Tuberculosis tricks the body's immune system to allow it to spread

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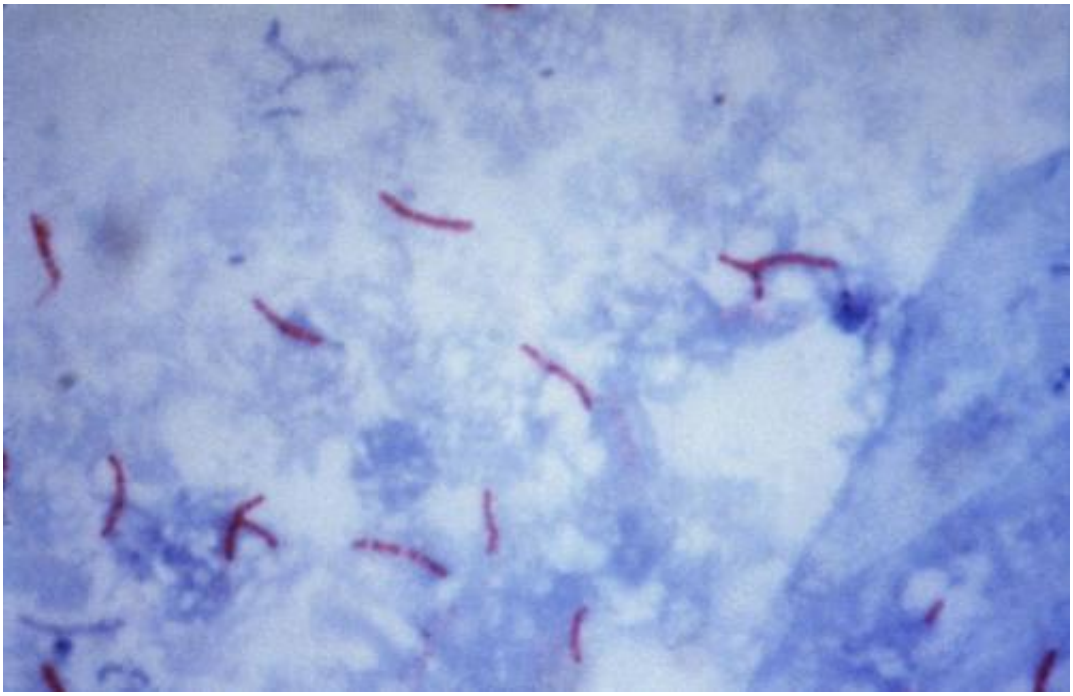

This photomicrograph reveals Mycobacterium tuberculosis bacteria using acid-fast Ziehl-Neelsen stain; Magnified 1000 X. The acid-fast stains depend on the ability of mycobacteria to retain dye when treated with mineral acid or an acid-alcohol solution such as the Ziehl-Neelsen, or the Kinyoun stains that are carbolfuchsin methods specific for M. tuberculosis. Credit: public domain

Tuberculosis (TB) tricks the immune system into attacking the body's lung tissue so the bacteria are allowed to spread to other people, new research from the University of Southampton suggests.

The concept, published in *Trends in Immunology*, proposes that current ideas about how tuberculosis develops in patients may be incomplete and that, in fact, infection causes autoimmunity, where the immune system reacts incorrectly to its own tissue.

Tuberculosis kills more people than any other infectious disease, and the causative bacterium, *Mycobacterium tuberculosis*, is becoming increasingly resistant to antibiotics used to treat the infection.

The Southampton research team conducted a review of published studies and found evidence suggesting that an autoimmunity process develops in TB.

Professor Paul Elkington, of the University of Southampton, who led the project, said "We are not disputing that the immune system mainly targets the bacteria to fight it off, but we are suggesting that there is more to the story.

"It seems that TB tricks the immune system into damaging our own lung tissue, which therefore makes the person highly infectious through coughing and the TB then spreads by aerosol droplets to other individuals.

"There is also a group of patients who develop a range of symptoms, such as eye inflammation, joint inflammation and skin rashes, that are not explained by current TB disease concepts. These symptoms are usually associated with diseases like rheumatoid arthritis and Crohn's disease, which led us to believe autoimmunity plays a key role in the TB disease process."

Professor Elkington highlights that more research is needed to investigate the hypothesis but if it is proved, the discovery could have major implications for the design of new vaccines and drug treatments.

The Southampton team are now undertaking a programme of work to investigate this new concept. Their approach is to combine the study of cells isolated from TB-infected patients with micro-engineering in 3D in the laboratory to investigate how TB damages the lungs.

More information: Tuberculosis: an Infection-1 Initiated Autoimmune Disease? *Trends in Immunology*, 2016.

Provided by University of Southampton