Poster Presentation

MS53.P24

Perturbation of host autophagy by the Irish potato famine pathogen

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Autophagy is a catabolic process involving degradation of dysfunctional cytoplasmic components to ensure cellular survival under starvation conditions. The process involves formation of double-membrane vesicles called autophagosomes and delivery of the inner constituents to lytic compartments. It can also target invading pathogens, such as intracellular bacteria, for destruction and is thus implicated in innate immune pathways [1]. In response, certain mammalian pathogens deliver effector proteins into host cells that inhibit autophagy and contribute to enabling parasitic infection [2]. Pyhtophthora infestans, the Irish potato famine pathogen, is a causative agent of late blight disease in potato and tomato crops. It delivers a plethora of modular effector proteins into plant cells to promote infection. Once inside the cell, RXLR-type effector proteins engage with host cell proteins, to manipulate host cell physiology for the benefit of the pathogen. As plants lack an adaptive immune system, this provides a robust mechanism for pathogens to circumvent host defense. PexRD54 is an intracellular RXLR-type effector protein produced by P. infestans. PexRD54 interacts with potato homologues of autophagy protein ATG8 in plant cells. We have been investigating the structural and biochemical basis of the PexRD54/ATG8 interaction in vitro. We have purified PexRD54 and ATG8 independently and in complex from E. coli. Using protein/protein interaction studies we have shown that PexRD54 binds ATG8 with sub-micromolar affinity. We have also determined the structure of PexRD54 in the presence of ATG8. This crystal structure provides key insights into how the previously reported WY-fold of oomycete RXLR-type effectors [3] can be organized in multiple repeats. The structural data also provides insights into the interaction between PexRD54 and ATG8, suggesting further experiments to understand the impact of this interaction on host cell physiology and how this benefits the pathogen.

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Keywords: Plant disease, effector protein, Autophagy