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UNIGE and NTU Singapore scientists find that silencing bacteria can worsen heart infections

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Is blocking bacterial communication always beneficial? A team from the University of Geneva (UNIGE) and Nanyang Technological University, Singapore (NTU Singapore) challenges a widely held assumption in infectious disease research.

By studying a bacterium responsible for a severe heart infection, the scientists show that disrupting bacterial communication can be associated with adverse clinical outcomes. Published in *Nature Communications*, these findings open the door to more targeted – and potentially more effective – therapeutic strategies against this type of infection.

Infectious endocarditis is a serious infection of the heart's inner lining, most often affecting the valves. It can be caused by several bacteria, including the particularly widespread Enterococcus faecalis.

These bacteria coordinate their collective behaviour through quorum sensing, a chemical communication system that enables them to gauge how many bacteria are nearby.

This process allows them to form dense clusters known as biofilms, which impair valve function and are highly resistant to antibiotics. As a result, infectious endocarditis is associated with high morbidity.

A team from NTU's SCELSE (Singapore Centre for Environmental Life Sciences and Engineering), a multidisciplinary biofilm and microbiome research centre, and UNIGE's Faculty of Medicine is challenging a widely held assumption in infectious disease research: that blocking bacterial communication is always beneficial.

While quorum-sensing inhibitors are actively explored as anti-virulence therapies, the study shows that suppressing bacterial signalling in cardiac infections may instead worsen the disease by promoting more aggressive forms.

Scientists found that when Enterococcus faecalis can no longer communicate with neighbouring bacteria, it forms larger, more resilient biofilms on heart valves, resulting in more severe clinical outcomes.

Blood flow silences bacterial communication

By combining devices that mimic blood flow with an animal model of cardiac infection, the team found that blood flow actively suppresses quorum sensing in the early stages of infection.

“On the surface of heart valves, bacteria are exposed to intense blood flow,” explains Dr Haris Antypas, Senior Research Fellow at SCELSE and lead author of the study. “This flow disrupts the chemical signals bacteria use to communicate, effectively shutting down quorum sensing.”

As the infection progresses, bacteria burrow deeper into the valve vegetation, where they are shielded from the bloodstream. At this stage, quorum sensing is normally activated, acting as a brake to limit excessive biofilm growth. Strikingly, bacteria that completely lack quorum sensing bypass this control. In animal models, these strains formed larger biofilms, showed greater antibiotic tolerance, and caused more severe disease.

The team attributed this effect to two key mechanisms: reduced production of bacterial proteases – enzymes that break down proteins – and a metabolic shift that allows the bacteria to use host nutrients more efficiently, fuelling persistent growth.

More unfavourable outcomes in affected individuals

The study also examined *E. faecalis* bacteria isolated from patients with infectious endocarditis in the United States and Switzerland. Nearly half of the clinical isolates lacked quorum sensing, and these cases were linked to longer-lasting bacteraemia, which is the presence of bacteria in the bloodstream despite active antibiotic treatment. “These are not rare mutants,” notes Dr Haris Antypas. “They are common in patients, and our data suggest they may actively contribute to poorer clinical outcomes.”

These findings challenge a widely held idea in infectious disease research: that blocking quorum sensing is always beneficial. “Our results show that in infectious endocarditis, inhibiting quorum sensing can actually harm the patient by promoting biofilm growth,” explains Kimberly Kline, full professor in the Department of Microbiology and Molecular Medicine at UNIGE's Faculty of Medicine, SCELSE Visiting Academic and senior author of the study.

“Understanding when and where bacterial communication helps or harms the patient will be essential for designing smarter therapies,” she added.

Giving an independent comment, Dr Matthaios Papadimitriou-Olivgeris, a senior physician and privat-docent at the Service of Infectious Diseases, Lausanne University Hospital (CHUV), commented on the findings of this paper, saying that it challenged “the prevailing paradigm that quorum-sensing inhibition is uniformly beneficial in bacterial infections”.

“In Enterococcus faecalis infective endocarditis, absence of the Enterococcus faecalis system regulator (Fsr) quorum-sensing system leads to enhanced biofilm formation, metabolic adaptation, and increased antibiotic tolerance, resulting in prolonged bacteraemia in patients, an effect that was also recapitulated in experimental strains with targeted fsr deletion,” Dr Papadimitriou-Olivgeris explained.

“While quorum-sensing inhibition is actively pursued as an anti-virulence strategy for several pathogens, these results underscore the need for caution when considering anti-quorum-sensing approaches in E. faecalis infective endocarditis, where such interventions may paradoxically worsen disease outcomes.”

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