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RelA participates in the regulation of the coupling between HCV RNA replication and HCV translation in huh7.5.1 cells

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Hepatitis C virus, HCV, is a positive-strand RNA. After released into cytoplasm, HCV RNA severs as a template for the viral translation and transcription. Therefore, the coupling between the viral translation and transcription plays an important role in the modulation of HCV replication. In order to escape the systemic surveillance, HCV develops a complexity strategy to coordinate these two processes. Of these, host factors have been implicated in involvement in the regulation of this adjustment. Although Huh7.5.1 cells is permissive for the study of HCV1a replication in culture system, a low viral production still restricts explore in the understanding of HCV living cycle. Recent studies suggest that a sustained NF-kB activation is a major factor for the impediment of viral replication. To further clarify the role of NF-kB in the HCV replication, we used shRNA to inhibit the activation of RelA. Intriguingly, we found that RelA silencing remarkably suppressed HCV IRES mediated translation. The expressions of viral proteins were also inhibited. Inversely, RelA silencing improved the production of HCV. The further investigation showed that this enhancement was mediated by the increment of HCV RNA replication through the inhibition of interferon beta response. In summary, our results suggest that RelA may participate in the coupling of HCV1a RNA translation and HCV1a RNA transcription, and so regulate HCV1a replication.

Biography

Lumin Zhang has completed his Ph.D at the age of 32 years from Nagoya University. Now, he is a visiting fellow in National Institutes of Health.

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