Study ties recently discovered immune cell to disease

Deficits in a recently discovered immune cell's function may trigger a rare age-related auto-inflammatory disease — and perhaps far more common ones, too.

By Bruce Goldman

Stanford University School of Medicine researchers have unraveled the workings of an important type of immune cell whose existence was unknown just a few years ago.

Connie Weyand, MD

The scientists found that this cell type keeps a lid on immune response, preventing runaway inflammation. But it becomes rare and malfunction-prone in even healthy people's bodies as they get older. That could help to explain why our immune systems go increasingly haywire with advancing age. The researchers identified the primary cause of these cells' malfunction and linked it to an auto-inflammatory disorder, giant cell arteritis. They suspect this connection may hold for some far more common age-related conditions, too.

The findings, described in a study published April 18 in the Journal of Clinical Investigation, suggest possible new approaches to restoring function in these cells.

Just as the immune system's assault brigades must expand and become warlike when confronting a pathogen or incipient tumor, they must contract and become peaceful afterward, lest they harm healthy tissues, said the study's senior author, Cornelia Weyand, MD, professor and chair of immunology and rheumatology. First authorship is shared by postdoctoral scholar Zhenke Wen, MD, PhD, and visiting scholar Yasuhiro Shimojima, MD, PhD.

"Fortunately, the immune system has built-in brakes," said Weyand. "We call them regulatory T cells, or Tregs."

When a pathogen invades the body or a cancerous cell emerges or a vaccine dose is administered, the immune system ramps up, producing antibodies and attacking suspected infected or tumorous cells, and secreting copious signaling substances that spur further attack-mode action.

If it weren't for Tregs, this chain reaction might go unchecked, resulting in chronic inflammation, said Weyand. That's what begins to happen in many people as they grow older. As we age, our immune response tends to grow both hyperactive and unfocused, like a car with lousy brakes, a distracted driver and a brick on the gas pedal. "The aging immune system becomes less focused — less capable of defending against cancers and infections or responding robustly to vaccinations — and much more inflammatory," Weyand said.

Tregs have long been known to exist. But until recently, the only ones known belonged to a category of immune cells called CD4 T cells. These cells have earned their nickname as "helper T cells" by participating in the immune response's expansion, as opposed to contraction, phase. But CD4 Tregs suppress the activation and proliferation of helper T cells by secreting anti-inflammatory substances, for example, or by soaking up growth factors.

For the full story: http://med.stanford.edu/news/all-news/2016/04/study-ties-recently-discovered-immune-cell-to-disease.html
For the journal article: https://www.ncbi.nlm.nih.gov/pubmed/27088800

Nanoscattering Patterning and Endothelial Function

Ngan Huang, MD

Using real time live-cell imaging, the Huang laboratory, tracked the alignment, migration trajectories, proliferation, and anti-inflammatory behavior of endothelial cells when cultured on parallel-aligned or randomly oriented nanofibrillar films.

The study provided new insights into how endothelial cells respond to opposing cues derived from nanotopography and mechanical shear force. This work has implications in the design of polymeric conduits and bioengineered tissues.

Authors of the study also include: Karina H. Nakayama, Vinay N. Surya, Monica Gole, Travis Walker, Weiguang Yang, Edwina S. Lai, Maggie Ostrowski, Gerald G. Fuller, Alexander R. Dunn, and Ngan F. Huang. *Nano Lett*. 2016 Jan 13;16(1):410-9. For the Journal Article: http://www.ncbi.nlm.nih.gov/pubmed/26670737

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Cathy Hutton

Ingrid Ibarra, PhD

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