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Tryptophan metabolism and immunogenetics in major depression: A role for interferon-γ gene

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ABSTRACT

The tryptophan metabolism and immune activation play a role in pathophysiology of major depressive disorders. The pro-inflammatory cytokine interferon-γ transcriptionally induces the indoleamine 2,3-dioxygenase enzyme that degrades the tryptophan and thus induces serotonin depletion. The polymorphism of certain cytokine genes was reported to be associated with major depression. We investigated the association between interferon-γ (IFNγ) gene CA repeat polymorphism, the profile of serotonin and tryptophan pathway metabolites and clinical parameters in 125 depressed patients and 93 healthy controls. Compared to controls, serum tryptophan and 5-hydroxyindoleacetic acid (5HIAA) concentrations in the patients were significantly lower and serum kynurenine concentrations were significantly higher at baseline ($p < 0.0001$). The presence of IFNγ CA repeat allele 2 homozygous has significant association with higher kynurenine concentrations in controls ($F = 4.47$, $p = 0.038$) as well as in patients ($F = 3.79$, $p = 0.045$). The existence of interferon-γ CA repeat allele 2 (homo- or heterozygous) showed significant association with increase of tryptophan breakdown over time during the study period ($F = 6.0$, $p = 0.019$). The results indicated the association between IFNγ CA repeat allele 2, tryptophan metabolism and the effect of medication.