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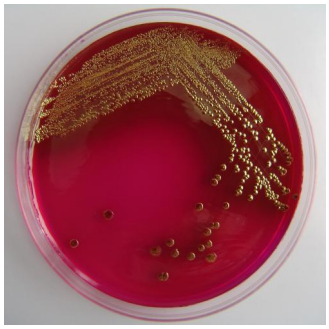
Antibiotic Resistance is not Just Genetic



Genetic resistance to antibiotics is not the only trick bacteria use to resist eradication– they also have a second defence strategy known as persistence that can kick in.

Researchers reporting in the Journal of Medical Microbiology have now demonstrated for the first time that interplay occurs between the two mechanisms to aid bacterial survival. The findings could lead to novel, effective approaches to treat multi-drug resistant (MDR) infections.

'Persister' bacterial cells are temporarily hyper-resistant to all antibiotics at once.

They are able to survive (normally) lethal levels of antibiotics without being genetically resistant to the drug. These cells are a significant cause of treatment failure yet the mechanism behind the persistence phenomenon is still unclear.

Scientists from Centre of Microbial and Plant Genetics, at the Katholieke Universiteit Leuven, Belgium found that the number of persister cells isolated from Pseudomonas aeruginosa infections decreases when the bacterial population shows genetic resistance to the antibiotic fosfomycin.

P. aeruginosa is an opportunistic human pathogen and a significant cause of hospital-acquired infections. It can cause fatal infections in people suffering from cystic fibrosis. The bacterium is notorious for its ability to develop resistance against commonly-used antibiotics and treatment failure is common.

Professor Jan Michiels who led the study explained that persister cells are a major contributor to treatment failure. "Persister cells are produced in low numbers, but nevertheless make it almost impossible to completely remove the bug from the patient. As a result, eradication of infections through antibiotic treatment usually takes a long time," he said. "Our work shows that antibiotic treatment may also influence the number of persisters formed."

Co-administration therapies are being developed to treat MDR infections, in which drugs targeting non-essential cellular functions are combined with antibiotics. Professor Michiels explained that targeting persistence is an attractive option. "Ideally both susceptible and persistent cells would be targeted in a single therapy, but firstly we need to understand more about the interplay between genetic resistance and persistence to avoid stimulating one or the other. Unravelling the mechanism behind bacterial persistence is really important to enable us to optimise treatments of chronic bacterial infections."

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