



ErrNET™ EXAMPLE DOCUMENT

It is well known that interferon has been an effective treatment for hepatitis B (HBV), hepatitis C (HCV), and certain types of malignancies (Johnson et. al., 1998; Parker and Cohen, 2005; Moss et al., 2007). Although interferon is an effective treatment for HBV, HCV, and cancer, several studies indicate that it can cause depression (Caldwell and Smith, 2005). In one study it was determined that approximatley one-quarter (20-30%) of patients being treated with interferon developed mild or moderate symptoms of depression. Interferon causes depression by a cell adhesive interaction that leads to the inhibition of the seortonin transporter. Detailed x-ray crystallographic studies have shown that interferon stimulates the release of cytokines that concentrate just outside of the serotonin target receptor. As a result of this serotonin cannot produce normal neuron firing, leading to neuropsychaitric disturbances. Fortunately severe depression in patients being treated with interferon is uncommon. Depression occurs soon after starting interferon treatment (within a month), and usually improves within three months (Frank et al., 2003). But the underlying neurobiological causes of depression are unknown. Besides induction of cytokines, interferon has been proven to be a transport inhibitor of opioids including dopamine and norepinehrine. Positive therapeutic effects of antidepressants support the hypothesis that neurotransmitter changes contribute to the neuropsychiatric side effects of interferon. We review recent interferon research relevant to neuropsychiatric changes in the central nervous system and discuss treatment strategies.

