Dear Joseph George:

Congratulations on having your abstract accepted for presentation at the The Liver Meeting® 2023 in Boston, MA, USA. Below you will find the date and time of your presentation along with other important information.

PRESENTATION DETAILS

Abstract Number: 2270-C, *A RAT MODEL FOR METABOLIC DYSFUNCTION-ASSOCIATED FATTY LIVER DISEASE*

Presentation Type: Abstract Poster

Presentation Date and Time: *Saturday, November 11, 2023, 8:00 AM*

Presentation Location: Poster Hall C

Poster Number (Final)	Poster Presentation Day	Poster Presentation Time	Letter Suffix
Poster #0000 - 0999	Oral Presentations	No physical poster needed	
Poster #1000 - 1999	Friday, November 10	12:00 PM – 1:00 PM	-A = Hall A (plaza level) -C=Hall C (second level)
Poster #2000 - 2999	Saturday, November 11	1:00 PM – 2:00 PM	-A = Hall A (plaza level) -C=Hall C (second level)
Poster #3000 - 3999	Sunday, November 12	1:00 PM – 2:00 PM	-A = Hall A (plaza level) -C=Hall C (second level)
Poster #4000 - 4999	Monday, November 13	1:00 PM – 2:00 PM	-A = Hall A (plaza level) -C=Hall C (second level)

ALL ABSTRACT PRESENTERS MUST:

- Register and attend The Liver Meeting® 2023. Registration is open!
- Book your housing online at onPeak.com . You may also speak to an onPeak agent by calling 1-888-254-0939 or 1-312-527-7300.
- In the **Speaker Center, accept** AASLD disclosure policies and provide your financial disclosure information if you have not already done so.
- ePosters this year will be a .pdf of your poster. Once that site is available for uploads, you will be provided those directions. ***Everyone must upload an ePoster (oral and poster).***

POSTER PRESENTER EXPECTATIONS

- Review all relevant guidelines, directions and resources found under Meeting Resources in your Speaker Center.
- Be present at your poster during the designated Presentation Time, noted above, to network and field questions about your abstract from attendees.
- Indicate all relevant financial disclosures on the bottom right-hand corner of your poster.
- Inform AASLD staff of any cancellation as soon as possible (Education@AASLD.org)
- Poster Printing Services will once again be offered through Genigraphics. Please visit www.genigraphics.com/tlm for more information.

PRESENTER EXPECTATIONS FOR PRESENTERS IN ORAL SESSIONS

- Review all relevant guidelines, directions and resources found under Meeting Resources in your Speaker Center.
- All Oral Presentations need to comply with AASLD's Disclosure Policy and the ACCME Standards for Integrity and Independence in Accredited Continuing Education for content and presentation.
- If requested, provide your slides in advance for review by the CME Committee for review for potential Conflicts of Interests.
- Inform AASLD staff of any cancellation as soon as possible (Education@AASLD.org)

You must register for the Annual Meeting on or before September 13, 2023 to receive early bird registration rates. Access into The Liver Meeting® sessions, poster or exhibit halls will not be permitted without an attendee badge. International Registrants may obtain a letter of invitation for visa purposes here.

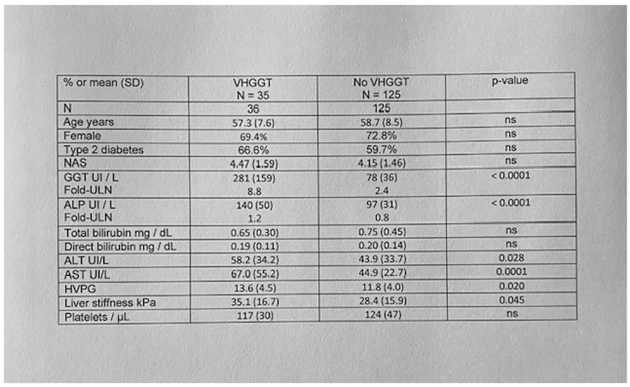
Poster printing services will be available to presenters; this offers a wonderful opportunity to upload your poster in advance of the meeting and to pick up onsite in the Poster Hall. No shipping, carrying or otherwise is needed! Please visit the Genigraphic website for more information.

AASLD Faculty Expectations and Conflict of Interest Policy
As an ACCME accredited provider offering continuing medical education for physicians, AASLD will ensure balance, independence, objectivity and scientific rigor in all of its directly and jointly provided educational activities. All educational programming is developed and must be presented in compliance with all ACCME accreditation requirements.

The Liver Meeting

Boston, Massachusetts

Nov 10-14, 2023



% or mean (SD)	VHGGT N = 35	No VHGGT N = 125	p-value
N	36	125	
Age years	57.3 (7.6)	58.7 (8.5)	ns
Female	69.4%	72.8%	ns
Type 2 diabetes	66.6%	59.7%	ns
NAS	4.47 (1.59)	4.15 (1.46)	ns
GGT UI / L Fold-ULN	281 (159) 8.8	78 (36) 2.4	< 0.0001
ALP UI / L Fold-ULN	140 (50) 1.2	97 (31) 0.8	< 0.0001
Total bilirubin mg / dL	0.65 (0.30)	0.75 (0.45)	ns
Direct bilirubin mg / dL	0.19 (0.11)	0.20 (0.14)	ns
ALT UI/L	58.2 (34.2)	43.9 (33.7)	0.028
AST UI/L	67.0 (55.2)	44.9 (22.7)	0.0001
HVPG	13.6 (4.5)	11.8 (4.0)	0.020
Liver stiffness kPa	35.1 (16.7)	28.4 (15.9)	0.045
Platelets / µL	117 (30)	124 (47)	ns

Disclosures: Eric Lawitz – 89Bio Inc., AbbVie, Akero Therapeutics, Allergan, Alnylam Pharmaceuticals Inc., Amgen, Ascelia Pharma, AstraZeneca, Axcella Health, Boehringer Ingelheim, Bristol-Myers Squibb, Conatus Pharmaceuticals, Cymabay Therapeutics, CytoDyn, DSM, Durect Corporation: Grant/Research Support (research funding from ineligible companies should be disclosed by the principal or named investigator even if that individual's institution receives the research grant and manages the funds), No, No; Akero, Boehringer Ingelheim, BMS, Intercept, Novo Nordisk, Metacrine, Sagimet, Terns: Advisor, No, No; Abbvie, Gilead Sciences, Intercept: Speaking and Teaching, No, No;
The following people have nothing to disclose: Pol Boudes, Naga P. Chalasani

2270-C | A RAT MODEL FOR METABOLIC DYSFUNCTION-ASSOCIATED FATTY LIVER DISEASE

Mutsumi Tsuchishima, Mikihiro Tsutsumi and Joseph George, Department of Hepatology, Kanazawa Medical University, Uchinada, Ishikawa 920-0293, Japan

Background: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a major health problem worldwide. It is characterized by intense deposition of fat globules in the hepatic parenchyma and is accompanied with fibrosis that could potentially progress to liver cirrhosis and hepatocellular carcinoma (HCC). We developed a rat model to evaluate the molecular mechanisms associated with the pathogenesis of MAFLD and to screen appropriate modalities to prevent MAFLD. **Methods:** SHRSP5/Dmcr rats (spontaneously hypertensive rats / stroke prone) were fed a high-fat and cholesterol (HFC) diet for a period of 12 weeks and evaluated for the development of steatosis, steatohepatitis, fibrosis, and cirrhosis. The same set of control animals received normal diet. A group of animals were sacrificed at the end of the 4th, 6th, 8th, and 12th weeks from the beginning of the experiment. Blood and liver samples were collected for biochemical and histopathological evaluations. Immunohistochemical staining was performed for α-SMA, TNF-α, 4-hydroxy-2-nonenal (4-HNE), collagen type I, and type III. **Results:** Histopathology and trichrome staining demonstrated steatosis at 4th week, steatohepatitis with progressive fibrosis at 6th week, advanced fibrosis with bridging at 8th week, and cirrhosis *at* 12th week of HFC diet administration. Biochemical markers and immunohistochemical staining for α-SMA, TNF-α, 4-HNE, and collagens type I and type III demonstrated the progression of MAFLD from simple steatosis to liver cirrhosis. The animals that received normal diet did not show any biochemical or pathological alterations. **Conclusion:** The results of the current study demonstrated that HFC diet administration to SHRSP5/Dmcr rats is a good and reproducible animal model for MAFLD and is suitable to study the molecular mechanisms associated with the pathogenesis of MAFLD and to screen appropriate therapeutic modalities. *Email: georgej@kanazawa-med.ac.jp*
Disclosures: The following people have nothing to disclose: Mutsumi Tsuchishima, Mikihiro Tsutsumi, Joseph George

2271-C | ABHD5 OVEREXPRESSION PROTECTS PNPLA3-148M PRIMARY HUMAN HEPATOCYTES FROM STEATOSIS IN LIVER CHIMERIC MICE WITH NAFLD

Ype P de Jong[1], Mohammad Kabbani[2], Aditya Upadhyay[1], Chenhui Zou[1], Corrine Quirk[3], Maha Maaliki[2], Antonis Athanasiadis[4], Luis Chiriboga[5], Fulmer G Clifton[6] and Eleftherios Michailidis[4], (1)Weill Cornell Medicine, NY, (2)Mhh, (3)Rockefeller University, (4)Emory, (5)NYU, (6)Cleveland Clinic

Background: The methionine variant at position 148 of patatin-like phospholipase domain-containing protein 3 (PNPLA3-148M) is associated with advanced fatty liver disease. Previous studies have proposed a mechanism by which PNPLA3-148M sequesters alpha/beta-hydrolase domain containing 5 (ABHD5),

Symbols: ♦, Poster of Distinction; ★, Foundation Award Recipient