Interleukin-2

Interleukin-2 (IL-2) is an interleukin, a type of cytokine signaling molecule in the immune system. It is a 15,5 - 16 kDa protein that regulates the activities of white blood cells (leukocytes, often lymphocytes) that are responsible for immunity. IL-2 is part of the body's natural response to microbial infection, and in discriminating between foreign (“non-self”) and “self”. IL-2 mediates its effects by binding to IL-2 receptors, which are expressed by lymphocytes.

Interleukin-2(IL-2): has shown minimal activity in brain Mets, and trials have usually excluded patients with untreated or uncontrolled brain Mets due to risk of cerebral edema and hemorrhage from the capillary leak.

Based on previous finding that IL-2 strongly elevated the expression of the checkpoint molecule Tim-3 in Treg cells, we examined the effect of IL-2 in the function of Treg cells from IA patients. External IL-2 significantly improved the proliferation of Treg cells, increased the expression of CTLA-4 and LAG-3, and enhanced Treg-mediated suppression of conventional T cell (Tconv) proliferation. Importantly, compared to the Tim-3- Treg cells, the Tim-3+ Treg cells presented comparable proliferation capacity, but significantly greater expressions of CTLA-4 and LAG-3 and significantly higher capacity to suppress Tconv proliferation. In addition, blocking Tim-3 abrogated IL-2-mediated enhancement of Tim-3+ Treg cells. We then investigated the IL-2 level in IA patients, and found that although IA patients and healthy controls presented similar serum IL-2 concentration, the concentrations of IL-1β and TNF-α were significantly higher in IA patients than in healthy controls, signaling a relative reduction in IL-2 abundance. Together, we found that IL-2 could significantly enhance the function of Treg cells from IA patients in a Tim-3-dependent manner [1].

1)
Zhang HF, Liang GB, Zhao MG, Zhao GF, Luo YH. Regulatory T cells demonstrate significantly increased functions following stimulation with IL-2 in a Tim-3-dependent manner in intracranial aneurysms. Int Immunopharmacol. 2018 Oct 23;65:342-347. doi: 10.1016/j.intimp.2018.10.029. [Epub ahead of print] PubMed PMID: 30366277.