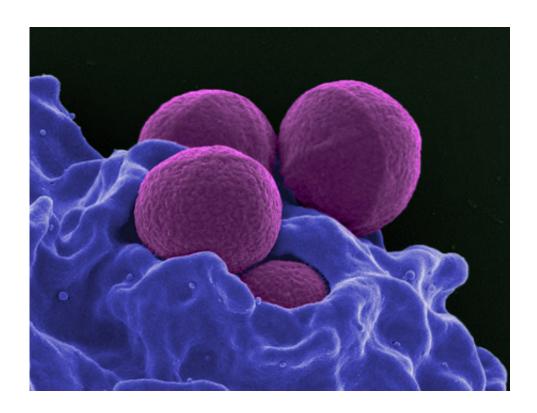


Genetic mutations help super bug become highly resistant to antibiotics

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A colorized scanning electron micrograph of MRSA. Credit: National Institute of Allergy and Infectious Diseases

Scientists from the University of Sheffield have found that genetic mutations in MRSA allow it to evolve and become more resistant to antibiotics such as penicillin.

The research, published in *PLoS Pathogens*, found that genetic mutations



in MRSA are allowing the bacteria to become highly resistant to <u>antibiotics</u> without reducing the bacteria's ability to cause disease.

Most clinical MRSA exhibits a low level of antibiotic resistance, due to the cells acquiring a <u>new gene</u> encoding a protein (MecA) that makes its cell wall, some strains can evolve high-level resistance and pose a serious threat.

Antibiotics such as penicillin and methicillin do not bind well to the new protein (MecA) meaning they cannot "kill" the bacteria. The next phase of this research is to understand how this protein works with other factors within the bacteria to allow a higher level of antibiotic resistance.

Findings from the research pave the way for greater understanding of the cause and evolution of antibiotic resistance, and will help researchers develop new treatments and drugs for MRSA.

Simon Foster, professor of molecular microbiology at the University and Principal Investigator of the research, said: "Antibiotics have been a mainstay of human healthcare for over 70 years, but the emergence of antimicrobial resistance is now a global catastrophe. In order to combat antimicrobial resistant organisms, we have to understand them. Our work uncovers the complex mechanisms that underpin resistance, giving insight into how we might tackle this global challenge."

Dr. Viralkumar Panchal, postdoctoral researcher at the University of Sheffield and leader of the research, said: "The research provides a new outlook into the process of evolution of resistance and reveals important details of how MRSA is so resistant. We can now exploit these findings to develop new cures."

More information: *PLoS Pathogens* (2020). <u>DOI:</u> 10.1371/journal.ppat.1008672



Provided by University of Sheffield

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