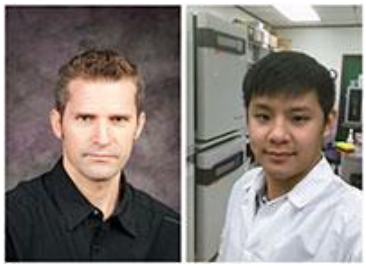
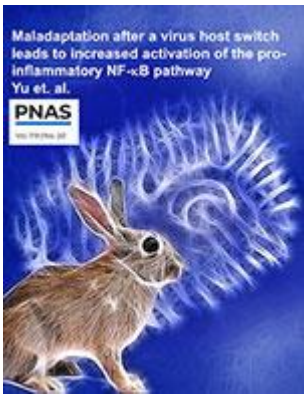
Maladaptation after a virus host switch leads to increased activation of the pro-inflammatory NF-κB pathway



New research from UC Davis scientists reveals that the maladaptation after a virus host switch, in which a poxvirus substantially gained in virulence, can have unexpected consequences such as activating pro-inflammatory pathways.

The poxvirus myxoma virus naturally infects American rabbit species (brush rabbit and tapeti), in which it causes a localized, benign infection. In European rabbits, which include the pet rabbits, myxoma virus causes a highly lethal disease called myxomatosis.



The work of former graduate student Huibin Yu from the laboratory of Stefan Rothenburg, showed that surprisingly the antiviral protein kinase R (PKR) from brush rabbits was inhibited better than PKR from European rabbits by a myxoma virus protein, thus not correlating with virulence. The intermediate inhibition of European rabbit PKR, however resulted in activation of the pro-inflammatory NF-κB pathway, which may contribute to rapid viral dissemination and increased virulence of myxoma virus in European rabbits.

The study demonstrates that maladaptation of viral immune antagonists can result in substantially different immune responses in aberrant hosts and might serve as a model that can explain increases in virulence after virus host switches.

The study was published in the Proceedings of the National Academy of Sciences of the United States of America and **can be found here »**