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# Dental researcher demonstrates how T cells cause inflammation during infections

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Case Western Reserve University dental researcher Pushpa Pandiyan has discovered a new way to model how infection-fighting T cells cause inflammation in mice.

The hope is that the discovery can lead to new therapies or drugs that jump-start weakened or poorly functioning immune systems, said Pandiyan, an assistant professor at Case Western Reserve School of Dental Medicine.

Pandiyan believes the process could lead to identifying and testing new drugs to replace antifungal medicines that have become ineffective as the fungi develop a resistance to them.

Pandiyan's findings are explained and demonstrated in the *Journal of Visualized Experiments* video and print article, "Th17 inflammation model of oropharyngeal candidiasis in immunodeficient mice."

The research advances Pandiyan's previous work on isolating different types of oral T cells for study. T cells are a type of white blood cell that is critical to the body's immune system.

In the newest research, she used T cells and injected them into mice genetically engineered with no immunity to test how the cells function when fighting a common thrush-like yeast infection found in the mouth, called *Candida albicans*. When the infection fighting cells are not controlled properly, they caused inflammation.

According to Pandiyan, about 60 percent of the population has the fungus, but a healthy immune system keeps it under control.

In humans with weak immune system, the fungal growth appears as a white coating on the tongue. Individuals with the infection report a painful burning sensation in the mouth. As the infection spreads, it causes inflammation of the mouth area, tongue and gums. Left untreated, it can spread to the throat and the food pipe.

The infection becomes a particular health problem for people with the HIV/AIDS infection, cancer patients with immune systems weakened by chemotherapy or those born with no immune defenses.

In her study, Pandiyan was specifically interested in how a type of T cells that secrete a cytokine IL-17a (T helper 17, or Th17 cells), and T regulatory cells (Tregs) controlled the fungal infection and inflammation, respectively.

"Although Th17 cells are required for antifungal immunity, uncontrolled Th17 cells have been implicated with such illnesses as multiple sclerosis, lupus, psoriasis, cancers and irritable bowel disease," she said.

## The process and findings

The immunodeficient mice were infected with the fungus and injected with Th17 cells.

One group of mice was also injected with Tregs, which are the main regulators of autoimmunity and critical to the immune system functioning properly.

Researchers then tracked how the cells functioned in controlling the infection.

The group that received both Th17 and Tregs fared better in stopping the infection and thriving during inflammation. Conversely, the mice that did not receive Tregs lost weight and began to waste away.

The researchers also found the immune system doesn't work well when the Th17 cells malfunction without appropriate control. "They can set into action a series of immune responses that develop into inflammation and greater health issues," Pandiyan said.

Pandiyan and her team provide visual instructions about lab techniques and special handling of the immunodeficient mice so other T-cell researchers can duplicate the process.

While Pandiyan studied Th17's role in fighting the yeast infection, she said other researchers could use the method to study Th17 cell functions in other areas of the body.

Case Western Reserve dental school researchers Aaron Weinberg, associate dean and chair of the Department of Biological Science, and Natarajan Bhaskaran, a research associate in biological sciences, contributed to the study.

Provided by Case Western Reserve University