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Researchers uncover cellular mechanism responsible for chronic inflammation, type 2 diabetes

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Researchers from Boston University School of Medicine (BUSM) have demonstrated that certain T cells require input from monocytes in order to maintain their pro-inflammatory response in people with type 2 diabetes (T2D). The study also showed, for the first time, how a loss in homeostasis in this group of T cells most likely promotes chronic inflammation associated with T2D.

Barbara Nikolajczyk, PhD, an associate professor of microbiology and medicine at BUSM, is the senior author of the study, which is currently featured in an online edition of the *Journal of Immunology*.

T2D is a chronic inflammatory disease in which the body has high levels of glucose in the blood due to the lack of insulin or the body's inability to use insulin efficiently. The incidence of T2D continues to rise at alarming rates in both children and adults in the United States.

Previous research done in mice has shown that T cells play a critical role in the development of insulin resistance in response to a high fat diet, often leading to T2D. Additional findings indicate that T cells exhibit a pro-inflammatory response more often than an anti-inflammatory response.

Working with human T cells, the team observed that in order for T cells to exhibit the pro-inflammatory response, they required constant

interaction with monocytes, indicating that monocytes play an indirect role in chronic inflammation and T2D.

While it is not known what the homeostatic balance levels are between pro-inflammatory and anti-inflammatory T cells, this study indicates the need to restore a balance in order to halt chronic inflammation and T2D.

"The true importance of our observations is the indication that altering balance among immune system cells could be a fundamentally novel treatment for T2D-associated inflammation and perhaps insulin resistance," said Nikolajczyk.

Provided by Boston University Medical Center