

Scientists discover endogenous dendritic cellderived interleukin-27 promotes tumor growth

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In a new report published in the *Journal of Leukocyte Biology*, scientists lay the groundwork for the development of novel tumor therapies that may help rid the body of cancer by inhibiting the recruitment of a specific suppressive immune cell type called "regulatory T-cells." The approach described in the report shows that an immune molecule, called interleukin-27, promotes the recruitment of regulatory T-cells. This suggests that by stopping IL-27's immunosuppressive function, cancer therapies can more effectively activate other T-cells to attack and destroy cancer tumors.

"Our study not only provides a new insight into the effects of interleukin-27 in regulatory T-cell biology, but also greatly improves our understanding of the physiological functions of interleukin-27, especially in tumor immunology," said Siyuan Xia, a researcher involved in the work from the State Key Laboratory of Medicinal Chemical Biology, College of Life Sciences, Nankai University, Tianjin, China. "We hope our study could shed new light on developing novel interventional therapies by targeting regulatory T-cells in cancer patients."

Scientists made this discovery by using mice deficient in a specific subunit of interleukin-27 called p28. They compared the tumor infiltrating lymphocytes between interleukin-27 p28 deficient mice and normal mice, and through their experiments, found a significant decrease of regulatory T cells, and other key immune mediators in the



absence of interleukin-27 p28.

"Suppressive and regulatory pathways in the immune system are incredibly important for normal health and preventing autoimmunity," said John Wherry, Ph.D., Deputy Editor of the *Journal of Leukocyte Biology*. "However, these pathways also get exploited by cancer to prevent immune responses leading to cancer progression. The current studies point to an important regulatory network centered on interleukin-27 that could be targeted to improve immunity to cancer in humans."

Provided by Federation of American Societies for Experimental Biology

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