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Interplay between genetic regulation of phosphate homeostasis and bacterial virulence

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Abstract


Bacterial pathogens, including those of humans, animals, and plants, encounter phosphate (Pi)-limiting or Pi-rich environments in the host, depending on the site of infection. The environmental Pi-concentration results in modulation of expression of the Pho regulon that allows bacteria to regulate phosphate assimilation pathways accordingly. In many cases, modulation of Pho regulon expression also results in concomitant changes in virulence phenotypes. Under Pi-limiting conditions, bacteria use the transcriptional-response regulator PhoB to translate the Pi starvation signal sensed by the bacterium into gene activation or repression. This regulator is employed not only for the maintenance of bacterial Pi homeostasis but also to differentially regulate virulence. The Pho regulon is therefore not only a regulatory circuit of phosphate homeostasis but also plays an important adaptive role in stress response and bacterial virulence. Here we focus on recent findings regarding the

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