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CHANGES IN THE IMMUNE SYSTEM AND GENE EXPRESSION IN BIPOLAR DISORDER

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The etiology of bipolar disorder is thought to involve multiple genes and environmental factors. Recently the immune system has been implicated in the pathogenesis. Various abnormalities indicate the presence of an activated inflammatory response system. In this presentation evidence will be presented on:

1. A higher susceptibility for autoimmune diseases (thyroiditis, gastritis, type 1 diabetes), not only in patients but also in first degree relatives independent from mood disturbances.
2. An inflammatory gene-expression signature comprising of 19 pro-inflammatory genes in monocytes, the monocyte (and its descendent cells) being important in the activation of the inflammatory response. The inflammatory gene-expression profile was also found in the monocytes of bipolar offspring, especially in those developing a mood disorder (prognostic value of the test?).
3. Common environmental factors (infection, stress and dietary components?) as the factors causing the inflammatory monocyte gene-expression signature (evidenced in a twin study).
4. A general T cell activation not only linked to the trait of the disorder, but also to the state of the disorder (i.e. with mania) and in part due to genetic factors (evidenced in a twin study). Also T cell activation is not linked to monocyte activation.
5. A poor T regulator activation which is genetically determined and correlates with the presence of above-described autoimmune diseases.

In sum, there is clear evidence for an activated inflammatory response system in bipolar disorder, yet different components are separately activated in a complex fashion linked to the phenotype of the disorder and involving both genes and environmental factors.

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