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# Modelling the spatial spread of hepatitis C virus infection in vitro

*Wednesday 18 June 2014 14:00 (15 minutes)*

There currently exists only one mathematical model describing the course of a hepatitis C virus (HCV) infection in vitro: a non-spatial ODE model. However, experiments have shown that the spread of HCV infection has an important spatial component: infection disseminates both distally via release and diffusion of virus through the medium, and locally via direct, cell-to-cell infection. Both infection modes appear to play an important role, yet could be differentially affected by antiviral therapy. Therefore, characterizing their relative contribution to infection kinetics has important implications for the control of HCV infections. We have developed an agent-based computer model which explicitly incorporates both distal and local modes of infection. The model consists of a two-dimensional, hexagonal grid in which each site corresponds to one, non-motile, hepatocyte (liver cell). Since experimental measures taken over the course of infection typically report both the concentration of extracellular infectious virus, as well as the count of intracellular viral RNA segments, our model also tracks both of these quantities. Within each cell, the concentration of HCV RNA is tracked and updated via an ODE model for intracellular viral replication. The intracellular concentration within each cell is, in turn, linked to the rates of extracellular release and cell-to-cell infection. In this presentation, I will showcase the range of kinetics exhibited by our model and its performance in reproducing data from experimental HCV infections in vitro.

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