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Actualización en Psicopatología, Neurofarmacología y Bases Biológicas de la Estimulación Neuronal.

DR. EDUARDO CALIXTO

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Tema 3

La psicopatía: diferencias/analogías entre la anatomía y neuroquímica de la psicopatía y sociopatía.

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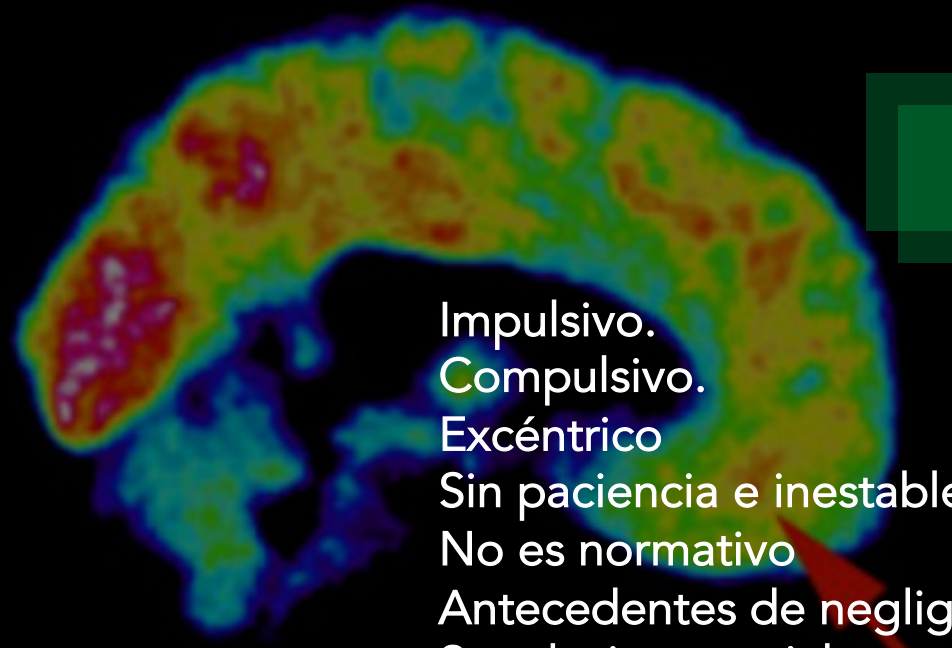
PSICÓPATA

0.2 a 3% población.
Calculador y detallista.
Insensible, despiadado.
Estrategia, manipulador.
Frío en relación a emociones.
Su origen relaciona más al cerebro.
No establecen relaciones interpersonales





SOCIÓPATA

Impulsivo.
Compulsivo.
Excéntrico
Sin paciencia e inestable
No es normativo
Antecedentes de negligencia en su crianza
Se relaciona socialmente (camuflaje).





Baja empatía
Sin culpas
Sin responsabilidad
Desprecio por lo convencional

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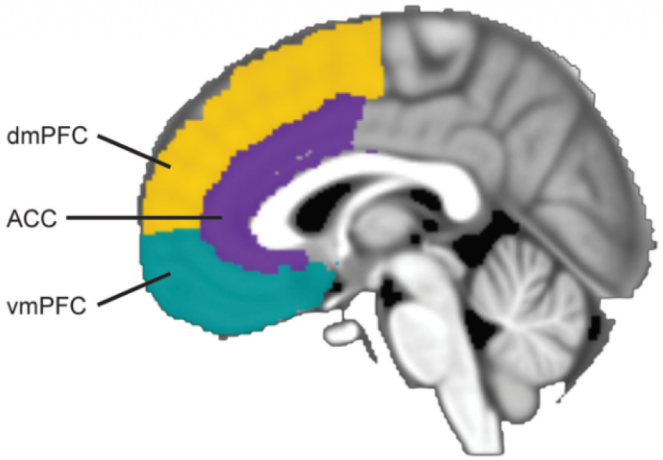
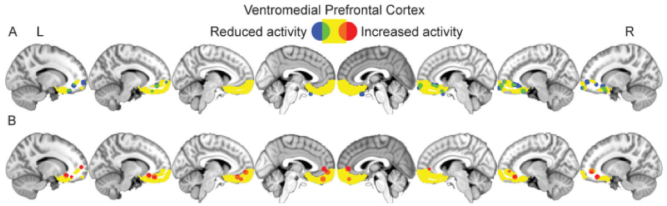
Neuroanatomía de la Psicopatía

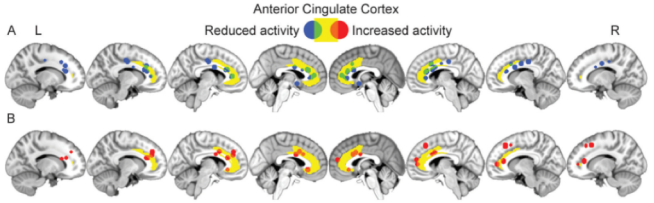


- La corteza prefrontal ventromedial (vmPFC) y la corteza cingulada anterior (ACC) muestran patrones de actividad alterados en individuos con rasgos psicopáticos.
- La actividad en estas regiones se relaciona con déficits en la toma de decisiones morales, regulación emocional y procesamiento de recompensas o castigos, tareas de juicio moral, empatía y reconocimiento emocional en psicópatas, afectando la percepción del dolor y la culpa.

Reducción en volumen y densidad de materia gris en vmPFC, ACC y la corteza temporal superior están asociados con rasgos psicopáticos.

Alteración en la Red Neuronal por Defecto y Red Neuronal de Prominencia.





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Psychopathy and medial frontal cortex: A systematic review reveals predominantly null relationships

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Abstract

Theories have posited that psychopathy is caused by dysfunction in the medial frontal cortex, including ventromedial prefrontal cortex (vmPFC), anterior cingulate cortex (ACC), and dorsomedial prefrontal cortex (dmPFC). Recent reviews have questioned the reproducibility of neuroimaging findings within this field. We conducted a systematic review to describe the consistency of magnetic resonance imaging (MRI) findings according to anatomical subregion (vmPFC, ACC, dmPFC), experimental task, psychopathy assessment, study power, and peak coordinates of significant effects. Searches of PsycInfo and MEDLINE databases produced 77 functional and 24 structural MRI studies that analyzed the medial frontal cortex in relation to psychopathy in adult samples. Findings were predominantly null (85.4% of 1,573 tests across the three medial frontal regions). Studies with higher power observed null effects at marginally lower rates. Finally, peak coordinates of significant effects were widely dispersed. The evidence failed to support theories positing the medial frontal cortex as a consistent neural correlate of psychopathy. Theory and methods in the field should be revised to account for predominantly null neuroimaging findings.

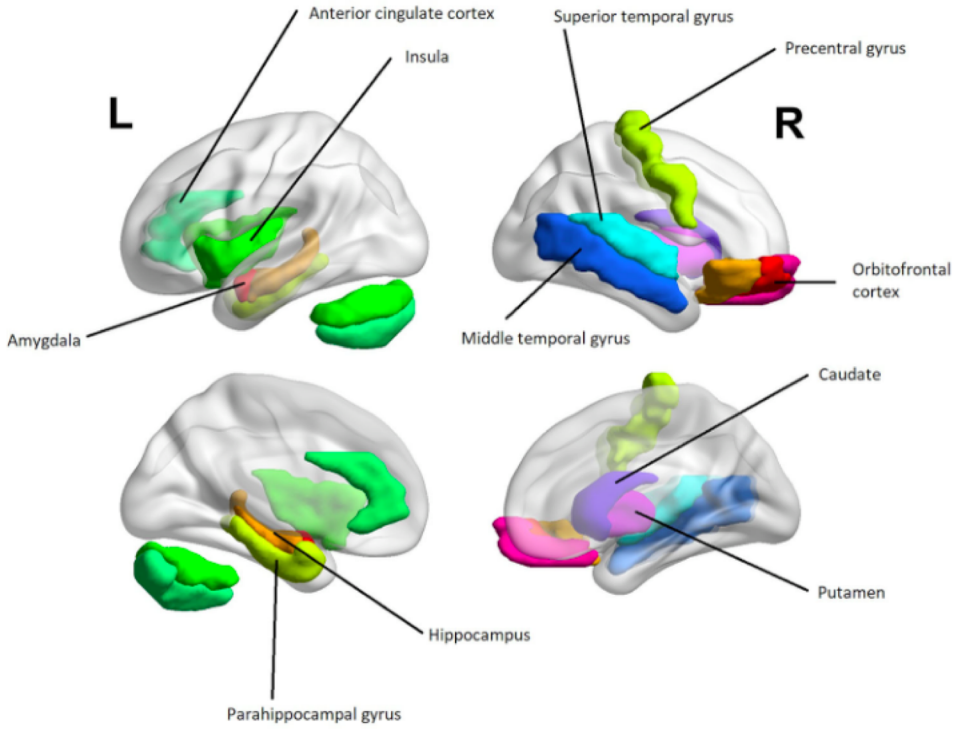
Keywords

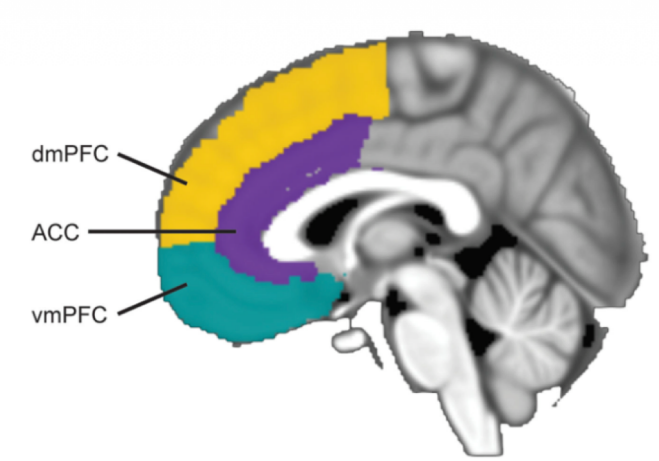
psychopathy; MRI; neuroimaging; prefrontal cortex; anterior cingulate cortex

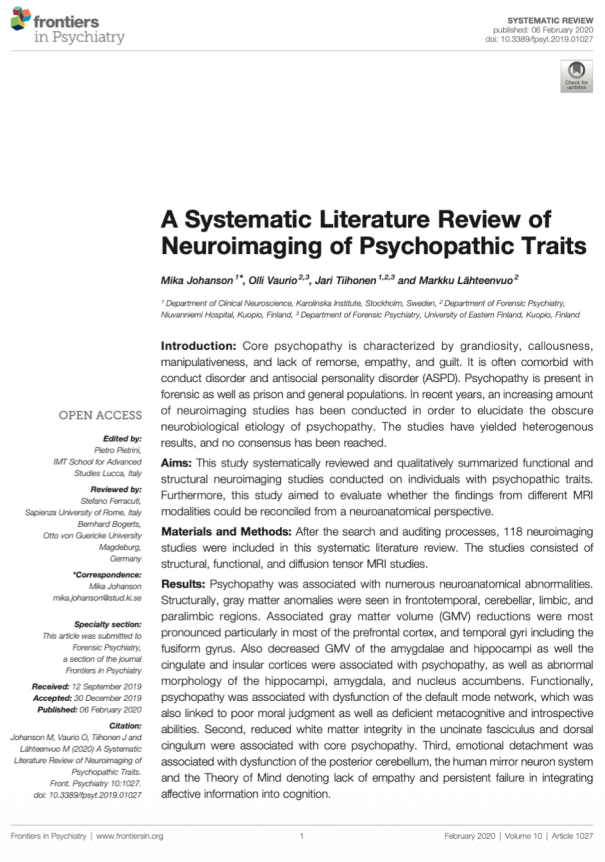
*Author for correspondence: p.deming@northeastern.edu.
Declarations of interest: none

Neuroanatomía de la Psicopatía

Reducciones en volumen de materia gris en la corteza orbitofrontal, dorsomedial y ventromedial, corteza temporal, incluyendo el giro temporal superior/ medio y la corteza fusiforme.



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A Systematic Literature Review of Neuroimaging of Psychopathic Traits

Mika Johanson[1], Olli Vaurio[2,3], Jari Tiihonen[1,2,3] and Markku Lähteenvuo[2]*

[1] Department of Clinical Neuroscience, Karolinska Institute, Stockholm, Sweden, [2] Department of Forensic Psychiatry, Niuvanniemi Hospital, Kuopio, Finland, [3] Department of Forensic Psychiatry, University of Eastern Finland, Kuopio, Finland

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Introduction: Core psychopathy is characterized by grandiosity, callousness, manipulativeness, and lack of remorse, empathy, and guilt. It is often comorbid with conduct disorder and antisocial personality disorder (ASPD). Psychopathy is present in forensic as well as prison and general populations. In recent years, an increasing amount of neuroimaging studies has been conducted in order to elucidate the obscure neurobiological etiology of psychopathy. The studies have yielded heterogenous results, and no consensus has been reached.

Aims: This study systematically reviewed and qualitatively summarized functional and structural neuroimaging studies conducted on individuals with psychopathic traits. Furthermore, this study aimed to evaluate whether the findings from different MRI modalities could be reconciled from a neuroanatomical perspective.

Materials and Methods: After the search and auditing processes, 118 neuroimaging studies were included in this systematic literature review. The studies consisted of structural, functional, and diffusion tensor MRI studies.

Results: Psychopathy was associated with numerous neuroanatomical abnormalities. Structurally, gray matter anomalies were seen in frontotemporal, cerebellar, limbic, and paralimbic regions. Associated gray matter volume (GMV) reductions were most pronounced particularly in most of the prefrontal cortex, and temporal gyri including the fusiform gyrus. Also decreased GMV of the amygdalae and hippocampi as well the cingulate and insular cortices were associated with psychopathy, as well as abnormal morphology of the hippocampi, amygdala, and nucleus accumbens. Functionally, psychopathy was associated with dysfunction of the default mode network, which was also linked to poor moral judgment as well as deficient metacognitive and introspective abilities. Second, reduced white matter integrity in the uncinate fasciculus and dorsal cingulum were associated with core psychopathy. Third, emotional detachment was associated with dysfunction of the posterior cerebellum, the human mirror neuron system and the Theory of Mind denoting lack of empathy and persistent failure in integrating affective information into cognition.

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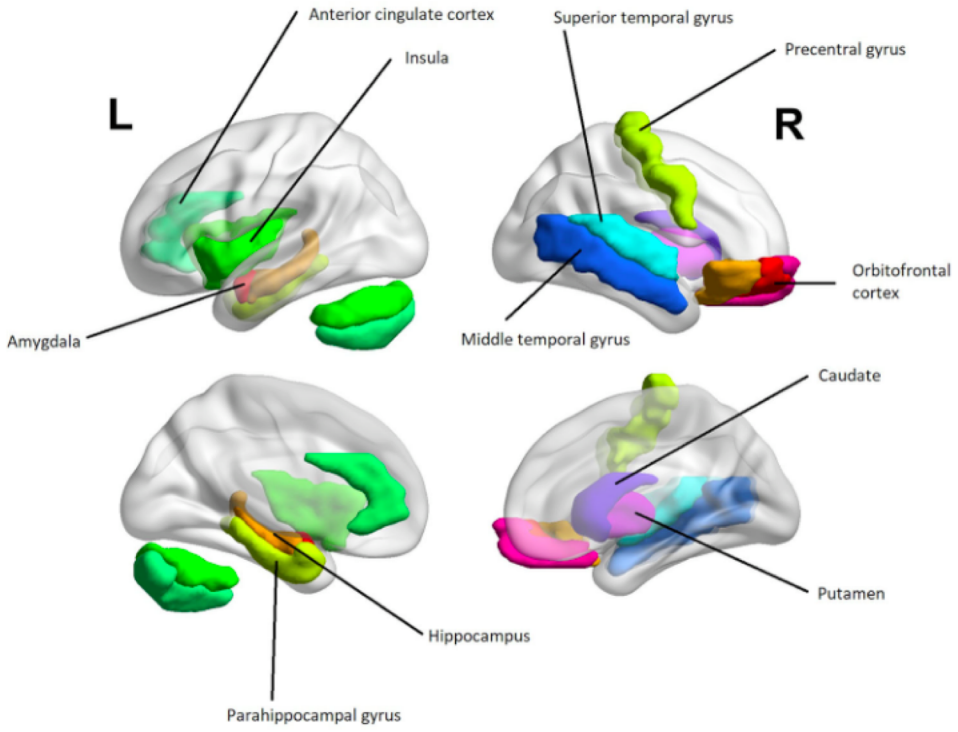
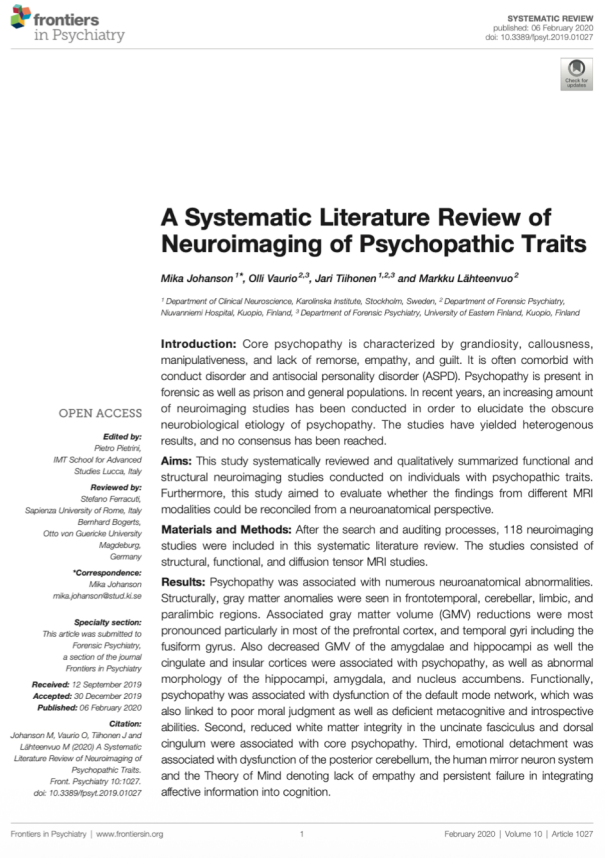
Reducciones en volumen en la corteza cingulada anterior, media y posterior.
Disminución de la corteza insular en individuos con rasgos psicopáticos.

Neuroanatomía de la Psicopatía

Disminución del volumen en la amígdala, hipocampo y giro parahipocampal, aunque algunos estudios muestran volúmenes mayores en la amígdala.

Aumento en volumen de los ganglios basales: globo pálido, putamen y núcleo accumbens.





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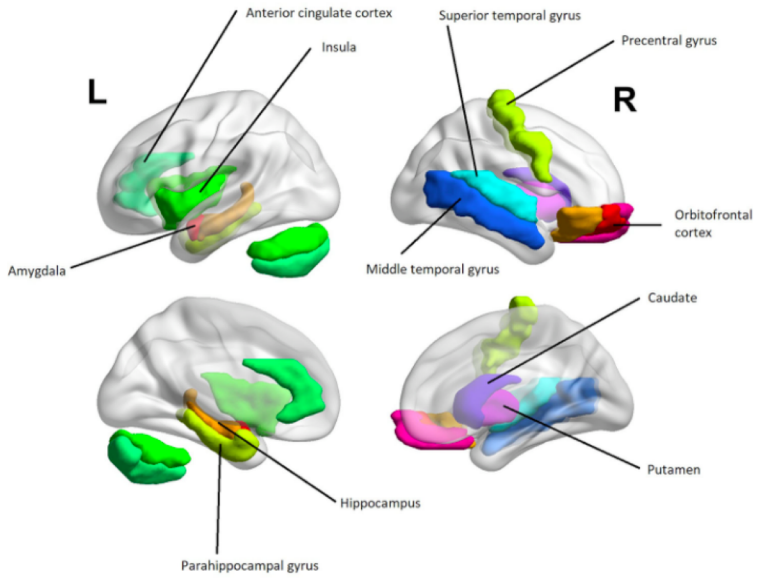
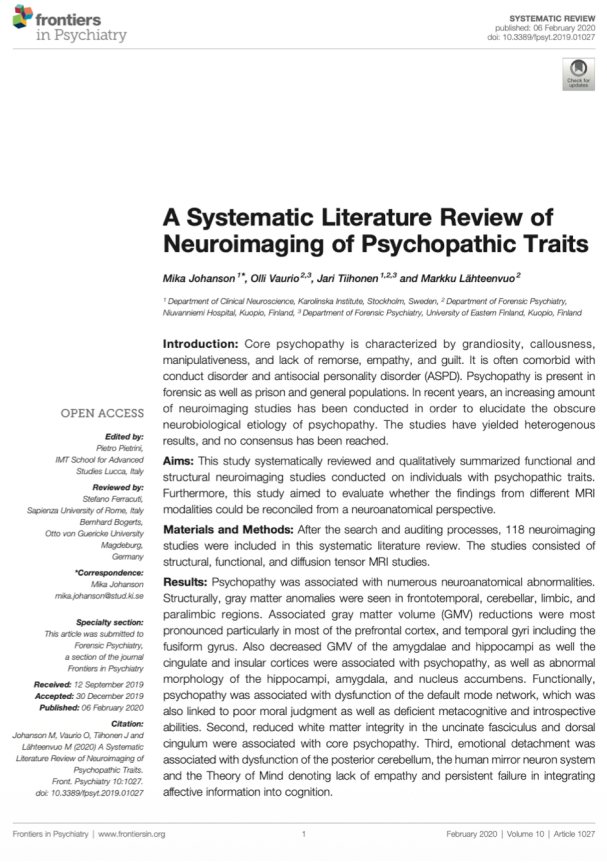
Neuroanatomía de la Psicopatía

Los psicópatas muestran mayor rendimiento en engañar, con reducción de actividad en la corteza dorsolateral prefrontal.
La actividad en regiones ejecutivas aumenta durante tareas de engaño, indicando entrenamiento cognitivo en estas habilidades.

Menor reconocimiento emocional.
La conectividad entre la corteza prefrontal y la amígdala está disminuida, afectando la integración emocional y cognitiva.





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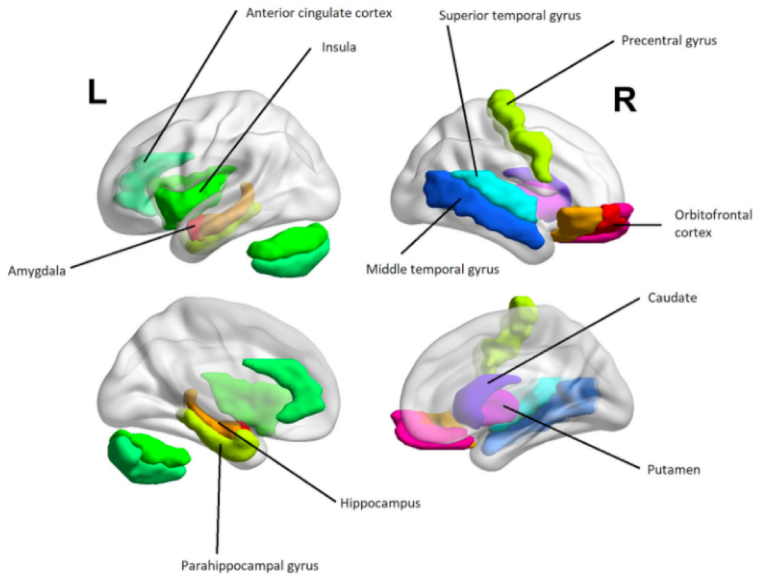
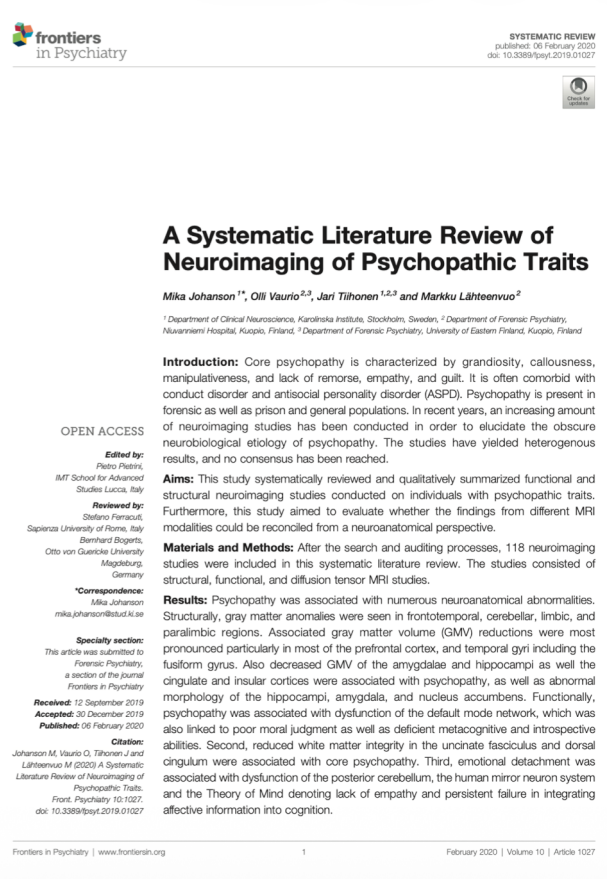
Neuroanatomía de la Psicopatía

Los psicópatas muestran mayor actividad en el núcleo accumbens en respuesta a recompensas, asociado a rasgos interpersonal-afectivos.

La conectividad entre la corteza prefrontal ventromedial y el núcleo accumbens está disminuida, vinculada a conductas criminales.





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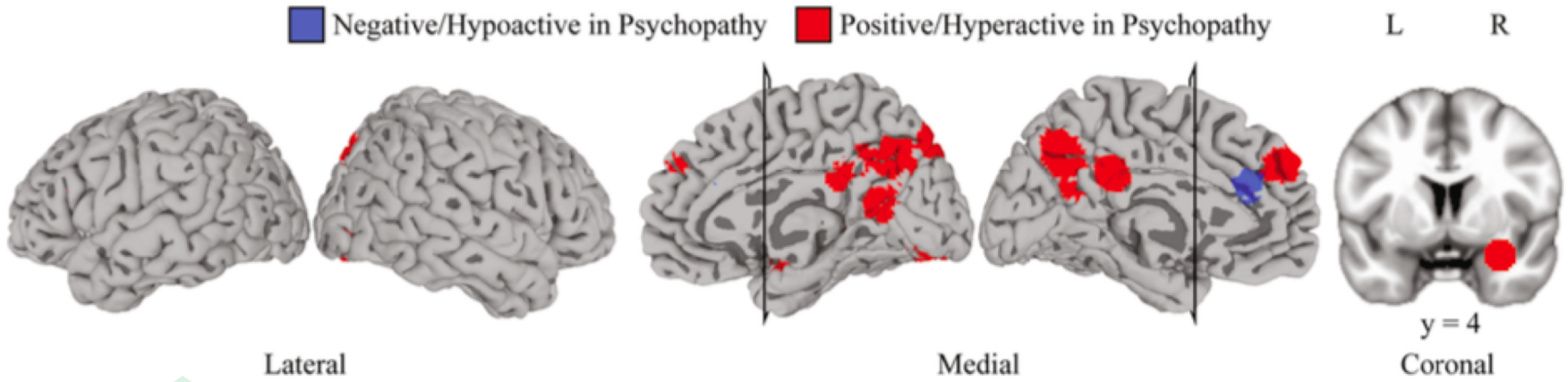
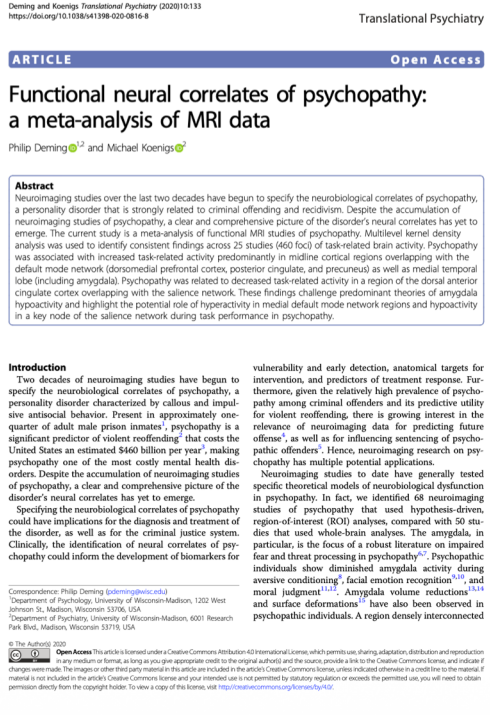
Sustrato neuronal de la Psicopatía



La psicopatía se asocia con incremento de la actividad en regiones corticales de la línea media, especialmente en áreas que se superponen con la red por defecto, como la corteza prefrontal dorsomedial, el cíngulo posterior, el precúneo, lóbulo temporal medial, incluyendo la **amígdala y el hipocampo**.

Significant associations between brain function and psychopathy.

Region(s)	Hemi.	Direction	Peak MNI coordinates (*x*, *y*, *z*)	Size (voxels)	Threshold
Anterior cingulate	L	–	−6, 32, 22	293	Extent
Dorsomedial prefrontal cortex	L	+	−2, 56, 28	1	Height
Dorsomedial prefrontal cortex	L/R	+	−2, 50, 34	347	Extent
Dorsomedial prefrontal cortex	L/R	+	2, 44, 32	69	Height
Inferior frontal gyrus (pars orbitalis)	R	+	40, 40, −8	301	Extent
Posterior orbitofrontal cortex	R	+	26, 8, −16	12	Height
Amygdala/temporal pole	R	+	30, 4, −20	399	Extent
Hippocampus/parahippocampal gyrus	R	+	24, −36, 2	105	Extent
Hippocampus/parahippocampal gyrus	R	+	30, −40, −4	337	Height
Cerebellum/cuneus/inferior occipital cortex/posterior cingulate/ precuneus/superior occipital cortex	L/R	+	12, −70, 16	4272	Extent



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Translational Psychiatry

ARTICLE Open Access

Functional neural correlates of psychopathy: a meta-analysis of MRI data

Philip Deming[1,2] and Michael Koenigs[2]

Abstract

Neuroimaging studies over the last two decades have begun to specify the neurobiological correlates of psychopathy, a personality disorder that is strongly related to criminal offending and recidivism. Despite the accumulation of neuroimaging studies of psychopathy, a clear and comprehensive picture of the disorder's neural correlates has yet to emerge. The current study is a meta-analysis of functional MRI studies of psychopathy. Multilevel kernel density analysis was used to identify consistent findings across 25 studies (460 foci) of task-related brain activity. Psychopathy was associated with increased task-related activity predominantly in midline cortical regions overlapping with the default mode network (dorsomedial prefrontal cortex, posterior cingulate, and precuneus) as well as medial temporal lobe (including amygdala). Psychopathy was related to decreased task-related activity in a region of the dorsal anterior cingulate cortex overlapping with the salience network. These findings challenge predominant theories of amygdala hypoactivity and highlight the potential role of hyperactivity in medial default mode network regions and hypoactivity in a key node of the salience network during task performance in psychopathy.

Introduction

Two decades of neuroimaging studies have begun to specify the neurobiological correlates of psychopathy, a personality disorder characterized by callous and impulsive antisocial behavior. Present in approximately one-quarter of adult male prison inmates[1], psychopathy is a significant predictor of violent reoffending[2] that costs the United States an estimated $460 billion per year[3], making psychopathy one of the most costly mental health disorders. Despite the accumulation of neuroimaging studies of psychopathy, a clear and comprehensive picture of the disorder's neural correlates has yet to emerge.

Specifying the neurobiological correlates of psychopathy could have implications for the diagnosis and treatment of the disorder, as well as for the criminal justice system. Clinically, the identification of neural correlates of psychopathy could inform the development of biomarkers for vulnerability and early detection, anatomical targets for intervention, and predictors of treatment response. Furthermore, given the relatively high prevalence of psychopathy among criminal offenders and its predictive utility for violent reoffending, there is growing interest in the relevance of neuroimaging data for predicting future offense[4], as well as for influencing sentencing of psychopathic offenders[5]. Hence, neuroimaging research on psychopathy has multiple potential applications.

Neuroimaging studies to date have generally tested specific theoretical models of neurobiological dysfunction in psychopathy. In fact, we identified 68 neuroimaging studies of psychopathy that used hypothesis-driven, region-of-interest (ROI) analyses, compared with 50 studies that used whole-brain analyses. The amygdala, in particular, is the focus of a robust literature on impaired fear and threat processing in psychopathy[6,7]. Psychopathic individuals show diminished amygdala activity during aversive conditioning[8], facial emotion recognition[9,10], and moral judgment[11,12]. Amygdala volume reductions[13,14] and surface deformations[15] have also been observed in psychopathic individuals. A region densely interconnected

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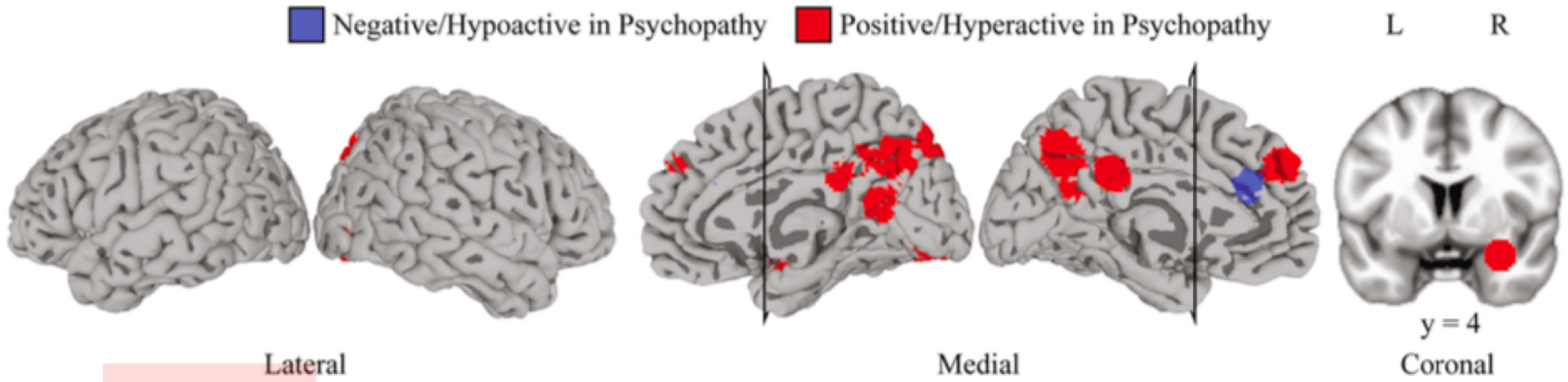
Sustrato neuronal de la Psicopatía



La psicopatía se asocia con disminución de actividad en la corteza cingulada anterior dorsal, una región clave de la red de saliencia.

Significant associations between brain function and psychopathy.

Region(s)	Hemi.	Direction	Peak MNI coordinates (*x*, *y*, *z*)	Size (voxels)	Threshold
Anterior cingulate	L	–	−6, 32, 22	293	Extent
Dorsomedial prefrontal cortex	L	+	−2, 56, 28	1	Height
Dorsomedial prefrontal cortex	L/R	+	−2, 50, 34	347	Extent
Dorsomedial prefrontal cortex	L/R	+	2, 44, 32	69	Height
Inferior frontal gyrus (pars orbitalis)	R	+	40, 40, −8	301	Extent
Posterior orbitofrontal cortex	R	+	26, 8, −16	12	Height
Amygdala/temporal pole	R	+	30, 4, −20	399	Extent
Hippocampus/parahippocampal gyrus	R	+	24, −36, 2	105	Extent
Hippocampus/parahippocampal gyrus	R	+	30, −40, −4	337	Height
Cerebellum/cuneus/inferior occipital cortex/posterior cingulate/ precuneus/superior occipital cortex	L/R	+	12, −70, 16	4272	Extent

Deming and Koenigs *Translational Psychiatry* (2020)10:133
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Translational Psychiatry

ARTICLE Open Access

Functional neural correlates of psychopathy: a meta-analysis of MRI data

Philip Deming[1,2] and Michael Koenigs[2]

Abstract
Neuroimaging studies over the last two decades have begun to specify the neurobiological correlates of psychopathy, a personality disorder that is strongly related to criminal offending and recidivism. Despite the accumulation of neuroimaging studies of psychopathy, a clear and comprehensive picture of the disorder's neural correlates has yet to emerge. The current study is a meta-analysis of functional MRI studies of psychopathy. Multilevel kernel density analysis was used to identify consistent findings across 25 studies (460 foci) of task-related brain activity. Psychopathy was associated with increased task-related activity predominantly in midline cortical regions overlapping with the default mode network (dorsomedial prefrontal cortex, posterior cingulate, and precuneus) as well as medial temporal lobe (including amygdala). Psychopathy was related to decreased task-related activity in a region of the dorsal anterior cingulate cortex overlapping with the salience network. These findings challenge predominant theories of amygdala hypoactivity and highlight the potential role of hyperactivity in medial default mode network regions and hypoactivity in a key node of the salience network during task performance in psychopathy.

Introduction

Two decades of neuroimaging studies have begun to specify the neurobiological correlates of psychopathy, a personality disorder characterized by callous and impulsive antisocial behavior. Present in approximately one-quarter of adult male prison inmates[1], psychopathy is a significant predictor of violent reoffending[2] that costs the United States an estimated $460 billion per year[3], making psychopathy one of the most costly mental health disorders. Despite the accumulation of neuroimaging studies of psychopathy, a clear and comprehensive picture of the disorder's neural correlates has yet to emerge.

Specifying the neurobiological correlates of psychopathy could have implications for the diagnosis and treatment of the disorder, as well as for the criminal justice system. Clinically, the identification of neural correlates of psychopathy could inform the development of biomarkers for vulnerability and early detection, anatomical targets for intervention, and predictors of treatment response. Furthermore, given the relatively high prevalence of psychopathy among criminal offenders and its predictive utility for violent reoffending, there is growing interest in the relevance of neuroimaging data for predicting future offense[4], as well as for influencing sentencing of psychopathic offenders[5]. Hence, neuroimaging research on psychopathy has multiple potential applications.

Neuroimaging studies to date have generally tested specific theoretical models of neurobiological dysfunction in psychopathy. In fact, we identified 68 neuroimaging studies of psychopathy that used hypothesis-driven, region-of-interest (ROI) analyses, compared with 50 studies that used whole-brain analyses. The amygdala, in particular, is the focus of a robust literature on impaired fear and threat processing in psychopathy[6,7]. Psychopathic individuals show diminished amygdala activity during aversive conditioning[8], facial emotion recognition[9,10], and moral judgment[11,12]. Amygdala volume reductions[13,14] and surface deformations[15] have also been observed in psychopathic individuals. A region densely interconnected

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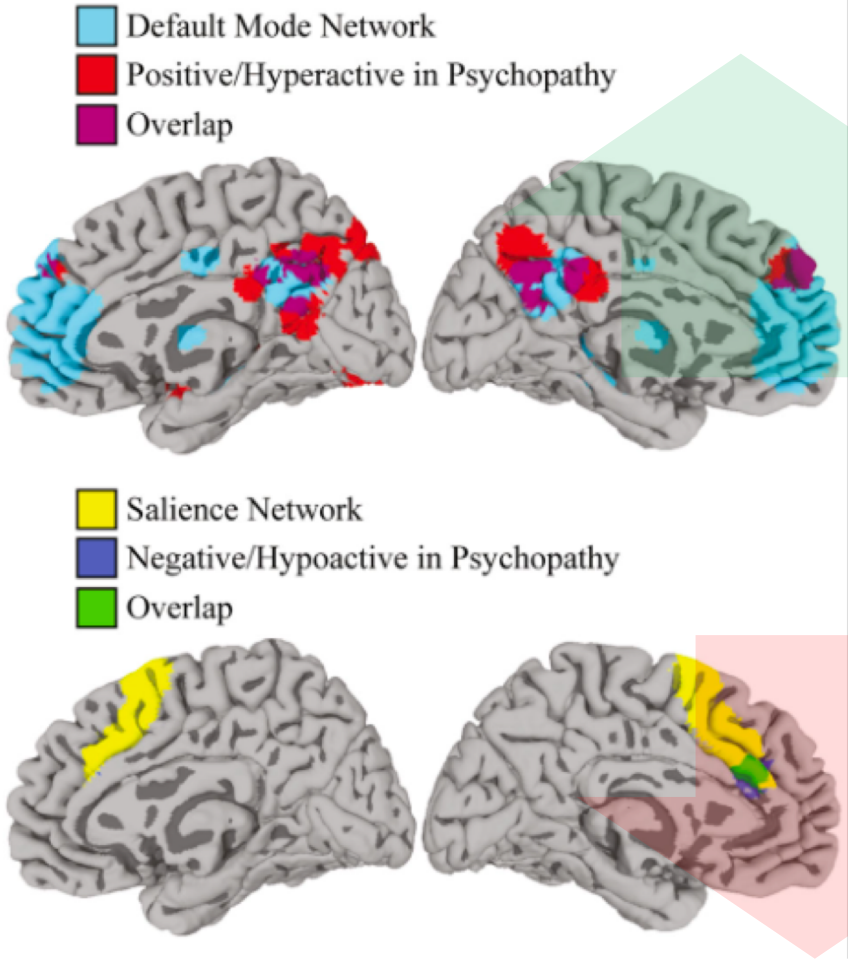
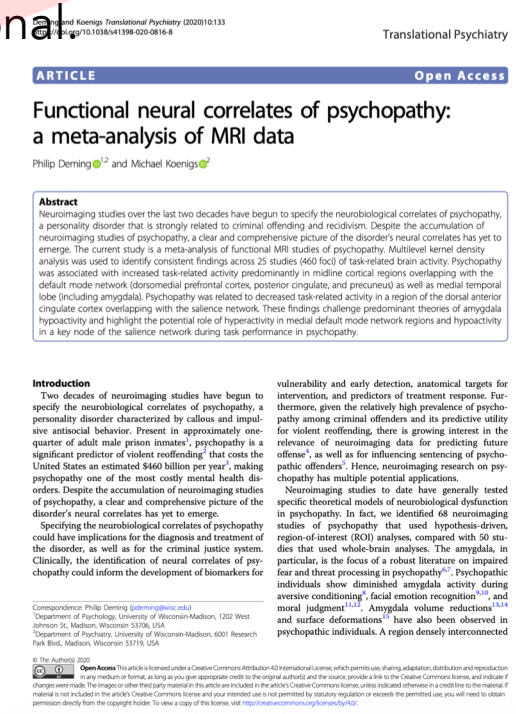


- La disfunción en la red **Neuronal por Defecto** indica dificultades para desactivar procesos autorreferenciales durante tareas externas, lo que REDUCE la atención y el rendimiento en tareas cognitivas.
- La hipoactividad en la red **Neuronal de Prominencia** sugiere una menor capacidad para cambiar entre redes cerebrales orientadas a tareas internas y externas, lo que podría contribuir a los déficits de control y regulación emocional.



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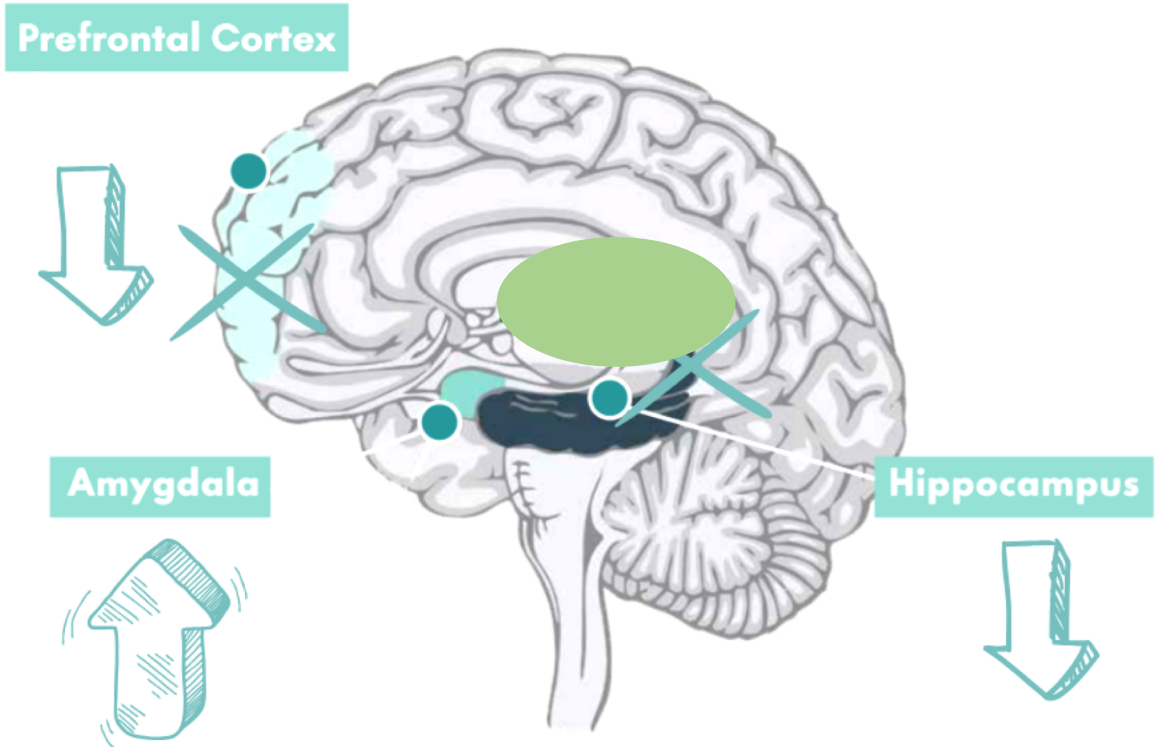
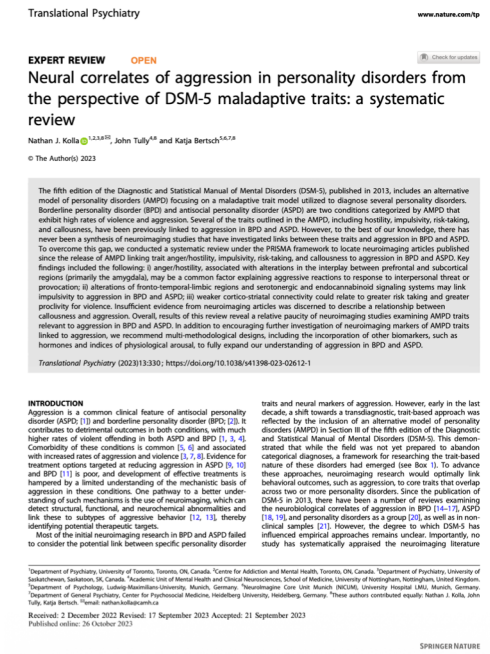
Alteraciones en la comunicación entre las regiones prefrontales y las subcorticales (amígdala)

- IRA muestran dificultades neurobiológicas para diferenciar correctamente las señales sociales neutras de las hostiles: sesgo en el proceso emocional.
- IMPULSIVIDAD: alteraciones estructurales y funcionales en las **regiones fronto-témporo-límbicas**. **Sistemas de señalización serotoninérgicos y endocannabinoides**, como intermediarios que vinculan la falta de control motor y cognitivo con la conducta violenta.





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EXPERT REVIEW OPEN

Neural correlates of aggression in personality disorders from the perspective of DSM-5 maladaptive traits: a systematic review

Nathan J. Kolla, John Tully and Katja Bertsch

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The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), published in 2013, includes an alternative model of personality disorders (AMPD) focusing on a maladaptive trait model utilized to diagnose several personality disorders. Borderline personality disorder (BPD) and antisocial personality disorder (ASPD) are two conditions categorized by AMPD that exhibit high rates of violence and aggression. Several of the traits outlined in the AMPD, including hostility, impulsivity, risk-taking, and callousness, have been previously linked to aggression in BPD and ASPD. However, to the best of our knowledge, there has never been a synthesis of neuroimaging studies that have investigated links between these traits and aggression in BPD and ASPD. To overcome this gap, we conducted a systematic review under the PRISMA framework to locate neuroimaging articles published since the release of AMPD linking trait anger/hostility, impulsivity, risk-taking, and callousness to aggression in BPD and ASPD. Key findings included the following: i) anger/hostility, associated with alterations in the interplay between prefrontal and subcortical regions (primarily the amygdala), may be a common factor explaining aggressive reactions to interpersonal threat or provocation; ii) alterations of fronto-temporal-limbic regions and serotonergic and endocannabinoid signaling systems may link impulsivity to aggression in BPD and ASPD; iii) weaker cortico-striatal connectivity could relate to greater risk taking and greater proclivity for violence. Insufficient evidence from neuroimaging articles was discerned to describe a relationship between callousness and aggression. Overall, results of this review reveal a relative paucity of neuroimaging studies examining AMPD traits relevant to aggression in BPD and ASPD. In addition to encouraging further investigation of neuroimaging markers of AMPD traits linked to aggression, we recommend multi-methodological designs, including the incorporation of other biomarkers, such as hormones and indices of physiological arousal, to fully expand our understanding of aggression in BPD and ASPD.

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INTRODUCTION

Aggression is a common clinical feature of antisocial personality disorder (ASPD; [1]) and borderline personality disorder (BPD; [2]). It contributes to detrimental outcomes in both conditions, with much higher rates of violent offending in both ASPD and BPD [1, 3, 4]. Comorbidity of these conditions is common [5, 6] and associated with increased rates of aggression and violence [3, 7, 8]. Evidence for treatment options targeted at reducing aggression in ASPD [9, 10] and BPD [11] is poor, and development of effective treatments is hampered by a limited understanding of the mechanistic basis of aggression in these conditions. One pathway to a better understanding of such mechanisms is the use of neuroimaging, which can detect structural, functional, and neurochemical abnormalities and link these to subtypes of aggressive behavior [12, 13], thereby identifying potential therapeutic targets.

Most of the initial neuroimaging research in BPD and ASPD failed to consider the potential link between specific personality disorder traits and neural markers of aggression. However, early in the last decade, a shift towards a transdiagnostic, trait-based approach was reflected by the inclusion of an alternative model of personality disorders (AMPD) in Section III of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). This demonstrated that while the field was not yet prepared to abandon categorical diagnoses, a framework for researching the trait-based nature of these disorders had emerged (see Box 1). To advance these approaches, neuroimaging research would optimally link behavioral outcomes, such as aggression, to core traits that overlap across two or more personality disorders. Since the publication of DSM-5 in 2013, there have been a number of reviews examining the neurobiological correlates of aggression in BPD [14–17], ASPD [18, 19], and personality disorders as a group [20], as well as in non-clinical samples [21]. However, the degree to which DSM-5 has influenced empirical approaches remains unclear. Importantly, no study has systematically appraised the neuroimaging literature

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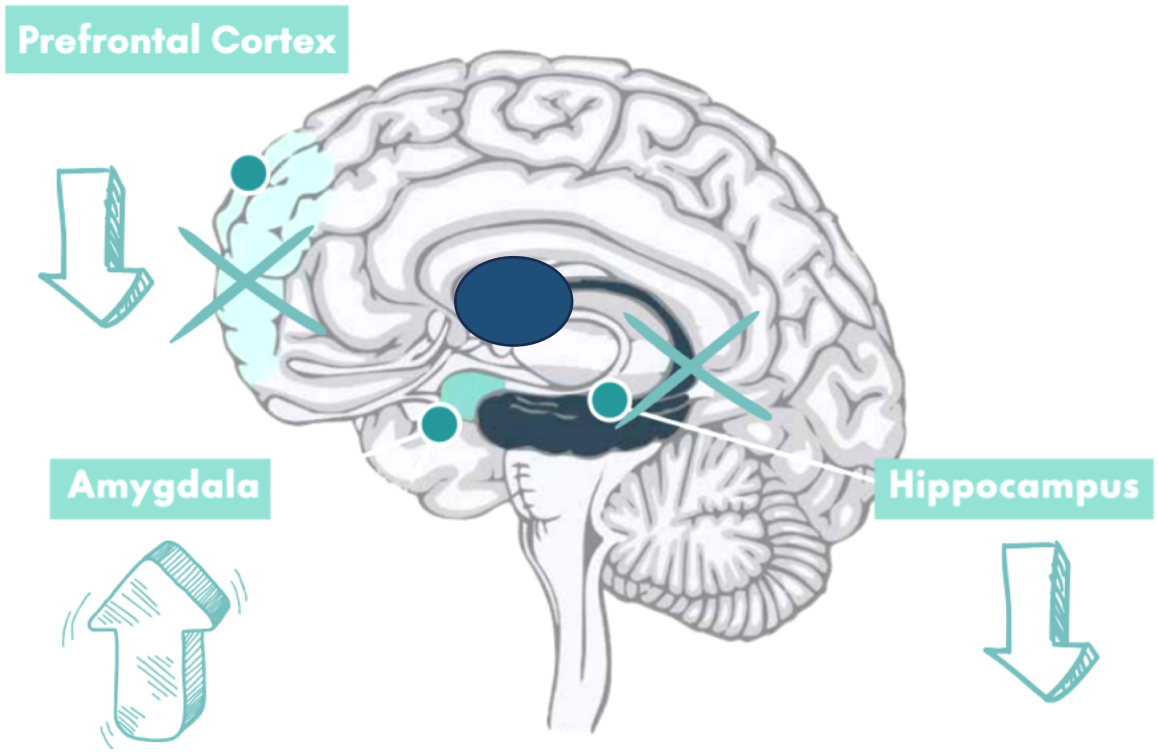
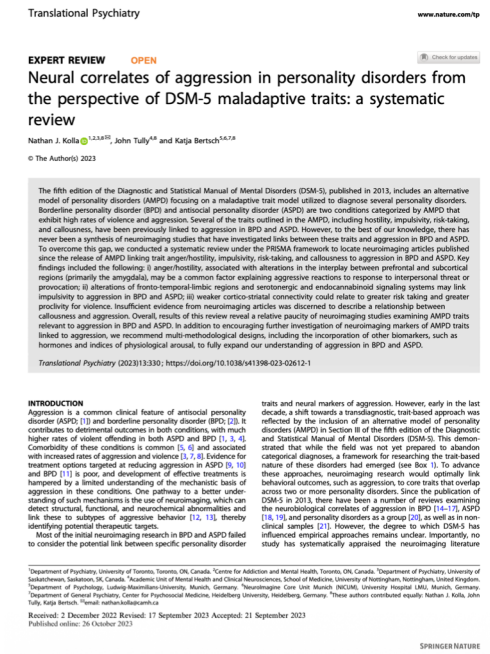
SPRINGER NATURE

Alteraciones en la comunicación entre las regiones prefrontales y las subcorticales

TOMA DE RISEGOS: **conectividad cortico-estriatal más débil** (núcleo accumbens y la corteza prefrontal ventromedial), lo que altera la evaluación del valor subjetivo y de las recompensas durante las decisiones financieras o de vida, impulsando una mayor propensión a la delincuencia y conductas de riesgo inconscientes.

Crueldad / Falta de empatía: La revisión determinó que **no existe evidencia neuroimagenológica suficiente** en la literatura actual para delimitar científicamente una relación directa entre la crueldad y las conductas agresivas.





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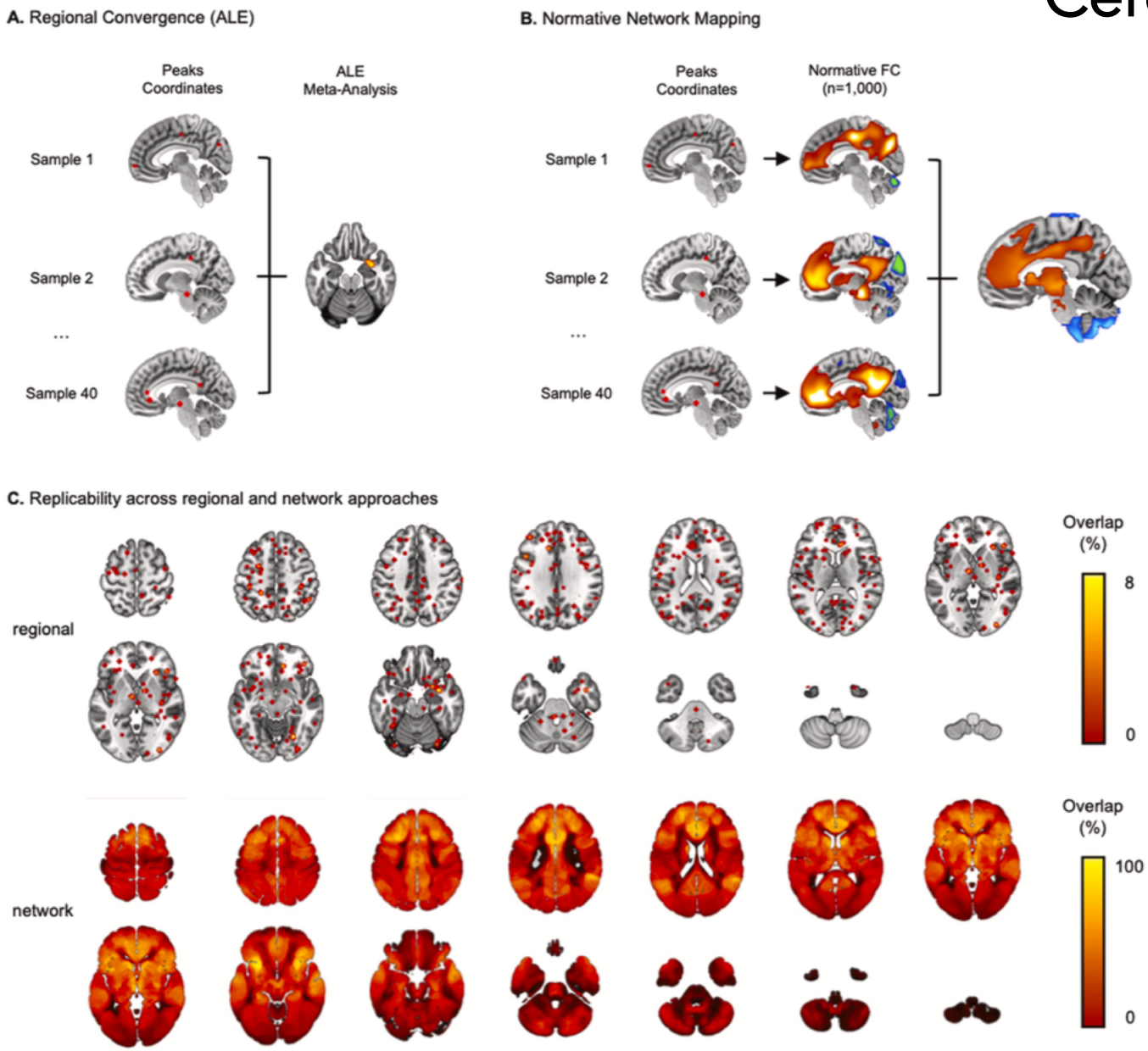
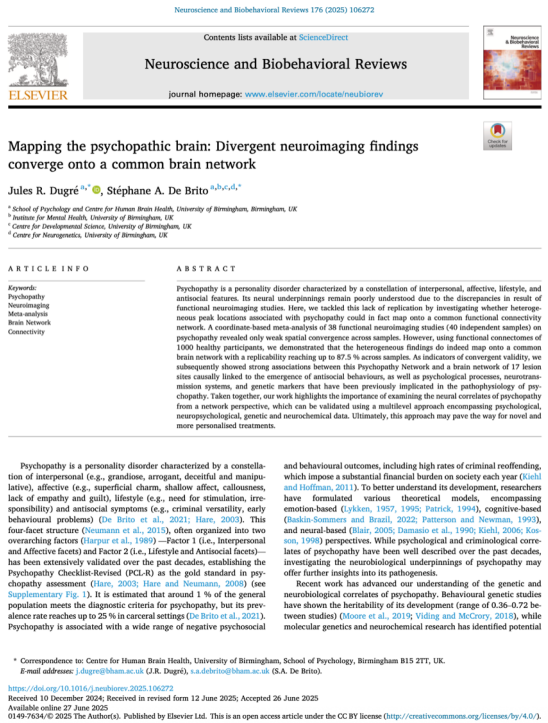
SPRINGER NATURE

Cerebro en Psicopatía



- Alteración de la red neuronal por defecto y prominencia.
- La actividad en regiones como el mPFC y la insula anterior se relaciona con los factores de interpersonales, afectivos y antisociales, control ejecutivo, atención y procesamiento emocional.



Neuroscience and Biobehavioral Reviews 176 (2025) 106272

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Mapping the psychopathic brain: Divergent neuroimaging findings converge onto a common brain network

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[a] School of Psychology and Centre for Human Brain Health, University of Birmingham, Birmingham, UK
[b] Institute for Mental Health, University of Birmingham, UK
[c] Centre for Developmental Science, University of Birmingham, UK
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Brain Network
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ABSTRACT

Psychopathy is a personality disorder characterized by a constellation of interpersonal, affective, lifestyle, and antisocial features. Its neural underpinnings remain poorly understood due to the discrepancies in result of functional neuroimaging studies. Here, we tackled this lack of replication by investigating whether heterogeneous peak locations associated with psychopathy could in fact map onto a common functional connectivity network. A coordinate-based meta-analysis of 38 functional neuroimaging studies (40 independent samples) on psychopathy revealed only weak spatial convergence across samples. However, using functional connectomes of 1000 healthy participants, we demonstrated that the heterogeneous findings do indeed map onto a common brain network with a replicability reaching up to 87.5 % across samples. As indicators of convergent validity, we subsequently showed strong associations between this Psychopathy Network and a brain network of 17 lesion sites causally linked to the emergence of antisocial behaviours, as well as psychological processes, neurotransmission systems, and genetic markers that have been previously implicated in the pathophysiology of psychopathy. Taken together, our work highlights the importance of examining the neural correlates of psychopathy from a network perspective, which can be validated using a multilevel approach encompassing psychological, neuropsychological, genetic and neurochemical data. Ultimately, this approach may pave the way for novel and more personalised treatments.

Psychopathy is a personality disorder characterized by a constellation of interpersonal (e.g., grandiose, arrogant, deceitful and manipulative), affective (e.g., superficial charm, shallow affect, callousness, lack of empathy and guilt), lifestyle (e.g., need for stimulation, irresponsibility) and antisocial symptoms (e.g., criminal versatility, early behavioural problems) (De Brito et al., 2021; Hare, 2003). This four-facet structure (Neumann et al., 2015), often organized into two overarching factors (Harpur et al., 1989) —Factor 1 (i.e., Interpersonal and Affective facets) and Factor 2 (i.e., Lifestyle and Antisocial facets)— has been extensively validated over the past decades, establishing the Psychopathy Checklist-Revised (PCL-R) as the gold standard in psychopathy assessment (Hare, 2003; Hare and Neumann, 2008) (see Supplementary Fig. 1). It is estimated that around 1 % of the general population meets the diagnostic criteria for psychopathy, but its prevalence rate reaches up to 25 % in carceral settings (De Brito et al., 2021). Psychopathy is associated with a wide range of negative psychosocial and behavioural outcomes, including high rates of criminal reoffending, which impose a substantial financial burden on society each year (Kiehl and Hoffman, 2011). To better understand its development, researchers have formulated various theoretical models, encompassing emotion-based (Lykken, 1957, 1995; Patrick, 1994), cognitive-based (Baskin-Sommers and Brazil, 2022; Patterson and Newman, 1993), and neural-based (Blair, 2005; Damasio et al., 1990; Kiehl, 2006; Kosson, 1998) perspectives. While psychological and criminological correlates of psychopathy have been well described over the past decades, investigating the neurobiological underpinnings of psychopathy may offer further insights into its pathogenesis.

Recent work has advanced our understanding of the genetic and neurobiological correlates of psychopathy. Behavioural genetic studies have shown the heritability of its development (range of 0.36–0.72 between studies) (Moore et al., 2019; Viding and McCrory, 2018), while molecular genetics and neurochemical research has identified potential

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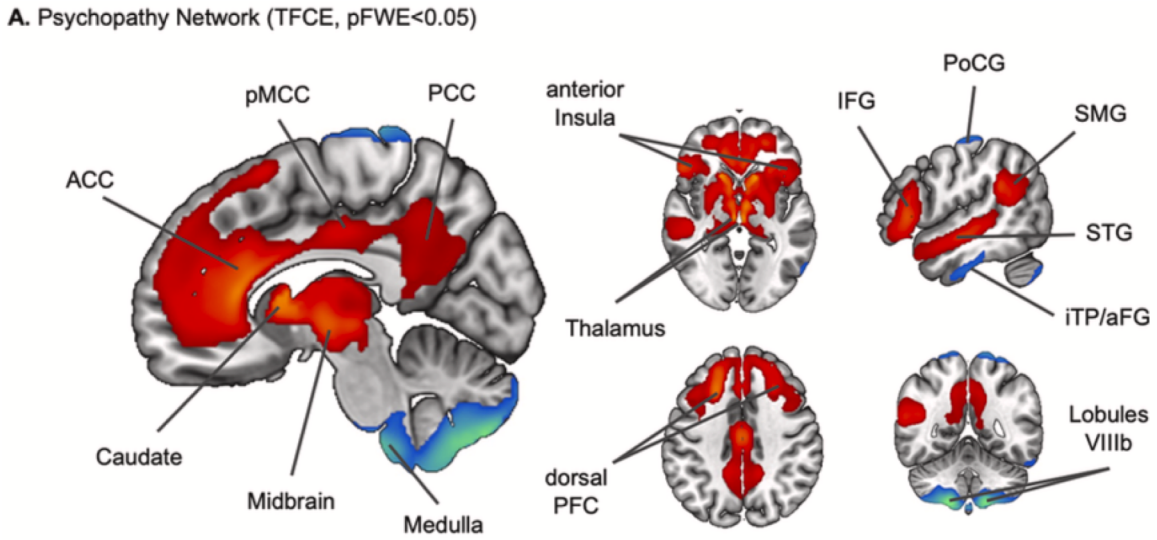
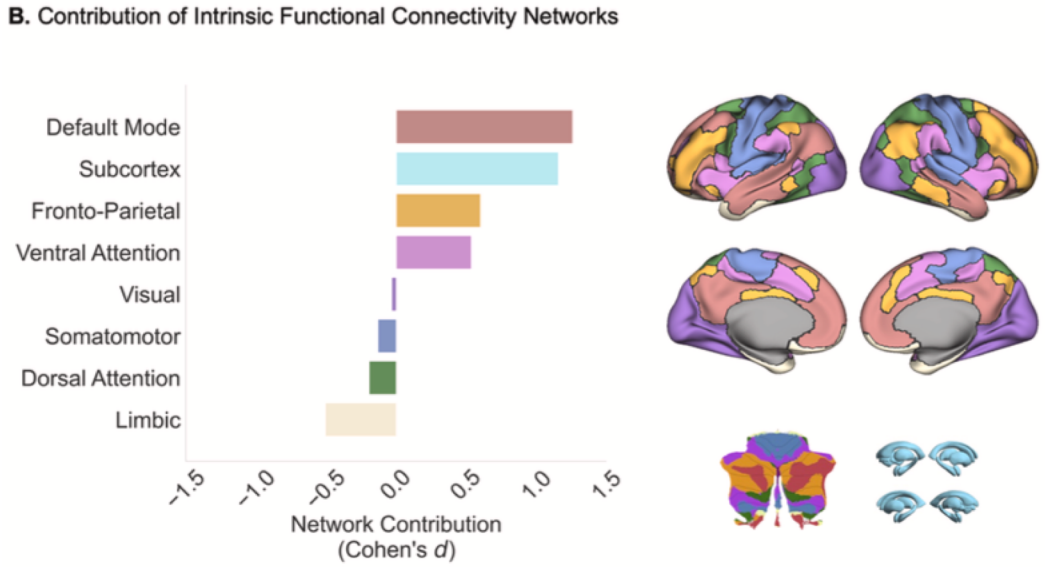
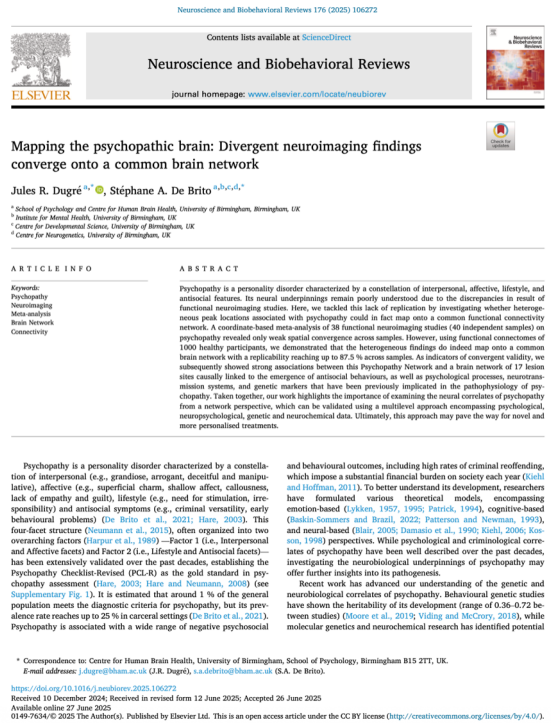
Cerebro en Psicopatía





- Cambios de dopamina, glutamato, histamina y serotonina (procesos socio-afectivos).
- Genes relacionados con la red incluyen BDNF, COMT, DRD2, MAOA, CNR1 y OXTR, con correlaciones espaciales significativas en regiones cerebrales.

corteza cingulada anterior dorsal, corteza cingulada media, núcleo caudado, ínsula anterior, claustro, corteza prefrontal dorsal/dorsolateral, tálamo/mesencéfalo, giro frontal inferior, giro supramarginal y giro temporal superior anterior.



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ABSTRACT

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Recent work has advanced our understanding of the genetic and neurobiological correlates of psychopathy. Behavioural genetic studies have shown the heritability of its development (range of 0.36–0.72 between studies) (Moore et al., 2019; Viding and McCrory, 2018), while molecular genetics and neurochemical research has identified potential

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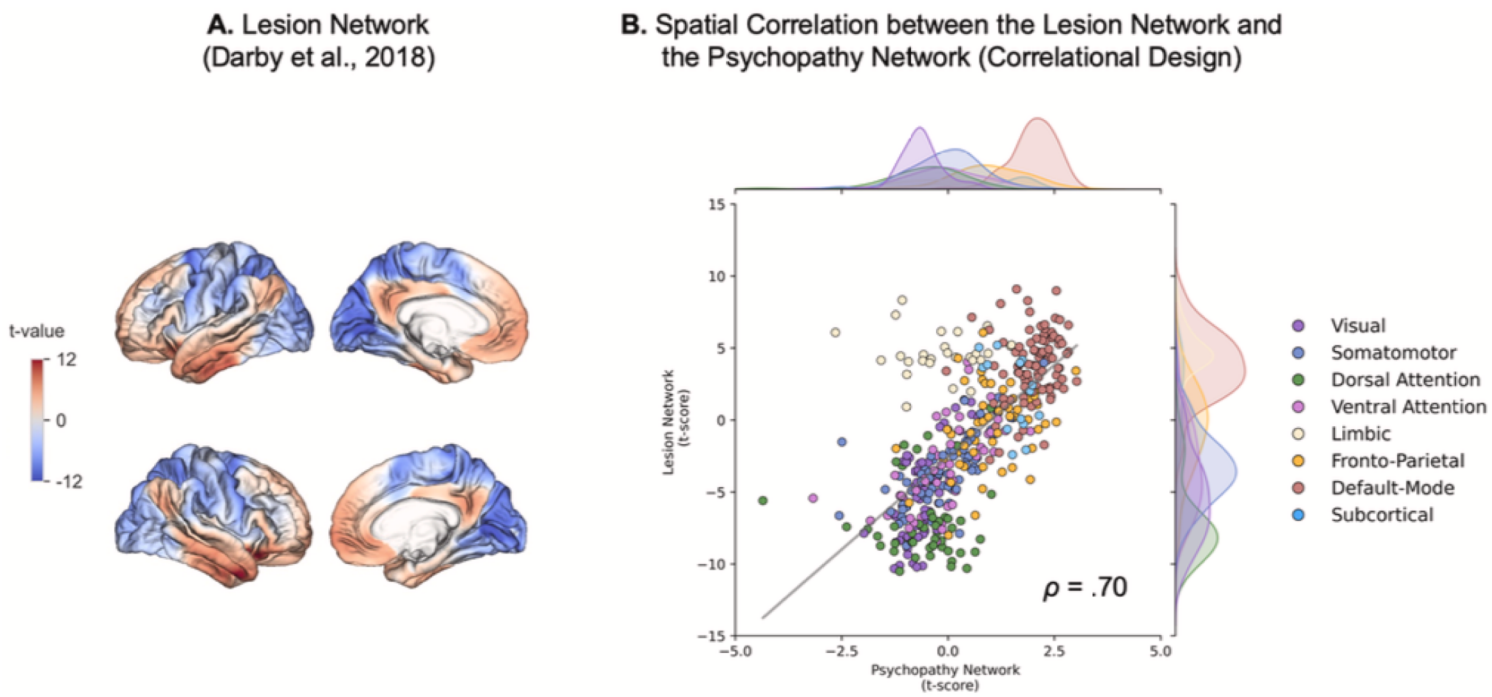
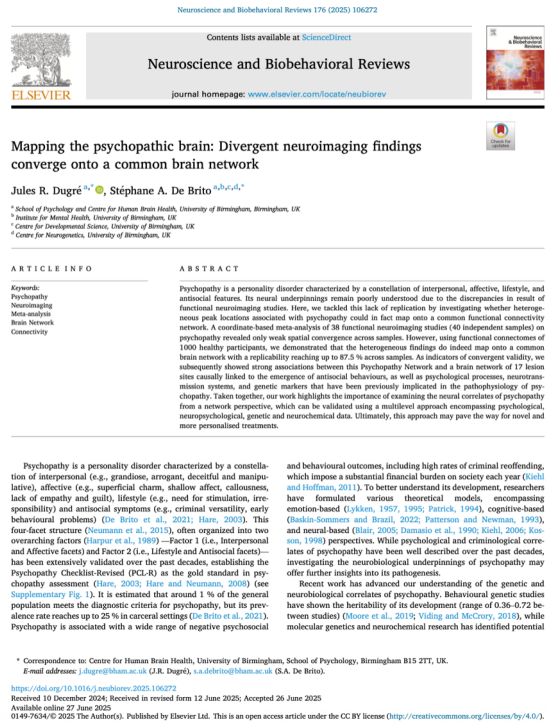
Cerebro en Psicopatía

- corteza cingulada anterior dorsal, corteza cingulada media, núcleo caudado, ínsula anterior, claustro, corteza prefrontal dorsal/dorsolateral, tálamo/mesencéfalo, giro frontal inferior, giro supramarginal y giro temporal superior anterior.

Alteraciones en la corteza prefrontal y temporal durante emociones y cognición.

La disfunción en estas áreas afecta la interacción entre emoción y comportamiento





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Mapping the psychopathic brain: Divergent neuroimaging findings converge onto a common brain network

Jules R. Dugré, Stéphane A. De Brito

ARTICLE INFO

ABSTRACT

Psychopathy is a personality disorder characterized by a constellation of interpersonal, affective, lifestyle, and antisocial features. Its neural underpinnings remain poorly understood due to the discrepancies in result of functional neuroimaging studies. Here, we tackled this lack of replication by investigating whether heterogeneous peak locations associated with psychopathy could in fact map onto a common functional connectivity network. A coordinate-based meta-analysis of 38 functional neuroimaging studies (40 independent samples) on psychopathy revealed only weak spatial convergence across samples. However, using functional connectomes of 1000 healthy participants, we demonstrated that the heterogeneous findings do indeed map onto a common brain network with a replicability reaching up to 87.5 % across samples. As indicators of convergent validity, we subsequently showed strong associations between this Psychopathy Network and a brain network of 17 lesion sites causally linked to the emergence of antisocial behaviours, as well as psychological processes, neurotransmission systems, and genetic markers that have been previously implicated in the pathophysiology of psychopathy. Taken together, our work highlights the importance of examining the neural correlates of psychopathy from a network perspective, which can be validated using a multilevel approach encompassing psychological, neuropsychological, genetic and neurochemical data. Ultimately, this approach may pave the way for novel and more personalised treatments.

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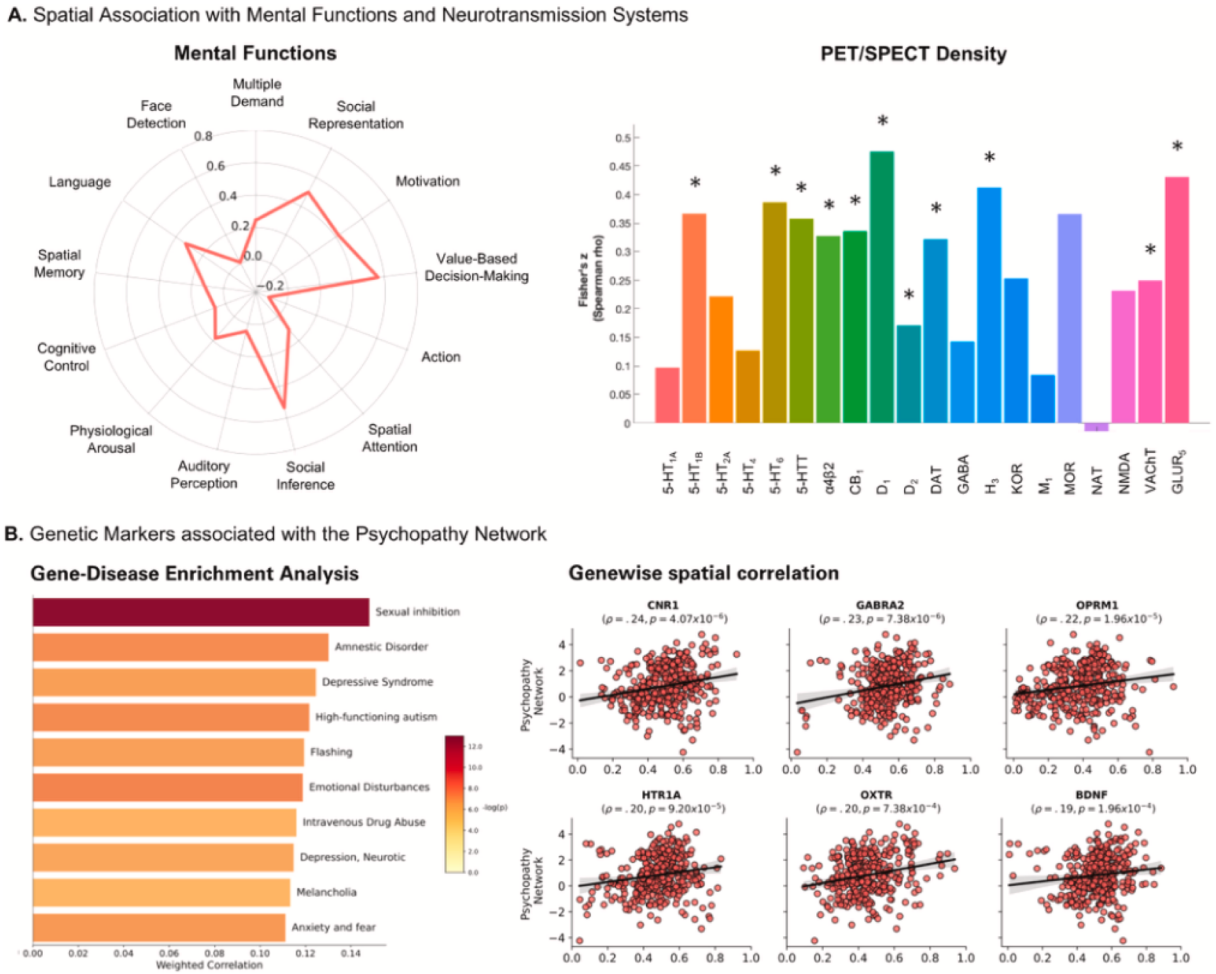
0149-7634/© 2025 The Author(s). Published by Elsevier Ltd. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

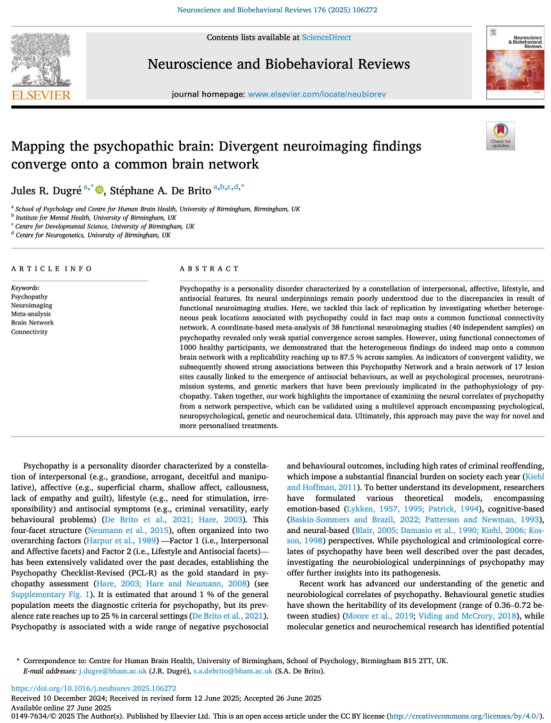
Cerebro en Psicopatía

La psicopatía mostró fuertes asociaciones espaciales con la toma de decisiones basada en el valor, la inferencia social y la representación social derivadas de la ontología cerebro-conducta.



112 trastornos fuertemente asociados con la red de la psicopatía

- Cambios de dopamina, glutamato, histamina y serotonina (procesos socio-afectivos).
- Genes relacionados con la red incluyen BDNF, COMT, DRD2, MAOA, CNR1 y OXTR, con correlaciones espaciales significativas en regiones cerebrales.



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Mapping the psychopathic brain: Divergent neuroimaging findings converge onto a common brain network

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Cambios en la mielinización conducta antisocial

Cíngulo

Fascículo fronto occipital

Fascículo longitudinal inferior

Fascículo longitudinal superior

Fascículo uncinar

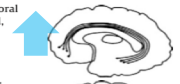
Cuerpo calloso

Proyecciones talámicas

Tracto cortico espinal

Tracto tálamo radial A/P

Summary of DTI findings by tract across studies youth and adults.

Tract and Description	Diagram of tracts	Adult findings	Youth findings
Association pathways Cingulum (CG): Connects frontal & temporal lobes, cingulate & medial gyri of frontal, parietal, occipital, & temporal lobes		*Greater diffusivity* ↓FA bilateral CG: interpersonal-affective traits (Sethi et al., 2015), AB + P+ vs. HC (Hoppenbrouwers et al., 2013) ↓FA left CG: AB + P+ vs. HC (Sethi et al., 2015) ↓AD bilateral CG: AB+ vs. HC (Lindner et al., 2016)	*Greater diffusivity* ↓AD bilateral CG: AB + vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↓RD bilateral CG: psychopathic traits (Pape et al., 2015)
Inferior fronto-occipital fasciculus (IFOF): Connects temporal lobe (medially) & frontal lobe (inferiorally).		*Greater diffusivity* ↓FA bilateral IFOF: AB + P+ vs. HC (Hoppenbrouwers et al., 2013; Sundram et al., 2012) ↓FA left IFOF: Interpersonal-affective traits (Hoppenbrouwers et al., 2013) ↓FA right IFOF: Lifestyle-antisocial traits (Hoppenbrouwers et al., 2013) ↑MD right IFOF: AB + P+ vs. HC (Sundram et al., 2012) ↓AD bilateral IFOF: AB + vs. HC (Lindner et al., 2016)	*Greater diffusivity* ↓FA bilateral IFOF: CD symptoms, AB+ vs. HC (Haney-Caron et al., 2014) ↓AD bilateral IFOF: AB+ vs. HC: (Haney-Caron et al., 2014) *Lower diffusivity* ↑FA & ↓RD bilateral IFOF: psychopathic traits (Pape et al., 2015)
Inferior longitudinal fasciculus (ILF): connects temporal pole & occipital pole.		*Greater diffusivity* ↓FA left ILF: AB + P+ vs. HC (Sundram et al., 2012)	*Greater diffusivity* ↓FA bilateral ILF: CD symptoms, AB+ vs. HC: (Haney-Caron et al., 2014 ↓AD bilateral ILF: AB+ vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↓RD bilateral ILF: psychopathic traits (Pape et al., 2015)
Superior longitudinal fasciculus (SLF): connects frontal lobe & parietal, occipital, & temporal lobes.		*Greater diffusivity* ↓FA & ↑RD right SLF: aggressive acts (Karlsgodt et al., 2015)	*Greater diffusivity* ↑RD right SLF: CD symptoms (Decety et al., 2015) ↓FA bilateral SLF: AB+ vs. HC (Haney-Caron et al., 2014; Li et al., 2005); ↓AD bilateral SLF: AB+ vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↑AD right SLF: CD symptoms (Decety et al., 2015) ↓RD bilateral SLF: psychopathic traits (Pape et al., 2015)
UF = uncinate fasciculus (UF): connects temporal pole & orbitofrontal cortex.		*Greater diffusivity* ↓FA bilateral UF: AB + P+ vs. HC (Hoppenbrouwers et al., 2013) ↓FA right UF: psychopathy (Wolf et al., 2015; Sobhani et al., 2015), interpersonal-affective traits (Wolf et al., 2015) & lifestyle-antisocial (Hoppenbrouwers et al., 2013), AB + P+ vs. HC (Craig et al., 2009; Sundram et al., 2012), AB + P+ vs. AB + P- (Motzkin et al., 2011) ↓FA left UF: lifestyle-antisocial (Hoppenbrouwers et al., 2013) AB + P+ vs. HC: ↓FA right UF (Craig et al., 2009; Sundram et al., 2012), ↑MD right UF: AB + P+ vs. HC (Sundram et al., 2012)	*Greater diffusivity* ↓FA bilateral UF: CU traits, AB + CU+ vs. HC (Breeden et al., 2015) ↓FA right UF: AB + CU- vs. HC: (Breeden et al., 2015) ↓AD bilateral UF: AB + vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↑FA bilateral UF: behavior problems: (Sarkar et al., 2013), psychopathic traits (Sarkar et al., 2013) ↑FA &↓RD bilateral UF: psychopathic traits (Pape et al., 2015) ↑FA left UF: AB + P+ vs. HC: (Sarkar et al., 2013); ↑FA, ↑AD, & ↓RD bilateral UF: AB+ vs. HC (Passamonti et al., 2012) ↓RD & ↓MD right UF: CU traits (Zhang et al., 2014a)
Commissural pathways Corpus callosum (CC), forceps major (Fmaj) & forceps minor (Fmin): Connects cerebral hemispheres: anterior (Fmin) & posterior (Fmaj)		*Greater diffusivity* ↓FA bilateral CC: AB + P+ vs. HC (Sundram et al., 2012) ↑MD right CC: AB + P+ vs. HC: (Sundram et al., 2012) ↓AD bilateral CC & Fmin: AB+ vs HC & clinical comparison (Lindner et al., 2016)	*Greater diffusivity* ↓FA left CC: CD symptoms (Haney-Caron et al., 2014) ↓AD bilateral CC & Fmin: AB+ vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↑FA & ↓RD bilateral CC & Fmin: psychopathic traits (Pape et al., 2015), AB+ vs. HC (Zhang et al., 2014b) ↑AD right Fmin: CD symptoms (Decety et al., 2015) ↓RD Fmaj: psychopathic traits (Pape et al., 2015)
Projection & thalamic pathways Fornix: Connects hippocampus & hypothalamus			*Greater diffusivity* ↓FA bilateral fornix: AB + CU+ vs. HC: (Breeden et al., 2015) ↓FA right fornix: CU traits, AB + CU- vs. HC (Breeden et al., 2015)
Cortico-spinal tract (CT), internal capsule (IC), external capsule (EC), peduncle, & corona radiata (ACR, PCR, SCR): Connects spinal cord & cerebral cortex		*Greater diffusivity* ↓FA bilateral IC: AB + P+ vs. HC (Sundram et al., 2012) ↓FA & ↑MD right ACR: AB + P+ vs. HC (Sundram et al., 2012). ↓AD left CR: AB+ vs. HC (Lindner et al., 2016)	*Greater diffusivity* ↓FA right ACR; bilateral SCR; left PCR: CD symptoms (Haney-Caron et al., 2014); ↓FA & ↓AD bilateral IC, peduncle, ACR, & SCR (Haney-Caron et al., 2014) ↓AD right PCR: AB+ vs. HC (Haney-Caron et al., 2014) *Lower diffusivity* ↑FA, ↑AD, & ↓RD bilateral CT: CU & psychopathic traits (Pape et al., 2015); right EC: AB+ vs. HC: (Passamonti et al., 2012) ↑AD left CT & right SCR: CD symptoms (Decety et al., 2015) ↑FA right SCR: AB+ vs. HC (Zhang et al., 2014b) ↑FA & ↓RD left ACR: AB+ vs. HC (Zhang et al., 2014b)
Anterior & posterior thalamic radiations (ATR & PTR): Connect the thalamus and visual, somatosensory, auditory, & pre-motor cortices		*Greater diffusivity* ↓FA bilateral ATR: AB + P+ vs. HC (Hoppenbrouwers et al., 2013) ↓FA left ATR: Interpersonal-affective (Hoppenbrouwers et al., 2013) ↓FA right ATR: Lifestyle-antisocial traits (Hoppenbrouwers et al., 2013) ↓FA left PTR: AB + P+ vs. HC (Sundram et al., 2012)	*Lower diffusivity* ↑FA in bilateral ATR: CD symptoms (Haney-Caron et al., 2014) & psychopathic traits (Pape et al., 2015)

La pubertad y las hormonas puede explicar las diferencias en la microestructura en la adolescencia.

Cambios en comunicaciones talámicas, incluyendo el tracto corticoespinal y radiaciones talámicas, afectan funciones motoras y sensoriales.



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White-matter tract abnormalities and antisocial behavior: A systematic review of diffusion tensor imaging studies across development

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ABSTRACT

Antisocial behavior (AB), including aggression, violence, and theft, is thought be underpinned by abnormal functioning in networks of the brain critical to emotion processing, behavioral control, and reward-related learning. To better understand the abnormal functioning of these networks, research has begun to investigate the structural connections between brain regions implicated in AB using diffusion tensor imaging (DTI), which assesses white-matter tract microstructure. This systematic review integrates findings from 22 studies that examined the relationship between white-matter microstructure and AB across development. In contrast to a prior hypothesis that AB is associated with greater diffusivity specifically in the uncinate fasciculus, findings suggest that adult AB is associated with greater diffusivity across a range of white-matter tracts, including the uncinate fasciculus, inferior fronto-occipital fasciculus, cingulum, corticospinal tract, thalamic radiations, and corpus callosum. The pattern of findings among youth studies was inconclusive with both higher and lower diffusivity found across association, commissural, and projection and thalamic tracts.

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Abbreviations: AB, antisocial behavior; AD, axial diffusivity; APD, antisocial personality disorder; CD, conduct disorder; CU, callous-unemotional; DMN, default mode network; DTI, diffusion tensor imaging; FA, fractional anisotropy; IFOF, inferior fronto-occipital fasciculus; ILF, inferior longitudinal fasciculus; MD, mean diffusivity; RD, radial diffusivity; SLF, superior longitudinal fasciculus; UF, uncinate fasciculus.

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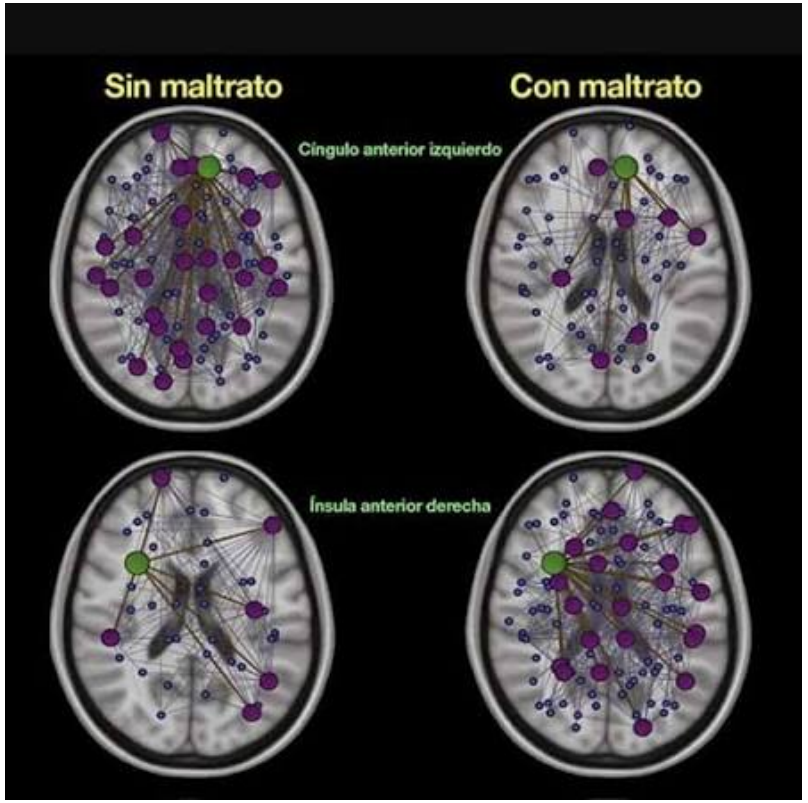
Maltrato infantil y daño cerebral

El maltrato infantil (abuso físico, emocional, la negligencia física, excepto abuso sexual), están significativamente relacionados con rasgos **insensibles y carentes de emociones.**

Mayores niveles de ansiedad
Aumento del riesgos de trastornos emocionales en la adultez.



29 estudios y casi 10,000 participantes.



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Review article

Association between childhood maltreatment and callous-unemotional traits in youth: A meta-analysis

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Psychopathy

ABSTRACT

Callous-unemotional (CU) traits (i.e., lack of remorse or guilt, callous lack of empathy, deficient concern for the feelings of others) in youth with conduct problems confer risk for a particularly severe and persistent form of antisocial behaviour. Previous research has linked childhood maltreatment as a potential risk factor for CU traits, both primary (i.e., genetically underpinned) and secondary (i.e., environmentally influenced) variants, but findings have been inconsistent, and the association has not yet been tested in a meta-analysis. To address this gap, we conducted a meta-analysis to assess the nature and strength of the associations between childhood maltreatment and its subtypes with CU traits and potential variants (i.e., primary and secondary CU traits). A systematic search identified 29 eligible studies including 9,894 participants (42% female) between the ages of 3 and 18 years (M_{age}=14.22 years, SD = 1.07). Results revealed a significant moderate positive association between childhood maltreatment and CU traits. All subtypes of maltreatment bar sexual abuse were significantly associated with CU traits. However, it was not possible to compare primary and secondary CU traits directly due to inconsistencies in how they are defined. The limitations posed by current research signal the need for clinical and operational guidelines on how to define primary and secondary CU traits. Additionally, prospective longitudinal, genetically informed research is needed to clarify if maltreatment is a causal risk factor for CU traits.

Conduct problems (CP) in childhood and adolescence delineate a group of youth with behaviours that include aggression and violence as well as overt and covert ways of violating the norms of society. CP are associated with high rates of crime and substance misuse as well as problems that persist throughout adulthood including compromised mental and physical health, reduced interpersonal functioning, and poor educational and economic outcomes (Erskine et al., 2014; Rivenbark et al., 2018). In the UK, the financial costs associated with CP include mental health referrals, hospital admissions due to accidents, supplementary educational and social services, as well as the high costs to the justice system (D'Amico et al., 2014; Romeo et al., 2006). Worldwide, CP carry a heavy global health burden (Erskine et al., 2014) and are a major predictor of how much an individual will cost society (Burt et al., 2018; Kassing et al., 2018; Scott et al., 2001).

Within CP, there is considerable heterogeneity, with different symptom clusters and developmental trajectories (Fairchild et al., 2019). Efforts to identify more homogenous subgroups of CP have resulted in different subtyping approaches within the DSM-5, one of which focuses on the presence of Limited Prosocial Emotions (LPE), indexing callous-unemotional (CU) traits (American Psychiatric Association, 2013). CU traits are defined by a characteristic lack of remorse or guilt, callous lack of empathy, and shallow or deficient concern for the feelings of others, which are the core affective features of psychopathy in adulthood (Frick et al., 2014b). CP co-occurring with elevated levels of CU traits have drawn considerable attention in recent years due to increased antisocial behaviours and reduced responding to socialization efforts and clinical interventions of this particular subgroup of antisocial youth (Viding and McCrory, 2012a). Indeed, there is now substantial evidence that CU traits identify a subgroup of youth with CP exhibiting particularly severe and persistent patterns of antisocial behaviour who might be at risk of developing the adult syndrome of psychopathy (Burke et al., 2007; Hawes et al., 2017). In addition, an extensive body of research indicates that youth with CP and elevated levels of CU traits i) are characterized by different environmental and genetic vulnerabilities (De Brito et al., 2021), and ii) differ behaviourally, emotionally, and neurocognitively from youth with CP and low

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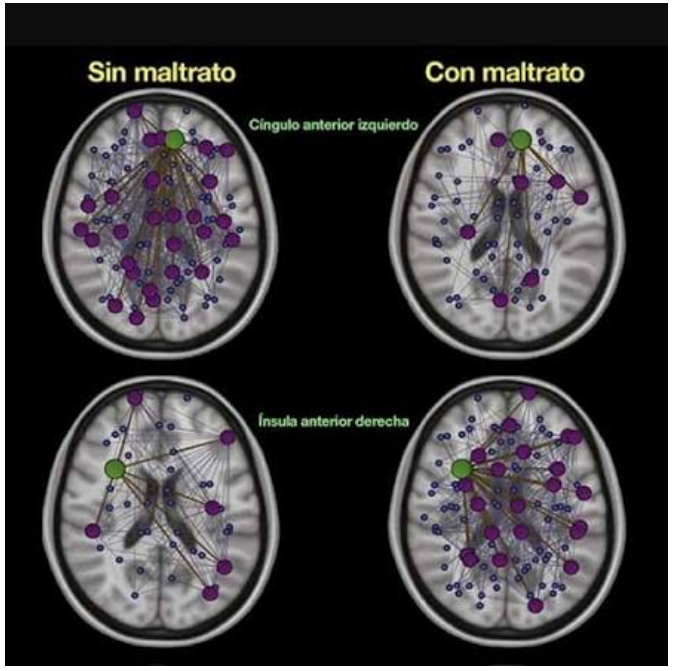
Maltrato infantil y daño cerebral

- La asociación fue más fuerte en las facetas conductuales y antisociales que en las afectivas e interpersonales.
- El maltrato en la infancia, especialmente en el contexto familiar, aumenta el riesgo de psicopatía en adultos.

El abuso infantil afecta la estructura, función y conectividad cerebral.
La neurobiología del maltrato deja cicatrices que dificultan la recuperación.



47 estudios con 12,737 participantes



PLOS ONE

RESEARCH ARTICLE

A meta-analysis of childhood maltreatment in relation to psychopathic traits

Corine de Ruiter[1]*, Matthias Burghart[1,2], Raneesha De Silva[1], Sara Griesbeck Garcia[1], Ushna Mian[1], Eoin Walshe[1], Veronika Zouharova[1]

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Abstract

Psychopathy is a personality disorder characterized by a mix of traits belonging to four facets: affective (e.g., callous/lack of empathy), interpersonal (e.g., grandiosity), behavioral instability (e.g., impulsivity, poor behavioral controls), and social deviance (e.g., juvenile delinquency, criminal versatility). Several scholars have argued that early childhood maltreatment impacts the development of psychopathy, although views regarding its role in the four facets differ. We conducted a meta-analysis including 47 studies comprising a total of 389 effect sizes and 12,737 participants, to investigate the association between psychopathy and four types of child maltreatment: physical abuse, emotional abuse, neglect, and sexual abuse. We found support for a moderate link between overall psychopathy and childhood physical abuse, emotional abuse, and neglect, as well as overall childhood maltreatment. The link between psychopathy and childhood sexual abuse was small, but still statistically significant. These associations were stronger for the behavioral and antisocial facets than for the affective and interpersonal facets of psychopathy, but nearly all associations were statistically significant. Our findings are consistent with recently developed theories on the role of complex trauma in the development of severe personality disorders. Trauma-focused preventive and therapeutic interventions can provide further tests of the trauma-psychopathy hypothesis.

Introduction

I had a kind of rough childhood. My Mom was admitted to a psychiatric institution many times, and my Dad was extremely harsh. He and my older brothers used to hit me with a broom. They also made me sit in a corner with hot peppers in my mouth. My memories of my childhood are rather chaotic, I do not like to think about that period. I was bullied in elementary school. But then, one day, I remember I hit this kid in the school yard during recess, and that's when I found out I could be a bully too. And from that time on, I switched from being a victim to being the perpetrator. (Patient A.)

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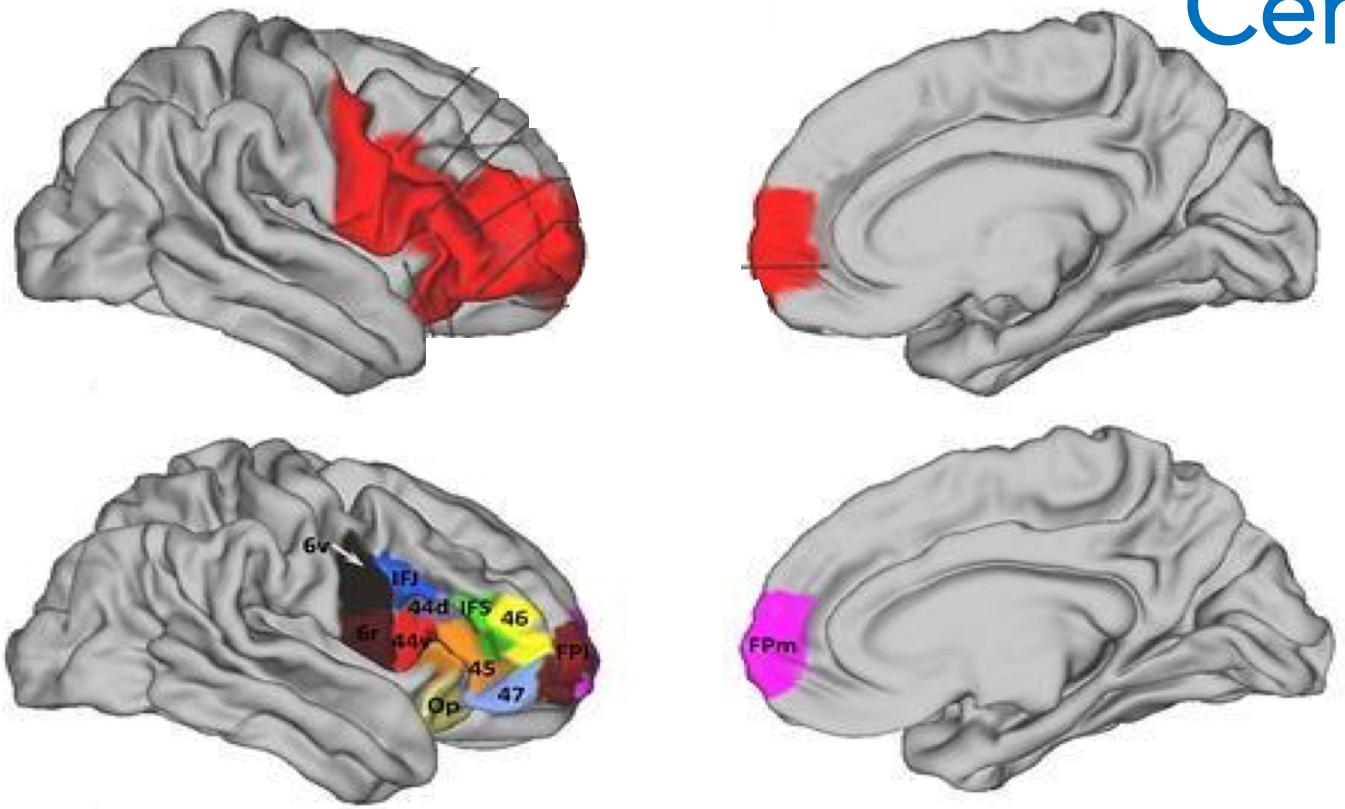
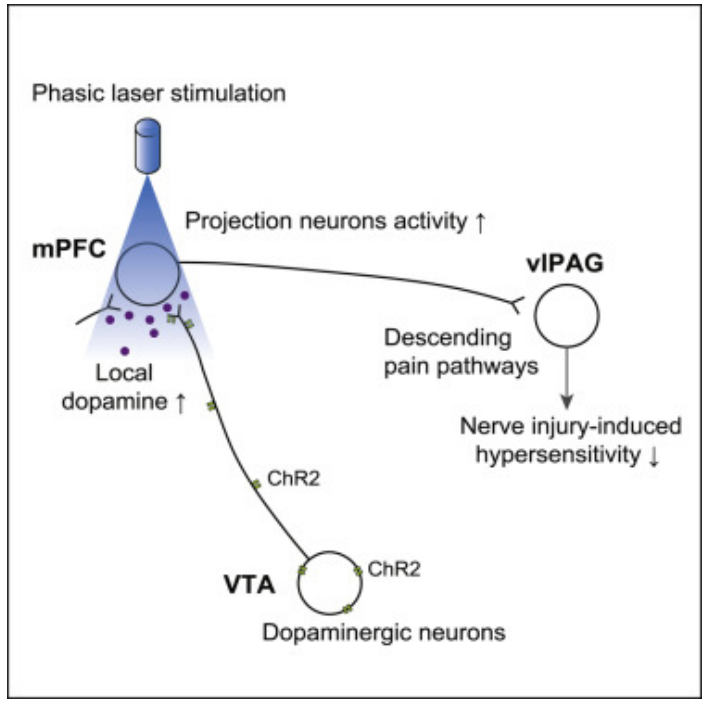
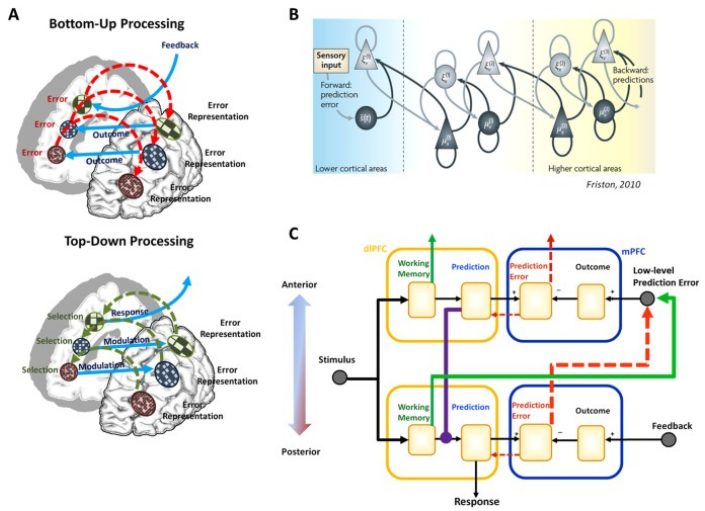
Cerebro Humano







TalenTU
Trasciende tu potencial, expande tu universo
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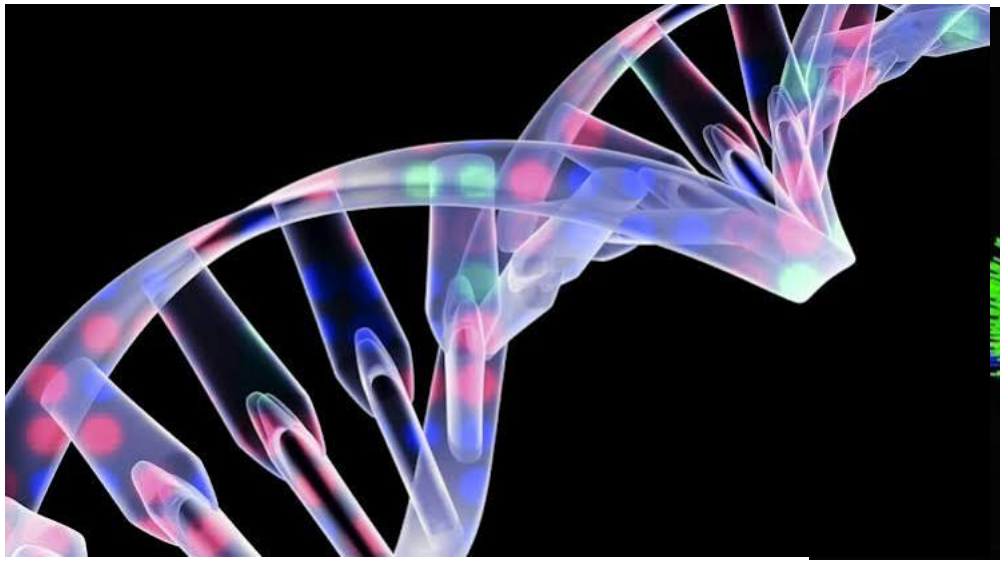
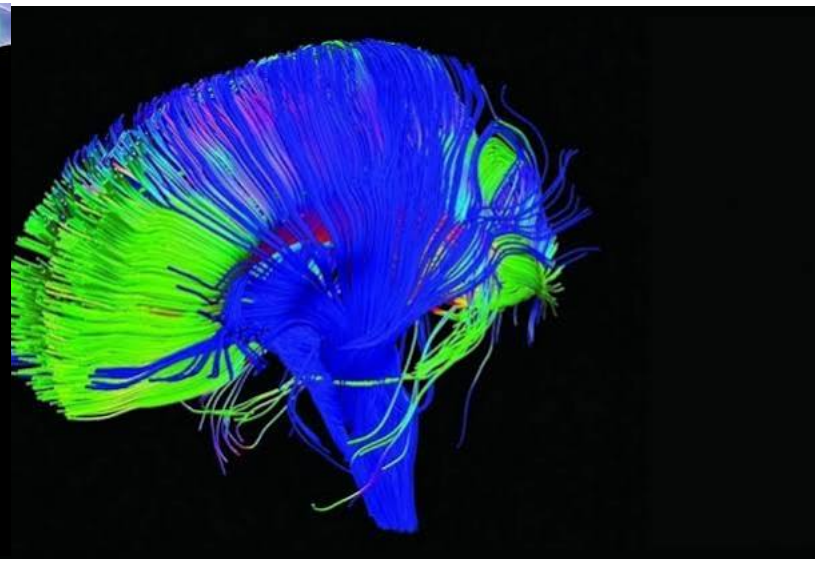
Drogas, herencia y Psicopatía

El comportamiento antisocial y el consumo de sustancias muestran alta comorbilidad y herencia genética, con estimaciones entre 40-60%.

Mecanismos comunes en impulsividad y sensibilidad a recompensas en ambos comportamientos.

En especial con uso de cannabis y cigarrillos.





Drug and Alcohol Dependence 187 (2018) 296–299

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Short communication

Genetic correlation of antisocial behaviour with alcohol, nicotine, and cannabis use

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Nicotine
Cigarette
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Antisocial behaviour
Genetic correlation
Substance

ABSTRACT

Background: There is high comorbidity between antisocial behaviour (ASB) and substance use, and twin studies have shown that part of the covariation is due to overlapping genetic influences. Here we used measured genetic effects to estimate the genetic correlations of ASB with nicotine, alcohol, and cannabis use.

Methods: We meta-analysed data from two genome-wide association studies for ASB and used existing summary statistics from the largest genome-wide association studies into substance use (ever smoking, cigarettes smoked per day, weekly alcohol consumption, and lifetime cannabis use). We performed cross-trait LD-score regression to estimate genetic correlations between ASB and substance use phenotypes explained by all single nucleotide polymorphisms (SNPs). When significant, we tested whether the signs of the regression coefficients of SNPs from the ASB and substance use phenotypes were in the same direction across multiple p-value thresholds and examined enrichment in overlap of the strongest associated SNPs.

Results: We found nominally significant genetic correlations of ASB with lifetime cannabis use (r_g = 0.69, p = .016) and cigarettes per day (r_g = 0.59, p = 0.036) but not with weekly alcohol consumption or ever smoking. Sign-tests revealed consistent directions of effect of SNPs for ASB and cannabis use for all p-value thresholds except the most stringent one, whereas for ASB with cigarettes per day no consistent evidence was found. We found no evidence of enrichment in overlap of the most associated SNPs across these traits.

Conclusion: Using measured genetic variants, we found preliminary support for a genetic correlation of ASB with lifetime cannabis use and cigarettes per day.

1. Introduction

Antisocial behaviours (ASBs, including conduct problems and antisocial personality) are characterised by irresponsible, impulsive, aggressive, and dishonest behaviours and pose a major burden on affected individuals and their families as well as on society as a whole (Foster and Jones, 2005; McCollister et al., 2010). The consequences of ASB—particularly violent behaviour—are severe and can be long lasting.

ASBs show substantial comorbidity with other psychiatric syndromes and maladaptive behaviours (Abram et al., 2015). Previous studies have shown that individuals with antisocial personality or conduct problems are at increased risk for substance (ab)use, including nicotine, alcohol, and cannabis use (e.g., Compton et al., 2005; Elkins et al., 2007; Fergusson et al., 2007; Goldstein et al., 2017; Palmer et al., 2013). In a 25-year longitudinal study, Fergusson et al. (2007) found that conduct problems during childhood and adolescence are related to later nicotine, alcohol, cannabis, and illicit drug use, abuse, and dependence (with the exception of alcohol use, probably as a result of the high rate of alcohol use in the cohort). The effects remained even after controlling for attentional problems and confounding social, family, and related factors (individual characteristics and behaviours).

Twin and family studies have shown that antisocial behaviours and substance use are heritable traits. Heritability estimates for conduct symptoms and conduct disorder generally range between 40% and 60% (Gelhorn et al., 2005; Miles et al., 2002; Polderman et al., 2015; Rhee

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Desregulación emocional es una característica transdiagnóstica de varios trastornos psiquiátricos y del neurodesarrollo:

1. Mayor reactividad emocional (cambios rápidos)
2. Inestabilidad del estado de ánimo (inapropiadas)
3. Dificultades para regular las respuestas emocionales (extremas).
4. Cambios de humor (arrebatos de ira)
5. Trastornos de alimentación.

Trastorno límite de la personalidad, trastorno por déficit de atención con hiperactividad y Trastorno bipolar.

Neurofunctional Correlates of Emotional Dysregulation: Systematic Review and ALE Meta-Analysis. Ern Wong; et al, 2026. *Brain and Behavior*. 16:e71376 https://doi.org/10.1002/brb3.71376

Desregulación emocional

NO es un diagnóstico: es un patrón transdiagnóstico

En adultos 6.6% y 11.3% en muestras comunitarias, con una tendencia a disminuir con la edad.
En contextos clínicos, la frecuencia es mucho mayor: hasta **62.3%** de los niños y adolescentes.



Dificultad persistente para **modular la intensidad, la duración y la expresión de las emociones** de forma flexible y adaptativa. Dificultad para **recuperar el equilibrio emocional** una vez activada una emoción.

Emociones muy intensas, cambios bruscos de ánimo, dificultad para calmarse, respuestas impulsivas o sensación de estar "desbordado" emocionalmente.



International Journal of *Environmental Research and Public Health*

MDPI

Review

Antisocial Disorders in Adolescence and Youth, According to Structural, Emotional, and Cognitive Transdiagnostic Variables: A Systematic Review

Ronald Toro [1], Juan García-García [2,*] and Flor Zaldívar-Basurto [2]

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Received: 27 March 2020; Accepted: 25 April 2020; Published: 27 April 2020

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Abstract: Transdiagnostic causal variables have been identified that have allowed understanding the origin and maintenance of psychopathologies in parsimonious explanatory models of antisocial disorders. However, it is necessary to systematize the information published in the last decade. The aim of the study was to identify through a systematic review, the structural, emotional and cognitive transdiagnostic variables in antisocial disorders of adolescence and youth. Recommendations for systematic reviews and meta-extraction and analysis of information according to the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA), the Cochrane Collaboration and Campbell were followed. We found 19 articles from 110 reviewed documents. The results indicated that at a structural level there is a general psychopathological factor (psychopathy or externalizing), non-emotional callousness and impulsivity from behavioral inhibition and activation systems, and negative affect traits as base structures. In the emotional level, the study found a risk component from emotional dysregulation and experiential avoidance. In the cognitive level, a key role of anger-rumination and violent ideation as explanatory variables of antisocial disorders. We concluded that the interaction of these identified variables makes it possible to generate an evidence-based transdiagnostic model.

Keywords: transdiagnostic; antisocial; externalizing; criminal behavior; juvenile delinquency; social deviance

1. Introduction

Antisocial disorders (ADs) are characterized by alterations in impulse control, emotional dysregulation and behavioral problems, manifested in behaviors that violate the rights of others such as delinquency, property damage, aggression, and conflicts with accepted social norms and authority figures [1,2]. ADs have been analyzed from a dimensional spectrum made up of a group of psychopathologies, which are organized according to their common etiologies and symptoms, resulting in the externalizing spectrum [3]. In DSM 5 [2], they appear in the group of ADs of childhood, adolescence, and adulthood, which includes conduct disorders (including specification: with limited pro-social emotions), intermittent explosive disorder and antisocial personality disorder, in addition to antisocial behavior in the child or adolescent located in other disorders associated with disruptive or impulsive behavior.

It is possible to develop models that guide the understanding of the origin and maintenance, from the common variables called transdiagnostic by using a spectrum as an indicator of AD in psychopathology research, following a dimensional perspective of the processes and factors underlying the different disorders. The transdiagnostic model (TM) "consists of understanding mental disorders

Desregulación emocional

NO es un diagnóstico: es un patrón transdiagnóstico

Presente en trastornos psiquiátricos y psicológicos, contribuyendo a su gravedad y persistencia:

- Trastornos del estado de ánimo (depresión, trastorno bipolar).
- Trastornos de ansiedad y trauma.
- Trastorno por déficit de atención e hiperactividad (TDAH).
- Trastornos de la personalidad.
- Trastornos de la conducta alimentaria.
- Trastornos del espectro autista y consumo de sustancias.





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Desregulación emocional

predice conductas agresivas y problemas internos.

La rumiación y la ideación violenta están relacionadas con la impulsividad, la agresividad y el consumo de sustancias.

La evitación experiencial y los sesgos perceptivos aumentan el riesgo de victimización y problemas conductuales.





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Desregulación emocional

La desregulación emocional y la evitación experiencial, se asocian con mayor riesgo de conductas antisociales, aumentan la probabilidad de victimización, problemas internos y externos.

Rumiación de ira y la ideación violenta, que explican comportamientos antisociales.

La percepción sesgada y la dificultad en la regulación cognitiva contribuyen a la agresividad y a la impulsividad.





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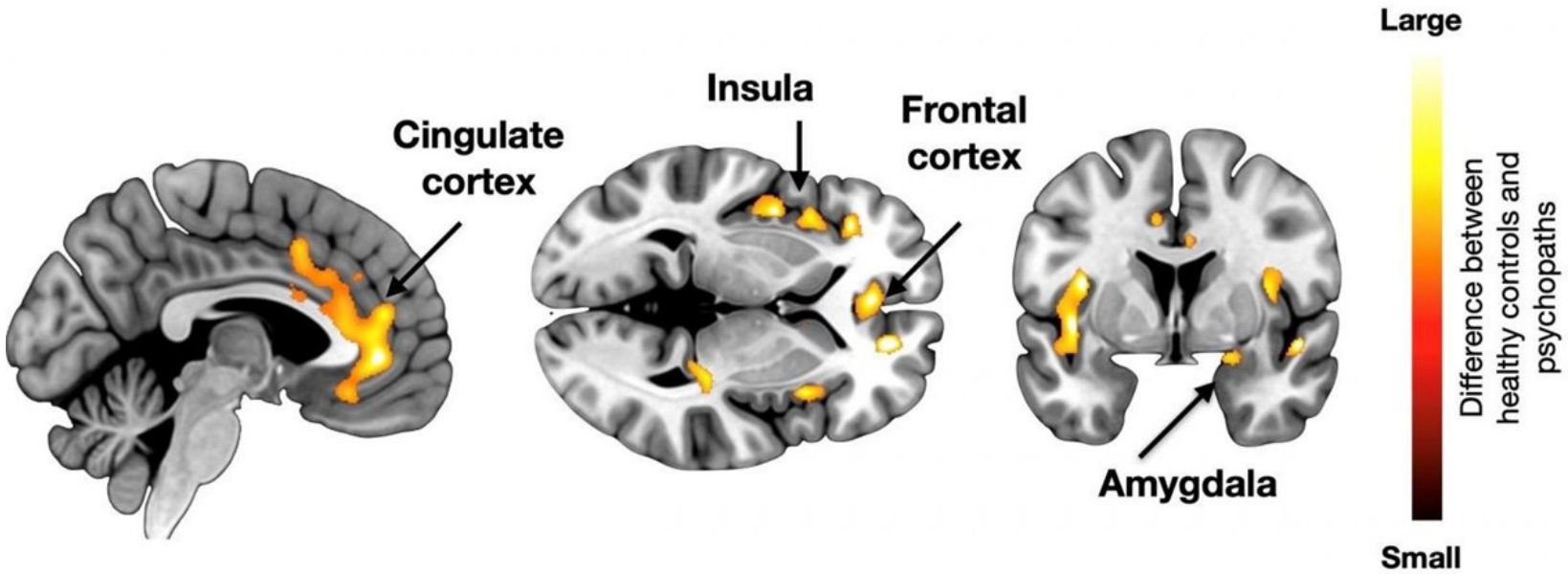
Psicología, Criminología y Salud mental

La psicopatía están más relacionadas con la agresión sexual.

Mezquindad y desinhibición: a mayor presencia de estos rasgos, más fuerte es la relación con la agresión sexual.

El abuso sexual infantil tiene mecanismos distintos a otras formas de agresión sexual en relación con la psicopatía.





ORIGINAL ARTICLE OPEN ACCESS

The Relation Between Psychopathy and Sexual Aggression: A Meta-Analysis

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Keywords: meta-analysis | personality | psychopathy | sexual aggression | triarchic model of psychopathy

ABSTRACT

Objective: Psychopathy is a multifaceted, hierarchical construct that has been linked to aggression and antisocial behavior. The triarchic model of psychopathy comprises three underlying, distinct trait domains: boldness, disinhibition, and meanness. Understanding how psychopathy at general and factor levels relates to sexual aggression is critical given its connection and the serious repercussions of sexual aggression.

Method: This preregistered meta-analysis ($k = 117$) examined the relationship between psychopathy at the total construct and triarchic levels and sexual aggression in adult samples. A relative weights analysis was used to produce triarchic psychopathy scores from commonly used psychopathy measures and the Triarchic Psychopathy Measure (TriPM).

Results: Psychopathy evinced a moderate, positive relationship with general sexual aggression as well as most specific forms of sexual aggression except for rape and child molestation. At the triarchic level, meanness and disinhibition related positively and boldness negatively to sexual aggression. Other moderation analyses revealed significant heterogeneity in study methods and characteristics that can explain variability in psychopathy's relations with sexual aggression.

Conclusions: Psychopathy relates moderately to various forms of sexual aggression. The relationship depends on multiple factors. Understanding these mechanisms can inform prevention, treatment, and risk assessment of damaging sexual acts.

Psychopathy is a personality construct associated with dysfunction in several domains, including perspective-taking (e.g., Brown et al. 2015), aggression (e.g., Walters 2003), and substance overuse (e.g., Lynam et al. 2013). Cleckley (1941) defined psychopathy by the "mask of sanity" which hid serious underlying pathology. Psychopathy is not an official diagnosis included in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association 2013), largely due to a lack of consensus on its conceptualization (e.g., Crego and Widiger 2015), but the Alternative Model of Personality Disorders Section III of the DSM-5 includes it as a specifier for the diagnosis of antisocial personality disorder.

The development of the Psychopathy Checklist and its revision (PCL, PCL-R; Hare 1980, 1991, 2003), which uses an extensive file review and interview, afforded a useful assessment that jumpstarted research on psychopathy (Hare and Neumann 2008). The PCL measures focus on traits such as grandiosity, impulsivity, irresponsibility, superficial charm, manipulativeness, callousness, and shallow affect. Stemming from a need to measure psychopathy outside of forensic settings, several self-report assessments have been developed, such as the Triarchic Psychopathy Measure (TriPM; Patrick 2010), Psychopathic Personality Inventory (PPI, Lilienfeld and Andrews 1996; Lilienfeld and Widows 2005), Levenson Self-Report Psychopathy Scale (LSRP; Levenson et al. 1995), Self-Report Psychopathy

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Trastorno Explosivo Intermitente







Agresión impulsiva, puede comenzar a los 6 años.
Curso crónico

Impulsos agresivos pueden desencadenarse por eventos menores.

El comportamiento es desproporcionado.

Agresiones físicas o daños a la propiedad.

Actos de agresión no físicos, como la agresión verbal.

Ocurre dos veces por semana durante 3 meses, o la agresión física debe ocurrir al menos 3 veces en un período de 12 meses.

Trastorno Explosivo Intermitente







4.5% de la poblacion
Suele iniciar a los 13 años
Más frecuente en varones

Sienten un profundo remordimiento, disgusto, arrepentimiento.

Síntomas fisiológicos como taquicardia, sudoración y disnea.

Tras el arrebato, pueden sentir alivio, en ocasiones: placer.

Trastorno Explosivo Intermitente









Reducción del volumen de la amígdala izquierda en comparación con los controles.

Menor actividad en la corteza orbitofrontal medial, la corteza cingulada anterior, corteza prefrontal ventrolateral izquierda.

Reducciones en el volumen de la ínsula izquierda.

El putamen y la ínsula anterior izquierda presentan una mayor actividad

Actualización en Psicopatología, Neurofarmacología y Bases Biológicas de la Estimulación Neuronal.

Fin del Tema 3

La psicopatía: diferencias/analogías entre la anatomía y neuroquímica de la psicopatía y sociopatía.

DR. EDUARDO CALIXTO

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