



Left: A single, inactivating point mutation was created in the LXCXD motif of the human cytomegalovirus pp71 protein. This DNA content histogram created from a flow-cytometric analysis of individual cells shows that wild-type pp71 can induce quiescent, G0 cells to enter the S phase when expressed from a recombinant adenovirus (as compared to mock infected cells), but the C219G mutant can not. **Right:** This western blot shows that pp71 degrades the growth-suppressive, hypophosphorylated form of the Rb protein (labeled Rb), but not the hyperphosphorylated form (*Rb) that does not inhibit the cell cycle. Also, it shows that the proteasome inhibitors lactacystin (L) proteasome inhibitor #1 (PI#1), ALLN (A), epoxomicin (EPO), and clasto-lactacystin (CL), each inhibit the ability of pp71 to degrade Rb, but the solvent DMSO (D) and the cysteine-protease inhibitor E64 do not. Thus pp71 degrades the hypophosphorylated forms of the Rb family members (Rb, p107 and p130) in a proteasome-dependent manner. The C219G mutant that doesn't stimulate the cell cycle, also can not degrade the Rb family members.