Recapitulation of the entire hepatitis C virus life in engineered mouse cell lines

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A US study presented today at The International Liver CongressTM 2012 demonstrates that the entire HCV lifecycle can be recapitulated in murine cells, implying that HCV permissive mouse models could soon be developed.

The data suggests that HCV replication in the murine environment is limited by innate immune responses. Inactivating these pathways and the expression of the appropriate entry factors and miR-122 creates murine fibroblasts that can be infected and support replication.

The study also corroborates previous data that the expression of apoE promotes production of infectious virus.

Although mouse orthologs of MAVS and TRIF could be cleaved by the HCV NS3/4A protease, this was not enough to establish robust HCV replication.

In contrast, impairment of type I and type III IFN signaling enhanced the permissiveness of murine fibroblasts for persistent HCV replication.

The in-depth study of hepatitis C pathogenesis and immune responses is currently hampered by the lack of suitable small animal models. This study is a positive step to addressing this issue.

More information: Vogt A, Recapitulation of the entire Hepatitis C virus life in engineered mouse cell lines. Abstract presented at the

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