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Nutritional intervention in chronic pain: an innovative way of targeting central nervous system sensitization?

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Abstract

Introduction: Few treatment programs for chronic pain nowadays take a dietary pattern or adipose status into account.

Areas covered: An important role of neuroinflammation in chronic pain is now well established, at least in part due to increased central nervous system glial activation. Based on preclinical studies, it is postulated that the interaction between nutrition and central sensitization is mediated via bidirectional gut-brain interactions. This model of diet-induced neuroinflammation and consequent central sensitization generates a rationale for developing innovative treatments for patients with chronic pain. Methods: An umbrella approach to cover the authors' expert opinion within an evidence-based viewpoint.

Expert opinion: A low-saturated fat and low-added sugar dietary pattern potentially decreases oxidative stress, preventing Toll-like receptor activation and subsequent glial activation. A low-saturated fat and low-added sugar diet might also prevent afferent vagal nerve fibers sensing the pro-inflammatory mediators that come along with a high-(saturated) fat or energy-dense dietary pattern, thereby preventing them to signal peripheral inflammatory status to the brain. In addition, the gut microbiota produces polyamines, which hold the capacity to excite N-methyl-D-aspartate receptors, an essential component of the central nervous system sensitization. Hence, a diet reducing polyamine production by the gut microbiota requires exploration as a therapeutic target for cancer-related and non-cancer chronic pain.

Keywords: Diet; brain; gut microbiota; lifestyle; microglia; pain; sensitization.

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