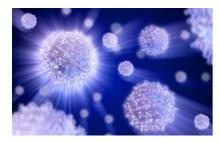


Autoimmune disease-causing cells can be controlled

A Yale University-led team of scientists has found that cells that drive certain serious, chronic autoimmune diseases can be redirected into the small intestine - and controlled there - potentially neutralising their propensity to trigger illness. The study appears in the advance online publication of *Nature*.



The researchers focused on the subset of T helper cells known as TH17, which secrete the cytokine interleukin-17. The inflammation-inducing TH17 cells play an important role in clearing infection, but in excess they drive chronic autoimmune disorders such as multiple sclerosis and rheumatoid arthritis.

The Yale team focused on normal mice as well as mice given human immune cells to mimic human clinical trials already underway of a CD3-specific antibody. The mice were given the CD3-specific antibody to begin the chain of biological events. Secretion of interleuken-17 induced the expression of molecules that attract T cells in the small intestine, which then facilitated the migration of TH17 cells to that part of the gastrointestinal system. Once in the small intestine, the TH17 cells acquired immuno-suppressive characteristics, and harmful cells were simultaneously eliminated from the body via the intestinal tract.

Unique properties

"Interestingly the TH17 cells were selectively redirected to the upper part of the small intestine. We believe that this part of the body has unique properties that allow it to confer immunological stability, and to quickly repair the damage caused by these initially inflammatory cells," according to lead author Richard Flavell, Ph.D., professor and chair of the Department of Immunobiology at Yale School of Medicine and a Howard Hughes Medical investigator.

The Yale team's findings suggest that it may be possible to avoid a life-threatening immune response caused by an overabundance of TH17 cells by redirecting them, and therefore controlling them. "There is currently no curative therapy available for autoimmune diseases. The key for a curative intervention might be to either eliminate or change the phenotype of these pathogenic T cells," said Flavell.

Other authors are Enric Esplugues of Yale, the German Rheumatism Research Centre and the Cluster of Excellence NeuroCure in Berlin; Samuel Huber of Yale and the I. Medizinische Klinik in Hamburg; Nicola Gagliani of Yale and the San Raffaele Diabetes Research Institute in Milan; Anja E. Hauser of the German Rheumatism Research Centre; Terrence Town of Cedars-Sinai Medical Centre and the David Geffen School of Medicine at UCLA; Yisong Y. Wan of the University of North Carolina; William O'Connor, Jr., Anthony Rongvaux and Kevan C. Herold, M.D., of Yale; Nico Van Rooijen of Vrije Universiteit in Amsterdam; Ann. M Haberman of Yale; Yoichiro Iwakura of University of Tokyo; Vijay K. Kuchroo of Brigham and Women's Hospital; Jay K. Kolls of LSU Health Sciences Centre; Jeffrey A. Bluestone of University of California San Francisco.

This study was supported by grants from Yale University, the National Multiple Sclerosis Society, a DFG (HU 1714/1-1) grant and a James Hudson Brown - Alexander B. Coxe Fellowship, and a Spanish Ministry of Science postdoctoral fellowship.

Source: Yale University

For more, visit: https://www.bizcommunity.com