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Inhibition of flavivirus infections by host ZFP36L1 protein through both of XRN1- and RNA exosome- dependent manners

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Abstract

Zinc finger protein 36, CCCH type-like 1 (ZFP36L1), a member of the ZFP36 family (also named as TIS11family) of CCCH-type zinc finger proteins, are generally thought of as RNA-binding proteins and identified to play an important role in the regulation of the cellular gene expression at post-transcriptional or translation levels. However, the antiviral potential of ZFP36L1 has not been identified so far. Here, we demonstrate for the first time that ZFP36L1 functions as a host innate defender against flaviviruses including Japanese encephalitis virus (JEV) and dengue virus (DENV). Ectopic overexpression of ZFP36L1 restricted JEV and DENV infection, and knockdown of ZFP36L1 enhanced viral replication. ZFP36L1 destabilized JEV genome by targeting and degrading viral RNA through recruiting both 5¢-3¢ XRN1-mediated and 3¢-5¢ RNA exosome-mediated RNA decay pathways. Mutation in both of the zinc-finger motifs of ZFP36L1 showed the disruption of antiviral activity.

Biography:

Assoc Prof Dr Ren Jye Lin studied in Taipei Medical University.

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