and stress, due to their anti-inflammatory and neuroprotective properties.

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98. Antidepressants from different classes reduce inflammation in human hippocampal stem cells

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Inflammation has been implicated as an important process in the pathogenesis of depression. The ability of antidepressants to suppress inflammation has been demonstrated in a variety of animal cell lines and human peripheral blood samples. Here we investigate the anti-inflammatory effect of antidepressants from different chemical classes in human hippocampal stem cells. Inflammation was induced by incubation with IL-1b for 24 h and inflammatory response was quantified by measurement of IL-6 secreted into the supernatant by means of ELISA. Three antidepressants from different chemical classes – moclobemide, agomelatine and venlafaxine all demonstrated a dose-dependent suppression of the inflammatory response to IL-1b upon co-incubation. Venlafaxine reduced IL-6 detected by 26% ($p < 0.01$) at 1 μM and 22% ($p < 0.05$) at 10μM; agomelatine reduced IL-6 by 31% ($p < 0.05$) at 1 μM and 29% ($p < 0.05$) at 10 μM. Moclobemide showed a trend towards decreasing IL-6 by 20% and 26% at doses of 1 and 10 μM, respectively. Preliminary data suggests that sertraline has no effect on the production of IL-6. Further preliminary data suggests that gene expression of IL-1 and IL-6 does not change with antidepressant co-incubation at 1, 12 or 24 h between suggesting antidepressants exert their effects through post-transcriptional means by, for example, decreasing mRNA stability or modulation of translation. Overall, our results add to the body of evidence suggesting that antidepressants have anti-inflammatory actions.

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99. Plasma markers of inflammation are elevated in intermittent explosive disorder and as a function of aggression in human subjects

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Behavior can influence immune function, inflammatory activity, and psychological functioning; these relationships are often bi-directional. Aggressive behavior is associated with elevations in inflammatory mediators in animals, but few studies have examined these connections in humans. The present study explored relationships between aggressive behavior and serum levels of interleukin-6 (IL-6), soluble interleukin-1B receptor type II (IL1BrII), and C-reactive protein (CRP) in patients with Intermittent Explosive Disorder (IED), controls with Axis I and/or Axis II, disorders (Psychiatric Controls: PC), and subjects with no evidence of psychopathology (Healthy Controls: HC). Inflammatory markers were highly correlated with aggressive behavior, and impulsivity and aggression were highly related in all subjects ($p < .05$). Each of the three inflammatory markers was correlated with life history of aggression (LHA) for all groups ($ps < .05$). Among the psychiatric (PC and IED) subjects, higher inflammatory markers were associated with life history of suicidal attempts although further analysis showed that this was accounted for by LHA. Finally, IED subjects had significantly elevated levels of all three inflammatory markers compared to PC and HC ($ps < .05$). These results suggest a relationship between aggressive behavior and inflammatory activity which is especially notable in patients with IED compared to PC and HC. Additional work is necessary to elucidate the significance of these findings for not only health and immunity but also for the management and treatment of IED.

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100. Leptin deficiency in maltreated children: Implications for psychoneuroimmunology

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Background: Childhood maltreatment is linked to multiple immunological abnormalities. Experimental research in animal models showed that stressful experiences in early life may also be associated with impaired leptin response to physiological stimuli, such as adiposity and inflammation. In turn, leptin deficiency is known to be associated with impaired adaptive immune system functioning and dysregulation in inflammation resolution. We therefore sough to translate these findings to human research and test if childhood maltreatment predicts leptin deficiency. *Methods:* We assessed leptin and C-reactive protein in dried blood spots and anthropometric measures from 170 12-year-old participants of the Environmental Risk (E-Risk) Study. Childhood maltreatment was prospectively assessed through repeated interviews with mothers in the first decade of study participants' life. *Results:* Maltreated children showed a trend towards lower leptin levels than nonmaltreated children. Furthermore, maltreated children showed reduced leptin response to increasing inflammation and adiposity levels. These findings could not be explained by key potential confounders or pre-existing abnormalities in energy homeostasis. *Conclusion:* Childhood maltreatment is associated with leptin deficiency, which could contribute to previously reported immune abnormalities.

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101. Asthma-associated IgE mechanisms are regulated by the beta-2-adrenergic receptor on human B cells

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Asthma affects 300 million people worldwide. Treatment involves the use of inhalers containing agonists that stimulate the beta-2-adrenergic receptor (b2AR) on lung smooth muscle cells. Norepinephrine, which is released in elevated amounts during a stress response, also stimulates the b2AR. Stimulation of the b2AR on a B cell responding to allergen results in an increase in IgE, which exacerbates bronchoconstriction and counteracts therapeutic benefit and worsens stress-related asthma effects. Our goal is to block the negative b2AR-induced effect on the B cell while retaining the positive effect on bronchodilation. Previously, we showed that when the B cell-associated b2AR is stimulated during antigen activation, hematopoietic protein tyrosine phosphatase (HePTP) is phosphorylated in a PKA-dependent manner to release bound p38 MAPK for phosphorylation, and to subsequently increase IgE transcription. Deletion of either b2AR or norepinephrine in mice results in a decrease in antigen-induced IgE and lung inflammation. It is unknown if HePTP functions similarly in human B cells. We found that anti-CD40/IL-4-primed human B cells exposed to a b2AR agonist