

Welcome to the Molecular-Machine-Shop

Date: Wednesday, Oct 4th, 2006, 4:00 pm

Venue: CMS Collaboratorium, 2nd floor

Speaker: April Burch,
DID, Wadsworth Center, NYSDOH

Title: Herpes Simplex Virus Type 1 induces changes
in the 26S proteasome during lytic infection

Herpes Simplex Virus Type-1 (HSV-1) is a significant pathogen in both immunocompetent and immunocompromised individuals and is a common cause of infectious corneal blindness. We have shown that several cellular pathways involved in protein quality control are activated in the HSV-1 infected cell. Specifically, we found that stress-activated chaperone molecules and the proteasomal core particle are sequestered within discrete compartments in the nucleus of the HSV-1 infected cell. Our goal is to understand this spatial reorganization vis-a-vis viral functions. We hypothesize that the core proteasome and associated proteins are recruited by the virus during lytic infection to process misfolded or unwanted viral/cellular proteins, but recent evidence indicates that a key regulatory subunit of the proteasome (Mss1), which can perform other cellular functions when not associated with the core proteasome, is required for viral replication.

