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CORRECTION

Correction to: The pathogenicity of Th17 cells in autoimmune diseases

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Unfortunately, an error occurred in the following passus of the article. The word "receptor" was missing in the sentence "Because T cells do not express GM-CSF receptor [41], GM-CSF affects non-T cells."

The corrected text is given below.

GM-CSF, a key pathogenic cytokine in autoimmune tissue inflammation

GM-CSF is recently highlighted as the pathogenic cytokine of Th17 cells. The role of GM-CSF in EAE model was first reported in 2001, in which blockade of GM-CSF showed resistance to the EAE induction, but the critical source of GM-CSF in immune cells was not investigated in detail [50]. There was the first report that among Th subsets infiltrating into the CNS after EAE induction, some of Th cells showed IL-17A+ GM-CSF+ double-positive producer [51]. The critical function of IL-23 signaling directing encephalitogenic Th17 cells has been reported to drive GM-CSF production, which causes local tissue inflammation [41, 52]. Because T cells do not express GM-CSF receptor [41], GM-CSF affects non-T cells. GM-CSF first acts on CNS-infiltrating myeloid cells such as dendritic cells (DCs), monocytes, and macrophages which in turn secrete pro-inflammatory cytokines such as IL-6 and IL-23, both of which upregulate IL-23R expression, amplifying IL-23-mediated pathogenic circuit to directly cause neurological pathogenicity and establishing local tissue inflammation by recruiting inflammatory macrophages in the CNS [51, 53]. GM-CSF also activates CCR2+ monocytes, monocyte-derived DCs, and microglia in the brain to produce IL-1β [54, 55]. Since microglia have a potential to produce IL-23, they could participate in the IL-23-IL-17 immune axis in Th17 cell–mediated tissue inflammation [56].

The original article has been corrected.

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