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TB harnesses part of immune defence system to cause infection, study finds

Scientists from the University of Cape Town's (UCT) Institute of Infectious Disease and Molecular Medicine (IDM) have contributed to an international study that shed new light on why humans and animals are so susceptible to contracting tuberculosis (TB) – and it involves the bacteria harnessing part of the immune system meant to protect against infection.

Despite more than 100 years of research, TB remains one of the deadliest bacterial infections in humans, resulting in 1.5 million deaths each year.

TB is caused by the bacterium *Mycobacterium tuberculosis* (MTB). Infection occurs when the bacteria are inhaled and taken up by specialist immune cells, such as macrophages, which recognise MTB and trigger a range of cellular and immune responses. These responses are mediated by receptors – molecules on the surface of immune cells that can recognise microbes. One such receptor is Dectin-1, which is best known for its role in anti-fungal immunity.

However, MTB has evolved a range of strategies to overcome these defences, manipulating host cells to enable survival and replication. The study found that MTB survives within its host cells by targeting Dectin-1. Published in *Science Immunology*, the finding gives new insight into how TB takes hold to cause disease.

"This research is a true international collaboration, with each institution bringing a distinct area of expertise. It's a fantastic example of the global partnerships required to tackle some of the greatest health challenges of our time," said Associate Professor Claire Hoving, associate member of the IDM and the Wellcome Trust Centre for Infectious Disease Research in Africa at UCT. She is also the local director of the AFRICA Unit for Medical Mycology, and an associate professor in the Division of Immunology, Department of Pathology.

In work supported by Wellcome and the Medical Research Council, the team showed that instead of protecting against infection, as occurs during fungal infection, MTB utilises the responses triggered by Dectin-1 to drive its own survival. When this Dectin-1 pathway was absent, both human and mouse cells could control MTB infection. Indeed, mice lacking Dectin-1 were much more resistant to MTB infection.

The team, made up of UCT, the University of Exeter, Osaka University, the Francis Crick Institute and others, also discovered that the bacteria produce a unique molecule alpha-glucan that targets Dectin-1 to induce these determinantal immune cell responses.

Dr Max Gutierrez, of the Francis Crick Institute, said: "TB is a major killer worldwide, yet we still know very little about how it is so effective at causing infections, in both humans and in animals. Our discovery of a new mechanism by which Mycobacterium tuberculosis is able to subvert host immunity is a key step in understanding the basis of susceptibility to TB."

Professor Sho Yamasaki, Osaka University, said: "Our results are surprising, because Dectin-1 is a key part of the body's defence system to protect against fungal infections, yet we've shown it's detrimental for MTB infections and actually promotes bacterial survival."

Professor Gordon Brown, of the University of Exeter's MRC Centre for Medical Mycology, added: "This discovery is the first step – and opens the door to exciting new prospects including, for example, if we could knock out this receptor in cattle to make them more resistant to infection."

The study is titled "Mycobacterial α-glucans hijack Dectin-1 to facilitate intracellular bacterial survival".

ENDS

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