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Molecular mechanism of restriction of influenza A virus by human annexin A6

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Elucidation of networks of interactions between viruses and host cells will pave the way towards the understanding of molecular determinants of cytopathogenicity and ultimately the design of novel therapeutics. Our strategy is to identify human restricting or enhancing factors of IAV infection. We specifically looked for interactions of cytosolic domains of IAV proteins with human factors using a yeast-two-hybrid screening approach. We found that the C-terminal domain of the highly pathogenic avian H5N1 virus M2 protein interacts with human annexin A6. We further confirmed the interaction of annexin A6 with the M2 protein of A/WSN/33 (H1N1) virus. Our initial study demonstrated that human annexin A6 restricts IAV infection *in vitro*. Interestingly, restriction was confirmed on all IAV strains tested. Using transmission electron microscopy, we found that annexin A6 expression alters IAV morphogenesis and restricts release of progeny virions. Our current investigations aim at understanding the molecular mechanism responsible for IAV restriction by annexin A6.

Biography

Stefan Diaz Gaisenband has completed a degree in biology from University of Balearic Islands and a Master's degree in Advanced Microbiology recognised by both University of Barcelona and University of Balearic Islands (Spain). He then enrolled for a PhD at Brunel University London and has learnt state of the art virology and cellular biology approaches, which has enabled him to study the molecular mechanism responsible for restriction of influenza viruses by human annexin A6.

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