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Differential Neuroanatomy of the Gyrus Rectus in Antisocial Disorder with and without Psychopathy: Structural and Functional Correlations using MRI, 99mTc-ECD SPECT, and 18F-SynVesT-1 PET

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ABSTRACT: The gyrus rectus is a key component of the medial orbitofrontal circuit and plays a central role in behavioral inhibition, moral judgment, and impulse control. Alterations in this region have been associated with impulse control disorders, psychopathy, and violent behavior; however, synaptic density in this area has not been examined in populations with antisocial personality disorder. The aim of this study was to describe structural, functional, and synaptic density differences in the gyrus rectus between individuals with antisocial personality disorder with and without psychopathy. Forty-three individuals with antisocial personality disorder were evaluated through psychopsychiatric and neuropsychological assessments, as well as neuroimaging studies (morphometric MRI, ^{99}mTc-ECD SPECT, and ^{18}F-SynVesT-1 PET). Statistical analyses were conducted in accordance with current ethical and legal standards. Both groups showed significant morphometric and functional alterations, with a more pronounced synaptic reduction in individuals with psychopathy. These findings suggest the presence of differential neurobiological patterns in the gyrus rectus, which are relevant for the neuroforensic understanding of inhibitory control and criminal responsibility.

KEY WORDS: neuroanatomy, synaptic density, antisocial personality disorder, psychopathy.

INTRODUCTION

The gyrus rectus is part of the medial orbitofrontal circuit involved in behavioral inhibition, moral judgment, and impulse control. Its study is relevant in psychiatric evaluations involving impulse control disorders (Berlin *et al*., 2004; Boes *et al*., 2009; Accolla *et al*., 2016), psychopathy (Moll *et al*., 2005), frontal damage, frontotemporal dementias (Seeley *et al*., 2008; Karaca *et al*., 2025), and violent behavior (Yang & Raine, 2009), where volumetric reduction or cortical thinning has been reported in correlation with neuropsychological assessments (Stroop, IGT, FrSBe, BIS-11), as well as alterations in the integrity of the fibers and tracts that interconnect it. Additionally, its anatomical and functional organization within the orbitofrontal cortex has been described in the literature (Ten Donkelaar *et al*., 2018). Despite substantial morphological and functional advances, including the use of PET in individuals with antisocial personality disorder and/or psychopathy with different tracers (Hsu, 2008; Cai *et al*., 2019), the study of synaptic density in this anatomical target has not (to date) been addressed in this population. The aim of this study was to describe structural, functional, and synaptic density alterations in the gyrus rectus between individuals with antisocial personality disorder with and without psychopathy.

MATERIAL AND METHOD

We studied 43 individuals aged 26–52 years (mean = 40.11, SD = 7.4) diagnosed with antisocial personality disorder (F60.2 ICD-10; 301.7 DSM-5), with and without psychopathic traits (DSM-5). Exclusion criteria included central neurological disorders and traumatic brain injury. Psychopsychiatric and neuropsychological evaluations were performed, along with neuroimaging studies (morphometric 1.5T magnetic resonance imaging with a cognitive protocol; 99mTc-ECD SPECT; and 18F-SynVesT-1 PET for neurodegeneration studies in neuropsychiatry).

Both groups underwent neuropsychological and neuropsychiatric assessment using the Mini-Mental State Examination by Folstein *et al*. (1975), the Global Deterioration Scale by Reisberg *et al*. (1982), the Handedness Test adapted from Tornese & Mascitti (1994), and the MINI International Neuropsychiatric Interview (Sheehan *et al*., 1998).

Subsequently, brain imaging studies were conducted in both groups using structural neuroimaging (MRI with a cognitive protocol for volumetric quantification) and functional neuroimaging (99mTc-ECD SPECT to assess perfusion and 18F-SynVesT-1 PET to study synaptic density).

The morphometric study was performed using a 1.5 T magnetic resonance scanner. Regions of interest were explored using sequences weighted for tissue relaxation times (T1, T2, and FLAIR), with acquisition in axial, coronal, and sagittal planes. T1-weighted images were obtained using 1.5 mm slices in the axial plane and sequences of 1.5 mm and 3 mm in the coronal plane, perpendicular to the temporal plane axis and the major brain axis. These were subsequently obtained through 3D gradient acquisition with artifact removal and reconstruction of stable static images.

Image quantification was carried out in three steps using a semi-automated data processing analysis, in which images were processed using the region of interest (ROI) option, manually corrected, and morphometry of the gyrus rectus was determined in both hemispheres. After MRI image normalization, image analysis was performed using atlas-based software, resulting in segmentation of the brain into anatomical structures. This atlas-based approach enabled automated and reproducible parcellation of cortical and subcortical regions. The atlases used for volumetric analysis allowed specific segmentation of the frontal lobe, including detailed segmentation of the gyrus rectus. The asymmetry index (AI) was calculated using the formula: $AI = [(L - R)/0.5 (L + R)] \times 100\%$, where L and R represent the volumes of the left and right brain structures, respectively.

Volumetric quantification of the gyrus rectus (Brodmann area 14, BA14) and its asymmetry index was obtained using the following boundaries: medially, the interhemispheric fissure; laterally, the olfactory sulcus; anteriorly, the frontal pole; posteriorly, the coronal plane corresponding to the posterior border of the genu of the corpus callosum; superiorly, the gray–white matter interface; and inferiorly, the orbitofrontal pial surface.

The neurofunctional study using Single Photon Emission Computed Tomography (SPECT) was performed with the administration of the radiotracer 99mTc-ECD to evaluate cortical cerebral perfusion (Fig. 1). Data were reconstructed using the OSEM method, with attenuation correction (Chang method) (Chang, 1979) and semi-quantitative analysis of cortical perfusion or cortical blood flow (CBF) (Tanaka *et al*., 2000), applied to the cortices of the gyrus rectus. For CBF classification, in accordance with

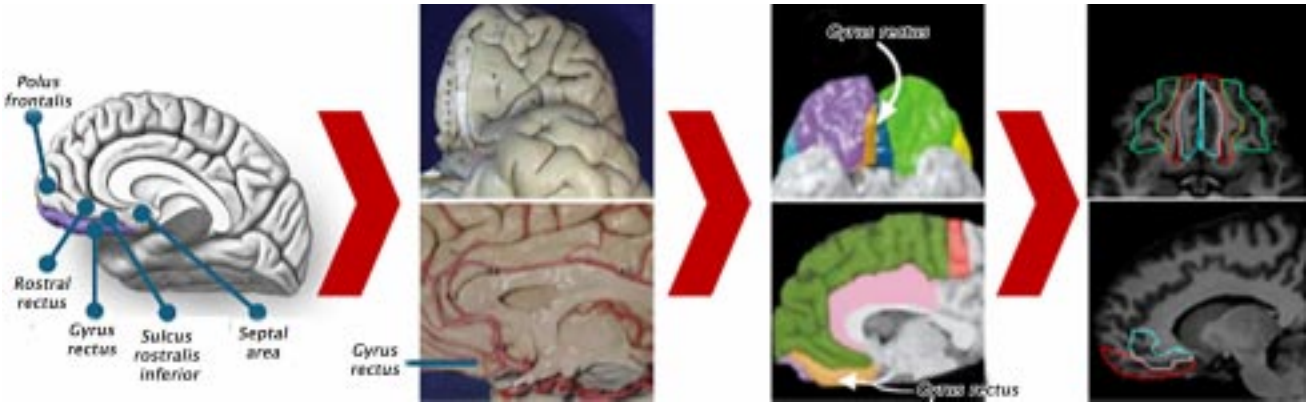

Fig. 1. Anatomical landmarks and MRI morphometry.

international reports, perfusion differences greater than 2 SD from the normalized perfusion for the anatomical region were considered absolute perfusion alterations (absolute hypo- or hyperperfusion), whereas those between 1 SD and 2 SD were considered relative perfusion alterations (relative hypo- or hyperperfusion). Subsequently, perfusion decreases were compared between the psychopathy and non-psychopathy groups (Fig. 2).

The neurofunctional study using Positron Emission Tomography (PET) was performed using the radiopharmaceutical ^{18}F-SynVesT-1, a PET tracer selective for the synaptic vesicle glycoprotein 2A (SV2A), an indirect marker of synaptic density. The uptake of ^{18}F-SynVesT-1 reflects SV2A density as an indirect marker of synapses, without allowing direct evaluation of dynamic synaptic or neurotransmitter activity. This radiopharmaceutical was synthesized with a radiochemical purity greater than 95% and administered intravenously (approximate dose: 185–370 MBq, adjusted to body weight), with dynamic acquisition for approximately 90 minutes. Participants remained at rest in a quiet environment, with eyes open or closed according to the standardized protocol. Corrections for attenuation, scatter, radioactive decay, and motion were applied. Image reconstruction was performed using iterative algorithms (OSEM), yielding corrected three-dimensional volumes.

PET images were co-registered to individual MRI images and normalized to a standard anatomical space. The region of interest, the gyrus rectus, was defined, and the signal of the ^{18}F-SynVesT-1 tracer was quantified using appropriate kinetic models. The main measure of synaptic binding was the total distribution volume (VT) or, alternatively, the binding potential (BP), using a reference region devoid of SV2A (e.g., central white matter). Brain function was inferred from regional synaptic density, estimated by ∂?F-SynVesT-1 uptake in the selected ROI.

After data acquisition, descriptive and inferential statistical analyses of anatomical parameters were performed for both groups (mean, standard deviation [SD], maximum, minimum, and percentage difference between group means). Statistical significance of the quantified parameters was assessed using Student's t-test, with significance defined as $p < 0.05$. Statistical analysis was performed using Microsoft Excel® 2010 for Windows XP.

Ethical considerations included obtaining informed consent from all participants prior to psychopatological testing and neuroimaging, ensuring confidentiality of their identity. This study complies with the requirements established by Good Clinical Practice (GCP), ANMAT Provision 6677/10, and adheres to the Ethical Principles originating from the Declaration of Helsinki.

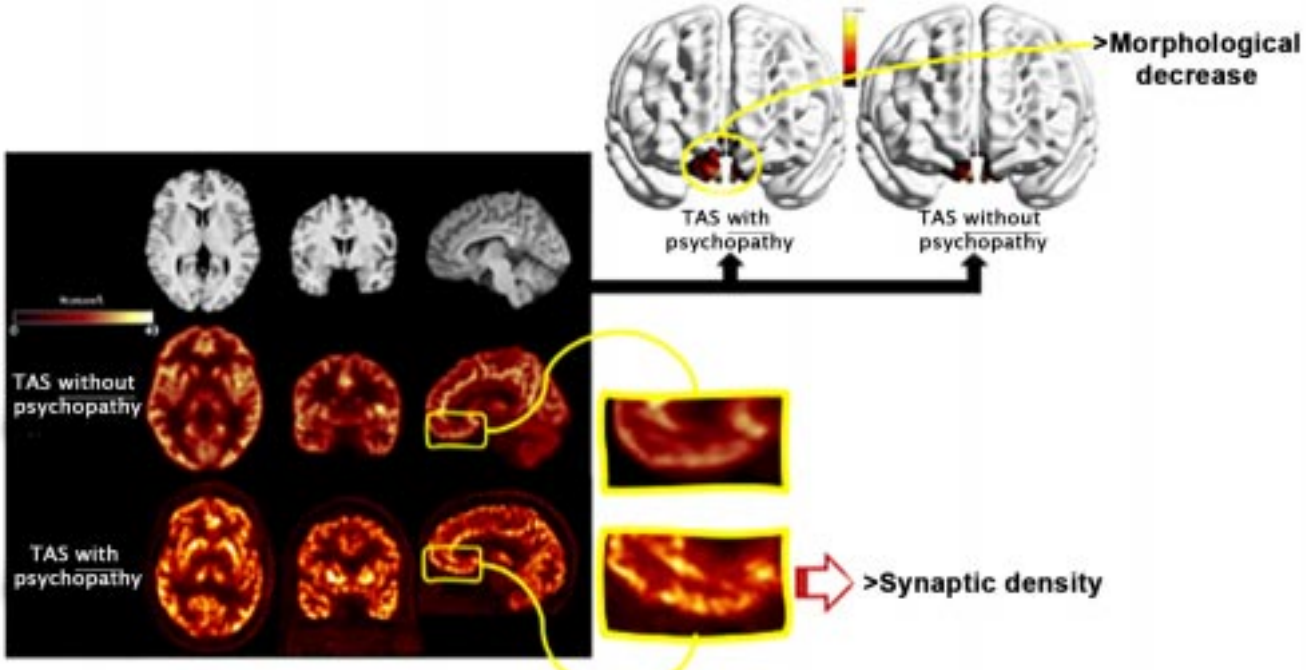

Fig. 2. Structural (MRI) and functional (SPECT/PET) neuroimaging with results by group.

RESULTS

Significant morphometric and functional alterations of the gyrus rectus were observed in antisocial personality disorder without psychopathic traits (volumetric: -18.4%; functional: -12.7%) and with such traits (volumetric: -11.5%; functional: 6.1%) ($p < 0.001$ and $p < 0.05$, respectively). Synaptic reduction was recorded in antisocial personality disorder without psychopathy (4.9%, $p < 0.05$), which was lower than in cases with psychopathy (-9.6%, $p < 0.01$) (Fig. 3).

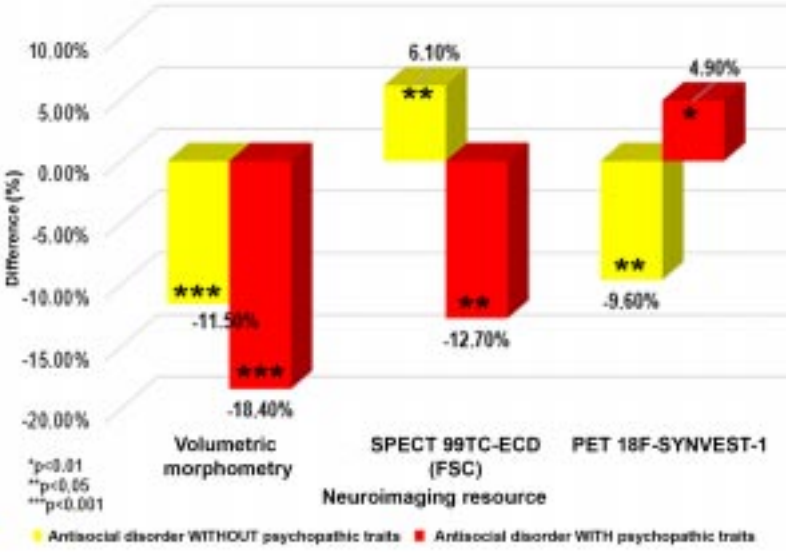

Fig. 3. Percentage variation in imaging studies by group.

DISCUSSION

The findings of this study on the gyrus rectus provide both novel and significant evidence and, in some aspects, are contradictory to the existing neuroanatomical and functional literature on antisocial personality disorder with and without psychopathy. The volumetric and functional decrease observed in both groups partially confirms previous studies that reported alterations in prefrontal and orbitofrontal regions in populations with antisocial behaviors and psychopathic traits (structural and functional changes in the orbitofrontal cortex, insula, and cingulate cortex), in both clinical and community samples (Rae *et al*., 2015; Atmaca *et al*., 2017; Li *et al*., 2023). However, the differential pattern of synaptic density found (lower in the group without psychopathy than in the group with psychopathy) introduces a microstructural dimension that has been scarcely addressed and may have implications for understanding the biology of behavioral inhibition and emotional regulation.

Classical literature has documented volumetric alterations in orbitofrontal and paralimbic regions in individuals with psychopathy and persistent antisocial behaviors, associating these patterns with deficits in impulse regulation, decision-making, and emotional processing (Ballmaier *et al*., 2004; Kringelbach & Rolls, 2004; Johanson *et al*., 2020). Our results of volumetric reduction (-18.4% in antisocial personality disorder without psychopathy and -11.5% in antisocial personality disorder with psychopathy) are consistent with these structural findings, suggesting that the gyrus rectus is compromised in both clinical phenotypes (Table 1). However, the observation of a less marked functional decrease in the psychopathy group (6.1%) compared to the non-psychopathy group (-12.7%) represents an interesting nuance: it may indicate compensatory mechanisms or functional reorganization in the presence of psychopathic traits that partially preserve activation in orbitofrontal circuits despite

Table 1. Structural alterations of the gyrus rectus / orbitofrontal cortex.

Study	Sample	Technique	Region	Main findings
Present study	Antisocial personality disorder with and without psychopathy (n = 43)	Morphometric MRI	Gyrus rectus	↓Volume in antisocial disorder without psychopathy (-18.4%); ↓moderate in with psychopathy (-11.5%)
Schiffer *et al.,* 2011	Antisocial disorder / psychopathy	Volumetric MRI	Medial orbitofrontal cortex	↓ Orbitofrontal cortex volume in both groups; greater association with impulsivity
Boccia *et al.,* 2015	Systematic review	Structural MRI	Orbitofrontal cortex / Ventromedial prefrontal cortex	Consistent reductions associated with moral and inhibitory deficits

volumetric reduction. Recent functional neuroimaging studies have proposed similar hypotheses, where individuals with psychopathic traits show altered, but not uniformly reduced, patterns of connectivity and activation in prefrontal and reward networks compared to controls without antisocial traits (Nummenmaa *et al*., 2021).

This apparently contradictory pattern (reduced hypofunction despite volumetric alterations) may be interpreted through neuroplasticity and neurocognitive compensation. For example, in other disorders of behavioral control, atypical functional reorganizations have been described in which non-traditional circuits partially assume compromised functions (e.g., increased frontoparietal activation for inhibitory tasks despite reduced prefrontal volumes). Although specific research in antisocial personality disorder and psychopathy remains limited, these models suggest that psychopathy may involve a reconfiguration of synaptic and functional networks that mitigates the structural impact on certain behavioral functions. This differential functional pattern, characterized by a milder impairment in the psychopathy group, has been interpreted in recent functional studies as a phenomenon of reorganization or instrumental efficiency of orbitofrontal circuits (Table 2).

The most novel finding of this study is the quantification of synaptic density, which introduces a microstructural dimension that few previous studies have directly addressed in antisocial personality disorder or psychopathy. The more marked synaptic reduction in the group without psychopathy (4.9%) compared to the group with psychopathy (-9.6%) suggests that the loss or reorganization of excitatory/inhibitory synapses in the gyrus rectus may play a differential role in antisocial phenotypes, influencing the efficiency or dysfunction of key circuits involved in emotional regulation, punishment learning, empathy, and moral decision-making. Although there are currently no published PET studies using synaptic density tracers in antisocial personality disorder and psychopathy, research in other neuropsychiatric conditions has shown correlations between synaptic density and cognitive and behavioral control functions, supporting the biological plausibility of these findings. The quantification of synaptic density using SV2A PET represents the most innovative contribution of this study and allows interpretation of differences between antisocial personality disorder with and without psychopathy at a microstructural level, a dimension that has been scarcely explored in previous literature (Table 3).

Table 2. Orbitofrontal functional alterations.

Study	Technique	Group	Functional findings
Present study	99mTc-ECD SPECT	Antisocial personality disorder without psychopathy	↓ Gyrus rectus function (-12.7%)
Present study	99mTc-ECD SPECT	Antisocial personality disorder with psychopathy	↓ Mild functional decrease (-6.1%)
Contreras-Rodríguez *et al*., 2015	fMRI	Psychopathy	Altered orbitofrontal cortex connectivity, not necessarily hypoactive
Rae *et al*., 2015	fMRI	Inhibitory control	Medial orbitofrontal cortex involved in behavioral inhibition

Table 3. Synaptic density (SV2A PET) and behavioral correlates.

Study	Technique	Population	Results
Present study	18F-SynVesT-1 PET	Antisocial personality disorder without psychopathy	↓ Synaptic density (-4.9%)
Present study	18F-SynVesT-1 PET	Antisocial personality disorder with psychopathy	↓ Greater synaptic density reduction (-9.6%)
Glenn & Raine, 2014	Review	Neurobiology applied to criminology	Synaptic microstructure relevant for criminal responsibility
Cai *et al*., 2019	Review	Applications in neuropsychiatry	SV2A biomarker is significant for synapse measurement
Kraguljac *et al*., 2021	SV2A PET	Psychiatric disorders	↓ Synapses associated with cognitive deficits
Howes *et al.,* 2025	Review	State of the art studies	SV2A quantification is related to neurological and psychiatric disorders, especially in the prefrontal cortex and hippocampus

From a neurobiological perspective of impulse control, the gyrus rectus is part of the medial orbitofrontal circuit involved in behavioral inhibition and moral processing (Glenn & Raine, 2009; Zhou *et al*., 2010; Rodrigues *et al*., 2015). Volumetric and functional alterations in this region have been associated with antisocial behaviors, deficits in behavioral change in response to punishment, and poor emotional regulation (Johanson *et al*., 2020). The integration of our data suggests that, beyond macroscopic features (volume) and functional activation, synaptic microstructure may be a key determinant of the efficiency of these circuits: lower synaptic density could be associated with reduced adaptive flexibility, hindering the integration of punishment/reward signals in decision-making.

This perspective also incorporates considerations of early neurodevelopment and inhibitory GABA/glutamate systems, which emerge as speculative hypotheses for psychopathy in genetic and neurochemical studies. Although the exact mechanisms are not yet clarified in the literature, studies on synaptic plasticity in the context of executive control and moral decision-making indicate that alterations in GABAergic and glutamatergic transmission may shape atypical behavioral patterns that persist into adulthood.

From a forensic neuroscience perspective—a construct that "meta-integrates" knowledge from basic, clinical, and translational neuroscience with psychiatric, psychological, and neuropsychiatric practice—these results imply that clinical and forensic evaluations should consider not only structural and functional deficits but also synaptic alterations that may influence inhibitory control and moral judgment. The distinction between antisocial personality disorder with and without psychopathy, in terms of synaptic density, could refine the understanding of voluntary choice capacity and criminal responsibility. However, larger sample sizes and longitudinal designs are required to assess causality and the stability of these biomarkers, as well as the multiple variables that shape human behavior. Neuroanatomical, functional, and synaptic differences between these antisocial phenotypes have direct implications for the assessment of inhibitory control and self-determination, which are central aspects in forensic psychiatric practice (Table 4).

Finally, this study has clear limitations related to sample size and the absence of a matched control group. In addition, the relationship between synaptic density measured by PET and specific behavioral assessments will require more robust correlations to establish conclusive causal links. Future studies should integrate multimodal paradigms (structural and functional MRI, DTI tractography, and PET with higher-resolution synaptic markers) and consider environmental and genetic factors that modulate neurodevelopment.

CONCLUSIONS

In antisocial personality disorder, morphological and functional reductions of the gyrus rectus were observed, greater in cases comorbid with psychopathy; however, synaptic density was lower in cases without psychopathy. These findings may reflect reduced efficiency of inhibitory/excitatory synapses in circuits responsible for fear regulation, guilt, punishment learning, moral decision-making, and cognitive empathy in individuals without psychopathy. The focal decrease reinforces the hypothesis that psychopathy involves not only functional but also microstructural alterations related to synaptic density and may be linked to early neurodevelopmental changes in the GABA/glutamate system, which, in a comorbid context, may "improve" or partially restore capacities related to social performance (empathy). Further studies with larger samples are required, as well as the (re)construction of new hypotheses within the neuro-law framework regarding these findings and their implications for punishability and inhibitory control.

Table 4. Comparative neuroforensic implications.

Dimension	ASPD without psychopathy	ASPD with psychopathy
Gyrus rectus volume	↓↓↓	↓↓
Orbitofrontal function	↓↓↓	↓
Synaptic density	↓	↓↓
Inhibitory control	Markedly impaired	Instrumental / selective
Legal implication	Regulatory deficit	Preserved capacity with emotional coldness

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