsamples of participants (n = 18 for half of the pictures; n = 13 for the other half) rated the arousal and the intensity of pain depicted in each picture on scales ranging from 1 to 9, under both the self-perspective and other-perspective instructions. The results confirmed higher ratings for *pain* ($Mean_{arousal}$ = 6.18; $Mean_{pain}$ = 6.22) compared to *no pain* pictures ($Mean_{arousal}$ = 2.50; $Mean_{pain}$ = 1.18; *F*s > 298.77; *p*s < .001, and for *self-* ($Mean_{arousal}$ = 4.66; $Mean_{pain}$ = 4.46) compared to *other-perspective* instructions ($Mean_{arousal}$ = 4.01; $Mean_{pain}$ = 3.57; *F*s > 39.12; ps < .001). No significant interactions were found (*F*s < 3.31; *p*s > .079).

2. LPP Analyses using Principal Component Analysis (PCA)

To isolate the Late Positive Potential (LPP) amplitude and remove the artifact, the LPP was calculated using temporal Principal Component Analysis (PCA; Dien, 2012; Dien & Frinschkoff, 2005) on the channels of interest (i.e., the 14-sensor centroparietal cluster; see ERP measurement on the Method section). PCA is a statistical technique that identifies linear combinations of data points that capture consistent patterns of electrocortical activity (Foti et al., 2009).

Given that the artifact occurred when stimulus was presented (i.e., at time point 0), while the LPP is a later potential, PCA can effectively separate these two types of EEG signals. We used the ERP PCA Toolkit version 2.93 (Dien, 2010) to perform a temporal PCA with Promax rotation on the 300 timepoints in the data, considering all subjects and conditions. The covariance matrix and Kaiser normalization were used, as described in Dien et al. (2005). After examining the resulting Scree plot, nine temporal factors were extracted for rotation. The LPP becomes evident on the second factor at 484 ms (see Supplemental Figure 1).

Supplemental Figure 1. *Grand Average Event-Related Potentials Waveforms for Pain (Purple) and No Pain (Green) under Self-Perspective (Dotted Lines) and Other-Perspective (Solid Lines)*



The results obtained from the LPP calculated using PCA are presented below:

Regarding **task effects**, a general linear model (GLM) analysis revealed significant main effects of *Pain*, $F(1, 99) = 12.45$, $p < .001$; $\eta_p^2 = .11$, $\varepsilon = 1$, indicating larger overall amplitudes for pain pictures compared to no pain pictures, and *Perspective*, $F(1, 99) = 19.84$, $p < .001$, $\eta_p^2 = .17$, $\varepsilon = 1$, indicating larger LPP overall amplitudes under the self-perspective compared to the other-perspective instruction. A significant *Pain* × *Perspective* interaction was also observed, $F(1, 99) = 8.12$, $p = .005$, $\eta_p^2 = .08$, $\varepsilon = 1$, revealing that LPP amplitudes were larger for pain pictures than for no pain pictures only under the other-perspective instruction, $t(99) = 4.04$, $p < .001$ (see Supplemental Figure 1).

Regarding the **effects** of **callousness**, a significant negative relationship was found between Callousness factor scores and LPP amplitudes for pain pictures in the other-perspective condition ($rs > -.23$, $ps < .02$; see Supplemental Table 1).

Supplemental Table 1. *Bivariate Correlations between Self-report and ERP Data for the Overall Sample (N = 100)*

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*
Self-Report Data								
1. ICU	-							
2. LSRP Primary	.50***	-						
3. TriPM Meanness	.63***	.54***	-					
4. Callousness Factor	.85***	.73***	.91***	-				
ERP Data								
5. LPP Pain You	-.16	-.05	-.10	-.13	-			
6. LPP Pain Other	-.25*	-.12	-.19	-.23*	.85***	-		
7. LPP No Pain You	-.13	.08	-.07	-.07	.80***	.79***	-	
8. LPP No Pain Other	-.20*	-.08	-.13	-.17	.76***	.73***	.72***	-

Note. TriPM = Triarchic Psychopathy Measure (Patrick, 2010); LSRP = Levenson Self-Report Psychopathy Scale (Levenson et al., 2995); ICU = Inventory of Callous-Unemotional Traits (Frick, 2004); LPP = Late Positive Potential
*$p < .05$, ** $p < .01$, *** $p < .001$

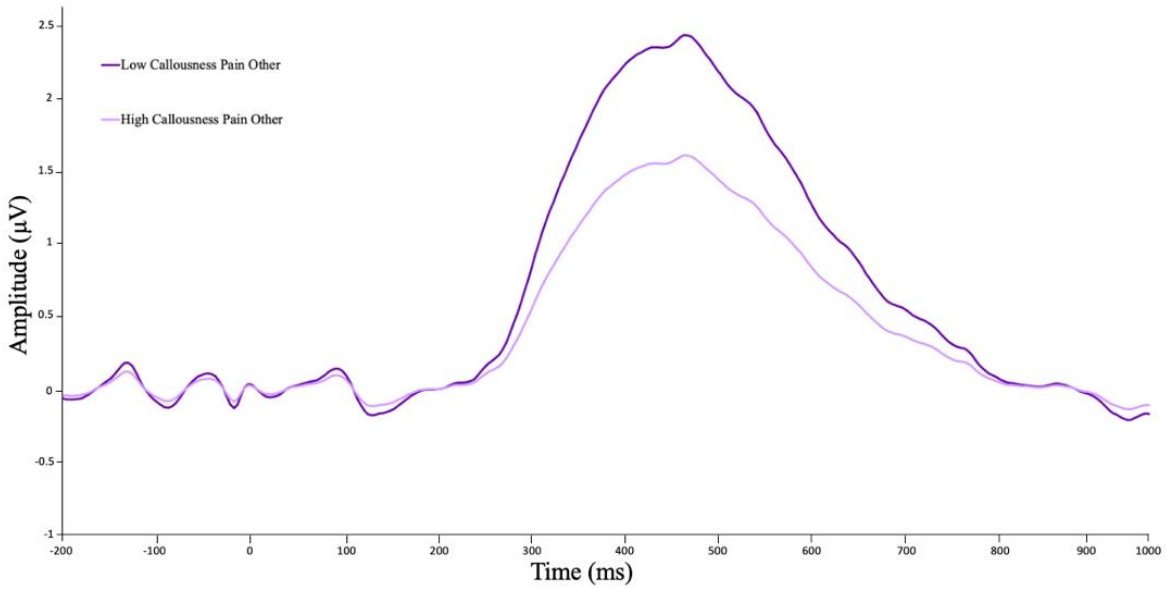
This association remained significant in the subsequent hierarchical regression analysis, where LPP amplitudes for self-perspective pain pictures and for self- and other-perspective no pain pictures were entered as predictors in Step 1, followed by Callousness factor scores in Step 2 (see coefficients in Supplemental Table 2). After controlling for the other LPP conditions, Callousness factor scores significantly contributed to the prediction of LPP amplitudes for other-perspective pain pictures, $\Delta F\,(1, 95) = 4.23$, $p = .043$, $R^2 = .019$, indicating smaller LPP responses during depictions of pain in others for participants with higher levels of callousness. Supplemental Figure 2 illustrates this finding.

Supplemental Table 2. *Standardized β Weights from Hierarchical Regression Predicting LPP Pain Other*

Step 1	β_1	$R^2 = .767$
LPP Pain Self	**.55*** **	
LPP No Pain Self	**.26** **	
LPP No Pain Other	.13	
Step 2	β_2	$\Delta R^2 = .014$*
LPP Pain Self	**.53*** **	
LPP No Pain Self	**.28** **	
LPP No Pain Other	.11	
Callousness Factor	**-.12***	

Note. β_x = standardized β for each predictor in the model at Step x; ΔR^2 = increase in explained variance at Step 2. Significant associations are highlighted in bold.
*$p < .05$, ** $p < .01$, *** $p < .001$

Supplemental Figure 2. *Grand Average Event-Related Potentials Waveforms for the Other-Perspective Pain Condition for Participants on the Bottom (Dark Purple) and Top (Light Purple) Median-Split Groups on the Callousness Factor*



3. Partial correlations between individual scale scores of callousness and LPP Pain Other controlling for Callousness factor scores

To examine the extent to which scales contributing to the callousness factor predicted unique variance in LPP reactivity to pain in others beyond the general factor explaining their covariance, a set of exploratory partial correlations were conducted. Residual LPP scores to pain in others were computed to account for its overlap with the other LPP conditions (akin to the hierarchical regression analyses reported in the main text and section 2 of this document). Then, partial correlational analyses between each callousness scale scores (TriPM Meanness, LSRP Primary, ICU) and this residualized LPP variable were conducted controlling for Callousness factor scores. After controlling for Callousness factor scores, correlations between individual scale scores and LPP Pain Other amplitudes were: r = .03 for TriPM Meanness, r = -.07 for LSRP Primary, and r = .03 for ICU; all ps > .474. These results indicate that the observed relationship between Callousness factor scores and LPP Pain Other amplitudes was accounted by the shared variance between the three callousness scales.

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CHAPTER 6

STUDY 4 Disinhibition and electrocortical correlates of inhibitory control

Study 4

Disinhibition and Electrocortical Correlates of Inhibitory Control

Victoria Branchadell, Rosario Poy, Pablo Ribes-Guardiola, Pilar Segarra, & Javier Moltó

Affective Neuroscience Lab, Department of Basic and Clinical Psychology, and Psychobiology. Universitat Jaume I, Castelló, Spain

Abstract

Some ERP components, such as P3 and ERN, have shown reduced amplitudes associated with trait disinhibition —the triarchic psychopathy dimension which is presumed to reflect impairments in fronto-cortical systems mediating executive functions. However, the specific neurocognitive process underlying ERP reductions in relation to disinhibition is unclear, given the variety of paradigms used and the inconsistent associations found between disinhibition and different variants of these ERPs. Therefore, this study aimed to clarify the different aspects of inhibitory control contributing to trait disinhibition by examining the associations between this triarchic domain and distinct ERP indicators that measure inhibitory control processing (Stop-P3) and error monitoring (Stop and Flanker ERNs) extracted from a modified flanker-stop-signal task administered to a large sample of undergraduates. The triarchic dimensions (i.e., boldness, meanness, disinhibition) were assessed using regression-based component scores derived from exploratory factor analysis on triarchic scores obtained from three self-report measures (TriPM, PPI-R, NEO PI-R). Analyses revealed diminished ERN amplitudes in relation to disinhibition factor scores, and this association was explained by the shared variance between both ERN variants. No significant association was found between Stop-P3 and disinhibition. These findings suggest that deficits in inhibitory control are primarily evident at earlier stages of error monitoring, rather than at later stages of elaborative processing of relevant stimuli. The difficulties in detecting and processing errors in higher disinhibited individuals may potentially underlie the difficulties in behavioral adaptation and self-regulation commonly observed in clinical conditions within the externalizing spectrum of psychopathology.

Keywords: Disinhibition, Inhibitory Control, ERN, P3

Introduction

Psychopathy is a multifaceted personality disorder characterized by significant behavioral deviance —including lack of behavioral restraint, impulsivity, poor decision-making, and difficulties to learn by experience, among others— within the context of distinct affective and interpersonal traits (Cleckley, 1976; Hare & Neumann, 2008; Patrick et al., 2009). In view of the longstanding debates about the nature, boundaries, and core traits of psychopathy (see e.g., Patrick, 2022; Skeem et al., 2011), the triarchic model (Patrick et al., 2009) was proposed as a conceptual framework that attempts to reconcile the different conceptualizations of psychopathy and, importantly, acts as a point of reference for coordinating research on neurobiological mechanisms contributing to this disorder.

The triarchic model defines psychopathy in terms of three distinct but interrelated trait constructs that correspond to distinct phenotypic expressions of psychopathy and relate to specific biobehavioral systems and processes (Patrick, 2022; Patrick et al., 2019; Patrick & Drislane, 2015). *Boldness* is characterized by social dominance, strong sense of self-worth, ability to stay focused under pressure, and quick recovery from stressful/threatening situations. *Meanness* involves attributes such as lack of empathy and close attachments with others, rebelliousness, empowerment through cruelty, dismissive attitude, and aggressive manipulation of others. Both boldness and meanness constitute the affective/interpersonal traits of psychopathy. Finally, *Disinhibition* entails traits such as boredom proneness, irresponsibility, deficient behavioral restraint, impulsivity, strong preference for immediate gratification, and lack of impulse control —representing the behavioral deviance features of the disorder (i.e., the so-called impulsive/antisocial traits).

Dual-process etiological models (cf. Fowles & Dindo, 2009; Patrick & Bernat, 2009) have proposed distinct neurobiological pathways for the different phenotypic trait constructs of psychopathy: *trait fearlessness (or threat sensitivity)*, reflecting an under-reactivity of the

brain's defensive motivational system, contributes to the affective/interpersonal features of psychopathy (although recent empirical research suggests that weak threat sensitivity plays a prominent role in boldness, while low *affiliative capacity* contributes to a greater extent to meanness; Patrick, 2022), and *externalizing proneness (or inhibitory control)*, which is presumed to reflect impairments in fronto-cortical systems mediating executive functions such as anticipation, planning, and behavioral control, contributes to the impulsive/antisocial features (i.e., disinhibition). Therefore, neurobiological research aims to identify neurophysiological and behavioral indicators to conform nomological networks for each triarchic disposition, understanding them as open-ended constructs that are subjected to revision based on accumulating new evidence.

One of the best-established physiological indicators of externalizing proneness is the reduced amplitude of the visually evoked P3 response (Euser et al., 2012; Gao & Raine, 2009; Iacono et al., 2003; Pasion et al., 2018), which is a large positive deflection that reaches its maximal amplitude between 250 and 600 ms after the stimulus presentation. The P3 response has been conceptualized as reflecting attentional resource allocation, memory and context updating, elaborative processing of motivational significance, and inhibitory control processing, depending on the specific nature of the paradigm used to elicit this brain response (Donchin & Coles, 1988; Friedman et al., 2001; Huster et al., 2013; Kok, 2001; Nieuwenhuis et al., 2005; Polich, 2007).

Given the variety of tasks/paradigms used to elicit the P3 response, it has been challenging for empirical studies to determine the precise neurocognitive process(es) that disinhibition-related reductions of P3 amplitudes reflect (Patrick, 2018). In turn, P3 amplitude reductions associated with disinhibition/externalizing traits have been observed using a wide variety of tasks, including oddball, novelty, flanker, gambling, as well as go/no-go tasks (Brennan & Baskin-Sommers, 2018; Nelson et al., 2011; Pasion et al., 2023; Patrick

et al., 2006; Ribes-Guardiola, Poy, Patrick et al., 2020; Venables et al., 2018; Yancey et al., 2013). Notably, some of these studies have demonstrated that the shared variance among distinct variants of P3 is inversely related to disinhibition/externalizing, suggesting the presence of a common process operating across different task contexts (Pasion et al., 2023; Ribes-Guardiola, Poy, Patrick et al., 2020; Venables et al., 2018). Although a few studies have pointed out to the possibility that reductions in P3 amplitudes in externalizing may reflect low inhibitory control capacity (Brennan & Baskin-Sommers, 2018; Venables et al., 2018), there is limited research examining how inhibition-specific variants of the P3 derived from stop-signal tasks relate to externalizing problems (Vilà-Balló et al., 2014). Therefore, investigating the inhibition-specific variants of P3 responses elicited in stop-signal tasks could offer new insights into the functional basis of disinhibition-related reductions of this brain response, as it might more directly assess evaluative-processing stages of salient inhibitory cues that prompt the cancelation of an already initiated behavioral response (Huster et al., 2013).

Another ERP component associated with externalizing proneness/disinhibition is the error-related negativity (ERN), which is a response-locked ERP that appears as an early negative deflection within the first 100 ms of an erroneous response in fronto-central sites (Falkenstein et al., 1991; Gehring et al., 1993, 2012, 2018). Due to its rapid onset, the ERN is thought to reflect early and automatic stages of error processing and has been interpreted as a signal for mismatch/error detection (Coles et al., 2001; Falkenstein et al., 2000; Gehring et al., 1993), a negative reinforcement learning signal (Holroyd & Coles, 2002), or a marker of post-response conflict processing (Yeung et al., 2004).

Previous research has found reductions in ERN amplitudes associated with externalizing problems and disorders (Hall et al., 2007; Lutz et al., 2021; Pasion & Barbosa, 2019), as well as specifically with the impulsive/antisocial traits of psychopathy (Vallet et

al., 2021). However, the extent to which distinct variants of the ERN response overlap in their relationships with disinhibitory/externalizing traits, and whether this association is task dependent, is less clear. Some studies have reported stronger associations between disinhibition/externalizing and no-go variants of the ERN (Pasion & Barbosa, 2019; Ribes-Guardiola, Poy, Patrick et al., 2020), while others have not found convincing evidence for a moderating role of task context (Heritage & Benning, 2013; Lutz et al., 2021; Vallet et al., 2021). In this regard, a recent study indicated that a common factor combining flanker and no-go variants of the ERN significantly related to externalizing (indicative of reduced amplitudes), suggesting that it is the shared variance across inhibitory and interference variants of this brain response that relates to externalizing psychopathology (Pasion et al., 2023).

The current study

The present study sought to clarify different aspects of *inhibitory control* contributing to trait *disinhibition*, by examining associations between this triarchic domain and distinct ERP indicators extracted from a modified flanker-stop-signal task administered to a large mixed-gender sample of undergraduates. Specifically, we focused on the Stop-P3 component, which reflects inhibitory control processing, as well as the Stop and Flanker ERNs, which are indicators of error monitoring. Additionally, we aimed to test for the unique association of trait disinhibition with these ERP responses while controlling for the other two dimensions of the triarchic model —given the moderate overlap between disinhibition and meanness scores (Drislane et al., 2014; Stanley et al., 2013; Strickland et al., 2013), as well as previous findings linking boldness to some task-based indicators of disinhibited performance (Ribes-Guardiola, Poy, Segarra et al., 2020; Snowden et al., 2017).

Psychopathic traits were assessed using a multi-measurement approach, by extracting scores on three factor indexes for each triarchic dimension from the triarchic

scales of the *Triarchic Psychopathy Measure* (TriPM; Patrick, 2010), the triarchic scales (Hall et al., 2014) of the *Psychopathic Personality Inventory-Revised* (PPI-R; Lilienfeld & Widows, 2005), and the triarchic scales (Drislane et al., 2018) of the *NEO Personality Inventory Revised* (NEO PI-R; Costa & McCrae, 1992).

Based on previous evidence (e.g., Pasion et al., 2018; Vallet et al., 2021), it was hypothesized that reduced amplitudes of P3 and ERN would be specifically associated with disinhibition factor scores, but not with meanness or boldness factor scores.

Method

Participants

Study participants were 258 undergraduates (204 women, 54 men; M_{age} = 19.9; SD_{age} = 3.8) recruited from the Universitat Jaume I of Castellón (Spain). Before the experimental session, three participants were discarded due to medical/psychiatric conditions (i.e., epilepsy or major depression). None of the participants presented uncorrected visual or auditory impairments. The experimental research procedures were approved by the Ethical Committee of the Universitat Jaume I and adhered to ethical principles for human research outlined in the Declaration of Helsinki. All participants provided written informed consent before self-report data collection and before the experimental session. They were compensated with course credits for their participation.

Out of the initial sample, 11 participants were excluded from all analyses due to their poor task performance: accuracy for flanker discrimination trials below 70% (n = 4), number of responses to flanker discrimination trials lower than the mean minus two standard deviations (n = 3), or both (n = 4). Additionally, 20 participants were excluded from the response-locked ERPs analysis due to having fewer than six incorrectly responded discrimination trials or less than 25% valid trials in some condition of interest (i.e., flanker discrimination trials and stop inhibition trials, and flanker and stop error trials).

Consequently, the final sample consisted of 244 participants for behavioral and stimulus-locked ERP analyses (195 women, 49 men; M_{age} = 19.9; SD_{age} = 3.6), and 224 participants for response-locked ERP analyses (181 women, 43 men; M_{age} = 19.9; SD_{age} = 3.7).

Self-report measures

Triarchic Psychopathy Measure (TriPM). The TriPM (Patrick, 2010; Spanish version, Poy et al., 2014) assesses the three trait constructs of boldness, meanness and disinhibition proposed in the triarchic model of psychopathy (Patrick et al., 2009). This self-report measure consists of 58 items that are answered using a 4-point Likert scale (0 = *false*, 1 = *somewhat false*, 2 = *somewhat true*, 3 = *true*). Scores for each TriPM scale are computed as the sum of the constituent items: TriPM-Boldness and TriPM-Meanness scores can range from 0 to 57 (19 items), and Tri-PM Disinhibition scores can range from 0 to 60 (20 items). All three scales showed good internal consistency reliability, with Cronbach's α coefficients in the final sample (N = 244) of .88, .72, and .77 for Boldness, Meanness, and Disinhibition scale scores, respectively.

Psychopathic Personality Inventory—Tri Scales (PPI-Tri). The PPI-Tri scales (Hall et al, 2014) assess the triarchic dimensions of boldness, meanness and disinhibition using items from the *Psychopathic Personality Inventory-Revised* (PPI-R; Lilienfeld & Widows, 2005; Spanish version, López et al., 2013), a 154-item self-report measure that evaluates psychopathic traits across eight content scales. Each item is assessed using a 4-point Likert type format scale (1 = *false*, 2 = *somewhat false*, 3 = *somewhat true*, 4 = *true*). PPI-Boldness consist of eight items from the PPI-R Fearlessness scale, seven from Social Potency, six from Stress Immunity, and one from Machiavellian Egocentrity content scales, and another one from the PPI-R Virtuous Responding validity scale; PPI-Meanness comprises thirteen items from PPI-R Coldheartedness and five from Machiavellian Egocentrity; and PPI-Disinhibition consists of three items from Rebellious Nonconformity,

five from Blame Externalization, and ten from Carefree Nonplanfulness. Scores for each PPI-based triarchic scale are computed as the mean of the constituent items. In the current sample, all three scales scores showed good internal consistency (PPI-Boldnes α = .84; PPI-Meanness α = .79; PPI-Disinhibition α = .72).

NEO Five Factor Inventory-Tri. The *NEO Personality Inventory Revised* (NEO PI-R; Costa & McCrae, 1992; Spanish version, Costa & McCrae, 1999) is a 240-item inventory that assesses the Five Factor Model (FFM) domains, including Neuroticism, Extraversion, Agreeableness, Conscientiousness, and Openness, as well as six lower-order facet scales for each one. Items are rated using a 5-point Likert scale from 1 *strongly disagree* to 5 *strongly agree*. In this study, the triarchic scales developed by Drislane et al. (2018) were used to assess the three triarchic dimensions. NEO-Boldness scale consists of twelve items from NEO-Neuroticism, nine from NEO-Extraversion and two from NEO-Agreeableness; NEO-Meanness is composed by seventeen items from NEO-Agreeableness, three from NEO-Openness, four from NEO-Extraversion, and one from NEO-Neuroticism; and NEO-Disinhibition comprises eleven items from NEO-Conscientiousness, seven from NEO-Neuroticism, and three from NEO-Agreeableness. Scores for each NEO-based triarchic scale were computed as the mean of the constituent items. Cronbach's α in the current sample were .89 for NEO-Boldness, .80 for NEO-Meanness, and .84 for NEO-Disinhibition.

Descriptive statistics (mean, standard deviation, range) for all triarchic scales are presented for all participants, as well as for men and women separately, in Table 1. Independent *t*-tests revealed that men scored significantly higher than women in all triarchic scales (ts > |1.99|; ps < .048), except for NEO-Disinhibition (ts < |.25|; ps > .806). Therefore, separate gender-corrected t scores were calculated for each triarchic scale using a mean of 50 and a standard deviation of 10. An exploratory factor analysis (EFA) was conducted on

the gender-corrected t scores of the triarchic scales to identify one factor representing each triarchic disposition. The results of the principal-axis factor analysis with Promax rotation (Barlett's $\chi^2 = 1262$, $p < .001$; KMO = .772) revealed three factors (eigenvalues = 2.40, 2.01, and 1.64), which accounted for 69.0% of the total variance (see the factor loadings in Table 2); Parallel Analyses corroborated the factor structure obtained by the EFA. An omnibus factor index for each triarchic dimension was computed for each participant by calculating regression-based component scores on each of the factors extracted from the EFA.

Procedure and experimental task

The experimental session was conducted individually in a dimly lit room, with participants seated 110 cm away from a PC Pentium Core 2 Duo (Intel) computer monitor where stimuli were presented. Electrophysiological responses were recorded while participants performed a modified version of the Eriksen flanker task (Eriksen & Eriksen, 1974), previously used in Vilà-Balló et al. (2014). The task consisted of a flanker task, in which the participants had to respond by pressing a button with their left or right index fingers to indicate the direction of the central arrow in a string of five horizontally presented arrows in the center of the screen. One third (33.3%) of trials were congruent (i.e., the central arrow pointed in the same direction as the four surrounding arrows), and 50% of trials were incongruent (i.e., the central arrow pointed in opposite direction to the four surrounding arrows).

Table 1. *Descriptive Statistics and Reliability for Triarchic Scale Scores in the Overall Sample, and for Women and Men Separately*

Triarchic scales	Overall (N = 258)		Women (N = 204)		Men (N = 54)		Gender Comparison	
	M (SD)	Min.-Max.	M (SD)	Min.-Max.	M (SD)	Min.-Max.	t	p
TriPM-Boldness	28.07 (9.98)	0-51	26.77 (9.84)	0-51	33 (9.00)	11-49	**-4.21**	< .0001
TriPM-Meanness	9.37 (6.33)	0-34	8.60 (6.02)	0-34	12.26 (6.66)	1-32	**-3.88**	.0001
TriPM-Disinhibition	12.91 (7.15)	0-34	12.19 (6.61)	0-34	15.63 (8.45)	0-33	**-3.20**	.002
NEO-Boldness	1.91 (.64)	.35-3.57	1.83 (.62)	.35-3.56	2.23 (.59)	.78-3.13	**-4.19**	< .0001
NEO-Meanness	1.22 (.46)	.32-3.04	1.27 (.45)	.32-3.04	1.56 (.43)	.64-2.64	**-4.24**	< .0001
NEO-Disinhibition	1.65 (.52)	.38-3.14	1.65 (.53)	.38-3.14	1.67 (.50)	.67-2.73	-.25	.806
PPI-Boldness	2.50 (.45)	1.39-3.70	2.44 (.45)	1.39-3.70	2.73 (.41)	1.61-3.57	**-4.35**	< .0001
PPI-Meanness	1.76 (.38)	1.06-3.78	1.71 (.36)	1.06-3.11	1.96 (.41)	1.28-3.78	**-4.45**	< .0001
PPI-Disinhibition	2.02 (.35)	1.22-2.94	2.00 (.36)	1.22-2.94	2.10 (.31)	1.5-2.94	**-1.99**	.048

Note. α = Cronbach's alpha; TriPM = Triarchic Psychopathy Measure; NEO = NEO Personality Inventory Revised; PPI = Psychopathic Personality Inventory-Revised. Significant comparisons are highlighted in bold.

Table 2. *Loadings of Triarchic Scales on the Three Factors Extracted from a Principal-Axis Factor Analysis*

	Factor Loadings		
	Factor 1 (Boldness)	**Factor 2 (Disinhibition)**	**Factor 3 (Meanness)**
TriPM-Boldness	**.93**	-.04	-.00
NEO-Boldness	**.90**	-.10	.09
PPI-Boldness	**.86**	.12	-.08
TriPM-Disinhibition	-.00	**.81**	-.02
NEO-Disinhibition	.01	**.85**	-.07
PPI-Disinhibition	-.02	**.69**	.07
TriPM-Meanness	.00	.25	**.65**
NEO-Meanness	.03	.13	**.61**
PPI-Meanness	-.03	-.16	**.88**

Note. TriPM = Triarchic Psychopathy Measure; NEO = NEO Personality Inventory Revised; PPI = Psychopathic Personality Inventory-Revised.

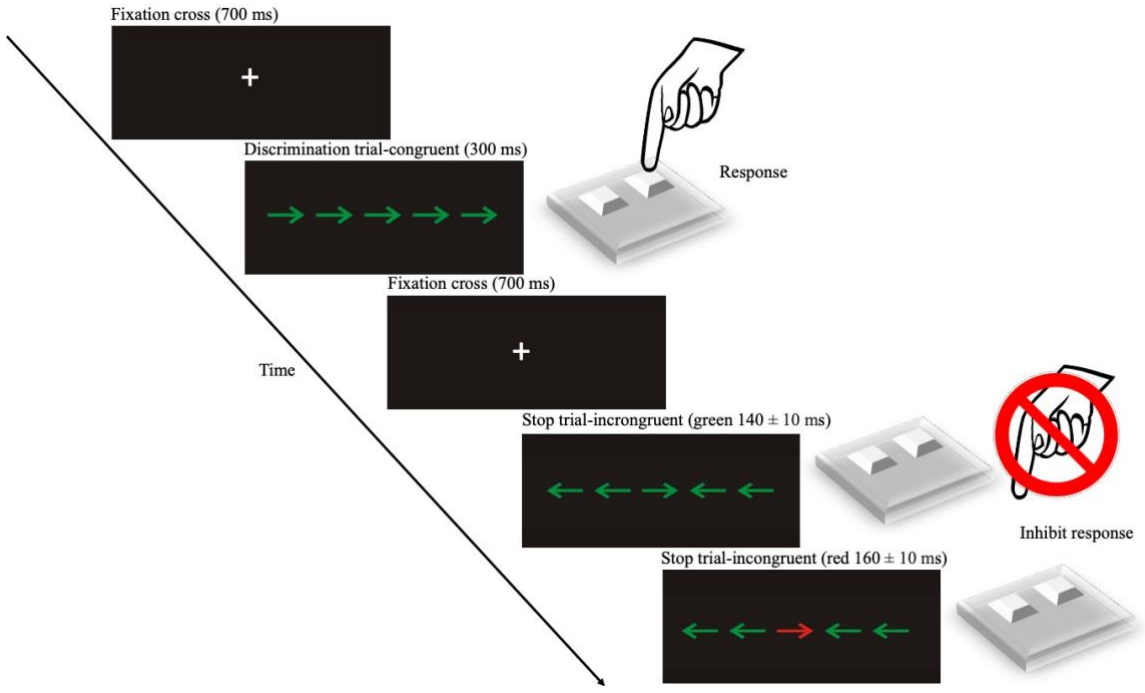
Factor loading values > .40 are highlighted in bold.

The remaining 16.7% of trials were stop trials —a variant of the stop-signal paradigm (Band et al., 2003)— in which the central arrow changed color from green to red after a variable delay (stop-signal delay), indicating the participant to inhibit their response. In order to achieve an inhibition rate of 50% approximately, the stop-signal delay was adjusted based on the participant's accuracy performance by means of a staircase tracking algorithm (Band & van Boxtel, 1999): following a failed inhibition, the stop-signal delay increased by 10 ms, while after a successful inhibition, the stop-signal delay decreased by 10 ms. Each trial began with a 700-ms fixation cross (+), followed by the presentation of the arrow-string stimulus for 300 ms. The initial stop-signal delay was set at 140 ms. The task was divided into five blocks of 240 trials, resulting in a total of 1200 trials. Participants had 7-seconds rest breaks every 20 trials, as well as 30-seconds rest breaks between blocks. Before starting the task, participants completed 40 training trials. The overall duration of the task, including practice trials and breaks, was approximately 25 minutes. A visual representation of the task can be

found in Figure 1. The order and timing of stimulus presentation were controlled using the Presentation® v.20.1 software (Neurobehavioral Systems, Inc. Albany, CA, USA).

Three measures of behavioral performance were computed for each participant: the mean reaction time for correct responses (RT Correct) and error responses (RT Error) in flanker discrimination trials, the percentage of discrimination errors (% Error), and the stop-signal reaction time (SSRT). The SSRT was computed by subtracting the mean stop-signal delay from the median reaction time for correct flanker discrimination responses (Band et al., 2003; Logan et al., 1997; Logan & Cowan, 1984; Vilà-Balló et al., 2014). All behavioral measures exhibited good split-half reliability (odd-even method, Spearman-Brown corrected): .99 for RT Correct, .60 for RT Error, and .98 for SSRT.

Figure 1. *Scheme of the Task Procedure*



Modified variant of the Eriksen flanker task (Eriksen & Eriksen, 1974), in which participants are asked to indicate the direction of the central arrow in the string of five arrows by making a left or right index-finger button press (discrimination trials: 83.3%) and to withhold responding —following a variant of the stop-signal paradigm (Band et al., 2003)— when the central arrow changes color from green to red (stop trials: 16.7%). The central arrow remains green throughout the stimulus presentation time (300 ms) in discrimination trials and changes to red after a variable delay (initially set at 140 ms and modulated in subsequent trials by the accuracy of participants responses by ± 10 ms) in stop trials.

Psychophysiological recording and data reduction

The EEG was recorded using a NetAmps 400 amplifier system with 257-channel high density Hydrocel Geodesic Sensor Nets from Electrical Geodesics Inc. (EGI; OR, USA). The continuous EEG recording was digitized at a sampling rate of 250 Hz using a 24-bit analog-to-digital converter. The data were referenced to the vertex scalp site (Cz) and recorded using the NetStation v5.4.1.2 software installed on a MacBook Pro (Apple) computer. As recommended by manufacturer's guidelines, scalp impedances were kept below 50 kΩ. Offline data preprocessing was conducted using Brain Electrical Source Analysis software (BESA v7.1.2.1; MEGIS software GmbH, Germany). First, a visual inspection of the raw recordings was performed to identify and interpolate data for electrodes with poor signal quality. Second, a principal component analysis-based adaptive artifact-correction was used in BESA to manually correct the eyeblink (EOG) and electrocardiogram (EKG) artifacts present in the continuous EEG data. The artifact-corrected data were low-pass filtered at 30 Hz.

The preprocessed data were segmented into epochs ranging from -100 to +1000 ms relative to stimulus presentation to extract stimulus-locked event-related potentials (ERPs). Additionally, epochs ranging from -100 to +600 around participant's response were extracted to capture response-locked ERPs. Finally, following epoch segmentation, a semi-automated procedure was used to detect and reject epochs that exhibit amplitude deflections greater than 75 μV between successive sampling points, exceeded an amplitude threshold of 120 μV, or fell below a low signal threshold of 0.01 μV. The accepted epochs were then converted to the average reference.

ERP measurement

The ERPs were extracted by using temporospatial Principal Component Analyses (PCA; Dien, 2012; Dien & Frischkoff, 2005) with the ERP PCA Toolkit version 2.93 (Dien,

2010). This method extracts linear combinations of all data points meeting certain criteria that tend to distinguish between consistent patterns of electrocortical activity (Foti et al., 2009). Three separate PCAs were conducted: one for stimulus-locked epochs and two for response-locked epochs. Due to latency differences between the ERN observed for erroneous responses in stop trials (Stop-ERN; Mean = 71 ms) and in flanker discrimination trials (Flanker-ERN; Mean = 55 ms), $t(223) = 8.6$, $p < .001$, $d = .57$, also found in previous studies (Vilà-Balló et al., 2014), both types of errors were analyzed independently using separate PCAs. Each PCA also included the averages of correctly responded trials as a control condition.

For the stimulus-locked data, a temporal PCA with Promax rotation (Dien et al., 2007) was performed on the 275 timepoints (from -100 ms pre to 1000 ms post stimuli), considering the 244 participants included for stimulus-locked ERPs analysis (see Participants section), both discrimination (flanker stimuli) and successful-stop trials condition averages, and all electrodes (257) as observations. Based on the Scree plot, 11 temporal factors were extracted for rotation. The covariance matrix and Kaiser normalization were used for the PCA, following the recommendations of Dien and Frischkoff (2005). Each temporal factor obtained can be understood as a *virtual epoch* and can be defined by its *factor loadings* (describing the time-course of each factor) and *factor scores* (providing a factor value for each combination of subject, condition, and electrode). The spatial information was preserved in the temporal PCA, allowing the reconstruction of scalp topographies for each participant, condition and timepoint by multiplying the corresponding factor scores by the factor loading and standard deviation (Dien, 1998). To further reduce the spatial dimensions of the data set, a spatial PCA was conducted for each temporal factor, using the temporal factor scores for each participant, condition, and electrode, as observations. The resulting Scree plots were averaged across all temporal factors such that

the same number of spatial factors was extracted for each temporal factor. Based on that, 10 spatial factors were extracted from each temporal factor using an Infomax rotation. Each spatial factor obtained represents a *virtual electrode* and, like temporal factors, could be described by its *factor loadings* —representing the scalp topography of each factor— and *factor scores* —providing a factor value for each combination of participant, condition, and timepoints (*virtual ERPs*). To accurately interpret the PCA results, each temporospatial factor (reconstructed in microvolts) was represented by multiplying the factor scores with the corresponding factor loadings, allowing the assessment of both the time course and scalp topography of EEG activity for each temporospatial factor. In total, the temporospatial PCA yielded 110 factor combinations (10 spatial factors extracted for each of the 11 temporal factors).

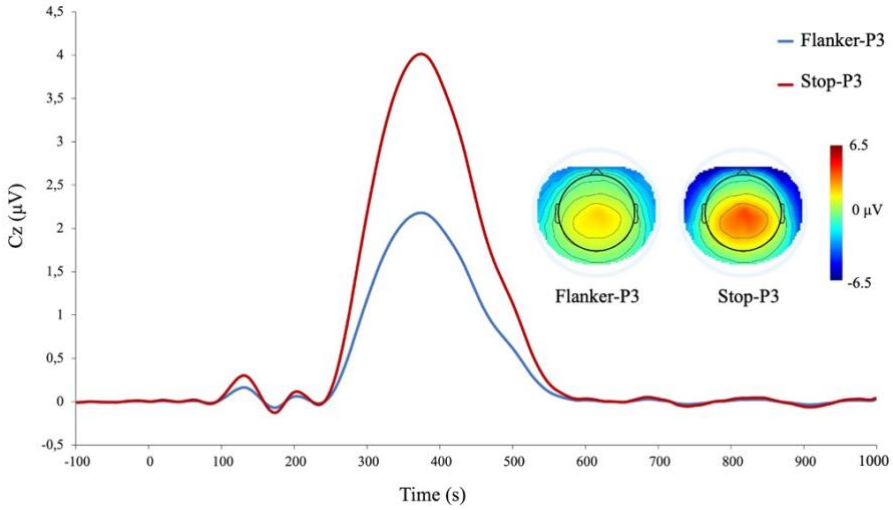
The same procedure was repeated twice for the response-locked data, separating the different types of error responses into different PCA, as mentioned above. First, a temporal PCA with Promax rotation was performed on the 175 timepoints (from -100 to 600 ms post-response), considering the 224 participants included for the response-locked ERPs analysis (see Participants section), the correct and stop error conditions, and all electrodes as observations. Based on the Scree plot, 7 temporal factors were extracted for rotation. Subsequently, a spatial PCA was conducted, from which 8 spatial factors were extracted. This temporospatial PCA resulted in a total of 56 factor combinations. Second, a PCA was performed including the flanker discrimination error (instead of the stop error) condition, along with the correct response condition. In this case, 8 temporal factors and 9 spatial factors were extracted, resulting in 72 factor combinations.

From these PCAs, we extracted the temporospatial factors scores representing the ERPs of interest for further analyses (i.e., Stop-P3; Stop-ERN; Flanker-ERN). From the stimulus-locked data PCA, the scores of the TF1SF1 best described the P3 ERP component,

with a peak latency of 372 ms, maximal activation at Cz, and accounted for the 16% of variance in stimulus-locked data (see Figure 2a). Regarding the response-locked data PCA, factor scores from the temporospatial factors representing the ERN were considered. For the first PCA, which included the stop error condition, the TF4SF1 factor showed a peak latency of 72 ms, maximal activity at Cz, and explainied 3% of the variance (see Figure 2b). As for the second PCA, which included the flanker error condition, the TF5SF1 factor exhibited a peak latency at 40ms, maximal activity at Cz, and explained 1.3% of the variance (see Figure 2b).

Figure 2. *Waveforms and Topographic Maps for the Temporospatial Factors Derived from Principal Component Analyses. (a) Flanker and Stop P3, (b) Stop ERN and CRN, and (c) Flanker ERN and CRN*

(a)



(b)

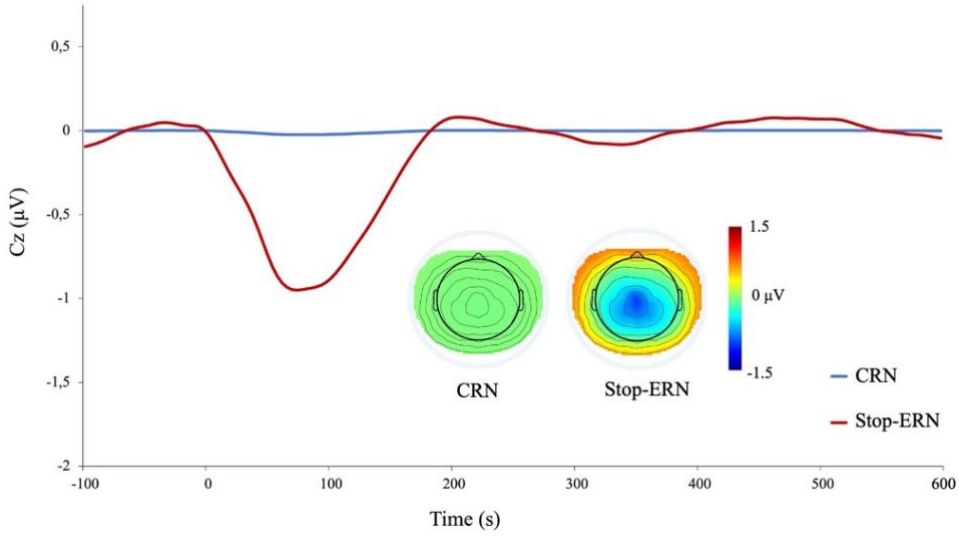
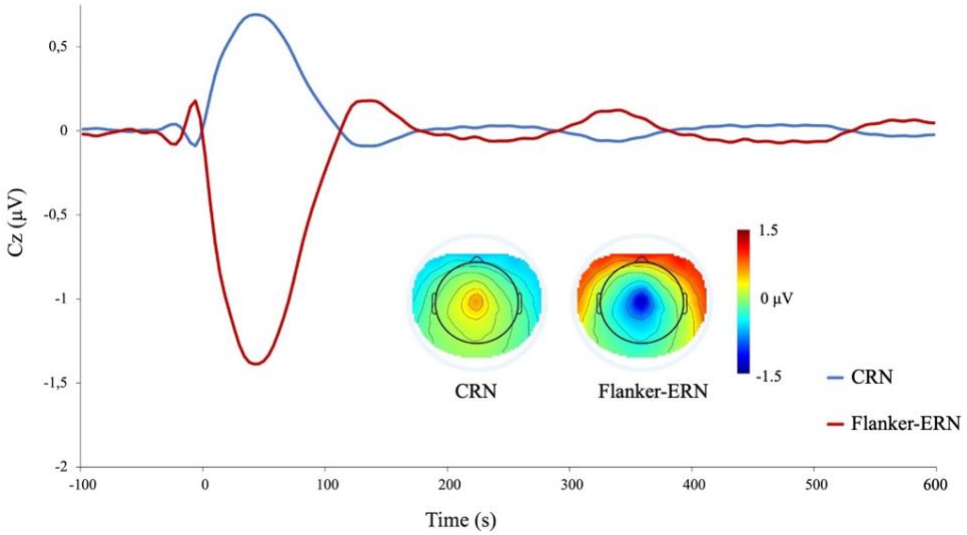


Figure 2. *(Cont.)*



Note. CRN = Correct-Related Negativity; ERN = Error-Related Negativity

Statistical analyses

The statistical analyses were conducted using Jamovi 2.3.21.0 software (The Jamovi Project, Sydney, Australia). To minimize the disproportionate influence of outlying ERP amplitude scores in subsequent statistical analyses, a winsorization procedure (Wilcox, 2012) was applied. Scores exceeding the 75th percentile plus 1.5 times the interquartile range of the distribution or falling below the 25th percentile minus 1.5 the interquartile range of the distribution were replaced by the maximum or minimum value within these ranges. This procedure corrected 6.79% of the total scores.

First, paired samples *t*-tests between the different ERPs derived from each PCA were computed to examine the differential brain reactivity during response inhibition (i.e., Stop-P3 vs. Flanker-P3) and error processing (i.e., Stop-ERN vs. Correct; and Flanker-ERN vs. Correct). Second, bivariate Pearson correlations were calculated to explore the associations between triarchic factor scores, temporospatial-PCA based ERP scores, and behavioral data. Hypothesized significant associations between trait disinhibition scores and Stop-P3 (TF1SF1), Stop-ERN (TF4SF1), and Flanker-ERN (TF5SF1) were followed up by

conducting partial correlational analyses on which relationships between disinhibition scores and each ERP of interest were examined when controlling for its overlap with boldness and meanness scores. Based on previous research indicating that trait disinhibition relates to different variants of the P3 response (Pasion et al., 2023; Ribes-Guardiola, Poy, Patrick et al., 2020; Venables et al., 2018), raw PCA-derived Stop-P3 factor scores were considered as the main stimulus-locked ERP variable. Given the evidence suggesting that disinhibition/externalizing relates to error-related brain activity but not to ERP activity on correct trials (Hall et al., 2007; Pasion et al., 2023), residualized PCA-based ERN factor scores were created from two separate regression models on which the error condition from each of the two response-locked temporospatial PCA analyses was used as the criterion (Stop-ERN [TF4SF1]; Flanker-ERN [TF5SF1]), and its corresponding factor scores from the correct condition as the predictor. The unstandardized residuals from each regression were saved (see Meyer et al., 2017). These variables thus better reflect the unique variance associated with error-processing of each type, independent from general correct-response monitoring activity.

Reliability of the ERP scores was estimated using the split-half method, which was calculated by re-computing the same PCA analyses described above for odd and even numbered trial averages. Factor scores corresponding to the factor combinations that best resembled the temporospatial factor combinations described above were saved, and correlations were run between these separate factor scores for each component (i.e., odd and even Stop-P3 factor scores; odd and even residual Stop-ERN factor scores; odd and even residual Flanker-ERN factor scores), which were corrected for attenuation using the Spearman-Brown prophecy formula. The resulting reliabilities were as follow: .94 for Stop-P3 tsPCA factor scores; .85 for Stop-ERN tsPCA factor scores; .44 for residualized Flanker-ERN tsPCA factor scores.

Results

Table 3 shows the descriptive statistics (mean, standard deviation, range) for ERP and behavioral measures for all participants, and separately for men and women, as well as independent *t*-tests between genders. Table 4 presents the bivariate correlations between self-report, ERP, and behavioral data.

Stimulus-locked ERPs: P3

P3 amplitudes were found to be significantly larger for stop than for flanker discrimination trials, $t(243) = 14.3$, $p < .001$, $d = .91$, indicating enhanced neural responding in the condition calling for inhibition (see Figure 2a). Contrary to our hypothesis, no significant correlations were observed between P3 amplitudes and triarchic factor scores, rs $< |.06|$, ps $> .352$ (see Table 4).

Response-locked ERPs: ERNs

For both temporospatial factor scores, larger amplitudes were observed for both types of erroneous responses compared to correct responses: Stop-ERN [TF4SF1], $t(223) = -12.0$, $p < .001$; $d = -.80$; Flanker-ERN [TF5SF1], $t(223) = -13.9$, $p < .001$; $d = -.93$ (see Figure 2b). Notably, a significant correlation was found between Disinhibition factor scores and both residualized Stop-ERN and residualized Flanker-ERN amplitudes rs $> .15$, ps $< .05$, indicative of reduced ERN responding in individuals with higher disinhibition scores. Figure 3 illustrates this finding by depicting the ERN waveforms for participants scoring in the upper and lower quartiles on the Disinhibition factor (for Stop-ERN, see Figure 3a; for Flanker-ERN, see Figure 3b). Boldness and Meanness factor scores did not exhibit significant associations with either ERN variant.

However, follow-up partial correlation analyses revealed that the association between Disinhibition factor scores and each variant of the ERN was not significant after accounting for the overlap with Meanness and Boldness factor scores: partial $r = .07$, $p = .299$, for Stop-ERN; partial $r = .07$, $p = .265$, for Flanker-ERN.

Table 3. *Descriptive Statistics and Reliability for ERP Components and Behavioral Data in the Overall Sample, and for Women and Men Separately*

	Overall		Women		Men		Gender Comparison	
	M (SD)	**Min.-Max.**	**M (SD)**	**Min.-Max.**	**M (SD)**	**Min.-Max.**	**t**	**p**
ERPs								
TF1SF1 Flanker-P3[a]	3.47 (2.5)	-.31-9.40	3.13 (2.3)	-.31-9.4	4.80 (2.7)	-.04-9.4	**-4.41**	< .0001
TF1SF1 Stop-P3[a]	6.44 (4.0)	-3.09-16.42	5.7 (3.6)	-3.09-16.42	9.39 (4.3)	.61-16.4	**-6.22**	< .0001
Residualized TFSF1 Stop-ERN[b]	1.00 (.8)	-2.89-2.22	.20 (.6)	-1.77-1.62	-.83 (1.2)	-2.89-2.22	**8.14**	< .0001
Residualized TF5SF1 Flanker-ERN[b]	5.95 (1.9)	-4.94-4.88	.19 (1.8)	-4.87-4.88	-.78 (2.1)	-4.95-2.51	**3.08**	.002
Behavioral								
% Error[a]	5.3 (3.5)	0.1-18.4	5.4 (3.5)	0.1-18.4	5.0 (3.8)	.2-15.5	.71	.478
RT Correct[a]	426.9 (48.8)	334.5-568.0	428.52 (48.7)	334.5-568.0	420.26 (50.0)	341.8-533.5	1.06	.291
RT Error[a]	330.2 (42.4)	190.9-591.4	328.49 (43.3)	190.9-591.4	336.82 (39.0)	272.8-447.7	-1.23	.220
SSRT[a]	237.9 (23.4)	183.8-317.6	239.00 (23.4)	191.4-317.6	233.70 (23.3)	183.8-290.7	1.42	.158

Note. α = Cronbach's alpha; CRN = Correct-Response Negativity; ERN = Error-Related Negativity; RT = Reaction Time; SSRT = Stop-Signal Reaction Time.
Significant comparisons are highlighted in bold.
Superscripts letters designate group sizes ([a] *Overall n* = 244, Women *n* = 195, Men *n* = 49; [b] *Overall n* = 224, Women *n* = 181, Men *n* = 43).

Table 4. *Bivariate Correlations between Self-report, ERP and Behavioral Data for the Overall Sample*

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*
Self-Report Data											
1. Boldness Factor [a]	-										
2. Meanness Factor [a]	.25***	-									
3. Disinhibition Factor [a]	.02	.68***	-								
ERP Data											
4. Stop-P3 [b]	.05	-.02	.00	-							
5. Flanker-P3 [b]	.06	-.02	-.02	.58***	-						
6. Residualized Stop-ERN [c]	-.08	.13	.16*	-.28***	-.12	-					
7. Residualized Flanker-ERN [c]	-.13	.11	.15*	-.16*	-.08	.30***	-				
Behavioral Data											
8. % Error [b]	.09	.12	.10	-.22***	.13*	.22**	.13	-			
9. RT Correct [b]	-.09	-.06	-.01	-.09	-.31***	.07	.12	-.49***	-		
10. RT Error [b]	-.07	.03	-.00	.01	-.09	.02	-.06	-.19**	.31***	-	
11. SSRT [b]	.00	-.04	-.04	-.19**	-.08	.23***	.20**	.19**	.22***	.02	-

Note. ERN = Error-Related Negativity; SSRT = Stop-Signal Reaction Time; RT = Reaction Time
*$p < .05$, ** $p < .01$, *** $p < .001$
Superscripts letters designate group sizes ([a] $n = 258$; [b] $n = 244$; [c] $n = 224$).

These results indicate that the relationship between disinhibition scores and blunted amplitudes of both ERN variants was partly explained by the overlap with the other triarchic dimensions, particularly meanness scores (which correlated highly with disinhibition scores, $r = .68$, and tended to be related to each ERN variant: .13 for Stop-ERN and .11 for Flanker-ERN; see Table 4).

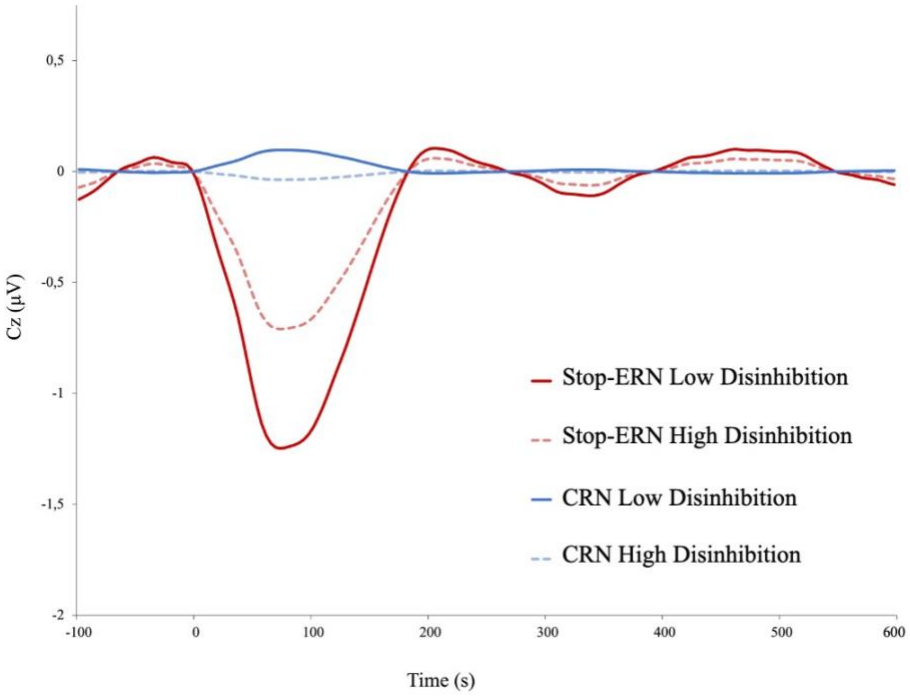
Given the significant relationships observed between Disinhibition factor scores and Stop and Flanker ERN amplitudes, a multiple regression analysis was conducted to examine the extent to which the unique variance of each ERN, as well as their shared variance, best accounted for these associations. The multiple regression analysis, using both residualized Stop and Flanker ERN amplitudes as predictors of Disinhibition factor scores, yielded a significant model ($p = .015$; $R^2 = .037$), but neither ERN variant showed a significant unique predictive contribution to disinhibition scores (βs < .12; ps > .095), suggesting that the shared variance between both ERN variants best accounted for the associations observed with this triarchic dimension.

Discussion

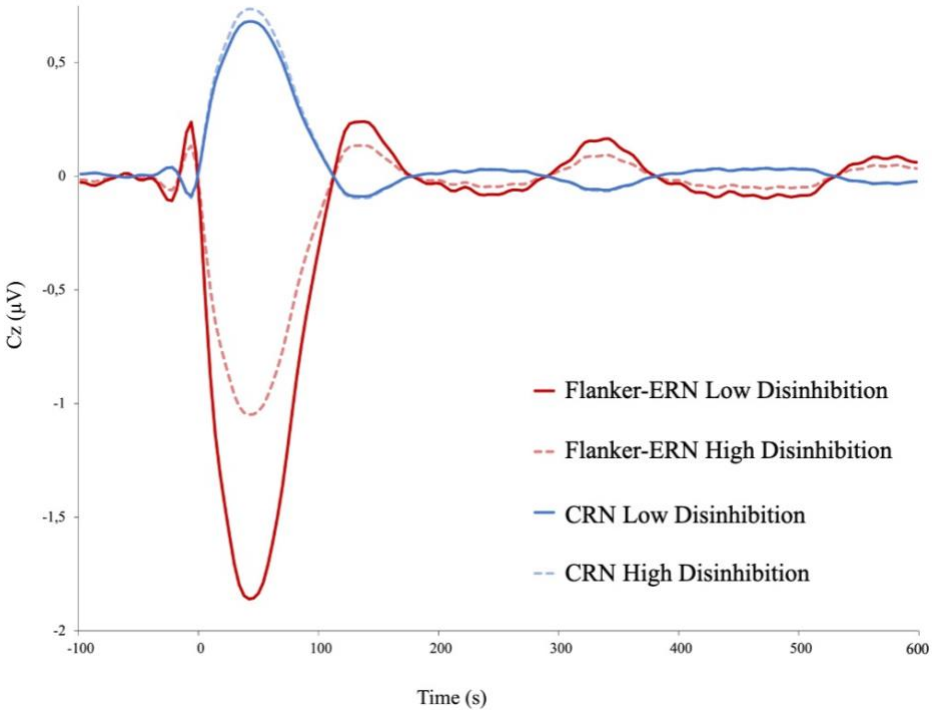
The current study aimed to investigate the relationship between electrophysiological indicators indexing different aspects of inhibitory control, derived from a modified flanker-stop-signal task, and the triarchic disposition of disinhibition, considering also the other triarchic dispositions. Our main hypotheses were partially supported, given that we found significant associations between disinhibition and ERP indices of error monitoring (Stop-ERN, Flanker-ERN), but not of inhibitory processing (Stop-P3). The implications of these findings for our understanding of the neurocognitive processing deviations underlying the disinhibition features of psychopathy are discussed in the following subsections.

Figure 3. *Waveforms for the Response-Locked Temporospatial Factors derived from PCAs for Participants Scoring in the Upper (Dotted Lines; N =56) and Lower (Solid Lines; N= 56) Quartiles on the Disinhibition Factor. (a) Correct Responses (CRN; Blue) and Inhibition Errors (Stop ERN; Red), and (b) Correct Responses (CRN; Blue) and Flanker Discrimination Errors (Flanker ERN; Red) in the Modified Flanker-Stop-Signal Task*

(a)



(b)



Note. CRN = Correct-Related Negativity; ERN = Error-Related Negativity; PCA = Principal Component Analysis

Disinhibition-related alterations in error processing

With regard to ERPs indexing error monitoring, both ERN variants (Flanker and Stop) measured in this study exhibited reduced amplitudes associated with disinhibition factor scores. These findings are consistent with previous studies that have reported reduced amplitudes of both inhibitory and choice/discrimination variants of the ERN in relation to impulsive/antisociality traits and behaviors using different versions of stop-signal tasks (Heritage & Benning, 2013; Vilà-Balló et al., 2014).

Furthermore, when both ERN variants were considered as joint predictors of disinhibition, neither variant significantly predicted disinhibition factor scores individually, but the overall model was significant, suggesting that it is the shared variance between discrimination and inhibitory variants of this brain response that relates to disinhibition. These results align with studies indicating that the shared variance across inhibitory and flanker discrimination tasks (Pasion et al., 2023) is more relevant to externalizing traits than the unique variance in inhibitory control variants of the ERN (Ribes-Guardiola, Poy, Patrick et al., 2020).

It is worth noting that while previous work has provided compelling evidence of externalizing-related reductions in ERN amplitude in inhibitory control tasks (Paiva et al., 2020; Ribes-Guardiola, Poy, Patrick et al., 2020), or combined tasks involving some form of inhibition (e.g., mixed no-go flanker tasks or choice-stop signal tasks; see Hall et al., 2007; Heritage & Benning, 2013; Nelson et al., 2011; Vilà-Balló et al., 2014), disinhibition-related reductions in ERN amplitudes have not been found in purely flanker discrimination tasks (Ribes-Guardiola, Poy, Patrick et al., 2020; Venables et al., 2018). The main difference between interference and inhibitory paradigms lies in the response requirements across trials. Interference tasks, such as the flanker task, require a motor response in all conditions, whereas inhibitory tasks involve trials that require either the execution of a motor response

or its inhibition (Gratton et al., 2018). Taken together, it is possible that some degree of inhibition of prepotent responding is necessary in the task in order to most reliably detect deficits in error processing associated with disinhibition. Some aspects of the current data support this interpretation. For example, both variants of the ERN were positively correlated with SSRT, a behavioral measure of inhibitory control capacity (Band et al., 2003; Logan et al., 1997; r = .23, p < .001, for Stop-ERN and r = .20, p = .003, for Flanker-ERN). This indicates that participants with poorer inhibitory control performance in stop trials also exhibited smaller ERN amplitudes in *both* types of error trials. It should be noted, however, that prior work has suggested that the SSRT might not be a suitable indicator of disinhibition-related impairments in inhibitory control capacity (see Venables et al., 2018), which is also consistent with the null relationship found between disinhibition scores and the SSRT in the current study (r = -.04, p = .581). Thus, although the SSRT is a widely used behavioral measure of action stopping abilities (Band et al., 2003; Logan et al., 1997), its validity as a behavioral indicator of disinhibition-related impairments in inhibitory control capacity is in need of further research.

Disinhibition and ERP reactivity to inhibitory cues (Stop)

Our study did not find evidence of impairments in the neural processing of response inhibition in high-disinhibited individuals in the context of a modified flanker-stop-signal task, as indicated by the lack of statistically significant correlations between Stop-P3 amplitudes and disinhibition factor scores (Table 4). This unexpected result is noteworthy, especially in light of prior work suggesting that disinhibition/externalizing reductions in P3 amplitudes reflect, at least in part, a process associated with low inhibitory control capacity (Brennan & Baskin-Sommers, 2018; Venables et al., 2018; see also Pasion et al., 2018 for a review). In contrast to these previous suggestions, we did not find evidence of disinhibition-related impairments at the P3 level in response to cues prompting the cancellation of already

initiated behavioral responses (i.e., Stop-P3 amplitudes).

The majority of studies linking P3 amplitude reductions to externalizing problems and traits have used oddball variants of this brain response (Carlson et al., 2009; Patrick et al., 2006; Venables et al., 2018; Yancey et al., 2013). Other work has likewise demonstrated that the *shared* variance between oddball and other P3 variants, including those from flanker and other cognitive processing tasks (e.g., choice-feedback, novel, noise-probe), is inversely related to externalizing (Nelson et al., 2011; Patrick et al., 2013; Venables et al., 2018). Still, however, less research has examined disinhibition-related reductions of P3 responses in response inhibition tasks specifically, generally finding amplitude reductions in no-go variants of the P3 as well (Brennan & Baskin-Sommers, 2018; Ribes-Guardiola, Poy, Patrick et al., 2020). Furthermore, there is also evidence supporting the idea that no-go variants of the P3 relate to disinhibition/externalizing problems and traits as a function of its shared variance with other P3 responses as measured in other tasks (see Pasion et al., 2023; Ribes-Guardiola, Poy, Patrick et al., 2020).

Considered in this context, it seems that Stop-P3 amplitudes reflect a distinct process that is less relevant to disinhibition compared to other variants of this brain response measured in oddball and other cognitive processing tasks, including go/no-go tasks. What could be the source of these inconsistencies?

Although both (no-go and stop-signal) tasks measure processes related to inhibitory control based on the repeated execution of motor responses to visual "go" signals that need to be inhibited on some trials ("no-go" trials or trials with a "stop" signal), they differ in the timing of the interruption signal. In go/no-go tasks, the "no-go" signal is presented at the same time or instead of the "go" stimulus. In stop signal-tasks, however, the "stop" signal appears after the "go" stimulus, so the response has already been initiated (Gratton et al., 2018). Therefore, although many studies use both tasks interchangeably (e.g., Huster et al.,

2013), they are likely measuring distinct aspects of inhibitory control. In this regard, Stop-P3 amplitudes in the current study were associated with shorter stop-signal reaction times (SSRT) and lower flanker discrimination errors, indicating that participants with larger Stop-P3 amplitudes demonstrated better inhibitory control (SSRT) and flanker discrimination accuracy (see Table 4). However, this pattern of brain-behavior associations differs from other inhibitory variants of this response (e.g., no-go P3), which have been found to covary primarily with reaction time (RT) speed, such that participants with faster RTs also exhibited enhanced no-go P3 activity when prompted to suppress their dominant response set (Albert et al., 2013). While these results provide support for the notion that Stop-P3 amplitudes in the current study assess individual differences in inhibitory control capacity, they also point out to some degree of dissociation in brain-behavior associations compared to other inhibitory variants of the P3 (i.e., no-go P3; see Albert et al., 2013) that have been linked to trait disinhibition in previous research (Brennan & Baskin-Sommers, 2018; Pasion et al., 2023; Ribes-Guardiola, Poy, Patrick et al., 2020).

Another factor that might explain the lack of significant associations between disinhibition and Stop-P3 amplitudes could be related to the difficulty of the task employed in our study. P3 amplitudes are modulated by different aspects of the task, including stimulus probability, task relevance, and task difficulty, which can limit the available resources for stimuli processing (e.g., attention allocation; working memory; see Kok, 2001 for a review). Consequently, the fast paced nature of our paradigm (i.e., fast inter-trial intervals: ~ 900 ms) and its complexity (i.e., requiring both discrimination and action stopping abilities), might have attenuated the overall amount of processing resources available for elaborative post-processing of task relevant stimuli to a greater extent than simpler paradigms where disinhibition-related reductions of P3 amplitudes are typically observed (e.g., oddball tasks). Therefore, in the context of the complex paradigm employed in this study, disinhibition-

related impairments in inhibitory control performance seem to emerge at earlier stages, indicating failures in reactive cognitive control processes triggered by a mismatch between intended and executed responses (i.e., ERN), rather than at elaborative processing stages of task relevant stimuli (P3; see Gratton et al., 2018 for a review).

Limitations and future directions

It is important to acknowledge some limitations that may restrict the generalizability of our results. First, the sample was composed exclusively of undergraduates, which might have limited the full range of psychopathic disinhibition scores. Follow-up research could address this limitation by preselecting the sample based on self-report scores (thus ensuring a broader representation of psychopathic traits) or by including different sample types (e.g., clinical, criminal) and a wider age range. Second, as discussed above, the high demanding task employed in this study may have hindered our ability to detect disinhibition-related deficits at the processing stages indexed by the P3. Therefore, it would be valuable for future studies to examine the effects of intertrial interval durations and overall task demands (e.g., combining discrimination and stopping abilities versus assessing only one of these domains) on externalizing-related differences in electrocortical measures of performance monitoring. A third issue to consider is the absence of significant associations between psychopathic traits and behavioral measures (i.e., RT, SSRT, and accuracy) in the current study, which does not align with the association found between disinhibition and ERP correlates of error processing. Prior evidence has also demonstrated mostly null associations between behavioral measures and externalizing/disinhibition traits (e.g., Hall et al., 2007; Ribes-Guardiola, Poy, Patrick et al., 2020). Therefore, future studies could consider collecting a wider range of behavioral measures of inhibitory control derived from other executive control tasks in order to better understand disinhibition-related impairments in inhibitory control capacity at the behavioral level (see, e.g., Venables et al., 2018). Finally, the elevated

correlation between disinhibition and meanness in our sample (r = .68), in comparison with meta-analytic evidence (r = .53; Sleep et al., 2019), may have contributed to the loss of statistical significance for disinhibition-related differences in ERN amplitudes when controlling for the other triarchic dimensions. Future research should aim to disentangle whether ERN reductions are specific of some triarchic dimension or, as Pasion et al. (2023) suggest, are related to a more broader externalizing dimension encompassing both disinhibition and meanness traits.

Despite these limitations, our study allows to conclude that deficits in inhibitory control are primarily detected at early processing stages indicating difficulties in error-monitoring processes, within the context of a complex hybrid paradigm that required multiple cognitive operations (e.g., interference control, action stopping). These findings support the idea that high-disinhibited individuals have difficulties in detecting and processing errors, which could underlie impairments in behavioral adaptation and self-regulation common to clinical conditions subsumed under the externalizing spectrum of psychopathology.

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CONCLUSIONS

CHAPTER 7. Discussion and conclusions

In the conceptual framework of the triarchic model of psychopathy (Patrick et al., 2009), the trait constructs of boldness, meanness, and disinhibition are formulated in biobehavioral terms. Self-report measures are merely used as provisional referents to identify potential physiological or behavioral indicators that can contribute to conform comprehensive nomological networks for each biobehavioral dimension corresponding to the triarchic trait constructs: **threat sensitivity** for boldness, **affiliative capacity** for meanness, and **inhibitory control** for disinhibition. Consequently, boldness, meanness, and disinhibition are open-ended constructs that can be reviewed based on new empirical evidence (Patrick et al., 2022). The main objective of this research was to provide additional evidence regarding the biobehavioral dispositions that underlie the trait constructs of the triarchic model of psychopathy by identifying new and distinct physiological correlates for each construct.

For **threat sensitivity,** which is presumed to reflect individual differences in the brain's defensive system reactivity, research has identified physiological indicators of reduced reactivity to threat (e.g., ASP; Esteller et al., 2016), as well as behavioral indicators of good adjustment under threatening situations (e.g., Yancey et al., 2019, 2022) in relation to boldness. With this in mind, the aim was twofold: to examine the association of boldness with (1) the resting vagally-mediated heart rate variability (vmHRV) as an indicator of adequate emotional self-regulation (Study 1), and with (2) the Cardiac Defense Response (CDR) and its second accelerative component (A2) as specific indicators of metabolic mobilization for active defense (Study 2).

In Study 1, the vmHRV recording was carried out in a large mixed-gender sample of undergraduates (N = 241) assessed via the Triarchic Psychopathy Measure (TriPM). As

hypothesized, boldness (but not meanness or disinhibition) showed a unique positive association with vmHRV, suggesting that effective emotional self-regulation ability is one of the adaptive features encompassed by the boldness trait construct. This finding aligns with previous studies that have demonstrated the protective effects of both boldness and vmHRV for internalizing symptomatology (e.g., Beauchaine & Thayer, 2015; Bertoldi et al., 2023; Latzman et al., 2020), as well as their links with well-adjusted personality traits such as emotional resilience, subjective well-being, and high self-esteem (Holzam & Bridgett, 2017; Sleep et al., 2019; Thayer et al., 2012), the high extraversion and low neuroticism personality traits characterizing boldness (Miller et al., 2016; Poy et al., 2014), and good task performance under threat conditions showed by high-boldness individuals (Yancey et al., 2019, 2022). These findings suggest that vmHRV can serve as a physiological indicator of the adaptive features of the boldness disposition, contributing to a better understanding of this triarchic trait construct in biobehavioral terms.

In Study 2, the CDR, a complex pattern of heart rate changes in response to an intense and unexpected aversive stimulus, was examined in another large mixed-gender sample of undergraduates (N = 156) assessed by the Psychopathic Personality Inventory-Revised (PPI-R). PPI-R Fearless Dominance (the component of the PPI-R most conceptually aligned with the boldness trait in the triarchic model) was related to a lower CDR in women, but not in men. Specifically, PPI-R Fearlessness (one of the scales conforming the PPI-R Fearless Dominance factor) was related to a reduced second accelerative component (A2) of the CDR only in women. This association was confirmed by the A2 index derived from a Principal Component Analysis (PCA), performed on the 80 second-by-second heart rate change scores conforming the CDR to obtain its four components (acceleration-deceleration-acceleration-deceleration) in a data-driven manner. The evidence of diminished cardiac reactivity to initial threat in women supports etiological models proposing the under-reactivity of the

brain's defensive motivational system as the mechanism contributing to boldness (Fowles & Dindo, 2009; Patrick & Bernat, 2009), as previously demonstrated with other physiological indicators of threat reactivity (Benning et al., 2005; Esteller et al., 2016; López et al., 2013). However, the specific relationship found between A2, which is presumed to reflect metabolic mobilization for active defense (Vila et al., 2007), and scores on the PPI-R Fearlessness scale suggests that, at least in women, the reduced mobilization of the organism's resources to give a defensive response is better captured by a narrower dimension of fear/fearlessness covering lack of fear when faced with physical threats and a propensity for engaging in risky activities, rather than by reduced distress in stressful situations (as indexed by PPI-R Stress Immunity scores) or by high social skills (as indexed by PPI-R Social Potency scores), which are traits also encompassed in the boldness disposition. Though further research on mixed-gender samples is needed, the fact that significant results were found only in women points to a gender-specific differential defense cardiac reactivity involving the fear/fearlessness dimension. In summary, these findings provide preliminary evidence for the CDR as a general measure of threat reactivity related to boldness, and the A2 as a specific correlate of reduced defensive mobilization specifically associated with psychopathic fearlessness in women. These results highlight the potential use of this cardiac response to better understand the physiological aspects of boldness and its potential distinct manifestations across genders.

Regarding **affiliative capacity,** which appears to reflect individual differences in empathy, social connectedness, and caring, previous evidence has shown reduced brain reactivity and recognition accuracy for emotional facial expressions, particularly fear, in relation to meanness (Brislin et al., 2018; Brislin & Patrick, 2019). Recently, it has been proposed that the brain's pain network also may play a significant role in empathic deficits associated with this dimension (e.g., Decety et al., 2013, 2015). Therefore, the aim of Study

3 was to investigate the relationship between callousness/meanness traits, assessed through a general factor obtained from an exploratory factor analysis (EFA) of scores on three well-established inventories of these traits, and electrocortical processing of pain through the Late Positive Potential (LPP). The LPP is a well-validated electrophysiological indicator of sustained attention to motivationally significant stimuli. EEG data were recorded while 100 female undergraduates viewed images depicting bodily injuries under an imagine-self (people in the picture is oneself) or either an imagine-other (people in the picture is someone unknown) perspective. The LPP amplitude was higher in response to pain compared to no pain pictures, but only in the imagine-other condition. As the LPP is an index of stimulus significance (Bradley et al., 2009; Hajcak & Foti, 2020), it seems that the self-relevant condition increased brain reactivity irrespective of the stimulus content (pain or no pain), whereas someone unknown in a neutral/non-painful situation would be the less motivationally relevant condition in terms of electrocortical responses. Consistent with our hypothesis, callousness was associated with reduced LPP amplitudes in response to pain pictures when imagining someone else (but not oneself) in a painful situation, indicating that high-callous individuals exhibit blunted elaborative processing of distress cues in others. This finding aligns with the theoretical characterizations of meanness as the triarchic disposition associated with empathic deficits. These results are consistent with prior evidence showing associations between callousness and reduced LPP amplitudes in conditions involving empathic concern (Decety et al., 2015), as well as reduced brain activations in relevant areas of empathy for pain (e.g., Decety et al., 2013; Lockwood et al., 2013). In addition to support the use of perspective-taking in electrocortical research on pain processing, these findings suggest that the LPP holds potential as an indicator for elucidating the biobehavioral processes associated with the selfish, disdain, and remorseless traits of psychopathy.

Finally, regarding **inhibitory control**, which is believed to reflect individual differences in the functioning of frontocortical systems that mediate executive functions, previous studies have consistently demonstrated an association between reductions in P3 amplitudes and disinhibition (Pasion et al., 2018). Research on response-locked event-related potentials (ERPs) has also indicated a relationship between disinhibition and diminished error monitoring (Vallet et al., 2021). Therefore, Study 4 aimed to investigate the association between disinhibition and ERP indicators from a modified flanker-stop-signal task indexing inhibitory processing (Stop P3) and error monitoring (Flanker and Stop ERNs). The study was conducted on a large mixed-gender sample of undergraduates (N = 258) assessed for triarchic traits using a multi-measurement approach (i.e., regression-based component scores from EFA on triarchic scales scores from three self-report measures). As expected, disinhibition (and not boldness or meanness) was found to be associated with reduced amplitudes of both Flanker and Stop ERNs, but not to Stop P3 amplitudes. These findings align with etiological models (Fowles & Dindo, 2009; Patrick & Bernat, 2009) and recent neurobiological research (Patrick, 2018b, 2022) that link disinhibition to reduced amplitudes of distinct ERN variants (Heritage & Benning, 2013; Vilà-Balló et al., 2014) or their shared variance (Pasion et al., 2023). The lack of association between Stop P3 and disinhibition observed in this study suggests that this particular brain response may reflect a distinct inhibitory process that is less relevant to disinhibition when compared to other P3 variants measured in go/no-go tasks (e.g., Paiva et al., 2020; Ribes, Poy, Patrick et al., 2020). This discrepancy could be attributed to the nature of our paradigm (involving higher complexity and shorter inter-trial intervals), which may have limited the available processing resources for stimuli processing (see Kok, 2001 for a review). The results of this study suggest a differential contribution of distinct aspects of cognitive control to the disinhibition trait construct, with reduced brain reactivity to error responses, presumed to reflect deficits

in the early stages of error-monitoring (e.g., Falkenstein et al., 2000; Holroyd & Coles, 2002), playing a more substantial role in the manifestation of disinhibition traits of psychopathy. Furthermore, these findings discourage the use of combined speed tasks in the study of individual differences in P3 responsiveness related to disinhibition.

In summary, the findings of this doctoral thesis contribute to advance in the understanding of the biobehavioral counterparts to the triarchic dispositions of psychopathy, by identifying distinct psychophysiological correlates: higher vmHRV (indexing suitable emotional self-regulation) and lower CDR and A2 (indexing low reactivity to threat) as new physiological indicators for boldness; a reduced LPP to pain in others (indicative of a reduced attribution of relevance to others' distress) as a physiological correlate of meanness/callousness; and a diminished ERN amplitude (indicative of blunted error processing) as an electrocortical indicator of disinhibition. By establishing these psychophysiological correlates, this research work provides valuable insights into the biobehavioral underpinnings of each triarchic disposition, thus contributing to the development of more comprehensive nomological networks for boldness, meanness, and disinhibition, and to better delineate this multifaceted personality disorder in biobehavioral terms.

Conclusions:

Study 1

1. Higher resting vagally-mediated heart rate variability (vmHRV), which serves as a physiological index of emotional self-regulation and mental health resilience, is associated with the trait of boldness.

2. Effective emotional self-regulation is identified as one of the adaptive features that underlie the trait of boldness.

Study 2

1. Reduced amplitude of the Cardiac Defense Response (CDR), which reflects reactivity of the defensive motivational system, is associated with Fearless Dominance, only in women.

2. Decreased amplitude of the second accelerative component (A2) of the CDR, serving as a more specific indicator of metabolic mobilization for active defense, is related to a narrower dimension of fear/fearlessness, only in women.

3. Principal Component Analysis is a valuable method for extracting the four components of the CDR in a data-driven manner.

4. There are gender-specific differences in defense cardiac reactivity involving fear/fearlessness traits.

Study 3

1. Reduced amplitude of the Late Positive Potential (LPP) in response to pain in others, reflecting a diminished attribution of relevance to others' distress, is associated with callousness traits.

2. The LPP is a valuable component of event-related potentials (ERP) for investigating perspective-taking in pain processing.

3. Perspective-taking in pain processing paradigms allows to find callousness-related differences in the LPP.

Study 4

1. Diminished amplitudes of the Error-Related Negativity (ERN), indicating blunted error monitoring, are associated with the trait of disinhibition.

2. The detection of disinhibition-related differences in the amplitude of the P3 component may depend on the specific experimental paradigm in which it is elicited.

Future considerations

1. Examining distinct indicators from different measurement domains (physiological, behavioral) contributes to better delineate the nomological networks for the triarchic dispositions in neurobiological terms.

2. Adopting a multi-measurement approach of self-report measures to assess the triarchic trait constructs theoretically enhances the assessment validity by minimizing the unique and error variance associated with each individual instrument.

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