Exploring the effects of non-equilibrium processes on virus assembly in vivo

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1. Abstract

Plus sense single-stranded RNA viruses represent one of the largest classes of viruses infecting all kingdoms of life. This class of viruses have an interesting problem in that their genomic material serves a dual purpose, it is a genome to be packaged into new viral progeny, but also it is an mRNA that needs to interact with host ribosomes to synthesize viral proteins. Because of this dual role, viral assembly needs to be carefully coordinated to ensure that it occurs on viral RNAs which are free from ribosomes. Thus (+)ssRNA viral assembly in vivo is inherently a non-equilibrium process, and the competition between replication/translation complexes (such as the ribosome and RNA dependent RNA polymerase) with viral encoded capsid proteins for the viral mRNA need to be taken into consideration when examining viral assembly in vivo. In this talk, I will discuss my recent work on developing a computation model capable of examining the competition between host ribosomes and coat proteins that takes place during the early stages of a viral infection. Using the ssRNA virus bacteriophage MS2 as a model system, I reveal some interesting insights into the ability of coat proteins to suppress ribosomes from synthesizing viral genes. This leads to some interesting questions regarding how viral assembly is eventually triggered during later stages of the viral infection.