Australian National University

Rapid spreading and immune evasion by vaccinia virus

Monday 16 December 2013 1pm

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Slatyer seminar room R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU

Vaccinia virus (VACV) is the vaccine that was used to eradicate smallpox and, like other poxviruses, replicates in the cell cytoplasm and encodes many proteins to suppress the host response to infection. My laboratory has long standing interests in how the virus replicates and spreads and how it madulates immunity. VACV is able to spread more rapidly than is predicted from its known replication kinetics. Video microscopy indicated that VACV spreads across a lawn of susceptible cells at 1.2 hour per cell, despite the replication cycle of the virus taking a minimum of 5 hours per cell. Investigation of the how VACV does this revealed a novel mechanism for rapid spread in which virus particles attempting to infect a cell that is already infected (super-infection) are repelled away from the infected cell to find new uninfected cells to infect. VACV immunomodulatory protein A49 that is expressed throughout infection and remains intracellular. It functions as an inhibitor of NF-κB activation by binding to the E3 ubiquitin ligase β-TrCP and thus prevents β-TrCP ubiquitinating and inducing degradation of phosphorylated IκBα. Thus p-IκBα remains bound to the NF-κB subunits p65 and p50 in the cytoplasm and NF-κB responsive genes remain silent. Despite the presence of nine other inhibitors of NF-κB encoded by VACV, loss of A49 caused virus attenuation *in vivo*, indicating these proteins have non-redundant properties.

Professor Geoffrey L. Smith FRS is Professor of Pathology and a Wellcome Trust Principal Research Fellow.
Read more about Professor Geoffrey Smith.

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