Receptor-mediated entry of hepatitis B virus particles into liver cells.

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Receptor-mediated entry of hepatitis B virus particles into liver cells.

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Abstract

In previous reports several receptors for either natural hepatitis B virus (HBV) particles or genetically engineered virus have been described, whereby endocytosis represents a putative uptake mechanism for HBV particles. We have found that HBV-particles from viremic carriers could bind to the human asialoglycoprotein receptor (ASGPR), which mediates glycoprotein uptake into liver cells. The HBV-ASGPR interaction was studied in a cell culture system using hepatoma HepG2 and HuH7 cells compared to COS cells as controls. About 50% of HBsAg-secretion into the cell culture supernatant after HBV-inoculation as a function of HBV-uptake could be inhibited by the specific ASGPR-ligand asialofetuin. COS-cells did not show HBsAg-secretion. If the cells were grown as clones, 15% of HepG2-cells demonstrated HBsAg-secretion but only 5% in the presence of asialofetuin. HBV-particle uptake was further confirmed by HBV-DNA analysis using PCR. HBV-ASGPR interaction was studied with purified, biotin-conjugated human ASGPR. Quantitative inhibition with asialofetuin indicated a high-affinity binding of HBV-particles to purified ASGPR. After denaturing polyacrylamid gel electrophoresis and transblotting of isolated HBV-particles a receptor-blotting system was established which identified distinct binding sites for biotinylated receptors. These results suggest that the ASGPR is capable of specifically binding HBV-particles and, moreover, to mediate their hepatic endocytosis which ultimately could be responsible for the HBV-infection of liver cells.

Reference

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