

## Microsymposium

### MS53.O05

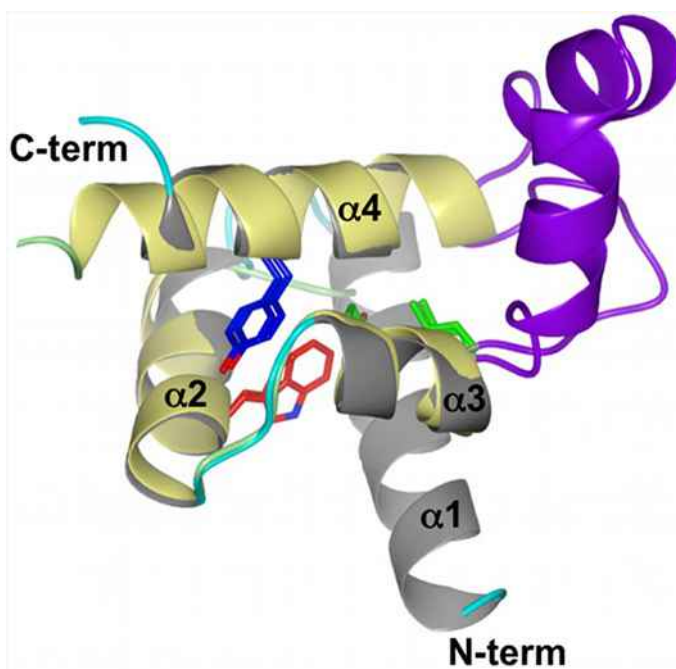
#### *Molecular mechanisms of host cell manipulation by plant pathogens*

R. Hughes<sup>1</sup>, S. King<sup>1</sup>, A. Maqbool<sup>1</sup>, H. McLellan<sup>2</sup>, T. Bozkurt<sup>3</sup>, Y. Dagdas<sup>3</sup>, S. Dong<sup>3</sup>, P. Birch<sup>2</sup>, S. Kamoun<sup>3</sup>, M. Banfield<sup>1</sup>

<sup>1</sup>John Innes Centre, Department of Biological Chemistry, Norwich, UK, <sup>2</sup>University of Dundee (at JHI), Dundee, UK, <sup>3</sup>The Sainsbury Laboratory, Norwich, UK

An estimated 15% of global crop production is lost to pre-harvest disease every year. New ways to manage plant diseases are required. A mechanistic understanding of how plant pathogens re-program their hosts to enable colonisation may provide novel genetic or chemical opportunities to interfere with disease. One notorious plant parasite is the Irish potato famine pathogen *Phytophthora infestans*. This pathogen remains a considerable threat to potato/tomato crops today as the agent of late blight. Plant pathogens secrete effector proteins outside of and into plant cells to suppress host defences and manipulate cell physiology. Structural studies have provided insights into effector evolution and enabled experiments to probe function [1-3]. Crystal structures of 4 *Phytophthora* RXLR-type effectors, which are unrelated in primary sequence, revealed similarities in the fold of these proteins. This fold was proposed to act as a stable scaffold that supports diversification of effectors. Further, molecular modelling has enabled mapping of single-site variants responsible for specialisation of a *Phytophthora* Cystatin-like effector, revealing how effectors can adapt to new hosts after a “host jump”. Structural studies describing how RXLR-effectors interact with host targets are lacking. We have used Y2H/co-IP studies to identify host proteins that interact with *P. infestans* effectors PexRD2 and PexRD54. PexRD2 interacts with MAPKKKe, a component of plant immune signalling pathways, and suppressed cell death activities of this protein. We used the structure of PexRD2 to design mutants that fail to interact with MAPKKKe, and no longer suppress cell-death activities. We found that PexRD54 interacts with potato homologues of the autophagy protein ATG8. We have obtained a crystal structure for PexRD54 in the presence of ATG8. We are now using X-ray scattering to verify the complex structure in solution prior to establishing the role of this interaction during infection.

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