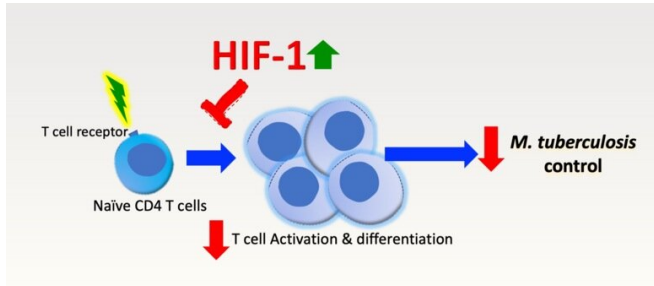


# Oxygen responses of T cells alter protection against tuberculosis

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T cell activation and differentiation. Credit: Karolinska Institutet

In a new publication in *Nature Communications*, Martin Rottenberg and Ruining Liu, professor and Ph.D. student at the Department of Microbiology, Tumor and Cell Biology, explain how T cell protection against tuberculosis is controlled by their oxygen responses.

Ten million individuals fell ill and 1.5 million died of tuberculosis (TB) in 2021, caused by [infection](#) with the intracellular *Mycobacterium tuberculosis* bacteria. Proper CD4 T cell responses are critical for the control of *M. tuberculosis* infection by activating intracellular bacterial killing.

The hypoxia-inducible factors (HIF-1 and HIF-2) control T cell metabolism as well as activation and differentiation in response to hypoxia or during inflammation.

"We showed that genetically modified [mice](#), in which HIF-1 expression T [cells](#) was stabilized by [genetic manipulation](#), were highly susceptible to the infection with *M. tuberculosis* and did not respond to vaccination. CD4 T cells from these mice were profoundly weakened in their early responses to mycobacteria-specific antigens," says Martin Rottenberg. "By impairing and or controlling HIF-1 stabilization in T cells, responses to vaccines

and protection against infections might be improved."

The studies were carried out on mouse models of *M. tuberculosis* infection. The mice used were genetically modified so that they lacked or overexpressed HIF-1 in T cells.

"The infection with *M. tuberculosis*, and the immune responses it generates in man, is fairly mimicked in the mouse infection. Our next step is to identify the molecular targets HIF-1 in T cells that account for their impaired activation, which could be targeted for improving T cell responses," Martin Rottenberg concludes.

**More information:** Ruining Liu et al, HIF-1 stabilization in T cells hampers the control of *Mycobacterium tuberculosis* infection, *Nature Communications* (2022). DOI: [10.1038/s41467-022-32639-9](https://doi.org/10.1038/s41467-022-32639-9)

Provided by Karolinska Institutet

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