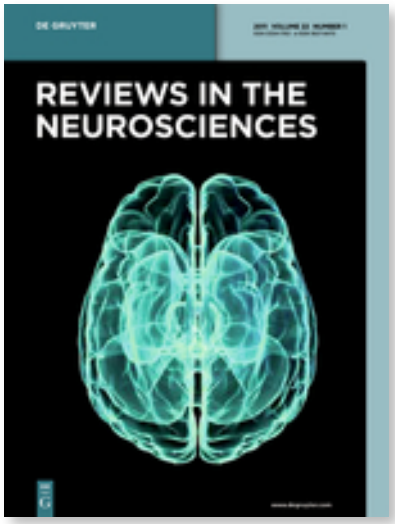

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Review on the Acute Phase Response in Major Depression

Michael Maes,

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A Review on the Acute Phase Response in Major Depression

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SYNOPSIS

There is some evidence that major depression is characterized by systemic immune activation with involvement of phagocytic cells, T cell activation, B cell proliferation and increased autoantibody production. This paper reviews that major depression may be accompanied by higher concentrations of positive and lower concentrations of negative acute phase proteins (APPs). The most prominent abnormalities of APPs in major depression are increased haptoglobin (Hp) plasma levels. The latter are significantly and positively correlated with interleukin (IL)-6 production, various indices of systemic immune activation (e.g. monocytosis, neutrophilia, T cell activation) and with the vegetative symptoms of depression (e.g. anorexia, weight loss, psychomotor retardation, sleep disorders, anergy). Major depression is characterized by an altered distribution of Hp phenotypes and genes suggesting that genetic variation on chromosome 16 may be associated with this illness. It is concluded that increased production of IL-6 and IL-1 in major depression may underlie both immune activation and the "acute" phase response in that illness, and that disorders in Hp may be related to the pathophysiology and pathogenesis of major depression.

KEY WORDS

depression, immune activation, acute phase proteins, interleukin-6 and -1, Hp phenotype, acute phase response, pathophysiology

1. INTRODUCTION

There is now some evidence that major depression is characterized by an *in vivo* immune response with involvement of phagocytic, T and B cells, exopeptidase enzymes, autoantibody and prostaglandin production. A brief synopsis of this evidence follows. Major depression is characterized by: (a) a moderate leukocytosis which is determined by neutrophilia and monocytosis /54,65/; (b) significantly higher antinuclear /10,24,50/ and antiphospholipid (i.e. anticardiolipin, antiphosphatidylserine and antithromboplastin) autoantibody /50,56/ titers; (c) T cell activation as indicated by a significantly increased number and percentage of activated T lymphocytes, i.e. $CD25^+$ (IL-2 receptor bearing cells) and $HLA\text{-}DR^+$ T cells /54,64/, significantly higher circulating levels of soluble interleukin-2-receptors/50/; (d) a significantly higher T helper / T suppressor cell (CD4/CD8) ratio /9,62/, which appears to be determined by increased absolute numbers and percentages of T helper cells, partly caused by an upregulation of $CD4^+CD45RO^+$ T memory cells, and by a reduction in $CD8^+CD57^-$ T suppressor cells /54,62/; (e) B cell proliferation as indicated by a higher number or percentage of various B cell subsets (i.e. $HLA\text{-}DR^+$, $CD19^+$, and $CD21^+$ B cells) /54,60/; (f) lower serum dipeptidyl peptidase IV activity, a phenomenon that is also observed in inflammatory and autoimmune disorders /53/; (g) increased prostaglandin (PGE_2) levels in peripheral blood /4/; and (h) a significantly increased production of IL-1β and IL-6 in culture supernatant of stimulated peripheral blood mononuclear cells /49,59/.

IL-1β and IL-6 are pleiotropic cytokines, which are considered to be major immune and inflammatory mediators /17/ that synergize strongly in T and B cell differentiation or proliferation /12,17,31,99/, induction of

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