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The role of IgG hypersensitivity in the pathogenesis and therapy of depressive disorders.

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Abstract

Depressive episodes are associated not only with changes in neurotransmission in the central nervous system, but also may lead to structural changes in the brain through neuroendocrine, inflammatory, and immunological mechanisms. The aim of this article is to present a new hypothesis connecting the inflammatory theory of depression with IgG food hypersensitivity and leaky gut syndrome. This new potential pathway that may mediate the pathogenesis of depression implies the existence of subsequent developmental stages. Overproduction of zonulin triggered, for example, by gliadin through activation of the epidermal growth factor receptor and protease-activated receptor causes loosening of the tight junction barrier and an increase in permeability of the gut wall ('leaky gut'). This results in a process allowing larger molecules that would normally stay in the gut to cross into the bloodstream and in the induction of IgG-dependent food sensitivity. This condition causes an increased immune response and consequently induces the release of proinflammatory cytokines, which in turn may lead to the development of depressive symptoms. It seems advisable to assess the intestinal permeability using as a marker, for example, zonulin and specific IgG concentrations against selected nutritional components in patients with depression. In the case of increased IgG concentrations, the implementation of an elimination-rotation diet may prove to be an effective method of reducing inflammation. This new paradigm in the pathogenesis of depressive disorders linking leaky gut, IgG-dependent food sensitivity, inflammation, and depression is promising, but still needs further studies to confirm this theory.