Obesidade e Depressão: Duas Faces da Mesma Moeda?

Obesity and Depression: Two Sides of The Same Coin?

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PALAVRAS-CHAVE: Depressão; Excesso de Peso; Obesidade; Saúde Mental

KEYWORDS: Depression; Mental Health; Obesity; Overweight

Dear Editor,

Obesity and depression are both common medical conditions with major public health implications and high worldwide prevalence, increasing in recent decades.¹⁻⁴ Obesity and depression frequently co-occur and epidemiological evidence strongly supports a bidirectional relationship between these conditions, with shared biological mechanisms and interventional opportunities.^{1,3} A better understanding of this comorbidity should be a priority, deserving our clinical attention.

Literature suggests that both obesity and depression are disorders of stress with dysregulation of the stress system.¹ Shared mechanisms involve genes, hypothalamic-pituitary-adrenal (HPA) axis dysregulation, inflammation, oxidative stress, endocrine and metabolic derangement and gut microbiota alterations.^{1,2} These biological pathways may act in two, non-mutually exclusive, ways: as common underlying mechanisms influencing the liability to both conditions, or as mediating mechanisms in causal links between them.³

The phenotypic relationship between obesity and depression is rooted in partially overlapping genetic bases, involving genes such as *NEGR1* – neuronal growth regulator-1 – modulating synaptic plasticity in brain areas

essential for mood and appetite regulation.³ Hyperactivation of HPA axis represents a potentially relevant mechanism connecting obesity and depression, determining hypercortisolism that, in the long-term, may lead to neuronal damage and loss in limbic regions vulnerable to stress and associated with depression, like hippocampus and amygdala.^{1,3} Depressive states and obesity are associated with chronic low-grade inflammation, with increased pro-inflammatory cytokines.^{1,3} Peripheral immune activation, as occurs in obesity, can be translated via humoral, neural and cellular pathways into brain inflammation, with impact in depression pathophysiological processes, such as monoaminergic neurotransmission alteration.³ Findings support a decrease in cytokines upon resolution of depressive symptoms, inhibitory effect on cytokine production of antidepressants, and persistent upregulation of pro-inflammatory cytokines in treatment resistant depression. Endocrine/metabolic dysregulation includes leptin, melanocortin and insulin alterations.3 Gut microbiota, a complex active network that directly affects host metabolic phenotypes, may trigger inflammation and depression-related brain processes, creating a gut microbiota-brain axis potentially impacting on mood states through its metabolites.^{2,3}

Recebido/Received: 20/01/2022 - Aceite/Accepted: 12/08/2022 - Publicado online/Published online: 14/09/2022 - Publicado/Published: 30/09/2022 © Autor (es) (ou seu (s) empregador (es)) e Gazeta Médica 2022. Reutilização permitida de acordo com CC BY-NC. Nenhuma reutilização comercial. © Author(s) (or their employer(s)) and Gazeta Médica 2022. Re-use permitted under CC BY-NC. No commercial re-use.

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Obesity and depression are thus interconnected through a vicious, mutually reinforcing cycle of adverse physiological adaptations.³ Reducing obesity-induced neuroinflammation may conduct to beneficial effects on depression.²

In patients with co-occurring obesity and depression, the inclusion of dietary interventions, with calorie-restricted diets, along with psychopharmacologic and/or psychotherapeutic treatments, may result in improvement of depression symptoms, in relation to immunoendocrine and psychosocial mechanisms. When treating this subgroup of patients, we must focus on multidisciplinary interventions and remember that obesity and depression appear to be two sides of the same coin.

RESPONSABILIDADES ÉTICAS

CONFLITOS DE INTERESSE: Os autores declaram não possuir conflitos de interesse.

SUPORTE FINANCEIRO: O presente trabalho não foi suportado por nenhum subsídio ou bolsa.

PROVENIÊNCIA E REVISÃO POR PARES: Não comissionado; revisão externa por pares.

ETHICAL DISCLOSURES

CONFLICTS OF INTEREST: The authors have no conflicts of interest to declare.

FINANCIAL SUPPORT: This work has not received any contribution grant or scholarship.

PROVENANCE AND PEER REVIEW: Not commissioned; externally peer reviewed.

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