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**UNDERSTANDING THE INTERPLAY BETWEEN MPYV INFECTION AND NUCLEAR LAMINA**

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The nuclear lamina (NL) is a dense meshwork of intermediate filaments V, type A lamins (lamin A/C), type B lamins and membrane associated proteins. It is located below the inner nuclear membrane. NL maintains the shape and structural integrity of the nucleus and plays an important role in fine-tuning of DNA related processes, e. g. replication or transcription. Also, it has been suggested to play a role in cell defence against pathogens. DNA viruses evolved mechanisms to exploit NL for their own purposes. In this study, we followed the changes of NL in cells infected with mouse polyomavirus (MPyV) and a possible role of lamins in MPyV replication. We examine the structure and integrity of the NL late times post infection (40h) and after transient expression of capsid proteins. Under both conditions, the major capsid protein VP1 significantly and non-randomly accumulates in close proximity of NL. Despite the irregularities in NL staining observed by confocal microscopy, and detected partial lamin A/C degradation, we proved that nuclear envelope remains intact. After in situ fractionation of infected cells (40h), MPyV DNA genomes, VP1 and LT (large T antigen necessary for viral genome replication) were found together with lamin A/C and lamin B1 in the last insoluble fraction, indicating possible complex formation. This phenomenon was later shown to be independent of the presence of lamin A/C. Further, we found that lamin A/C was solubilized during the infection progress probably due to its detected hyperphosphorylation. Given that lamin A/C was detected in virus transcription/replication centres and that in the absence of lamin A/C, slight reduction of LT andVP1 gene transcription were observed (24 hpi), it seems that lamin A/C supports viral gene transcription at early stage of infection. Despite that, the level of LT and VP1 proteins finally increased in cells lacking lamin A/C, probably by their stabilization caused by stronger binding to lamin B. This assumption is supported by observation, that VP1 together with lamin B1, was found in the insoluble fraction after in situ fractionation of cells with lamin A/C knockdown.

Altogether, these data suggest that lamins are affected by MPyV replication and are involved in the formation of the viral DNA replication/transcription centres and/or virus assembly. Mechanisms and importance of these processes have to be further elucidated.

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