EPV0390

The Effects of a Chatbot-Based Interpretation Bias Modification on Early Adulthood Depression

J. Lee* and D. Lee

Kangwon National University, Chuncheon, Korea, Republic Of
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1105

Introduction: Depression, particularly in early adulthood, presents a significant mental health challenge with far-reaching implications. Innovative approaches to address and alleviate depressive symptoms are of paramount importance in this context. One such approach involves the utilization of technology, specifically chatbot-based programs, to target specific cognitive biases associated with depression.
Objectives: The central objective is to empirically examine whether this program can effectively influence depressive mood and negative cognition in individuals grappling with depressive symptoms.
Methods: To ascertain the program's efficacy, participants were divided into two groups: the CBM-I group (n=20), which underwent interpretation bias modification training, and the Mood Check group(n=20), which served as a control and engaged in a simple mood-checking exercise. A battery of psychological measures was employed, including assessments of depression, interpretation bias, suicidal ideation, resilience, and attention control.
Results: Analysis results showed that the CBM-I group had a significant reduction in depression (PHQ-9, CES-D) compared to the Mood Check group in the post-measurement. Moreover, resilience (CD-RISC) and attention control (ACQ) significantly improved in the CBM-I group.
Conclusions: This research serves as a stepping stone towards a deeper understanding of how chatbot-based interventions can contribute to the management of early adulthood depression, offering new perspectives and possibilities in the realm of mental health support and treatment.

Disclosure of Interest: None Declared

EPV0391

Obesity and depression: Review on common neurobiological mechanisms and identification of potential drug targets.

J. R. Lopez[1,2]* and R. Zaghi-Lara[3]

[1]Posgrado de psiquiatría, Universidad de San Carlos de Guatemala; [2]President, Asociación Psiquiaátrica de Guatemala and [3]CIGua, Comunidad de Investigación de Guatemala, Guatemala, Guatemala
*Corresponding author.
doi: 10.1192/j.eurpsy.2024.1106

Introduction: This review aimed to identify common pathophysiological mechanisms that exist between depression and obesity, as well as pharmacological strategies used in clinical trials and animal models. It is necessary to carry out larger studies that integrate the multiple neurobiological processes of these phenomena and the search for therapeutic targets that affect these pathways.

Objectives: Conduct a literature review on the common neurobiological aspects that exist between depression and obesity. Compare pharmacological and therapeutic strategies in the management of depressive patients by means of common neurobiological mechanisms.
Methods: We used the Pubmed search engine to search for the keywords: ((depression and obesity) OR (common pathways for depression and obesity) OR (therapeutic targets for depression and obesity) OR (neurobiology of depression and obesity) OR (treatments for depression and obesity))Once the predefined screening was carried out, 68 studies were identified. 54 articles were left for review and analysis. At the end of the review, 25 studies were discarded, including 29 studies with relevance to the objectives described in the study. These articles were selected when they provided information with adequate, concrete and specific reasoning towards the scientific and methodological elements of the review.
Results:
Table 1: Findings related to the NEGR1 gene and its involvement in processes associated with alterations associated with depression and obesity.
Table 2: Findings of inflammation associated with depression and obesity in clinical trials and animal models.
Image:

AUTHORS	STUDY SUBJECTS	FINDS
Lee et al. (2012)	Mouse deficient NEGR1 constitutive line and mutagenic ENU line with loss-of-function mutation (Negr1-I87N)	Expression analyses confirmed the brain-specific distribution of NEGR1, strong expression in the hypothalamus. In vitro assays demonstrated NEGR1 promoted cell adhesion and neurite growth of hypothalamic neurons.
Carboni et al. (2020)	FSL Rats	Antidepressant treatments were able to influence the Negr1-Fgfr2 pathway and support the hypothesis that Negr1-mediated modulation of neuronal plasticity is activated by antidepressant treatment.

Image 2:

AUTHORS	OBJECTIVE OF THE STUDY	SUBJECT STUDIO	FINDS
Visser et al. (1999)	Assessment of overweight and obesity associated with low-grade systemic inflammation as measured by C-reactive protein level	16,616 non-pregnant men and women aged 17 years or older.	Elevated CRP level of 0.22 mg/dL or higher and a clinically elevated stricter CRP level of more than 1.00 mg/dL. A higher BMI is associated with higher concentrations of CRP, suggesting a state of low-grade systemic inflammation in overweight and obese individuals.
Capuron et al. (2008)	Relationship between MS and depressive symptoms in a population of 323 male twins, using the Beck Depression Inventory (BDI) and analyzing plasma CRP and IL-6 levels	323 male twins, with and without MS and free of symptomatic cardiovascular disease, drawn from the Vietnam Era Twin Registry.	MS is associated with a higher depressive symptomatology characterized mainly by neurovegetative traits. Inflammation is a determinant of depressive symptoms in people with MS.
You et al. (2011)	Detecting depression-like behavior in a rat animal model induced swelling in the spleen and brain by CMS	Wistar Rats	High expression of pro-inflammatory cytokines IL-1β, TNF-α and IL-6, and low expression of anti-inflammatory cytokines TGF-β and IL-10. Decreased BDNF mRNA in hippocampus and hypothalamus of stressed rats.
Soto et al. (2018)	To evaluate behaviors and insulin action in the brains of diet-induced obesity (OID) mice with and without antibiotic treatment.	Male mice C57BL/6J,	OID mice exhibit insulin resistance in the brain, depression-like behaviors, and anxiety, and these improve with antibiotic treatment.

Conclusions: Alterations in the NEGR1 gene, inflammatory markers, HPA axis and microbiota demonstrate multiple pathophysiological mechanisms in the clinical pictures associated with obesity and depression.
Infliximab, pioglitazone, ondansentron, BVT.2733 and palmitoylethanolamide showed anti-inflammatory regulatory effects with reduction in depressive symptoms and multiple anti-inflammatory markers.
Animal models for obesity and depression present ample and reliable evidence regarding the use of drugs that direct their