

Solution sought to 'sticky' staph situation

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Microbiology and Immunology professor David Heinrichs focuses on MRSA, the drug-resistant variety of staphylococcus aureus. Credit: Paul Mayne

David Heinrichs faces a potentially deadly adversary found on the skin of almost 30 per cent of the population – and the battle just got a whole lot stickier.

"Staphylococcus aureus is tricky in how it evades the immune system. It is a 'professional pathogen.' It knows how to cause disease and it does it very well," said the Microbiology and Immunology professor. "The overwhelming majority of micro-organisms don't do anything to us; they don't make us sick because we have good immune systems. But there are a few good at preventing being killed by the [immune system](#); [staphylococcus aureus](#) is one of them."

In a recent study, Heinrichs discovered some variants of [staph aureus](#) have the potential to cause more severe infections. These variants do so by over-expressing surface proteins that bind to our cells in the bloodstream – they simply 'stick' to us better than other variations.

That might seem like bad news if not for the further discovery these findings unlocked.

As a rule, prior exposure to staph aureus does not prevent subsequent infections. In other words, as opposed to many other infectious diseases, an immune response to this organism is not protective. This is why it has been exceedingly difficult to make a vaccine against staph aureus.

In his work, however, Heinrichs showed that pre-existing antibodies produced by our bodies to fight previous staph aureus infections can inhibit the 'sticking.' Therefore, prior exposure to staph aureus – in this case – may be beneficial to preventing serious infections should these hyper-virulent variants arise.

"We came across this almost by chance. We were making certain mutations in staphylococcus aureus and happened to cross this observation where the [bacteria](#) started sticking together when we grew them in blood. 'What's going on?' we asked. 'This is not supposed to happen like this,'" he said.

"It turned out that during our growth of the bacteria in the lab, they had picked up another mutation, which happens all the time. You don't see it unless you stress the bacteria, and when we did, that caused the mutation to occur, and what we saw was the bacteria sticking together."

Shown so far only in mouse models, the bacteria were sticking together so fervently, and in such big clumps, they were probably blocking the vasculature and causing a stroke in the mice.

Fortunately, for humans, because we staphylococcus aureus all the time, we get exposed to it from skin-to-skin contact and develop an immune response. That response, Heinrichs said, should actually be protective against this super virulent form of the bacteria. Tests, however, have not been done in humans.

Staph infections are caused by staphylococcus bacteria – germs commonly found on the skin, perineum or in the nose of even healthy individuals. Most of the time, these bacteria cause no problems or, if they do, result in minor skin infections treatable with antibiotics.

Staph infections can turn deadly, however, if the bacteria invade deeper into the body, entering the bloodstream, joints, bones, lungs or heart. A growing number of otherwise healthy people can develop life-threatening staph infections.

"Our skin is very good at protecting us. Your immune cells try to kill it. If they can't control it, you get an [infection](#). But it's when you have a breach in the skin that will cause a problem," Heinrichs said. "If you get a cut or abrasion and you have staphylococcus aureus on your [skin](#) and it gets underneath that, into the underlying areas, it can stick to and start to grow."

Heinrichs and his team focus their research on MRSA, the drug-resistant

variety of [staphylococcus aureus](#).

"We focus our research on the [molecular mechanisms](#) that allow the bacteria to grow in our body – what genes are needed, when do they get expressed, and what are they doing during the infection when they do get expressed," he said.

His paper, Stress-induced inactivation of the Staphylococcus aureus purine biosynthesis repressor leads to hypervirulence, was published this month in *Nature Communications*.

More information: Mariya I. Goncheva et al. Stress-induced inactivation of the Staphylococcus aureus purine biosynthesis repressor leads to hypervirulence, *Nature Communications* (2019). [DOI: 10.1038/s41467-019-08724-x](#)

Provided by University of Western Ontario

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