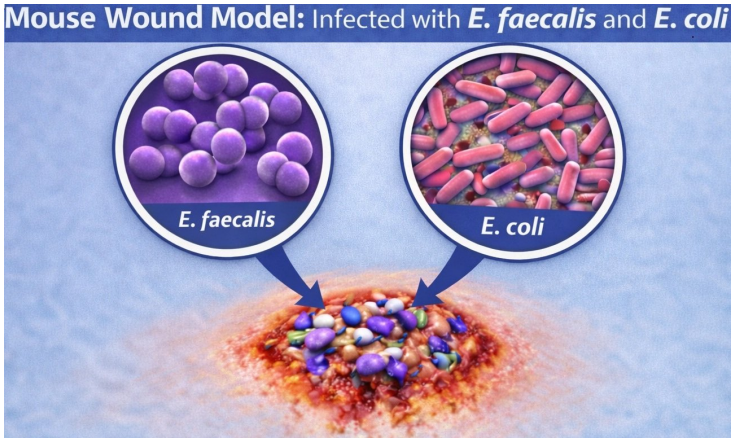
BioSpectrum
the business of Bio & Health Sciences
ASIA EDITION

Singapore researchers uncover bacterial immunity suppression in stubborn wound infections

18 March 2026 | News

The bacterium releases large amounts of lactic acid in the wound, which lowers pH, weakens immune cells and prevents them from fighting an infection



Researchers from the Singapore-MIT Alliance for Research and Technology (SMART) Antimicrobial Resistance (AMR) group, in collaboration with SCELSE at Nanyang Technological University (NTU Singapore), Massachusetts Institute of Technology (MIT), and University of Geneva (UNIGE), have uncovered how the bacterium Enterococcus faecalis (E. faecalis) disrupts immune responses to cause persistent wound infections.

The study reveals that *E. faecalis* releases lactic acid during infection, acidifying the wound environment. This suppresses macrophage activation—immune cells critical for clearing infections—by silencing the NF-kB alarm signal. Lactic acid enters macrophages via the MCT-1 transporter and binds to the GPR81 receptor, blocking immune signaling and preventing the cells from sending "danger" signals. This two-step mechanism allows E. faecalis to persist in wounds, creating conditions for multi-species infections that are harder to treat.

Using a mouse wound model, researchers demonstrated that E. faecalis strains unable to produce lactic acid were quickly cleared, with stronger immune responses observed. Infections involving both E. faecalis and Escherichia coli (E. coli) showed that lactic acid-driven immune suppression enabled E. coli to thrive, explaining why chronic wounds often involve multiple bacterial species.

"Chronic wound infections fail not because antibiotics are ineffective but because the immune system is 'switched off' at the site. E. faecalis floods wounds with lactic acid, lowering pH and muting the NF-kB alarm in macrophages," said Dr. Ronni da Silva, first author of the study.

This discovery highlights new treatment approaches targeting immune suppression rather than relying solely on antibiotics. Potential strategies include reducing wound acidity or blocking lactic acid's immune-dampening signals.

The research, supported by Singapore's National Research Foundation under the CREATE programme, paves the way for therapies to improve wound healing and reduce complications. Future work will validate findings in additional pathogens and

human wound samples before advancing to preclinical models.

Image Caption: A mouse wound infection model demonstrated that wounds infected with E. faecalis had dampened immunity, allowing E. faecalis to persist and even enabling co-infecting bacteria like E. coli to thrive. The model allowed researchers to study how lactic?acid?driven immune suppression promotes persistent, polymicrobial infections.