

Hrp-dependent Biotrophic Mechanism of Virulence: How Has It Evolved in Tumorigenic Bacteria?

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A mechanism of virulence mediated by *hrp*-genes is present in many Gram-negative bacterial pathogens. It involves delivery of effector proteins into host cells *via* the type III secretion system (TTSS) and the interaction of TTSS effectors with plant proteins. These interactions may either promote responses beneficial to the pathogen or trigger the hypersensitive response if an effector is recognized by corresponding resistance protein. *Pantoea agglomerans*, which is widespread in nature mainly as an epiphyte, has evolved into a *hrp*-dependent and host-specific tumorigenic pathogen by acquiring a plasmid containing a pathogenicity island (PAI). This PAI harbors a *hrp*-gene cluster, and genes encoding for TTSS effector proteins and biosynthesis of IAA and cytokinins. The results reviewed describe how the interplay between negative-acting and positive-acting TTSS effectors determines the transformation of *P. agglomerans* into two related pathovars. Furthermore, the PAI's structure supports the premise that these pathovars are recently evolved pathogens. Finally, the possible interaction between TTSS effectors and phytohormones for gall formation is proposed.

KEY WORDS: Type III secretion system; Type III effectors; biotroph; necrotroph; plasmid; *hrp*-gene cluster; galls; IAA; cytokinins; virulence; pathogenicity island; IS elements.

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