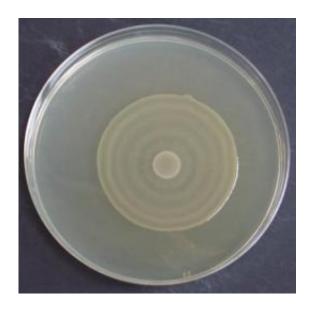


Discovery of controlled swarm in bacteria

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This is an example of swarm movements in a bacteria population. Credit: Universitat Autňnoma de Barcelona

A study led by Spanish researchers from Universitat Autonoma de Barcelona (UAB) describes one of the mechanisms in which pathogenic bacteria populations control the way they spread over the surface of the organs they infect and stop when they detect the presence of an antibiotic, only to resume again when the effect wears off. The star of this process is the RecA protein, which significantly increases its concentration at the start of the bacteria DNA repair mechanism induced by antibiotics. The research was published in *Infection and Immunity*.

In order to develop the infectious process, many pathogenic bacteria



move collectively along the surface of the organ they infect until growing into a massive colony, and consequently produce toxins and substances that harm host tissues. This movement is known as swarming, similar to the movement of bee colonies and other animals. Parts of the molecular process taking place during this movement already have been described, but the mechanism controlling activation or inhibition was not yet known.

The research reveals for the first time the relation between the bacteria DNA repair system, known as SOS response, and swarming. Researchers demonstrated that the presence of antibiotics activates SOS response and thus increases concentration of RecA protein. This interferes with the action of the CheW protein, essential for swarming, and thus causes the bacterial colony to stop moving. When the concentration of this antibiotic decreases, the amount of RecA protein reduces and CheW once again can continue its task of spreading the bacteria.

The results obtained indicate that given the special characteristics of this type of collective movement, <u>antibiotics</u> only affect outer cells of the swarm, which in turn act as sensors and activate the aforementioned molecular repair system. This action thus cancels out the effect of the drug on the rest of the bacteria population.

Jordi Barbé, Laura Medina Ruiz and Susana Campoy, researchers at UAB's Department of Genetics and Microbiology and directors of the study, highlight the importance of this basic discovery since it will allow for the design of targets blocking RecA action and thus increase antibiotic sensitivity of bacteria.

The research was carried out with Salmonella enterica, member of a bacteria group found in several pathogenic species responsible for diseases in the digestive and respiratory system, such as septicaemia and systemic infections.



Provided by Universitat Autonoma de Barcelona

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