Cytokines and Depression: A neurochemical hypothesis

Propositions

Up to 40% of HCV infected patients treated with IFN-alpha will develop major depression.

A new psychometric scale is needed to measure depression in the IFN-alpha treated HCV patients since physical symptoms are more prevalent in those patients than in endogenous depressed patients.

Early treatment of depression with SSRI in HCV patients treated with IFN-alpha therapy allows continuation of the treatment.

SSRI have anti-inflammatory effects.

Interferon-alpha (IFN-alpha) induced depression is caused by a IFN-alpha induced depletion of tryptophan, the precursor of serotonin.

Interferon-alpha (IFN-alpha) induced depressive symptoms is related to the development of neurotoxicity by tryptophan toxic catabolites.

The activation of the inflammatory response system is triggered in major depression.

Disorders in the HPA axis and in serotonin in conjunction with immune activation are specific to major depression.

The increase in depressive symptoms during IFNalpha-ribaverin treatment may correspond to less viral clearance.

In HCV patients treated with IFN-alpha, there are significant differences in cytokine patterns between responders and non-responders to IFN-alpha treatment.

The "Fight Club" philosophy applied to HCV patients. It hurts but it's necessary!