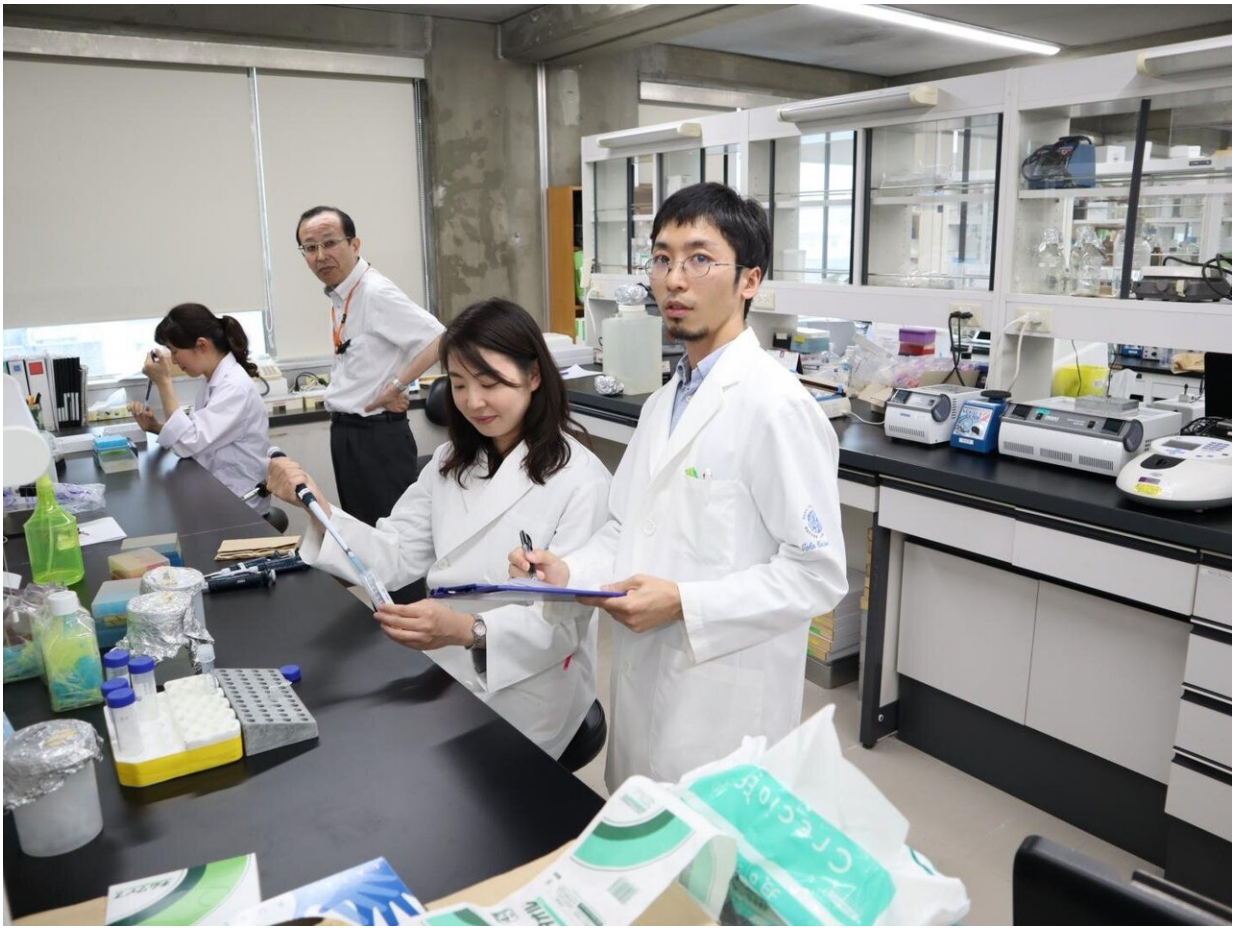
How to equip the brake of immunity

July 9 2019



Takaharu Katagiri, M.D., standing on the front and Hiroyasu Nakano,M.D.,Ph.D., Professor of the Toho University Medical School, standing on the back with other lab members in his lab. Credit: Toho University Medical School

The immune system is indispensable for defense against invading pathogens, but its aberrant activation may lead to autoimmune diseases. Regulatory T cells play a crucial role in preventing excess immune activation; mice without enough Treg function develop autoimmune disorders and are susceptible to immune diseases.

"Treg cells have been attracting much attention due to their unique suppressive function, but how Treg cells are generated in our bodies is not fully understood. In a mouse model for human ulcerative colitis, mice lacking JunB developed more severe symptoms due to reduction of Treg cell number. JunB-deficient T cells exhibited an impairment of IL-2 production and IL-2 signaling. In addition, injection of a high dose of IL-2 into JunB-deficient mice mitigated colitis by expansion of Treg cells," explained the lead author of the paper, Takaharu Katagiri, M.D., who is a graduate student at Toho University working to become a physician-scientist.

"We hope these findings will lead to the development of a novel strategy to treat inflammatory diseases by manipulating the function of JunB," said the senior authors of the study, Soh Yamazaki, Ph.D, Associate Professor, and Hiroyasu Nakano, M.D., Ph.D. Professor of the Toho University School of Medicine. Their finding was published online on July 8th 2019 in the journal *Mucosal Immunology*.

More information: Takaharu Katagiri et al, JunB plays a crucial role in development of regulatory T cells by promoting IL-2 signaling, *Mucosal Immunology* (2019). DOI: 10.1038/s41385-019-0182-0

Provided by Toho University

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