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Mathematical modeling of HBV-induced hepatocellular carcinoma with taking into account regulatory role of both virus and liver miRNAs

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Chronic HBV infection leads to virus-induced hepatocellular carcinoma. The genome of the HBV encodes miRNA. The mechanism of microRNA action is not yet fully understood. We consider interconnected activity between molecular-genetic systems of liver and HBV with taking into account regulatory role of both virus and liver microRNA's. Mathematical modeling of intracellular processes in hepatocytes is realized on the basis of firmly established biological facts relative to control mechanisms of cells functioning, cooperative nature of biological processes, end product inhibition effect and combined feedback features in living processes. The function-differential equations for molecular-genetic systems regulatorica of cells of multicellular organisms are used as class of mathematical equations for the quantitative analysis of activity regularities of "hepatocytes-hepatitis B viruses" genetic system. The results have shown that there are stable stationary, self-oscillating solutions inherent to the normal functioning of the liver; chaotic solutions and "black hole" effect that can be identified with an abnormal functioning of the genetic system of the liver. The suppression of the functioning of the molecular genetic system of the hepatocyte is directly proportional to the level of activity of the molecular genetic system of the virus (in particular, the level of microRNA concentration). Results of researches and the revealed regulatory mechanisms allows, at computer support of laboratory and clinical researches of infectious process at hepatitis B, to define molecular-genetic bases of pathogenesis at different level of microRNA concentration, to carry out early diagnostics and forecasting of hepatocellular carcinoma during hepatitis B.

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