

عنوان مقاله:

ALTERATIONS OF NEUROTRANSMITTER SYSTEMS AND BRAIN ACTIVITY IN PATHOLOGICAL GAMBLING: A MULTIDISCIPLINARY APPROACH TO UNFOLD THE PLEXUS OF THE ADDICTIVE GAMBLING NEUROBIOLOGY

محل انتشار:

سيزدهمين كنگره بين المللي دانش اعتياد (سال: 1398)

تعداد صفحات اصل مقاله: 1

نویسنده:

Bahman Sadeghi - Department of Biochemistry, Institute of Biochemistry and Biophysics (IBB), University of Tehran,
Iran

خلاصه مقاله:

Background and Aim: Notwithstanding the roughly notable prevalence of 5%, and remarkable comorbidity and mortality of pathological gambling (PG), little evidence has unfolded the convoluted plexus of neurotransmitters such as norepinephrine in arousal, glutamate in cognitive flexibility, serotonin in impulse control, dopamine in rewarding or reinforcing, and opioids in pleasure-seeking with disparate brain regions such as ventromedial prefrontal cortex (vmPFC), insula and ventral striatum in terms of connectivity and function. So far, evidences from blood, urine and CSF analyses allude elevated levels of norepinephrine in PG subjects versus non-PG men. Methods: fMRI results indicated differential amygdala activation to yohimbine (as an alpha-2 adrenergic antagonist) in PG men. Also, decreased serotonin levels in CSF have been reported in PG cases that clarify different behavioral responses in them to meta-chlorophenylpiperazine, as a partial agonist of serotonin receptors (5HT1 and 5HT2). Moreover, increased dopamine is observed in PG cases during gambling or card-game tasks as D2-like dopamine antagonists are associated with increased gambling motivations in PG, and ventral striatal discrepancies in D2 receptor affinity was observed in PG. Results: Also, glutamatergic antagonists such as memantine with constant doses showed to help enhance cognitive flexibility in PG cases. On the other hand, simulated gambling task in fMRI studies indicated a decreased vmPFC and striatal activation in PG, though differential thalamocortical and temporal gyrus patterns has been observed. In other cognitive processes such as cue exposure, loss chasing, near miss identification and risktaking paradigms in fMRI studies, a remarkable recruitment of gain circuitry in insula and ventral striatum as well as rostral anterior cingulate was less activated in PG cases compared to non-PG ones. Conclusion: However, all of these findings have not led to a unanimously accepted and efficacious approach to treat PG in a long-term with possible withdrawal of risk-taking symptoms. Further research in future is needed to mainly focus on the integrative genetics investigation of multiple SNPs or CNVs of a specific gene, and enhanced neuroimaging modalities such as diffusion tensor imaging and PET to portray the shortcomings in the way of realizing next-generation biomarkers for .PG, and a more auspicious approach to control PG

کلمات کلیدی:

Pathological gambling; Neurotransmission; fMRI; Brain regions

لینک ثابت مقاله در پایگاه سیویلیکا:

https://civilica.com/doc/974971



