

'Lonely' bacteria increase risk of antibiotic resistance

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A photo of antibiotic resistant bacteria. Credit: Rok Krašovec, University of Manchester

Scientists from The University of Manchester have discovered that 'lonely' microbes are more likely to mutate, resulting in higher rates of antibiotic resistance.

The study, published today in *Nature Communications* and jointly funded by The Wellcome Trust and Engineering and Physical Sciences Research Council, explored the mutation rates of *E. coli*.

Researchers found out that the rate of mutation varied according to how many of the [bacteria](#) there were. Surprisingly, they discovered that more bacteria gave fewer mutations.

Meanwhile more 'lonely' bacteria developed greater resistance to the well-known antibiotic Rifampicin, used to treat tuberculosis.

Dr Chris Knight joint lead author on the study with Dr. Rok Krašovec from The University of Manchester, said: "What we were looking for was a connection between the environment and the ability of bacteria to develop the resistance to antibiotics. We discovered that the rate at which *E. coli* mutates depends upon how many 'friends' it has around. It seems that more lonely organisms are more likely to mutate."

This change of the [mutation rate](#) is controlled by a form of social communication known as quorum sensing – this is the way bacteria communicate to let each other know how much of a crowd there is. This involves the release of signalling molecules by bacteria when in a dense population to help the organisms understand their surrounding environment and coordinate behaviour to improve their defence mechanisms and adapt to the availability of nutrients.

Dr. Krašovec said: "We were able to change their mutation rates by changing who they shared a test tube with, which could mean that bacteria manipulate each other's mutation rates. It also suggests that mutation rates could be affected when bacteria are put at low densities for instance by a person taking antibiotics."

The rate of mutation was found to be dependent on the gene luxS which

is known to be involved in quorum sensing in a wide range of bacteria.

The team now hopes to find ways to control this signalling for medical applications in a future study funded by the Biotechnology and Biological Sciences Research Council.

"Eventually this might lead to interventions to control mutation rates, for instance to minimise the evolution of [antibiotic resistance](#), allowing antibiotics to work better," said Dr Knight.

Dr Mike Turner, Head of Infection and Immunobiology at the Wellcome Trust said: "Antibiotic resistance is a real threat to disease control and public health today. Any insight into the origins of such resistance is valuable in the fight to prevent it. Chris Knight and his team have gained a fundamental understanding of bacterial communication and the development of mutations which in the long run could contribute to more potent antibiotics and better control of bacterial disease".

More information: "Mutation rate plasticity in rifampicin resistance depends on Escherichia coli cell–cell interactions' by Rok Krašovec, Roman V. Belavkin, John A. D. Aston, Alastair Channon, Elizabeth Aston, Bharat M. Rash, Manikandan Kadirvel, Sarah Forbes and Christopher G. Knight, *Nature Communications*, 2014.

Provided by University of Manchester

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