

Identification of SV40 Vp1-Vp2/3 interactive interface important for the assembly of infectious particles



A Nakanishi¹ and H Kasamatsu²

¹Department of Bioscience and Biotechnology, Tokyo Institute of Technology Yokohama, Japan and ²Molecular Biology Institute, Department of Molecular, Cellular, and Developmental Biology, University of California-Los Angeles, Los Angeles, California, USA

Interaction of SV40 major capsid protein Vp1 and the minor capsid proteins, Vp2 and Vp3, is an integral aspect of the SV40 structure, though its role during the viral life cycle is yet to be understood. We have examined the domains that involve the interaction by biochemical approach and found Vp3 (155–190) that resides in the Vp2/3 common region, and Vp1 (215–275) are important for the Vp1-Vp2/3 binding. Based on this knowledge structural model of SV40 Vp1 pentamer-Vp3 complex was generated and four sets of residues, Vp3 Phe157-Ile158, Vp3 Pro-Gly-Gly (164–166), Vp3 Leu 177- Leu181, and Vp1 Val-Leu-Leu (243–245), were predicted important for the Vp1 to Vp2/3 interaction. Disrupting these motifs by altering the original residues to bulky charged residues block the interaction *in vitro*. The respective mutations were introduced to the viral genome and examined their effect on the viral life cycle. All the mutants formed virion-like particles and, surprisingly, packaged viral genome. However, the extent of Vp2/3 in the mutant particles was much reduced or undetectable. The loss of the Vp2/3 in the VLP led to reduction of the viability, and the mutants whose particles did not contained detectable Vp2/3, such as those harboring alteration of either Vp1 Val 243, Leu 245, or both, to glutamate were nearly non-viable. The results imply that Vp2/3 may not be necessary for packaging the viral genome into the Vp1 capsid, though the Vp2/3s are essential for the early phase of infection.