# Interleukin-1β subverts hippocampal synaptic plasticity by promoting long-term potentiation in experimental multiple sclerosis

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Approximately half of all patients with multiple sclerosis (MS) experience cognitive dysfunction including learning and memory impairment. Recent studies suggest that hippocampal pathology is involved, although the synaptic mechanisms underlying these deficits still remain unclear. Herewe provide evidence that in the hippocampus of experimental autoimmune encephalomyelitis (EAE), an established mouse model of MS, long-term potentiation (LTP) is favored over long-term depression (LTD) during the acute phase of disease.Remarkably, this effect is dependent on suppression of GABAergic activity mediated by IL-1β released from infiltrating lymphocytes or activated microglia. We hypothesizethat LTP-like phenomena occurring during immune attacks on the brain might underlierecovery of function,as well as cognitive deficits and excitotoxic neurodegeneration. Having identified that pro-inflammatory cytokines such as IL-1β can influence synaptic function in early MS, it is hoped that new treatments targeted toward preventing synaptic pathology can be developed.