

P014 Interactions of influenza A virus with the ESCRT pathway
**Emily A. Bruce¹, Liz Medcalf¹, Colin M. Crump¹,
Katherine Bowers², Paul Digard¹**

¹Division of Virology, Department of Pathology, University of Cambridge, ²Institute of Structural and Molecular Biology, Division of Biosciences, University College London

Use of the cellular ESCRT pathway by influenza A virus has been the subject of controversy. We confirm previous work showing that *in vitro*, the influenza virus matrix 1 (M1) protein binds VPS28, a member of the ESCRT pathway. However, using immunofluorescent confocal microscopy to examine infected cells, we observed no colocalisation between influenza virus structural proteins and VPS28, Alix or VPS4 ESCRT proteins. Trafficking of viral HA and M1 proteins to the plasma membrane appeared undisrupted, even when endosomal trafficking was greatly impaired by the presence of catalytically inactive VPS4. Using siRNA sequences targeting VPS28, we showed that depletion of endogenous VPS28 had no significant effect on viral replication, as measured by plaque assay of released virus particles. Finally, using a stably transfected cell line expressing wildtype or trans-dominant VPS4, we demonstrated that influenza virus release was not dependent on a functional VPS4 protein, as is the case for all viruses currently known to use the ESCRT pathway. Based on our results, we see no obvious role for the ESCRT pathway in influenza virus budding.