P-13
ISO-ALPHA-ACIDS FROM HOPS ATTENUATE ACUTE ALCOHOL-INDUCED LIVER STEATOSIS IN MICE
M. Landmann¹, C. Sellmann¹, C. J. Jin¹, D. Ziegenhardt¹, C. Hellerbrand², and I. Bergheim¹
¹Institute of Nutritional Sciences, S.D. Model Systems of Molecular Nutrition, Friedrich-Schiller-University Jena, 07743 Jena, Germany, and ²Department of Internal Medicine I, University of Regensburg, D-93042 Regensburg, Germany

Results of in vitro and in vivo studies suggest that consumption of beer is less harmful for the liver than intake of spirits. It further has been suggested that secondary plant compounds derived from hops like xanthohumol but also iso-α-acids may have beneficial effects on the development of liver diseases of various etiologies. In the present study, the protective effects of iso-α-acids derived from hops (0.75 mg/ kg BW) were assessed in a mouse model of acute ethanol-induced steatosis (6 g/ kg BW once intragastric) and in J774A.1 macrophages used as a model for Kupffer cells. Markers of liver damage, Kupffer cell activation and lipid peroxidation were determined by immunohistochemical methods and real-time RT PCR. Acute ethanol administration led to a significant accumulation of fat (~10-fold) in the liver, which was accompanied by a significant increase in protein levels of the inducible nitric oxide synthase (iNOS), 4-hydroxynonenal protein adducts, and plasminogen activator inhibitor 1 protein when compared to controls. In mice pre-treated with iso-α-acids these effects of ethanol were significantly attenuated. Pre-treatment of J774A.1 macrophages with iso-α-acids significantly inhibited LPS-induced mRNA expression of iNOS and IL-6 as well as the release of reactive nitrogen species. Taken together, our data suggest that iso-α-acids may attenuate the development of acute ethanol-induced liver damage in mice.