

# How bacteria talk to each other and our cells

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Bacteria can talk to each other via molecules they themselves produce. The phenomenon is called quorum sensing, and is important when an infection propagates. Now, researchers at Linköping University in Sweden are showing how bacteria control processes in human cells the same way.

The results are being published in the journal [PLOS Pathogens](#) with Elena Vikström, researcher in Medical Microbiology, as the main author.

When an infection is signaled, more and more bacteria gather at the site of the attack – a wound, for example. When there are enough of them, they start acting like [multicellular organisms](#). They can form biofilms, dense structures with powers of resistance against 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 – bind to receptors inside the bacterial cells; as a consequence various genes are turned on and off.

AHL can migrate 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.

A team at Linköping University is the first research group in the world to show how AHL can influence their host cells. 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.

"The protein can both listen in on the bacteria's communication and change the functions in its host cells," Vikström says.

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:** The *Pseudomonas aeruginosa* N-acylhomoserine lactone quorum sensing molecules target IQGAP1 and modulate epithelial cell migration by T Karlsson, M V Turkina, O Yakymenko, K-E Magnusson and E Vikström. *PLOS Pathogens*, Vol 8 issue 10, October 2012. [www.plospathogens.org/article/info%3Adoi%2F10.1371%2Fjournal.ppat.1002953](http://www.plospathogens.org/article/info%3Adoi%2F10.1371%2Fjournal.ppat.1002953)

Provided by Linköping University

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