Research team identifies mutations key to antibiotic resistance

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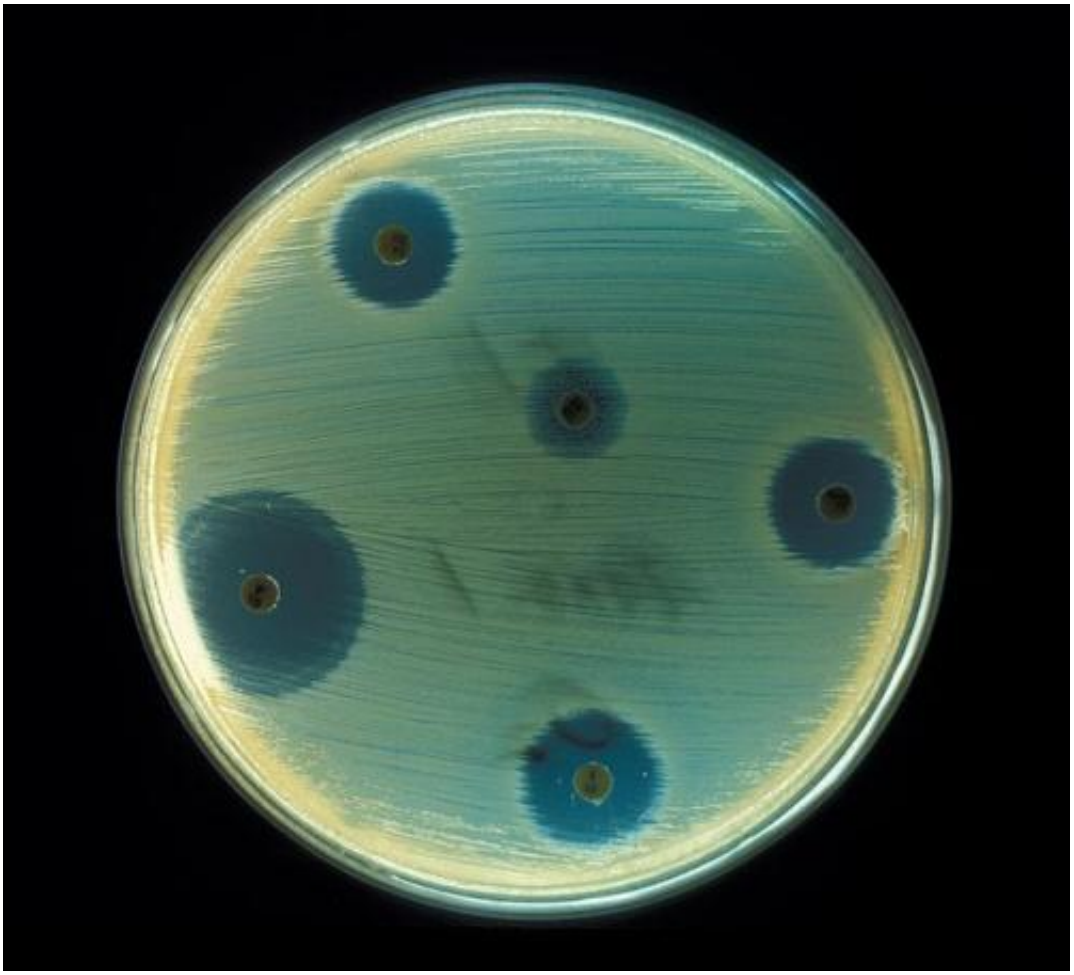

Staphylococcus aureus - Antibiotics Test plate. Credit: CDC

Two genetic mutations could be key to understanding how bacteria retain antibiotic resistance, according to a team led by University of Idaho

researchers.

How those mutations improve antibiotic resistance is the subject of a paper published this month in the journal *Nature Ecology & Evolution*. The paper, "Compensatory mutations improve general permissiveness to antibiotic resistance plasmids," was written in partnership with researchers from the University of Florida, the University of Washington and Point Loma Nazarene University.

Leading the UI team was Eva Top, professor in the Department of Biological Sciences in the College of Science, a researcher at UI's Institute for Bioinformatics and Evolutionary Studies (IBEST) and director of the Bioinformatics and Computational Biology graduate program. Other UI participants involved in the project were Wesley Loftie-Eaton, Jack Milstein, Samuel Hunter, Kelsie Bashford and Kieran Dong.

The World Health Organization has declared antibiotic resistance among bacterial pathogens a significant human health crisis. The changing nature of bacteria in order to resist antibiotic treatments can result in little or no treatment options for someone infected with certain severe bacterial infections.

Top and her team's research focuses on one of the major contributors to antibiotic resistance: pieces of DNA called plasmids that can transfer between cells of bacteria and provide that resistance. Before this research, it was unknown how or why some bacteria hold on to these plasmids and others don't.

By studying the evolution of bacteria in her laboratory, the team found two genetic mutations that led bacteria to hold on to the resistance plasmids. These bacteria were also able to retain other plasmids they had not seen before, making them resistant to even more drugs.

"Any bacteria can mutate," Top said. "They always find a way. We're looking for a mechanism in the cell that improves retention, so we can block it. We're seeing general patterns of adaptation that allows the cell to hold on to the plasmid."

The study, which was funded by a grant from the National Institutes of Health, will act as a springboard for additional research that will attempt to force bacteria to "drop" the antibiotic-resistance plasmids.

More information: Wesley Loftie-Eaton et al. Compensatory mutations improve general permissiveness to antibiotic resistance plasmids, *Nature Ecology & Evolution* (2017). DOI: 10.1038/s41559-017-0243-2

Provided by University of Idaho