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[Biochemistry] Academia Sinica - Institute of Biological Chemistry Researchers Detail Mechanism Involved in Staph Resistance to Antibiotics

[Biochemistry] Academia Sinica - Institute of Biological Chemistry Researchers Detail Mechanism Involved in Staph Resistance to Antibiotics (Chinese Version)

Academia Sinica Newsletter (2010/04/28) The Staphylococcus bacteria are notorious for their ability to develop resistance to antibiotics almost as soon as they are approved for clinical use, and are the scourge of hospitals where they can cause deaths in patients admitted for other diseases. A research team from the Institute of Biological Chemistry led by Academia Sinica Vice President Andrew H.-J. WANG has revealed a molecular mechanism of antibiotic resistance in Staphylococcus epidermidis, a species of Staphylococcus that lives on the skin. Their research was published in the scientific journal, Proceedings of the National Academy of Sciences (PNAS) on April 27, 2010.

There are 32 species and eight sub-species of Staphylococcus. They cause a wide range of diseases in humans and animals such as skin infections, pneumonia, food poisoning and blood poisoning. Among them, the S. epidermidis species usually lives off skin causing no harm to healthy people; however, it can cause severe infections in immune-suppressed patients and those with catheters or other surgical implants.

S. epidermidis protects itself from the host immune system and enhances its resistance to antibiotics by producing a biofilm. Andrew H.-J. WANG and his team revealed the 3D structure of a protein component (named Teicoplanin-Associated Locus Regulator, TcaR), both in isolation and in complex with several antibiotics, which effectively prevents the formation of the biofilm. The clinically-important results increased understanding about how exposure to antibiotics affects bacterial resistance. To solve the structure of TcaR, researchers employed x-ray crystallography, molecular biochemistry, and microbiology.

The group discovered that under normal conditions TcaR binds to DNA to prevent the synthesis of biofilm. Upon the addition of antibiotics to the cell, significant conformational changes in the DNA binding domains of TcaR occur, inducing the inactivation and the departure of TcaR from DNA, thus increasing the expression of biofilm to resist antibiotics treatment. The study successfully delineated the antibiotic binding site of TcaR and the mechanism by which antibiotics induce biofilm formation.

The research was conducted and financed by Academia Sinica and the provision of the protein crystallography synchrotron facility was supported by the National Genomic Medicine Research Program, a project funded by the National Science Council of Taiwan.

The full article entitled "Structural Study of TcaR and its Complexes with Multiple Antibiotics from Staphylococcus epidermidis" is available at the PNAS journal website at: http://www.pnas.org.

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