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Role of HIV-1 Nef in acceleration of HCV-mediated liver disease progression

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The precise molecular mechanisms on how HIV-1 co-infection accelerates HCV-mediated liver disease progression are currently unknown. Our data showed that infectious HIV-1 virus particles failed to enter into human hepatocytic cell lines, and discernible virus replication was not observed, even when the hepatocytes transfected with HIV-1 proviral DNA were co-cultured with Jurkat T cells, suggesting that liver deterioration in the co-infected patients is not due to the replication of HIV-1 in the hepatocytes. Instead, HIV-1 Nef protein can be transferred from Nef-expressing or HIV-1-infected cells to hepatocytes through conduits, not exosomes, and the transferred Nef in the target hepatocytes altered lipid droplet formation, up-regulated subgenomic replicon expression of HCV, augmented reactive oxygen species (ROS) production, and enhanced ethanol-mediated up-regulation of HCV replication. Taken together, these data indicate that HIV-1 Nef plays an integral role in expedition of liver pathogenesis in the HIV-1/HCV co-infected hosts.

Biography

In-woo Park is currently working as Associate Professor in department of Cell Biology and Immunology, UNTHSC (University of North Texas Health Science Center). 2011 - 2013 Research Associate Professor Cell Biology and Anatomy, UNTHSC, 2007 - 2011 Research Assistant Professor Microbiology and Immunology, Indiana Uni. Sch. of Medicine, 1996 - 2007 Instructor Experimental Medicine, Harvard Medical School. He is also a member of Radiation Safety Committee.

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