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LS-6-P-2204 Interactions of Mouse polyomavirus structural proteins with host cell structures

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Mouse polyomavirus (MPyV) is a small non-enveloped double-stranded DNA virus. Virions of MPyV are composed of three structural proteins – the major protein, VP1, and two minor structural proteins, VP2 and VP3. Seventy-two VP1 pentamers create viral capsid and each pentamer is associated with one molecule of either VP2 or VP3. A great deal is known about functions of the Mouse polyomavirus capsid proteins in the virion structure as well as in early stages of virus infection. However, interactions of these proteins with cellular proteins and structures in late stages of infection are not well characterised.
We studied properties of VP1 after its individual expression and also after co-expression with VP2 and VP3 in mammalian cells using confocal and electron microscopy. Although VP1 possess nuclear localization signal, it was localised preferentially in the cytoplasm. In situ fractionation of transfected cells revealed that VP1 protein formed fibres in the cytoplasm. These fibres were sensitive to nocodazol, drug destabilizing microtubules. Moreover, in a comparison with control cells, tubulin was retained in insoluble fraction in cells transiently expressing VP1. Immunoelectron microscopy of thin sections of cells after in situ fractionation showed co-localization of VP1 and tubulin cytoskeleton. Co-expression of VP1 with VP2 or VP3 led to nuclear localisation of structural proteins. Two phenotypes of VP1 staining were observed – difuse localization and spot-like phenotype. Nuclear lamina staining (with an antibody against lamin A/C or lamin B) was disturbed in infected cells and in cells co-expressing VP1/2 and VP1/3. A possible meaning of these interactions in viral life cycle context will be discussed.

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