New findings link estrogen and T cell immune response to autoimmune inflammation

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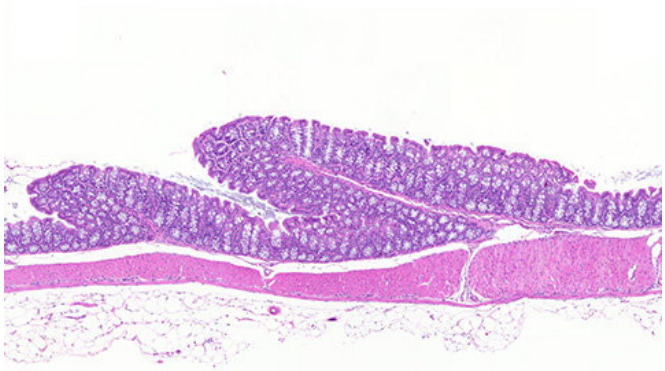

Histological image of colon sections from mice that received T cells from CD4-creERafl/fl mice. Credit: University of Turku

Women are more prone to the development of autoimmune diseases. The female hormone estrogen is likely to affect the immune system. A team of scientists from Turku Center for Biotechnology and University of Georgia reported new findings related to the involvement of estrogen hormone receptor in autoimmune diseases.

The incidence of autoimmune diseases such as MS, RA, and SLE is higher in women than in men. The estrogen hormone secreted in women may contribute to the pathogenesis of these diseases. A research team led by Docent Zhi Chen from Turku Center for Biotechnology of the University of Turku has collaborated with researchers from the University of Georgia, United States to address the long-standing issue of hormonal effect on autoimmune diseases.

Estrogen hormone shows its action on cells mostly through estrogen receptor alpha (ER?). Researchers from Turku generated mice with ER? protein specifically deleted in T cells.

"The eureka moment of our research is that in a mouse model of human inflammatory bowel disease, transfer of naive T helper cells from ER? deficient mice did not succumb to colitis, unlike transfer from their counterparts," Docent Zhi Chen says.

"Furthermore, using cutting-edge technique RNA sequencing approach combined with in vitro and in vivo experiments, we discovered that ER? regulates multiple aspects of T cell function, including T cell activation, proliferation and survival," Chen adds.

Regulatory T cells are group of T cells that help in preventing autoimmune diseases. The researchers found that ER? influences the function and differentiation of regulatory T cells.

More information: Imran Mohammad et al, Estrogen receptor ? contributes to T cell–mediated autoimmune inflammation by promoting T cell activation and proliferation, *Science Signaling* (2018). DOI: 10.1126/scisignal.aap9415

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