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Role of HCV capsid protein on cellular lipid droplets content and localization during HCV infection

Muhammad Sohail Afzal[1,2], Najam us Sahar Sadaf Zaidi[2], Jean Dubuisson[1] and Yves Rouille[1]
[1]Université Lille Nord de France, France
[2]National University of Science and Technology, Pakistan

Hepatitis C virus (HCV) infects approximately 3% of the world's population, with more than 10 million chronically infected persons in Pakistan; establishing chronic infection in the majority of cases. HCV life cycle and lipid metabolism are tightly linked, resulting in steatosis for many patients. As lipid droplets (LDs) have emerged as crucial cellular organelles, which are necessary for persistent viral propagation and virion production, the study was designed to evaluate the role of HCV core protein in lipid droplets morphology and lipid metabolism disruption. Huh-7 cells were transfected with core expression vectors and the effect of core on cellular LDs was monitored by confocal microscopy. Core protein was localized on LDs surface and interfered with lipid droplets morphology. Core protein after localization on LDs surface increased intracellular lipid content. Lipid contents in cells expressing wild type core was higher as compared with cells transfected with a mutated core (double mutant P138A, P143A), which is deficient for LDs localization. Attachment of core to LDs induced a redistribution of LDs. The redistribution induced by core protein aggregated LDs around the nucleus in HCV-transfected cells, in a manner very similar as during HCV infection. The study confirms the role of HCV core protein in the disruption of lipid metabolism and in the redistribution of LDs during viral infection, which might be a pathway for HCV persistence and pathogenesis.

sohail.ncvi@gmail.com