

## **International Conference on Flu**

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## Subversion of autophagy by viral LC3 interacting regions

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A utophagy can provide a cell with nutrients in time of starvation by engulfing cytoplasmic contents in a double membrane targeting the autophagosome to a lysosome and utilizing the resultant lipids and amino-acids for metabolism. This process has been adapted to form a critical component of cellular self-defence with invading micro-organisms targeted for autophagy by adaptor proteins that can bind both to danger signals in the cytosol and to the LC3 molecules that decorate the autophagosomal membrane (Boyle and Randow, 2013). Binding to LC3 typically takes place via a LIR (LC3 Interacting Region), a short motif comprising a hydrophobic beta-strand and surrounding acidic residues. We have discovered that influenza M2 contains a LIR at its C-terminal tail. This enables influenza to subvert autophagy and promotes the formation of stable virions. M2 also has a LIR independent function to subvert autophagy inhibiting fusion of autophagosomes with lysosomes. We propose that influenza M2 targets autophagosomes to the plasma membrane to increase resources for budding and present evidence that other enveloped viruses also encode LC3-interacting regions.

## Biography

Rupert Beale began his interest in host pathogen interactions when he worked with Professor Douglas Fearon during his Medical Degree. He subsequently completed his PhD on the AID and APOBEC cytidine deaminase proteins with the late Professor Michael Neuberger at the MRC Laboratory of Molecular Biology. During his higher Medical Training in Nephrology, he returned to the LMB to work with Dr. Felix Randow collaborating with Professor Paul Digard's group to investigate the role of autophagy in influenza replication. He is now starting his own group in the Division of Virology in Cambridge, funded by a MRC Clinician Scientist Fellowship.

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