# Scientists discover how some bacteria survive antibiotics

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Researchers at the University of Illinois at Chicago have discovered how some bacteria can survive antibiotic treatment by turning on resistance mechanisms when exposed to the drugs. The findings, published in the April 24 issue of the journal *Molecular Cell*, could lead to more effective antibiotics to treat a variety of infections.

"When patients are treated with antibiotics some pathogenic microbes can turn on the genes that protect them from the action of the drug," said Alexander Mankin, professor and associate director of the University of Illinois at Chicago's Center for Pharmaceutical Biotechnology and lead investigator of the study. "We studied how bacteria can feel the presence of erythromycin and activate production of the resistance genes."

Erythromycin and newer macrolide antibiotics azithromycin and clarithromycin are often used to treat respiratory tract infections, as well as outbreaks of syphilis, acne and gonorrhea. The drugs can be used by patients allergic to penicillin.

Macrolide antibiotics act upon the ribosomes, the protein-synthesizing factories of the cell. A newly-made protein exits the ribosome through a tunnel that spans the ribosome body. Antibiotics can ward off an infection by attaching to the ribosome and preventing proteins the bacterium needs from moving through the tunnel.

Some bacteria have learned how to sense the presence of the antibiotic in the ribosomal tunnel, and in response, switch on genes that make them

resistant to the drug, Mankin said. The phenomenon of inducible antibiotic expression was known decades ago, but the molecular mechanism was unknown.

Mankin and his team of researchers -- Nora Vazquez-Laslop, assistant professor in the Center for Pharmaceutical Biotechnology, and undergraduate student Celine Thum -- used new biochemical and genetic techniques to work out the details of its operation.

"Combining biochemical data with the knowledge of the structure of the ribosome tunnel, we were able to identify some of the key molecular players involved in the induction mechanism," said Vazquez-Laslop.

"We only researched response to erythromycin-like drugs because the majority of the genetics were already known," she said. "There may be other antibiotics and resistance genes in pathogenic bacteria regulated by this same mechanism. This is just the beginning."

Source: University of Illinois at Chicago