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CHRONIC ETHANOL CONSUMPTION: ITS EFFECTS ON LIVER INFLAMMATION

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Alcohol-induced alterations in cell function, hepatic inflammation, and fibrosis are prominent features of liver disease in general and of alcoholic liver injury in particular. The link between these processes, however, remains unclear. A virtually universal characteristic of liver injury and subsequent inflammation is the induction of hepatocellular damage, and work from our laboratory has extensively studied the effect of ethanol administration on the hepatocyte and the process of endocytosis by these cells, using the asialoglycoprotein receptor (ASGP-R) pathway as a model. Our recent studies have shown that impaired uptake of several ligands by the ASGP-R (cellular fibronectin, carcinoembryonic antigen, and apoptotic bodies) leads to an ethanol-induced accumulation which then contributes to enhanced activation and cytokine production by non-parenchymal cells such as Kupffer cells and liver endothelial cells. The interaction of these ligands with the sinusoidal cells of the liver, as well as the cooperation and regulation between the different cell types after ethanol administration warrants further investigation and is the focus of talk. In our work we aim to acquire a better understanding of the cross-interactive associations that occur between the cell types following chronic ethanol administration, and which contribute to inflammation.