Quorum sensing: Researchers examine bacteria communication

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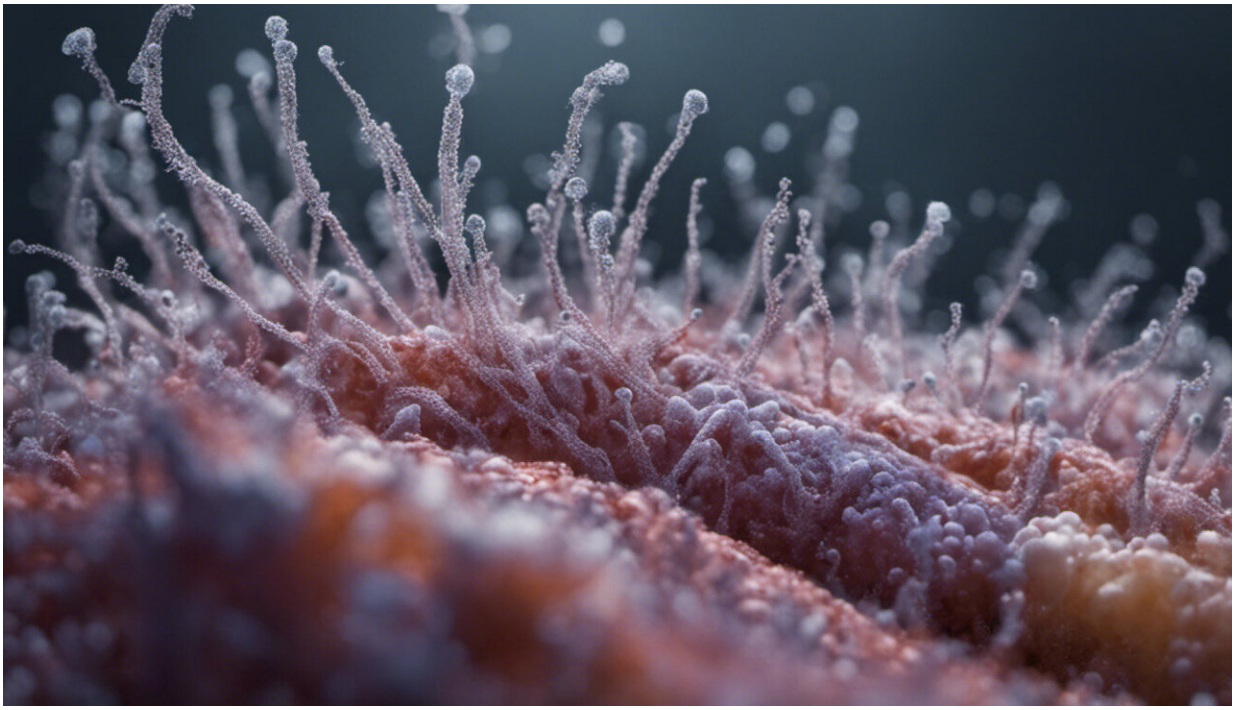

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European researchers at Linköping University in Sweden are showing how bacteria control processes in human cells through a process called quorum sensing. This phenomenon is where bacteria talk to each other via molecules they themselves produce and is an important process during their proliferation. The study, whose results were published in the

journal *PLOS Pathogens*, was made possible thanks to grants from the European Science foundation, TraPPs Euromembrane project, the Swedish Research Council, the King Gustaf V 80-Year Foundation, and the Faculty of Health Sciences, Linköping University.

According to their study, 'The human pathogen Pseudomonas aeruginosa and other bacteria communicate with each other using quorum sensing. This is important for their growth, virulence, motility and the formation of biofilms. Furthermore, eukaryotic cells "listen and respond" to QS signalling, but the exact mechanisms and receptors on mammalian cells have not been identified.'

If the body is injured and a wound is caused, a call goes out and more and more bacteria gather at the site of the attack. When the bacteria gather in sufficient numbers, they start acting like multicellular organisms. They can form biofilms, which are dense structures with the ability to resist both antibiotics and the body's immune defence system. At the same time, they become more aggressive and increase their mobility. All these changes are triggered when the communication molecules - short fatty acids with the designation AHL - fasten to receptors inside the bacterial cells; as a consequence, various genes are turned on and off.

AHL can wander freely through the cell membrane, not just in bacterial cells but also our own cells, which can be influenced to change their functions. In low concentrations, white blood cells, for example, can be more flexible and effective, but in high concentrations the opposite occurs, which weakens our immune defences and opens the door for progressive infections and inflammations.

The team from Linköping University is the first research group in the world to show how AHL can influence their host cells. According to their report, their study builds on their past research: 'We have

previously shown that N-acylhomoserine lactones (AHL) alter epithelial barrier functions and increase chemotaxis in human neutrophils.'

Now, using biochemical methods, the researchers have identified a protein, designated IQGAP, which they single out as the recipient of the bacteria's message, and something of a double agent. Elena Vikström, researcher in medical microbiology and the main author of the study, explains, 'The protein can both listen in on the bacteria's communication and change the functions in its host cells.'

Their laboratory studies were carried out on human epithelial cells from the intestines, which were mixed with AHL of the same type produced by Pseudomonas aeruginosa, a tough bacterium that causes illnesses in places like the lungs, intestines and eyes. With the help of mass spectrometry, they have been able to see which proteins bind AHL.

'We have proof that physical contact between bacteria and epithelial cells is not always required; the influence can happen at a distance,' Vikström says.

The team's discovery can open the door to new strategies for treatment where antibiotics cannot help. One possibility is designing molecules that bind to the receptor and block the signal path for the bacteria - something like putting a stick in a lock so the key won't go in. It's a strategy that could work with cystic fibrosis, for example, an illness where sticky mucus made of bacterial biofilm and large amounts of white blood cells is formed in the airways.

More information: Karlsson, T., et al. 'The Pseudomonas aeruginosa N-acylhomoserine lactone quorum sensing molecules target lQGAP1 and modulate epithelial cell migration', *PLOS Pathogens*, 2012.

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