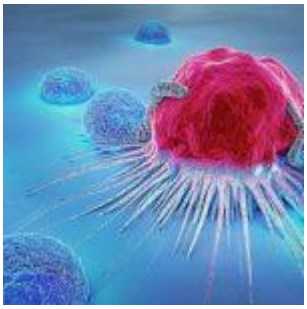
T-cell protection against pneumococcal disease



In a recent study, scientists demonstrated how a distinct subgroup of lung T regulatory cells provides resistance to bacteraemic pneumonia.

The most common cause of community-acquired pneumonia is Streptococcus pneumoniae (the pneumococcus), with a considerable percentage of cases progressing to bloodstream infection. One of the main unresolved concerns in pneumococcal research for decades has been why certain people are more prone to invasive illness and other people are more resistant.

A subpopulation of white blood cells in mice that give resistance to bacteraemic pneumonia was identified in this study. When pneumococci attack the lungs, these cells—which have been identified as TNFR2 expressing Tregs—are essential for maintaining and managing the host immune system's frontline responses. When the function of this particular subset of Tregs is compromised or absent, the immune response to infection becomes dysregulated, resulting in excessive and uncontrolled inflammation that damages tissue and allows bacteria to pass through compromised lung tissue barriers into the bloodstream, leading to sepsis, a serious and fatal condition.

Their research demonstrates that TNFR2-expressing Treg cells are extremely necessary for regulating lung inflammation and limiting pneumococcal translocation from the lung to blood,

which provides resistance to invasive illness. Yet, these cells are either missing or functionally compromised in vulnerable hosts, predisposing them to the onset of sepsis.

Journal article: Rong X, et al., 2023. TNFR2+ regulatory T cells protect against bacteremic pneumococcal pneumonia by suppressing IL-17A-producing γδ T cells in the lung. *Cell Reports*.

Summary by Stefan Botha