techman / October 28, 2012 06:25PM

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Academia Sinica Newsletter (2012/10/26) A research team led by Dr. Erh-Min LAI, Associate Research Fellow at the Institute of Plant and Microbial Biology, Academia Sinica, recently reported the regulatory mechanisms of a bacterial protein secretion system named type VI (T6SS). The research team used a soil bacterium Agrobacterium tumefaciens, a causal agent of crown gall disease (a common plant disease) and an important gene transfer tool, as a model system to unravel the molecular mechanisms underlying how T6SS is activated by acid. This work was published in the Sept. 27 issue of the scholarly journal PLoS Pathogens.

The laboratory of Dr. Erh-Min LAI previously discovered T6SS in Agrobacterium tumefaciens and has since focused on understanding the mechanistic and biological functions of T6SS in this disease-causing bacterium. Agrobacterium tumefaciens is capable of transferring cancer-causing genes from bacterial cells into plant cells to induce plant tumors. Because of this unique interkingdom DNA transfer ability, Agrobacterium tumefaciens has become the most popular gene transfer agent for creating transgenic plants for research and agriculture. Structural and functional studies revealed that T6SS assembles into a phage tail-like needle structure to inject cytotoxic effectors into host cells to allow disease development and/or increase bacterial survival. Thus, a smart bacterium must properly control the expression and activity of T6SS to cope with diverse environments when necessary. However, the signals and regulatory mechanisms of most T6SSs remain largely unknown.

In this report, the research team discovered that T6SS is activated by acidity via an ExoR-ChvG/ChvI cascade. In a neutral pH (7.0) growth environment, T6SS is off as the sensor kinase ChvG is inactivated by binding with ExoR repressor. When Agrobacterium tumefaciens senses the acidic signal (pH 5.5), ExoR is rapidly degraded and thereby derepresses ChvG to activate T6SS. This is the first report to unravel the molecular mechanisms underlying the acid-activated T6SS.

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Further Information:
Academia Sinica Newsletter 2012/10/26

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Edited 1 time(s). Last edit at 10/28/2012 06:28PM by techman.