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DOPAMINE TRANSPORTER AVAILABILITY IN GAMBLING DISORDER: PRELIMINARY ¹²³I-FP-CIT SPECT RESULTS

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BACKGROUND-AIM

Gambling disorder (GD) has recently been acknowledged as a behavioural addiction instead of an impulse control disorder, as the clinical phenomenology and neurobiological profile share many common features with substance-related disorders. Multiple neurotransmitters have been implicated in the pathophysiology of GD. Current research has highlighted an abnormal functioning of mesolimbic structures and an altered neurotransmitter regulation of the reward pathways, particularly of dopamine (DA). The role of DA is also supported by the associations between dopaminergic medications and impulse control disorders in Parkinson's disease patients. We investigated striatal DAT availability in GD using ¹²³I-FP-CIT SPECT.

METHODS

We enrolled 7 male patients (mean age: 48±13 years) with a DSM-V diagnosis of GD, mainly addicted to slot machine gambling. Exclusion criteria were substance dependence other than nicotine, major Axis-I disorder other than GD, clinically relevant medical conditions, brain MRI abnormalities, medications affecting central dopamine functions. Severity of GD was assessed by the Gambling Symptom Assessment Scale and Pathological Gambling-Yale Brown Obsessive-Compulsive Scale. The Gambling Timeline Followback (G-TLFB) was used to estimate gambling behaviour in the last 4 weeks before enrolment. Anxiety, depression and anhedonia levels were evaluated by the Hamilton Anxiety Rating Scale, Hamilton Depression Rating Scale and Snaith Hamilton Pleasure Scale. The Temperament and Character Inventory and Barratt Impulsiveness Scale were used to assess personality dimensions and impulsivity traits. SPECT was carried out 3 hours after 185 MBq ¹²³I-FP-CIT intravenous injection. Specific to non-specific ¹²³I-FP-CIT binding ratios in the basal ganglia were computed using the automated BasGan V2 software. The control group consisted of 14 healthy subjects (HC; 11 M, mean age: 48±17 years).

RESULTS

In comparison with HC, GD patients showed decreased ¹²³I-FP-CIT binding ratios in the right caudate ($p < 0.05$, Mann-Whitney U test). Significant inverse correlations were found between the number of days spent gambling (as assessed by G-TLFB) and ¹²³I-FP-CIT binding ratios in the caudate and putamen, bilaterally ($p < 0.05$, Spearman's correlation analysis). The severity of gambling behaviour did not significantly correlate with ¹²³I-FP-CIT binding ratios. No correlations were found between ¹²³I-FP-CIT binding ratios and other psychometric measures.

CONCLUSION

We provided evidence of a lower DAT availability in the right caudate of pathological gamblers, as well as an inverse association between DAT levels in the bilateral caudate and putamen and the number of gambling days, thus supporting the hypothesis of disordered DA transmission in GD pathophysiology. Although our findings are consistent with some previous reports in various types of addictions, either with or without substances, the small sample size hinders any definite conclusion. A functional down-regulation of DAT may be hypothesized even though a genetically-determined lower membrane expression leading to higher synaptic DA levels cannot be ruled out.