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Dental researchers find some immune cells change to prolong inflammation

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Researchers at Case Western Reserve University School of Dental Medicine have unraveled one of the mysteries of how a small group of immune cells work: That some inflammation-fighting immune cells may actually convert into cells that trigger disease.

Their findings, recently reported in the journal *Pathogens*, could lead to advances in fighting diseases, said the project's lead researcher Pushpa Pandiyan, an assistant professor at the dental school.

The cells at work

A type of white blood cell, called T-cells, is one of the body's critical disease fighters. Regulatory immune cells, called "Tregs," direct T-cells and control unwanted immune reactions that cause inflammation. They are known to produce only anti-inflammatory proteins to keep inflammation caused by disease in check.

But using mouse models, the researchers studied how the body fights off a common oral fungus that causes thrush. They found that these harmful invaders activate a mechanism in Tregs that could transform the inflammation-fighting cells into cells that allow the disease to flourish.

The study

When the immune system functions normally, disease-fighting T-cells

produce inflammatory secretions—proteins that can cause symptoms, such as soreness or swelling at the infected site. This process is evident, for example, when a cut or glands swell from the infection's inflammatory reaction.

Once the invader is gone, the disease-fighting cells—with help from Treg cells—normally shut down those proteins to control long-term inflammation.

But the researchers found that, during oral thrush, yeast sugars on the surface of the disease-causing fungus act as a binding agent and can activate a small population of Treg cells to make inflammatory proteins themselves. (The researchers are calling this novel subset of malfunctioning cells Treg-17 cells).

"An excess of these malfunctioning cells can lead to the inflammatory disease process instead of stopping it," she said.

Other binding agents normally found in the body may create these cells and contribute to continued inflammation, the researchers concluded.

Other researchers have reported the presence of these cells in many human inflammation conditions, such as psoriasis, periodontitis and arthritis. Until now, however, the mechanisms of how these cells developed were not completely understood, Pandiyan said.

The implications

The findings will help researchers understand the origin of cells they suspect may keep the disease active or, at a minimum, don't battle inflammation. Pandiyan believes the knowledge could lead to new ways to fight diseases, such as:

- Using the converting Tregs (Treg-17) to identify chronic inflammation, including oral inflammation.
- Using the persistence of Treg-17 cells to indicate an excessive amount of the inflammatory proteins.
- Using the presence of the binding agent that triggers the cell's conversion as a point to use medicines to block its connection to Tregs.

Future studies will investigate whether these cells are actually perpetrating inflammation.

More information: The study, "TLR-2 Signaling Promotes IL-17A Production in CD4+CD25+Foxp3+ Regulatory Cells during Oropharyngeal Candidiasis," was recently reported in *Pathogens*.

Provided by Case Western Reserve University