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Liver spatial heterogeneities and HBV

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The liver is a spatially complex and heterogeneous network of blood and bile flows coupled with metabolic processing and a favoured target for infection by hepatic viruses. We present here a mathematical model aimed at investigating these intrinsic heterogeneities and their impact on the dynamic of the Hepatitis-B variant (HBV). Dramatic spatio-temporal scaling from individual hepatocytes to the entire liver organ invites multi-scale approaches inspiring assembly of sinusoid-level 'unit models' into a whole organ representation. Each 'unit-model' sinusoid combines individual hepatocytes communicating with blood flow in the sinus in turn connected with other 'unit-model' sinusoids aggregated into a whole liver modelling scheme. This permits investigating impacts on the whole organ of precisely distributed spatial heterogeneities such as varying HBV uptake mechanisms (e.g., the sodium-taurochloriate cotransporter or NTCP), immune cell responses (e.g., cytolytic or interferon-based) and simple efficiency of HBV replication. We present our results showing how heterogeneities of, in particular, HBV replication efficiencies may be responsible for persistent chronic infections.

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