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Role of monocyte mRNA in Mood Disorders

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Accumulating evidence supports the view that deregulation of the immune system represents an important vulnerability factor for mood disorders and that this may be a common pathophysiological mechanism that underlies the development and progression of these disorders, at least in a subset of patients. Furthermore, mood disorders are also often accompanied by chronic medical conditions related to immune dysfunction such as autoimmune diseases, diabetes and atherosclerosis. The reviewed data suggest that if immune biomarkers exist for such immune abnormality they may be found in raised macrophage/monocyte inflammatory activation patterns (monocytosis, high inflammatory gene expression, raised GRβ/GRα ratio, and high levels of pro- and anti-inflammatory monocyte/macrophage derived cytokines in serum/plasma). This activation of the inflammatory response system may be suggestive for microglia activation as these cells are the macrophages of the brain, although evidence from human studies is still lacking. However, animal models of mood disorders suggest that such monocyte/microglia activation could be seen as the result of a combination of genetic predisposition and environmental factors. It has been proposed that the abnormal immune activation state of these cells is reached after an aberrant development/maturation from precursors. In the case of microglia, this in turn may lead to impaired neuronal development. This developmental/maturation abnormality may render the cells more vulnerable to subsequent stressors such as inflammatory stimuli thereby leading to increased incidence of mood disorders.

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