Experimental evolution of bacterial virulence functions

Abstract

Pathogenic bacteria have evolved their virulence functions through a long period of host-pathogen interaction. The difference between pathogenic bacteria and non-pathogenic bacteria has been investigated by various approaches including comparative genomic analysis. However, the molecular mechanism how non-pathogenic bacteria acquire the pathogenic functions is not well understood. To elucidate the molecular mechanisms, we searched highly pathogenic mutants of Escherichia coli through an experimental evolution in a silkworm infection model. Silkworms are larvae of Bombyx mori, a lepidopteran insect, and the infection model enables a large scale screening of bacterial mutant strains because of low cost and little ethical constraints. We found that gene mutations in LptD and LptE (LPS transporter), OpgG and OpgH (synthase for osmoregulated periplasmic glucan), or MlaA (maintenance of outer membrane lipid asymmetry) increased killing activity of E. coli against silkworms. In addition, all of the gene mutants were resistant not only to the silkworm antimicrobial peptide, cecropin, but also to several antibiotics including vancomycin. These results suggest that the acquisition of resistance to a common antibacterial action between host antimicrobials and antibiotics produces highly pathogenic and drug-resistant bacteria. This seminar focuses on how we can utilize experimental evolution to understand pathogenic bacteria and develop clinical strategies against them.

Date

2025 8/7 Monday
16:00 ▶17:00

Online

https://00m.in/fSJbm

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Venue

Hiroshima University, Graduate School of Advanced Science of Matter Build A, 6F Seminar room601-(2)

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