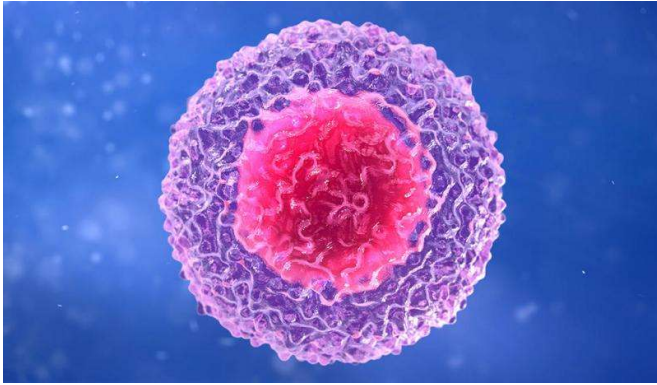


Study describes key RNA epigenetic marker's role in immune system

11 August 2017



A T cell is a type of white blood cell that fails to differentiate in mice lacking the m6A genetic marker. Credit: Yale University

which could have a profound impact on the research field, they said.

The finding provides new insight into this genetic marker's role in development and human health. It also points to the potential for developing drugs to target m6A to alleviate [autoimmune diseases](#), said first author and immunobiologist Huabing Li.

More information: Hua-Bing Li et al. m6A mRNA methylation controls T cell homeostasis by targeting the IL-7/STAT5/SOCS pathways, *Nature* (2017). [DOI: 10.1038/nature23450](https://doi.org/10.1038/nature23450)

Provided by Yale University

The white blood cells known as T cells regulate our body's response to foreign substances—our adaptive immune response. In a new study, Yale scientists have learned how changes in a recently discovered RNA epigenetic marker regulate T cells and the immune response. Their finding could lead to new approaches to treating autoimmune diseases.

The Yale-led research team focused on an important genetic marker, m6A, which modifies RNA. Prior to this study, it was known that m6A affected RNA and stem cells, but its role in biology was not understood. To investigate, the researchers deleted one of the genes that produce m6A in T cells, and tested m6A-deficient mice using various mice disease models.

The researchers found that the m6A-deficient T cells lost the ability to differentiate, or further develop into specialized [immune cells](#); thus the cells were unable to cause autoimmune disease. The authors further revealed detailed molecular pathways that undermine T cell differentiation,

APA citation: Study describes key RNA epigenetic marker's role in immune system (2017, August 11) retrieved 16 August 2017 from <https://medicalxpress.com/news/2017-08-key-rna-epigenetic-marker-role.html>

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