Lecture

Toward Cellular and Metabolism Hallmarks in Mental Disorders: Focus on the Link Between Obesity, Depression and Alzheimer's Disease

Vera-Salazar, Roberto

Neuroscience Researcher at CAREPUC Clinical Fellow Research Exercise Physiology. Associate Professor on Neurophysiology and Neuroscience at FACIMED-USACH, Chile

Abstract

Obesity condition in humans triggers a decline of several neuronal functions including cognitive performance, an increase of brain inflammation, decrease of neuronal plasticity and metabolism of glucose, all of which has been related to neuritis architecture changes such a decrease in the formation of new spines. In this regard, obesity has been related to increasing in Alzheimer´s disease (AD) hallmarks and depression behavior, which at turns, implicates changes in brain structure including decreased total and gray matter volumes, increased white matter lesions, and reduced white matter integrity. Our laboratory had demonstrated, in a murine model, that the induction of obesity, produces a decrease in cognitive performance and an increase of several histopathological markers of AD in brain, including the rise of inflammation, oxidative stress, and astrocyte activation. At the same time, we have described using a transgenic model mouse of AD, and a protocol to induce like-behavior depression in mice, that several histopathological markers of AD could be improved by voluntary running which is able to partially recover the adult neurogenesis in AD and depression, respectively. Altogether, our result suggests a link between obesity, AD, and depression and an eventual therapeutic tool based on exercise to these mental health pathologies. The goal of this study is to show how some cellular signaling including Wnt and cytokines pathways, could be linked to AD, depression, and obesity in a rational and causal manner in human mental health contexts.

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