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Silver nanoparticles inhibit hepatitis B virus replication.

Lu L[1], Sun RW, Chen R, Hui CK, Ho CM, Luk JM, Lau GK, Che CM.

Author information

Abstract

BACKGROUND: Silver nanoparticles have been shown to exhibit promising cytoprotective activities towards HIV-infected T-cells; however, the effects of these nanoparticles towards other kinds of viruses remain largely unexplored. The aim of the present study was to investigate the effects of silver nanoparticles on hepatitis B virus (HBV).

METHODS: Monodisperse silver nanoparticles with mean particle diameters of approximately 10 nm (Ag10Ns) and approximately 50 nm (Ag50Ns) were prepared from AgNO3 in HEPES buffer. The in vitro anti-HBV activities of these particles were determined using the HepAD38 cell line as infection model.

RESULTS: Ag10Ns and Ag50Ns were able to reduce the extracellular HBV DNA formation of HepAD38 cells by >50% compared with the vehicle control (that is, HepAD38 cells in the absence of silver nanoparticles). Silver nanoparticles had little effect on the amount of HBV covalently closed circular DNA (cccDNA), but could inhibit the formation of intracellular HBV RNA. Gel mobility shift assays indicated that Ag10Ns bound HBV double-stranded DNA at a DNA:silver molar ratio of 1:50; an absorption titration assay showed that the nanoparticles have good binding affinity for HBV DNA with a binding constant (Kb) of (8.8 +/- 1.0)x10(5) dm(3)mol(-1). As both the viral and Ag10Ns systems are in the nanometer size range, we found that Ag10Ns could directly interact with the HBV viral particles as revealed by transmission electronic microscopy.

CONCLUSIONS: Silver nanoparticles could inhibit the in vitro production of HBV RNA and extracellular virions. We hypothesize that the direct interaction between these nanoparticles and HBV double-stranded DNA or viral particles is responsible for their antiviral mechanism.

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