



archives

May 18-21, 2013
Exhibit Dates: May 19-21, 2013
Orange County Convention Center
Orlando, FL

[Welcome \(index.cfm\)](#)[Search
\(epsSearchDDW.cfm\)](#)[Top Ten
\(epsViewsDDW.cfm\)](#)[Help \(epsHelpDDW.cfm\)](#)

Tu2047 — 2013

Acetaldehyde-Derived Advanced Glycation End-Products Promote Alcoholic Liver Disease[View ePoster](#)*Liver and Biliary*

Presented on Tuesday, May 21, 2013 8:00 AM

Author(s): Joseph George¹, Nobuhiko Hayashi¹, Masayoshi Takeuchi², Tomiyasu Arisawa¹, Mikihiro Tsutsumi¹**Abstract**

Background: Chronic ingestion of ethanol increases acetaldehyde and leads to the production of acetaldehyde-derived advanced glycation end-products (AA-AGE). We evaluated the cytotoxicity of AA-AGE on hepatocytes and examined the role of AA-AGE in the pathogenesis of alcoholic liver disease (ALD). **Methods:** Rat hepatocyte cultures were treated with N-ethyllysine (NEL) or AA-AGE and the cell viability was evaluated using MTT assay. Male Wistar rats were fed with liquid diet containing 5% ethanol for 8 weeks followed with normal diet for 12 weeks. A group of animals were sacrificed at 4, 6, and 8th week and the remaining animals at 12, 14, 16, 18 and 20th week. The liver sections were stained for AA-AGE and 4-hydroxynonenal (4-HNE). Liver biopsy obtained from ALD patients was also stained for AA-AGE and 4-HNE. **Results:** The hepatocyte viability was significantly reduced in cultures treated with AA-AGE compared to control cultures or with NEL. Severe fatty degeneration was observed during the chronic administration of ethanol increasing from 4-8 weeks. The staining of AA-AGE and 4-HNE was strongly correlated with the degree of ALD in both rat and human. In rats, hepatic fatty degeneration was completely disappeared and the staining for both AA-AGE and 4-HNE returned to normal at 12th week of abstinence. Staining for AA-AGE and 4-HNE was completely absent in normal human liver.

Conclusions: The data demonstrated that AA-AGE is toxic to hepatocytes, but not NEL. Chronic ethanol ingestion produces AA-AGE and reactive oxygen species (ROS) contributing to the pathogenesis of ALD. Abstinence of alcohol results in complete disappearance of both AA-AGE and 4-HNE suggesting that AA-AGE may play a significant role in the pathogenesis of ALD.

* Presenting author

Disclosure: The following people have nothing to disclose: Joseph George, Nobuhiko Hayashi, Masayoshi Takeuchi, Tomiyasu Arisawa, Mikihiro Tsutsumi