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Researchers closer to the super bug puzzle

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Infectious diseases specialists from Austin Health are working closely with Microbiologists from the University of Melbourne to understand how Staph is becoming resistant to all antibiotic therapies.

The treatment of serious infections caused by Staphylococcus aureus (Golden Staph) is complicated by the development of antibiotic resistance. Seriously ill patients, vulnerable to infections can be at additional risk if antimicrobial agents become less effective in fighting infections.

Published today in the journal PLoS Pathogens, a new piece has been added to the puzzle, making the picture clearer. By using whole genome DNA sequencing of strains obtained from patients during persistent blood stream infections, Dr Timothy Stinear and Associate Professor Ben Howden, senior research fellows from the Department of Microbiology and Immunology have discovered how Staph can make one small change to its DNA and then develop resistance to the last-line antibiotic, vancomycin.

"We have applied the latest genome sequencing technology to show that Staph can readily become vancomycin (antibiotic) resistant by acquiring a single mutation in its DNA. When the bacteria mutate, they are reprogramming themselves, changing their cell walls to resist the action of our antibiotics" said Dr Stinear.

Associate Professor Howden, who is also the head of Microbiology at Austin Health, is concerned by the implications of this discovery for

patients. "Worryingly, this mutation also makes Staph more resistant to another last-line antibiotic, daptomycin, even though this drug had never been used for treatment. These last-line therapies are more toxic and cause additional side-effects in already compromised patients." Associate Professor Howden said.

"This study highlights the high adaptability of Staph in the face of antimicrobial treatment and suggests we need to improve the way in which we use antibiotics to treat serious bacterial infections." he said.

Provided by University of Melbourne