

Title: Tobacco Mosaic Virus: Infection, Disease and Energy Storage

Author: Jim Culver, Dept of Plant Sciences and Landscape Architecture, U of Md, College Park

Abstract:

The capacity of a viral pathogen to cause disease is often attributed to its ability to reprogram its host's cellular machinery to facilitate its own replication and spread. Reprogramming cellular processes is likely driven by multiple virus-host interactions that combine to produce a successful infection and subsequent disease. However, the complexity of virus-host interactions has hindered efforts to understand the molecular mechanisms whereby viruses commandeer host machinery. The approach we have taken to address this complexity is to identify individual virus-host interactions and the role each plays in infection and disease development. Using a *Tobacco mosaic virus* (TMV) host pathosystem we have identified interactions that lead to the disruption of host factors involved in plant development and defense. These interactions are responsible for a significant portion of the disease symptoms induced by TMV and contribute to the ability of the virus to move systemically and avoid host defenses. Understanding the combined contributions of these individual virus-host interactions has revealed new insights into the molecular mechanisms needed to establish an infection.

For more information see:

Wang X, Goregaoker SP, and Culver JN. 2009. Interaction of the Tobacco mosaic virus replicase protein with a NAC domain transcription factor is associated with suppression of systemic host defenses. *J. Virol.* 83, 9720-9730.

Padmanabhan MS, Kramer SR, Wang X and Culver JN. 2008. TMV-Aux/IAA interactions: reprogramming the auxin response pathway to enhance virus infection. *J. Virol.* 82:2477-2385.

Culver, J.N. and Padmanabhan, M.S. 2007. Virus-induced disease: altering host physiology one interaction at a time. *Annu. Rev. Phytopathol.* 45:221-243.