

Poster presentation

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Electrostatic repulsion regulates hiv-1 capsid hexamer plasticity and assembling capacities into core-like structures

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Capsid protein (CA) is the major component of the HIV-1 core. Three major phosphorylation sites have been identified at positions S₁₀₉, S₁₄₉ and S₁₇₈ in amino-acid sequence of CA. Here we investigated the possible consequences of phosphorylation at these sites on CA hexamer formation and core assembly. Using *in silico* modeling approaches we found that the presence of a phosphate group at each position impacts CA hexamer organization and flexibility. The biological relevance of molecular modeling was evaluated analyzing *in vitro* assembly properties of bacterially expressed CA bearing S₁₀₉D, S₁₄₉D or S₁₇₈D substitutions that mimic constitutive phosphorylation at these sites. Constitutive negative charge at positions 109, 149 but not at position 178 impaired *in vitro* assembly of mature CA. *In vivo*, the corresponding mutations were found to impact the core morphology of HIV-1 mutants bearing the corresponding mutation. Moreover, these viruses lacked infectivity since they were unable to produce proviral DNA. Altogether our data indicate that phosphorylation may impact structural properties of CA hexamers and of HIV-1 cores. Its contribution in HIV-1 replication appears to play an important role in HIV-1 life cycle.