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Effector translocation in soil beneficial bacteria

Wei Qian[1*]

Unlike eukaryotes, bacteria are single-celled organisms whose lives prone to be endangered by various stresses; therefore, it is crucial to understand how bacterial cells establish intra-specific or inter-specific communication for survival. Quorum sensing (QS), a cell–cell communication process that mediated by small-molecule signaling chemicals, confers this ability to bacteria. Acyl-homoserine lactones (AHL) are the most extensively studied QS signals. Usually, AHL mediated quorum sensing is required for full virulence of pathogenic bacteria so that it is ideal target for novel antibiotics (Fuqua et al. 2002; Schaefer et al. 2008). Bacteriologists have developed several effective strategies to interfere or disrupt QS, a process known as quorum quenching (QQ), in order to prevent bacterial infection and suppress antibiotic resistance (Zhang et al. 2001; Grandclement et al. 2016). The most common mechanism of QQ is degradation of AHL by enzymes (Dong et al. 2000; Zhang et al. 2001). To date, several AHL-degrading enzymes have been identified, including the well-known enzyme AiiA from *Bacillus* sp. 240B1, AttM from *Agrobacterium tumefaciens* C58, MomL from *Muricauda olearia*, and AiiD from *Ralstonia* sp. XJ12B (Zhang et al. 2001, 2002; Lin et al. 2003; Tang et al. 2015) (Fig. 1A).

In a recent study, Qian and colleagues identified a novel QQ mechanism. They revealed that a bacterial effector protein that is secreted by type IVA secretion system (T4ASS), interferes AHL biosynthesis of competitive bacteria (Fig. 1B). T4ASS is mainly employed for DNA delivery and is represented by the VirB/D4 system of *Agrobacterium tumefaciens*. However, this system of the pathogenic bacteria *Xanthomonas citri* and *Stenotrophomonas maltophilia* also inject toxic effector proteins into susceptible competitor cells, casing cell lysis and death (Souza et al. 2015). Unlike these earlier findings, the present study demonstrated that a non-AHL-producing antifungal bacterium, *Lysobacter enzymogenes* OH11, could utilize T4ASS to deliver an effector protein LqqE1 into the cell of another soil bacterium, *Pseudomonas fluorescens* 2P24 that could produce AHL. Interestingly, the delivery of LqqE1 did not cause growth inhibition of *P. fluorescens* 2P24, which is a bacterial competitor. Alternatively, LqqE1 acted as an interspecies AHL quorum quencher, but its function is independent from degrading AHL. LqqE1 binds to the competitor' AHL synthase PcoI, preventing the synthase from recognizing *S*-adenosy-L-methionine, an essential substrate for AHL synthesis. Consequently, the LqqE1-triggered QQ is ecologically important because its presence in the native *L. enzymogenes* or delivery into *P. fluorescens* enhanced the killing efficiency of *L. enzymogenes* against *P. fluorescens*. This work is fascinating because it reveals a novel mechanism by which bacteria compete with each other in the soil microbiome by secreted effectors just likes the molecular interaction between microbes and their hosts. It also highlights that T4ASS-translocated effectors have a unique function, which is typically thought to involve transferring DNA or toxins into competitor cells, causing cell death.

In translocation, the authors present shreds of evidence showing the potential application of LqqE1-triggered QQ to block AHL signaling in both the human pathogen

*Correspondence:
Wei Qian
qianw@im.ac.cn
[1] State Key Laboratory of Plant Genomics, Institute of Microbiology, Chinese Academy of Sciences, Beijing 100101, China

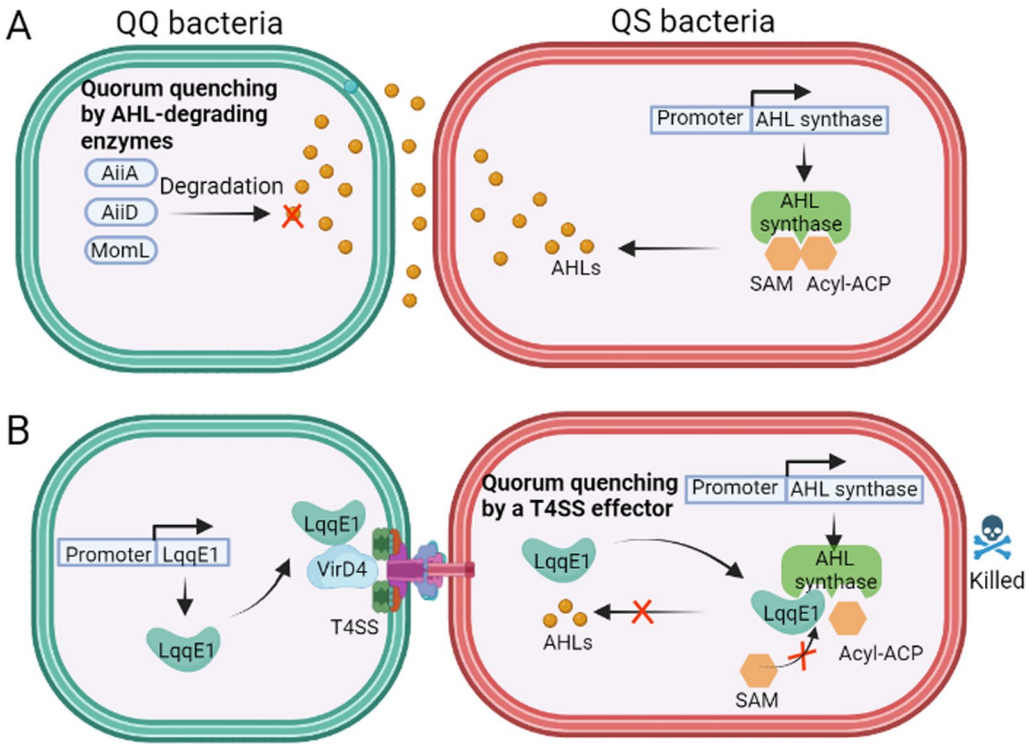

Fig. 1 LqqE1-triggered interspecies quorum quenching in soil microbiome. **A** Canonical quorum quenching inhibits bacterial virulence. Canonical AHL-quenching proteins AiiA from *Bacillus* sp. 240B1, MomL from *Muricauda olearia*, and AiiD from *Ralstonia* sp. XJ12B could degrade AHLs produced by AHL synthases. To achieve this, these canonical AHL-quenching proteins do not need to interact with AHL synthases. **B** Novel quorum quenching controls interspecies bacterial killing. OH11 can transport the T4E protein LqqE1 into the cytoplasm of 2P24. LqqE1 does not degrade AHL, but it is able to bind to AHL synthetase through direct protein-protein interactions, causing interference with the recognition of SAM, the substrate required for AHL synthesis, thus achieving naturally occurring quorum quenching and gaining a survival advantage

Pseudomonas aeruginosa and the plant pathogen *Ralstonia solanacearum*, providing a feasible avenue to expand the scope of traditional biocontrol agents from agriculture to medical therapy.

Finally, it is noteworthy that during plant-pathogen interactions, it is well known that pathogens could deliver diverse effector proteins into hosts to subvert plant immunity and hence promotes infection. The work presented by Qian and colleagues uncovered that soil beneficial bacteria can also transfer effector proteins into other bacteria, indicating that the effector-triggered immune process evolved anciently, and is currently in progress underground.

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