

Immune cell study prompts rethink on how to tackle infections

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Fresh insights into how immune cells are regulated could signal a new approach to tackling infections.

Researchers have found that <u>oxygen levels</u> in the <u>body</u> can profoundly change the way <u>immune cells</u> behave.

Scientists say the findings pave the way for new therapies that target the <u>immune response</u> to infection, with the potential to boost existing antibiotic treatments.

The research in mice found that bacterial infections have vastly different outcomes depending on levels of oxygen in the body when the infection takes hold.

If oxygen levels are low when infection strikes, the immune system launches a massive overreaction. A fatal illness ensues even though the bacteria have been cleared from the body.

Exposure to low oxygen before infection, however, seems to protect the body from illness without compromising its ability to fight off bacteria.

Researchers say the effects are caused by changes to the way the cells use energy, which reprogrammes their response.

If human cells are found to behave in the same way, tweaking their oxygen sensing mechanisms could hold the key to tackling infections,



the team says.

The findings are particularly relevant for people with chronic lung conditions, such as emphysema. They often have low levels of oxygen in their body and are more vulnerable to infections.

The study was carried out by scientists in the Medical Research Council Centre for Inflammation Research at the University of Edinburgh and is published in the journal *Science Immunology*.

Dr Sarah Walmsley, of the MRC Centre for Inflammation Research at the University of Edinburgh, said: "We are excited by our observation that oxygen levels can regulate <u>immune</u> cell responses to <u>infection</u>. Targeting these pathways could have the potential to improve outcomes from infections where oxygen is limited."

More information: A. A. Roger Thompson et al, Hypoxia determines survival outcomes of bacterial infection through HIF-1 α -dependent reprogramming of leukocyte metabolism, *Science Immunology* (2017). DOI: 10.1126/sciimmunol.aal2861

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