



**Novel treatment of acute on chronic liver failure
using synergistic action of G-CSF and TAK
H2020 – 945096**

D7.10 Consensus conference for the use of G-TAK in subjects with steroid resistant alcoholic hepatitis (srAH) and acute-on chronic liver failure (ACLF)

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Contributors

Contributor	Contribution	Estimate of invested person months (PM)
UCL (coordinator)	<i>Speaker / Presenter, 1st draft ppt slide deck, strategy approval of Deliverable Report D7.10</i>	<i>Rajiv Jalan (0.05 PM)</i>
Charité (clinical study supervisor)	<i>Speaker / Presenter, 2nd draft ppt slide deck, approval of Deliverable Report D7.10 for submission</i>	<i>Cornelius Engelmann (0.05 PM)</i>
EASL (consortium partner)	<i>Provision of the venue and time slot for the Networking Session, promotion of the event via the EASL website and their social media channels</i>	<i>Wouter Kruijs (0.1 PM)</i>
concentris (WP7 lead)	<i>Planning of the event, final revision of ppt slide deck, social media announcements and promotion of the event on social media and website; writing and submission of Deliverable Report D7.10</i>	<i>Nina Donner (0.2 PM)</i>

Abbreviations

ACLF	Acute-on-chronic liver failure
Charité	Charité – Universitätsmedizin Berlin
CO	Confidential
concentris	concentris research management gmbh
D	Deliverable
EASL	European Association for the Study of the Liver
EASL-CLIF	European Association for the Study of the Liver and Chronic Liver Failure (Research Consortium)
EF CLIF	European Foundation for the Study of Chronic Liver Failure
ELPA	European Liver Patients Association
G-CSF	Granulocyte-Colony Stimulating Factor
G-TAK	combinatorial therapy of A-TANGO using G-CSF and TAK-242
LUMC	Leiden University Medical Center
OF	Organ Failure
PM	Person Months
Q&A	Questions and Answers
SC	Steering Committee
srAH	steroid-resistant Alcoholic Hepatitis
TAK-242	Antagonist of toll-like receptor 4 (TLR4)
TLR4	Toll-Like Receptor 4
UCL	University College London
URL	Uniform Resource Locator
WP	Work Package

Executive Summary

As a replacement of the originally planned Consensus Conference, the A-TANGO consortium held an interactive **Networking Session at EASL Congress 2026** in Barcelona, Spain, on **Friday, 29 May 2026**. The Networking Session was entitled *“The A-TANGO Score: A Novel Tool for Risk Stratification and Clinical Endpoints in Acute-on-Chronic Liver Failure (ACLF)”*. The session was organised and planned by WP7 leader concentris research management gmbh (concentris) together with the European Association for the Study of the Liver (EASL), and featured presentations by A-TANGO coordinator **Prof. Rajiv Jalan** (University College London, UCL) and **Prof. Cornelius Engelmann** (Charité – Universitätsmedizin Berlin), followed by questions and answers (Q&A) and a lively discussion with the audience during and after the event.

The session presented the rationale, development, and validation of the novel A-TANGO Organ Failure (OF) Score (Engelmann et al. 2026) and compared its performance with existing ACLF models and proposed harmonised ACLF criteria. The presentation highlighted limitations of current ACLF definitions, including difficulties in applying organ-failure criteria, inconsistencies in diagnosing ACLF grade 1, the broad mortality range associated with grade 3, and challenges in using existing scores for clinical trials and regulatory endpoints.

The **A-TANGO OF Score** was presented as a refined approach incorporating graded dysfunction across six organ systems: Liver, kidney, brain, coagulation, circulation, and respiration. Evidence suggests **improved risk prediction** and a **more differentiated 28-day mortality stratification** compared to the commonly used model by the EASL-CLIF¹ consortium. The session also described the overall **goal and approach of A-TANGO**, particularly the use of the so-called **combinatorial G-TAK² therapy** in subjects with **steroid-resistant alcoholic hepatitis (srAH) and ACLF**, compared currently **existing international ACLF definitions**, and proposed novel consensus criteria - highlighting the differences in diagnostic approaches and the need for evidence-based harmonisation.

Overall, the Networking Session highlighted the potential relevance of the novel A-TANGO OF Score for more consistent ACLF diagnosis, risk stratification, and clinical trial endpoints, as well as the potential advantages of improved **evidence-based harmonised definitions** for regulatory agreement, drug development, and clinical decision-making.

Apart from the Networking Session, consortium members also contributed **more than 20 topic-related activities for EASL Congress 2026**, including posters, scientific sessions, and talks. Together, these activities greatly increased the visibility of A-TANGO and facilitated scientific exchange within the international hepatology community.

¹ EASL-CLIF, European Association for the Study of the Liver and Chronic Liver Failure (Research Consortium)

² G-TAK, combinatorial therapy using Granulocyte-Colony Stimulating Factor (G-CSF) and TAK-242, an antagonist of toll-like receptor 4 (TLR4)

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1 Planning and Rationale

More than 10 million people worldwide suffer from decompensated cirrhosis. In its final stages, decompensated cirrhosis leads to ACLF, a syndrome characterised by systemic inflammation, fluid accumulation in the abdomen (ascites), impaired brain function (hepatic encephalopathy), internal bleeding (gastro-intestinal haemorrhage), and multi-organ failure, often leaving liver transplantation as the last resort to save a patient's life. Therefore, effective treatment of ACLF remains an urgent and unmet need.

The A-TANGO consortium set out to perform a Phase 2 clinical study of an innovative therapeutic strategy aiming to improve hepatocyte proliferation through granulocyte colony-stimulating factor (G-CSF) and to reduce systemic inflammation by repurposing TAK-242, an antagonist of toll-like receptor 4 (TLR4) – a novel combinatorial therapy, named “G-TAK”.

However, over the duration of the 5.5 years of the A-TANGO project, the consortium experienced a multitude of technical and logistical set-backs that resulted in a substantial delay of the clinical study. In practical terms, this meant that the G-TAK study did not start until July 2026 - with the first patient being screened and included in the study during the week of 6th July 2026.

1.1 Reasoning against the originally planned Consensus Conference

By the end of 2025, it became clear that the total number of patients would likely not be sufficient to justify the organisation of a meaningful *Consensus Conference on the use of G-TAK in subjects with srAH and ACLF* by 31st August 2026, as this would have required a) a statistically relevant number of patients, and b) sufficient time to analyse the data and samples collected from these patients – neither of which we were able to achieve before 31st August 2026.

Without the prospect of meaningful results from the clinical G-TAK study, the Steering Committee (SC) therefore decided against the originally planned format of a scientific Consensus Conference. Nevertheless, the newly developed A-TANGO OF Score, the overall aim of the A-TANGO project, and the mechanistic approach of the G-TAK study remained important topics to be shared and discussed with the hepatology community.

1.2 Rationale behind the Networking Session as a Replacement Event

Therefore, the SC decided on an in-depth *Networking Session during EASL Congress 2026 about the novel A-TANGO OF Score* in Barcelona, Spain, as a replacement event. The intention was an interactive, thought-provoking presentation - unafraid of scientific controversy and discussion. Aims of the session were a) to describe the rationale and validation of the A-TANGO OF Score, b) evaluate the performance of currently existing ACLF models, and c) evaluate the performance of the Harmonisation Consensus Criteria with the A-TANGO OF Score.

1.3 Planning of the Event

The logistics, planning, and promotion of the 30-min long event were a collaborative effort between consortium partner EASL and WP7 leader concentris, while the topic and content of the Networking Session were proposed and presented by the project coordinator, Prof. Rajiv Jalan (UCL), and clinical study supervisor Prof. Cornelius Engelmann (Charité). The SC oversaw and approved the activity, while the European Liver Patients Association (ELPA) supported the event's promotional activities.

The Networking Session took place on the third day of the international [EASL Congress 2026](#) (more than 8,500 attendees in total between 27-30 May) in Barcelona, Spain (venue: “Fira Gran Via” Conference Centre), namely on **Friday, 29 May 2026, from 10:00 – 10:30 h at the Cirrhosis & Complications Track Hub**. The track hub format was introduced by EASL a couple of years ago to facilitate less formal and more interactive session, especially for the presentation of novel data and results. Since then, it has proven quite successful, also because the track hubs are commonly located within the same hall as the industry booths and in direct vicinity of **topic-related poster presentations** (in our case “Cirrhosis & Complications”, see also **Chapter 5**) – making the track hubs easy to find and access.

Later the same day, consortium member **Prof. Minneke Coenraad** from Leiden University Medical Center (LUMC) chaired a topic-related poster tour about “*Cirrhosis and its complications – Experimental and Pathophysiology*”

2 Promotion of the Event

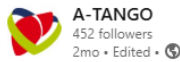
2.1 Via the Project Website



Figure 1. Announcement of the Networking Session on the *Homepage* (landing page) and the *Project News* subpage of the project website a-tango.eu.



2.2 Via the Project's Social Media Channels



Excited to announce the A-TANGO [#Networking](#) Session at [#EASLCongress](#) 2026 in Barcelona - Join us and engage in the discussion!

Friday, 29 May 2026
10:00 – 10:30 h (CEST)
Cirrhosis & Complications (Track Hub 6)

Speakers:
[Cornelius Engelmann](#) (Charité - Universitätsmedizin Berlin)
[Rajiv Jalan](#) (UCL, Yaqrıt)

Session title: The A-TANGO Score: A Novel Tool for Risk Stratification and Clinical Endpoints in [#ACLF](#)

The networking session will highlight emerging data behind the A-TANGO organ failure score and its implications for how we define, stratify, and study acute-on-chronic liver failure (ACLF). Recent multinational studies from the A-TANGO collaboration have shown that outcome-calibrated approaches may identify clinically actionable high-risk patients more effectively than current consensus-based definitions - with important consequences for bedside decision-making, trial design, and regulatory endpoints. The discussion comes at a particularly important moment for the field, alongside ongoing international efforts to harmonise ACLF definitions globally ahead of the "EASL Global ACLF Consensus Meeting".

Join us to discuss:

- ✓ Novel approaches to ACLF risk stratification
- ✓ Outcome-based clinical endpoints
- ✓ Implications for future clinical trials
- ✓ The evolving global ACLF consensus landscape

Looking forward to seeing colleagues, collaborators, and friends from across the hepatology community in Barcelona!

[#EASL](#) [#EASL2026](#) [#Hepatology](#) [#LiverDisease](#) [#Cirrhosis](#) [#AlcoholicHepatitis](#)
[#PatientStratification](#) [#DecompensatedCirrhosis](#) [#ClinicalResearch](#)
[#TranslationalMedicine](#) [#CriticalCare](#) [#ATANGO](#)



A-TANGO Project @a-tango.bsky.social · 2mo
Join the A-TANGO Networking Session at [#EASLCongress](#)!

Fri, 29 May 2026
10:00–10:30
Cirrhosis & Complications Track Hub 6

The A-TANGO Score: A Novel Tool for Risk Stratification & [#ClinicalEndpoints](#) in [#ACLF](#)

[@rajivjalan.bsky.social](#)
[@easlnews.bsky.social](#)
[@europeliver.bsky.social](#)



4

Figure 2. Posts announcing the Networking Session on the project's social media channels, [LinkedIn](#) (upper part) and [Bluesky Social](#) (lower part, left side), as well as [EASL's LinkedIn account](#) (lower part, right side).



You and 40 others

2 comments · 4 reposts



EASL | The Home of Hepatology
15,856 followers
3mo ·

Meet A-TANGO at [#EASLCongress](#) 2026! Representatives Cornelius Engelmann and Rajiv Jalan will be discussing the A-TANGO Score: A Novel Tool for Risk Stratification and Clinical Endpoints in ACLF, and answer all your questions!

Friday, 29 May 10:00-10:30
Track Hub 6

Register to EASL Congress: <https://lnkd.in/e-gipwBx>

[#LiverTwitter](#) [#LiverX](#)



Friday, 29 May



Track Hub 6

10:00–10:30



Cornelius Engelmann
Rajiv Jalan

7

3 reposts

3 Execution of the Networking Session

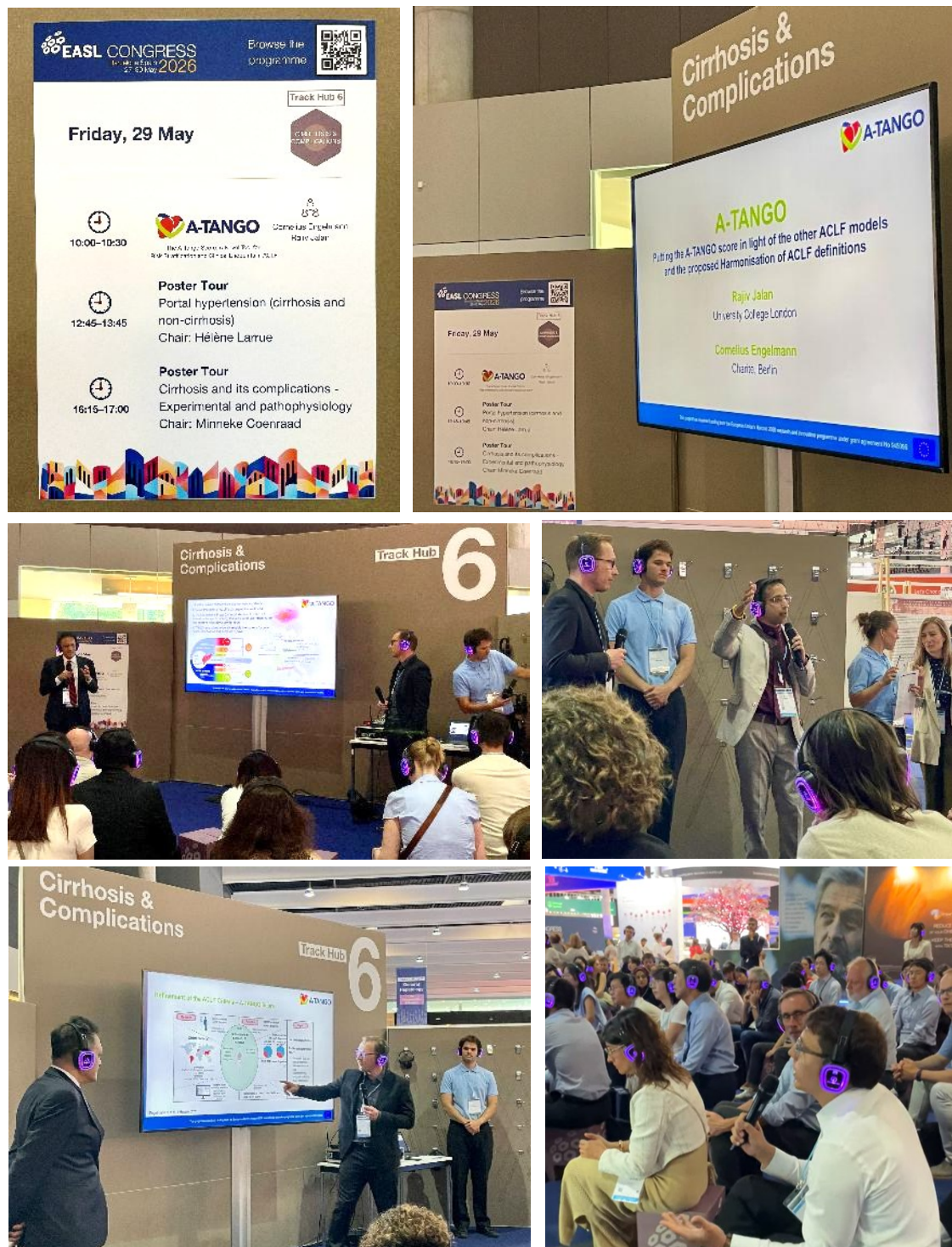


Figure 3. Photographs of the on-site promotional poster by EASL (upper left) and of the actual Networking Session at EASL Congress 2026 (other photos). All seats (> 50 cubic chairs) were filled and several more people had grabbed headphones to listen to the session while standing. The presentation by Rajiv Jalan (UCL) and Cornelius Engelmann (Charité) generated a lively discussion with the audience, including - for example - Nipun Verma, a hepatology professor from Chandigarh, India.

4 Social Media Coverage



Thanks for the great attendance & active discussion this morning at #EASLCongress! It was a very insightful & interactive A-TANGO #NetworkingSession on the evolving definition and #RiskStratification of acute-on-chronic liver failure (#ACLF), highlighting the development of the new #ATANGOScore and the urgent need for international #Harmonisation of ACLF criteria.

The presentation reviewed limitations of current ACLF classifications, including diagnostic complexity, variability between regions, and challenges for clinical trials, regulatory approval, and patient selection. The proposed A-TANGO #OrganFailure (OF) Score introduces refined and more reproducible criteria for liver, kidney, brain, coagulation, circulatory, and respiratory dysfunction, aiming to improve prognostic accuracy and clinical applicability.

Key findings that were presented by project coordinator **Rajiv Jalan** (UCL) and clinical study supervisor **Cornelius Engelmann** (Charité - Universitätsmedizin Berlin):

- The A-TANGO score demonstrated improved prediction of 28-day mortality compared with existing models.

- Simplified and quantitative definitions may reduce diagnostic uncertainty and improve trial design.

- Comparative analyses showed important differences between current ACLF definitions (EASL-CLIF, COSSH, APASL, NACSELD).

- Proposed harmonisation strategies may help align global ACLF diagnosis and facilitate drug development and regulatory acceptance.

- An important takeaway was that harmonisation must remain evidence-based and carefully integrated into clinical practice, particularly given implications for liver transplantation prioritisation and therapeutic innovation.

- An exciting glimpse into the future direction of ACLF research, precision risk stratification, and international collaboration.

#EASL2026 #Hepatology #LiverDisease #ClinicalResearch #TranslationalMedicine #Cirrhosis



A-TANGO Project @a-tango.bsky.social · 2mo
Great attendance & active discussion at #EASLCongress today! Very insightful & interactive Networking Session on the evolving definition and #RiskStratification of ACLF, highlighting the new #ATANGOScore & the urgent need for international #Harmonisation of #ACLF criteria.
@rajivjalan.bsky.social



Figure 4. Live coverage posts about the Networking Session on the project's social media channels, [LinkedIn](#) (upper part) and [Bluesky Social](#) (lower part).

5 Related Activities of A-TANGO Partners at EASL Congress 2026

In addition to the Networking Session, members of the A-TANGO consortium, particularly from consortium partners LUMC, UCL, and Charité also contributed **more than 20 other activities to EASL Congress 2026, including posters, scientific sessions, and talks.** Together, these activities greatly increased the visibility of A-TANGO and its consortium partners - facilitating scientific exchange within the international hepatology community.

Shown below are **11 exemplary scientific contributions** that were highlighted on the project's social media channels as their topics were related to the overall scientific scope and interest of the A-TANGO project. Also, to increase visibility, the >20 activities were announced in a [preview post](#) and also in the early morning hours of the respective congress day via a short promotional video for each day.




A-TANGO organ failure (OF) score performs better than EASL-CLIF, NACSELD and APASL scores for the diagnosis of acute-on-chronic liver failure (ACLF) in the independent COSHH cohort

Comparison of Diagnostic Models and Prognostic Scores of ACLF: Evidence Towards Global Harmonization

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Introduction
 Acute-on-chronic liver failure (ACLF) is associated with high short-term mortality, yet existing diagnostic models show substantial heterogeneity in patient identification across regions. This inconsistency leads to discordant risk stratification and may directly influence clinical decision-making, including intensive care allocation and liver transplantation prioritisation. A systematic comparison across major diagnostic frameworks is therefore needed to inform harmonization.

Aim
 To perform a head-to-head comparison of ACLF diagnostic and prognostic models to evaluate concordance, mortality prediction, and clinical utility, and to inform harmonization.

Method
 We analysed 3,370 patients with acute decompensation of cirrhosis in the COSHH cohort, with external validation in an independent Aalto-Spectre cohort from India (n=2,855). Five diagnostic models were assessed for 28-day mortality risk. Reclassification was evaluated using net reclassification improvement, and prognostic scores were compared using concordance index, integrated discrimination improvement, calibration, and decision-curve analysis.

Results
 A total of 3,370 patients were included. The five diagnostic models identified markedly different proportions of ACLF: COSHH-ACLF (n=1,155), A-TANGO (n=1,134), EASL-CLIF (n=796), APASL-ACLF (n=497), and NACSELD-ACLF (n=511). A-TANGO identified most consistently with COSHH-ACLF (n=941), and this showed subgroup consistency in 15-day, 28-day and 90-day (15-day mortality 15.8% and 28-day mortality 18.8% and 90-day mortality 25.8%).

Conclusions
 A-TANGO and COSHH-ACLF showed consistent performance across cohorts, while their discordance identified a clinically meaningful intermediate-risk group. Outcome-based models demonstrated robust predictive ability, supporting a data-driven framework for global harmonization of ACLF evaluation.

References
 1. Hu M, et al. (2025) A-TANGO vs COSHH-ACLF for ACLF diagnosis. *Journal of Hepatology*.
 2. Hu M, et al. (2025) A-TANGO vs EASL-CLIF for ACLF diagnosis. *Journal of Hepatology*.
 3. Hu M, et al. (2025) A-TANGO vs APASL-ACLF for ACLF diagnosis. *Journal of Hepatology*.
 4. Hu M, et al. (2025) A-TANGO vs NACSELD-ACLF for ACLF diagnosis. *Journal of Hepatology*.

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at **EASL CONGRESS**
Barcelona Spain 2026
27-30 May



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Body Composition is a Predictor of Treatment Response and Survival in Hepatorenal Syndrome (HRS-AKI)

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CHARITÉ
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INTRODUCTION

Advanced liver cirrhosis is associated with complex systemic complications, including hepatorenal syndrome (HRS-AKI) and sarcopenia. Despite frequent co-occurrence, the relationship between body composition, treatment response and survival remains unclear. Terlipressin plus albumin is standard of care, yet 30-40% of patients do not respond. AI-based CT body composition analysis (BCA) may help identify risk factors in this vulnerable population.

AIM

To investigate BCA-derived predictive markers for treatment response and 90-day transplant-free survival in patients presenting with HRS-AKI.

METHOD

CONSORT
Retrospective screening of admissions at Charité Campus Virology (2016-2022) for liver cirrhosis and HRS.

INCLUSION CRITERIA
- Cirrhosis (EASL criteria)
- Treatment with terlipressin plus albumin
- Radiologic CT scans (no metal)

EXCLUSION CRITERIA
- Stage 4/5 chronic kidney disease
- Malignant tumor (except HCC)
- Age < 50

ENDPOINTS
- Treatment response: 225% reduction in peak creatinine on day 7
- 90-day transplant-free survival

BODY COMPOSITION ANALYSIS
- Multivariate regression of lean mass at L3
- Height-adjusted indices (SMI, SMI²)

STATISTICS
- Regression and correlation analysis
- Independent predictors of treatment response and 90-day mortality
- ROC curve analysis for C-statistic

RESULTS

Parameter	Response	Non-Response	p-value
Age	60.4 (SD 10.5)	60.4 (SD 10.5)	0.95
Sex	48 (88.9%)	48 (88.9%)	0.95
Child-Pugh	10 (18.5%)	10 (18.5%)	0.95
Crackles	10 (18.5%)	10 (18.5%)	0.95
Body Composition			
SMI	12.5 (SD 1.5)	12.5 (SD 1.5)	0.95
SMI ²	156.2 (SD 25.0)	156.2 (SD 25.0)	0.95

PREDICTORS OF MORTALITY

In a multivariate competing risk model, day 7 treatment response was a significant protective factor for 90-day mortality (HR 0.42; p = 0.025). While SMI, SMI², and VFI also showed no association with transplant-free survival, a lower SMI was associated with higher mortality risk, reaching borderline statistical significance.

PREDICTORS OF TERLIPRESSIN RESPONSE

Multivariate Cox-Regression models identified higher SMI (HR 1.55; p = 0.02) and higher VFI values (HR 1.30; p = 0.02) as independent predictors of terlipressin response (figure 3).

CONCLUSIONS

Reduced muscle mass (SMI) and altered muscle-to-fat distribution (VFI) are independent predictors of terlipressin non-response which is a critical determinant of 90-day transplant-free survival. BCA-based stratification may help guide early treatment strategies in HRS-AKI.

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Tacke F et al. "Effect of terlipressin on survival in patients with cirrhosis: a meta-analysis." *J Hepatol*. 2022; 76(3):539-546.



Sina Oesinghaus, Charité, Berlin, Germany



at **EASL CONGRESS**
Barcelona Spain 2026
27–30 May



Loss of barrier-supportive RELM α macrophages promotes gut barrier failure and organ injury in alcohol-related hepatitis

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Introduction

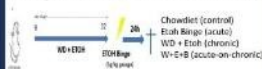
Patients with alcohol-related hepatitis (AH) are at high risk for progression to acute-on-chronic liver failure (ACLF). Intestinal dysbiosis and gut barrier dysfunction contribute to organ injury and inflammation, yet the underlying mechanisms remain poorly understood.

Aim

We investigated gut barrier dysfunction and the role of intestinal macrophages in organ injury during acute-on-chronic metabolic injury.

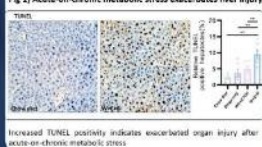
Methods

Spectral flow cytometry; macrophage phenotyping
Multiplex IHC; spatial organ zonation
Multiphoton intravital microscopy; barrier dysfunction & in vivo model
16S rRNA amplicon sequencing; dysbiosis
Single-cell RNA sequencing; transcriptional profiling
Colon organoids & Cx3CR1 cell co-culture; functional in vitro assays



Results: From Liver Injury...

Fig 1) Acute-on-chronic metabolic stress exacerbates liver injury



Results: ...To Gut Dysfunction

Fig 2) Metabolic stress disrupts colonic barrier, reduces Cx3CR1-GFP⁺ cell size and accumulates of Ly6C⁺ monocytes

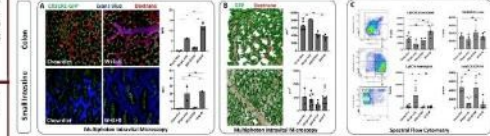


Fig 3) Dysfunctional CX3CR1⁺ cells fail to promote colon organoid regeneration, which is linked to reduced epithelial YAP signaling in vivo

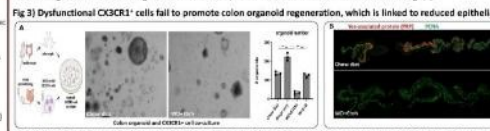


Fig 4) Loss of RELM α ⁺ macrophages and inflammatory M1 polarization

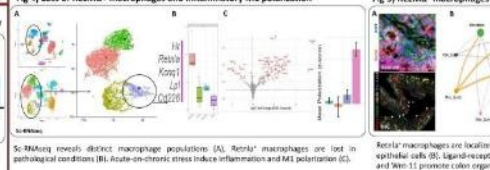
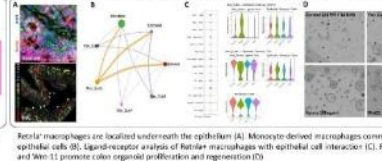


Fig 5) RELM α ⁺ macrophages promote epithelial regeneration via RELM α , Fc γ 1 and Wnt11



Conclusion

The colon is a central driver of organ injury in metabolic and alcohol-related liver disease. Loss of gut function and altered colonic macrophage composition emerge as key contributors, highlighting potential targets to prevent progression to ACLF. We identify a crypt-adjacent, barrier-supportive RELM α ⁺ macrophage population whose loss under metabolic stress is associated with increased gut permeability and susceptibility to secondary insults in the gut and liver. We further elucidate the ligand-receptor crosstalk by which RELM α ⁺ macrophages signal to the epithelium to promote proliferation and regeneration.



Joscha Vonderlin et al., Charité, Berlin, Germany



at **EASL CONGRESS**
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Reduced thrombin potential is associated with bleeding events and short-term mortality in acute decompensation and acute-on-chronic liver failure

EASL CONGRESS
THIRTY-SEVEN

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Background

In liver cirrhosis, haemostasis is rebalanced through concomitant alterations of pro- and anticoagulant systems, which limits the assessment of plasmatic procoagulation by conventional coagulation tests (e.g. INR, platelet count). Thrombin generation assays (TG) allow an integrative assessment of the plasmatic capacity for thrombin formation.

Method

In 136 participants (AD n = 76, ACLF grades I-III n = 46; controls n = 13), Elap S[®] Peak, peak thrombin, velocity index (VI), and endogenous thrombin potential (ETP) were determined using platelet-poor plasma thrombin generation (PPP-TG) without thrombomodulin.

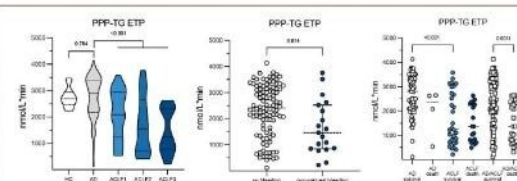
Results

Compared with AD, patients with ACLF exhibited more pronounced laboratory abnormalities (INR, bilirubin, creatinine; all $p < 0.01$) and greater disease severity (MELD 25 vs. 14; CLIF-C OF 9 vs. 5, $p < 0.001$). The onset of thrombin generation was delayed in ACLF (median lag 5.0 minutes) compared with AD (3.85 min) and controls (3.8 min, $p = 0.007$), with a comparable tPeak ($p = 0.148$). Compared with controls, patients with AD already showed a reduced peak thrombin generation (253.4 vs. 187.5 nM, $p < 0.05$). However, overall plasmatic thrombin potential (ETP) was preserved between AD and controls (2669.6 vs. 2714.4 nM, $p = 0.784$). In ACLF, ETP was markedly reduced (1266.9 nM, $p < 0.001$ vs. AD/controls). CLIF-C organ failures (liver, kidney, brain, circulation) correlated with low ETP (all $p < 0.05$). In patients with AD/ACLF, reduced ETP was associated with an increased 30-day risk of (non-variceal) bleeding and 90-day mortality (both $p < 0.05$). Higher ETP values were, in univariate analyses, associated with a lower hazard ratio (HR) for non-variceal bleeding within 30 days and for 90-day mortality. An exploratory elastic net-weighted TGA model (peak, VI, ETP) provided robust prognostic performance (HR 0.546; 95% CI 0.403–0.739; $p = 0.001$) and remained independently significant after adjustment for platelet count, INR, age, or aetiology.

Cox proportional hazard models for 90-day mortality in patients with acute decompensation (AD) and acute-on-chronic liver failure (ACLF).

	Multivariate model 1	
Linear TG-Score (ETP/peak-height/VI)	0.621 (0.45–0.857)	0.004
Adjusted for platelet count	0.618 (0.449–0.852)	0.003
	Exploratory multivariate model 1	
Predictive TG-model (Elastic net regression, outcome-weighted)	0.546 (0.403–0.739)	0.001
Adjusted for platelet count	0.529 (0.391–0.716)	0.001
	Exploratory multivariate model 2	
Adjusted for INR	0.645 (0.451–0.921)	0.016

Variables for multivariate model selection are based on multiparametricity to avoid overfitting. Variables selected for elastic net regression are identical to linear TG-score and additionally outcome-weighted (0.5–2) for predictive fitness (elastic net).



Plasmatic capacity is reserved in AD, but markedly reduced in ACLF

Violin plots of PPP thrombin generation (PPP-TG) endogenous thrombin potential (ETP) values stratified by disease group (Healthy controls HC, Acute decompensation AD, acute-on-chronic liver failure ACLF). Central lines indicate medians with interquartile ranges. P values were calculated using the Kruskal-Wallis test with Dunn's post hoc multiple comparisons.

ETP is reduced in patients with non-variceal bleeding within 30 days after TG. Dot plots of PPP thrombin generation (PPP-TG)

endogenous thrombin potential (ETP) values in patients with acutely decompensated cirrhosis (AD) and acute-on-chronic liver failure (ACLF) who developed a clinically significant, non-variceal bleeding event within 30 days following TG assessment. P values were calculated using the Mann-Whitney U test.

ETP is reduced in patients with AD/ACLF who died within 90 days after TG. Dot plots of PPP thrombin generation (PPP-TG)

endogenous thrombin potential (ETP) values in patients with acutely decompensated cirrhosis (AD) and acute-on-chronic liver failure (ACLF) grouped into survival or death within 90 days. P values were calculated using the Kruskal-Wallis test with Dunn's post hoc multiple comparisons.

Conclusions

While plasmatic thrombin-generating reserve is preserved in AD, the development of ACLF represents a turning point characterized by a marked reduction in endogenous thrombin potential. Individual and composite TGA parameters may be suitable plasmatic coagulation markers for risk stratification in acutely ill patients with cirrhosis.



Alex Zubko, Charité, Berlin, Germany



at  **EASL CONGRESS**
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Why does ammonia damage hepatocyte mitochondria?

Because it activates a TLR4-dependent pathway that impairs Complex IV.

YAQ005 preserved Complex IV activity and respiratory capacity despite ammonia exposure

These findings identify TLR4 as a direct mediator of ammonia-induced mitochondrial dysfunction.

Contact information
Supachaya Sriphoosanaphan, M.D., Ph.D.
Email: S.Sriphoosanaphan@ucl.ac.uk

Ammonia-Induced Mitochondrial Bioenergetic Failure is Driven Through a TLR4-dependent Mechanism that Specifically Targets Complex IV

Supachaya Sriphoosanaphan^{1,2,3}, Francesco De Chiara⁴, Fausto Andreola¹, Harry Horsley⁵, Nicola Mordan⁶, Jan-Willem Taanman⁷, Rajiv Jalan¹

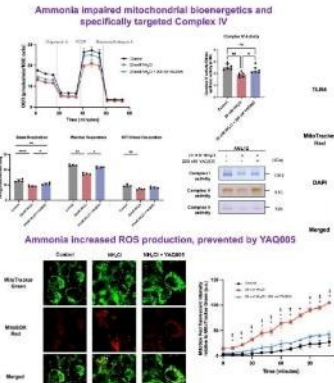
¹ Liver Failure Group, Institute for Liver and Digestive Health, University College London, London, UK; ² School of Hepatology, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand; ³ Centre of Excellence in Liver Diseases, King Chulalongkorn Memorial Hospital, The Rajabhat Siriraj, Bangkok, Thailand; ⁴ Institute for Biomechanics of Cellular Responses, Spain; ⁵ Centre for Mitochondrial Research, University College London, London, UK; ⁶ Biomedical Research Unit, University College London, London, UK; ⁷ Department of Clinical and Molecular Biomedicine, Queen's Medical Centre, University of Nottingham, Nottingham, UK

Introduction

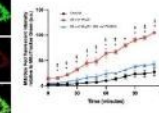
- Hyperammonemia causes mitochondrial dysfunction, but the mechanism linking ammonia to organelle failure remains incompletely defined.
- We previously identified TLR4 as a critical mediator of ammonia-induced liver injury and hypothesized that TLR4 may directly drive hepatocyte mitochondrial dysfunction^{1,2}.

Results

Ammonia impaired mitochondrial bioenergetics and specifically targeted Complex IV



Ammonia increased ROS production, prevented by YAQ005



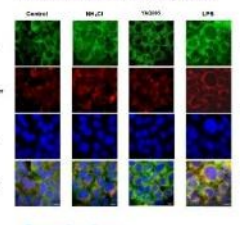
Aim

- To define the role of TLR4 in ammonia-induced mitochondrial dysfunction and identify the mitochondrial processes affected by TLR4 signalling.

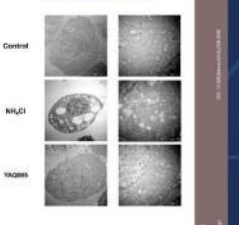
Methods

- AML12 hepatocytes were exposed to NH₄Cl with or without YAQ005.
- Mitochondrial respiration, OXPHOS activity, ROS, membrane potential, TLR4 localisation, and ultrastructure were assessed using Seahorse, spectrophotometry, BN-PAGE, live-cell confocal imaging, immunofluorescence, and TEM.

Ammonia increased TLR4 recruitment to the mitochondrial network, reduced by YAQ005



Ammonia induced mitophagy, attenuated by YAQ005



Conclusions

- Ammonia directly induced mitochondrial dysfunction in hepatocytes.
- This effect was associated with TLR4 upregulation, mitochondrial recruitment, and specific inhibition of Complex IV.
- TLR4 blockade with YAQ005 preserved mitochondrial bioenergetics, supporting TLR4 as a therapeutic target in hyperammonemia.

References: 1. Kerbert A et al. Sci Adv. 2025 2. Sriphoosanaphan S et al. EASL 2025



Supachaya Sriphoosanaphan et al., UCL, London, UK



at  **EASL CONGRESS**
Barcelona Spain 2026
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Why does ammonia lowering fail in ACLF?

Because ammonia is only part of the problem.

YAQ006 + YAQ005 achieved 100% coma-free survival in ACLF

This synergistic effect was seen in inflammatory ACLF but not in OTC deficiency.

Contact information
Supachaya Sriphoosanaphan, M.D., Ph.D.
Email: S.Sriphoosanaphan@ucl.ac.uk

Targeting Disrupted Immunometabolism-Induced Mitochondrial Bioenergetic Failure Using YAQ006 and YAQ005 Reduces Mortality in ACLF

Supachaya Sriphoosanaphan^{1,2,3}, Annarein Kerbert⁴, Tingting Qi¹, Abeba Habtesion¹, Fausto Andreola¹, Jan-Willem Taanman⁴, Francesco De Chiara⁵, Rajiv Jalan¹

¹ Liver Failure Group, Institute for Liver and Digestive Health, University College London, London, United Kingdom; ² Division of Gastroenterology, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand; ³ Centre of Excellence in Liver Diseases, King Chulalongkorn Memorial Hospital, Thai Red Cross Society, Bangkok, Thailand; ⁴ Department of Clinical and Biomedical Neurosciences, Queen Square Institute of Neurology, University College London, London, United Kingdom; ⁵ Institute for Biomedical Research, University of Valencia, Spain

Introduction

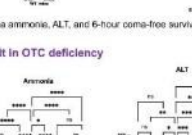
- Hyperammonaemia contributes to organ dysfunction and poor outcomes in ACLF.
- Our prior multi-omic work suggests that ammonia drives mitochondrial bioenergetic dysfunction through a TLR4-dependent immunometabolic pathway¹.
- Aim:** To test whether combining ammonia scavenger (Ornithine Phenylacetate, YAQ006) with TLR4 inhibitor (YAQ005) produces synergistic benefit in ACLF.

Methods

ACLF model



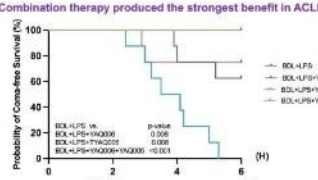
OTC-deficient model



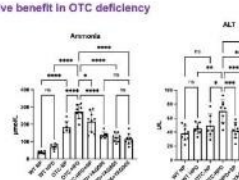
Endpoints: plasma ammonia, ALT, and 6-hour coma-free survival.

Results

Combination therapy produced the strongest benefit in ACLF



No additive benefit in OTC deficiency



Conclusions

- YAQ006 + YAQ005 produced synergistic benefit in inflammatory ACLF.
- No additive benefit was seen in OTC deficiency, suggesting disease-context-specific synergy.
- Dual targeting of ammonia and TLR4 may offer a new therapeutic strategy for ACLF.

References: 1. Sriphoosanaphan S et al. EASL 2025



Supachaya Sriphoosanaphan et al., UCL, London, UK



at **EASL CONGRESS**
Barcelona Spain 2026
27–30 May

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4. Department of Pathology, Affiliated Hospital of Nantong University, Nantong, 226001, China

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Acknowledgments

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Modulation of the gut microbiome by Yaq-001 ameliorates fecal microbiome transplant (FMT) induced liver injury from cirrhotic Specific Pathogen-Free mice to Germ-Free Mice

Jinxia Liu^{1,2*}, Fernando Garcia-Gonzalez³, Haining Wu⁴, Qin Jin⁴, Hanyan Jia⁵, Saad Shoukri⁴, Rajiv Jalan^{1*}

Introduction

The gut microbiome is critically involved in the pathogenesis of liver diseases. Yaq-001, a non-absorbable, gut-restricted microbiome therapeutic, has demonstrated potential in restoring microbiome balance, mitigating endotoxemia, systemic inflammation, hepatic fibrosis, and liver cell death in specific pathogen-free (SPF) mice with liver cirrhosis.

Aim

This study aims to investigate the mechanisms through which Yaq-001 modulates the liver-gut axis using FMT from SPF to germ-free (GF) mice.

Method

Two mouse models of cirrhosis were utilized: (a) SPF mice: carbon tetrachloride (CCl₄)-induced cirrhosis for 6-weeks in SPF mice; 4 groups: control (n=6), control-Yaq-001 (n=6), CCl₄ (n=12), and CCl₄-Yaq-001 (n=12); and (b) GF mice: Fecal samples from SPF mice were used for FMT in GF mice for 8-weeks, combined with CCl₄ administration for 3 weeks; 4 groups: FMT from SPF control (n=6), FMT from SPF control-Yaq-001 (n=6), FMT from SPF CCl₄ (n=10), and FMT from SPF CCl₄-Yaq-001 (n=10). Yaq-001 (0.4 g/100 g body weight per day) was administered to SPF mice for 6-weeks. Blood and tissue samples were collected for subsequent analysis of biochemistry, colonic and liver bulk RNA sequencing, and fecal samples for 16S RNA microbiome analysis.

Results

GF mice that received FMT from Yaq-001-pretreated CCl₄-exposed SPF mice donors exhibited markedly attenuated liver damage, as evidenced by significantly reduced serum ALT, hepatic fibrosis (CPA), apoptosis (TUNEL), and endotoxemia, compared to those receiving microbiota from untreated SPF-CCl₄ donors. Microbiome analysis confirmed that Yaq-001 pretreatment reshaped microbial communities, enriching for beneficial taxa negatively correlated with injury biomarkers and depleting harmful taxa positively correlated with damage. Transcriptomic profiling revealed that Yaq-001's effects extended to host gene expression, modulating pathways involved in metabolism and stress response in both the liver and intestine. A key finding was the upregulation of intestinal Cyp2e1 following Yaq-001, which correlated negatively with injury markers.

Conclusions

The core mechanisms underlying the protective effect of Yaq-001 on inflammation and liver injury involves restructuring the gut microbiome, which in turn regulates host gene expression programs both locally and also in distant organs such as the liver. The data support its future development as a microbiome therapeutic for diverse diseases associated with dysbiosis.

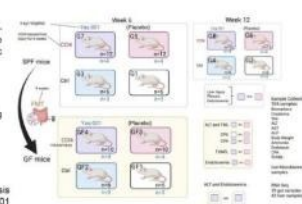


Figure 1: Experimental design

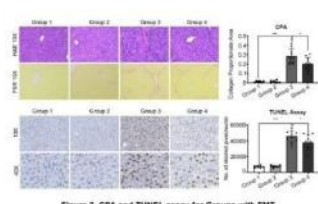


Figure 2: CPA and TUNEL assay for Groups with FMT.



Jinxia Liu et al., UCL, London, United Kingdom

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EASL CONGRESS
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27–30 May **2026**

Poster tour: Cirrhosis and its complications - Experimental and pathophysiology



Chaired by Minneke Coenraad,
LUMC, Leiden, The Netherlands



0:13

1x





A-TANGO



EASL CONGRESS

Barcelona Spain 27-30 May 2026

Incidence and implications of abstinence-induced recompensation in alcohol-related cirrhosis






Frank Tacke (Charité, Berlin) chairing the session

Annalisa Berzigotti (Switzerland) presenting as "challenger"

Play

0:08 1x

6 Conclusion

In summary, the Networking Session at EASL Congress 2026 successfully replaced the originally planned Consensus Conference and provided an effective platform to disseminate and discuss key A-TANGO results, particularly the A-TANGO Organ Failure (OF) Score, with the international hepatology community. The session presented the rationale and validation of the novel A-TANGO OF Score, highlighted its potential for improved ACLF diagnosis and risk stratification, and addressed the need for more consistent and evidence-based ACLF definitions for clinical trials and regulatory purposes.

The strong attendance of 50-75 hepatologists (including both clinicians as well as basic researchers) and lively discussion with the audience demonstrated substantial interest in the topic, while more than 20 additional A-TANGO-related scientific contributions at EASL Congress further increased the project's visibility and facilitated scientific exchange about the most recent scientific outputs by consortium partners.

Although the originally envisaged Consensus Conference could not be conducted as planned due to the limited availability of clinical study data within the project timeframe, the replacement Networking Session ensured continued dissemination of the project's scientific achievements and supported dialogue on the future harmonisation of ACLF definitions, risk stratification, and clinical endpoints.

7 References

Engelmann C and Berg T (2023). Clinical practice guidelines for acute-on-chronic liver failure: are we ready for reaching global consensus? *HepatoBiliary Surgery and Nutrition*, 12(2): pp. 239-243. DOI: 10.21037/hbsn-23-6

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Verma N et al. (2026). Consensus versus Outcome-based Definitions of Acute-on-chronic Liver Failure: Implications for Risk Stratification and Case Identification. *Journal of Clinical and Translational Hepatology*, 14(5): pp. 525–540. DOI: 10.14218/JCTH.2026.00267

8 Relevant Cohorts, Data Sources, and Clinical Trials

NCT04620148 – clinical trial identifier cited in the discussion of the limitations of the CLIF-C ACLF score for G-CSF trials.

COSSH cohort (China), n=3,370 – used to compare the performance of different ACLF models for 28-day mortality.

TIH cohort (India), n=2,398 – used in the comparison of consensus criteria and the A-TANGO Score.

CATCH-LIFE cohort (China), n=2,568 – used in the comparison of consensus criteria and the A-TANGO Score.

9 Acknowledgement and Disclaimer

This project has received funding from the European Union’s Horizon 2020 research and innovation programme under grant agreement No 945096. This report reflects only the author’s view and the Commission is not responsible for any use that may be made of the information it contains.

10 APPENDIX

10.1 Slide Deck of the Networking Session



A-TANGO

Putting the A-TANGO score in light of the other ACLF models and the proposed Harmonisation of ACLF definitions

Rajiv Jalan

University College London

Cornelius Engelmann

Charite, Berlin

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



- **10 million cases** of decompensated cirrhosis **worldwide**
- Effective **treatment of ACLF** is an **urgent & unmet need**
- A-TANGO performs **Phase 2 clinical studies** of G-TAK, an innovative therapeutic strategy that aims to **reduce inflammation** and **improve hepatocyte proliferation**
- A-TANGO also strives to identify **reliable biomarkers** for better patient stratification and increased survival



www.a-tango.eu

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



We tango together to fight ACLF



 Rajiv Jalan UCL, Hepyx, EF CLIF Scientific Coordinator	 Cornelius Engelmann Charité Clinical Study Lead	 Anna Bosch EF CLIF General Manager	 Lidia Garcia-Campmany EF CLIF Science Communication
 Cristina Sánchez EF CLIF Data Manager	 Carrie Morgan Yaqrit Clinical Study Design	 Christiana Krammer concentris Project Management	 Nina Donner concentris Science Communication
 Michał Kowalski Hepyx Drug Manufacturing	 Troels Jordansen Yaqrit CEO	 Bircu Kiper CrowdHelix Liver Health Helix	 Fausto Andreola UCL Translational Research
 Thomas Berg ULEI, EASL Hepatology Expert	 Minneke Coenraad LUMC Clinician	 Louise Perrault IMAC Market Access	 Isabelle Durand-Zaleski APHP Health Economics
 Wouter Kruijs EASL Event Management	 Annarein Kerbert LUMC Early-Career Scientists' Speaker	 Marko Korenjak ELPA Patient Advocacy	 Veronika Vsetickova ELPA Patient Advocacy

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Aims of this session

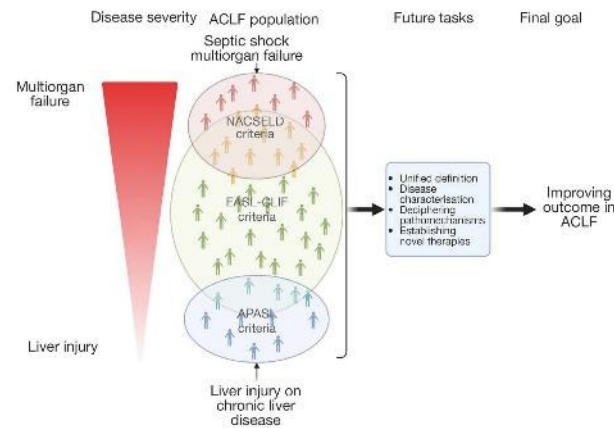


- Describe the rationale and validation of the ATANGO score
- Evaluate the performance of the current ACLF models
- Evaluate the performance of the Harmonisation Consensus criteria with the ATANGO score

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ACLF criteria – the current status



Engelmann C et al. HepatoBiliary Surg Nutr 2023

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Rationale

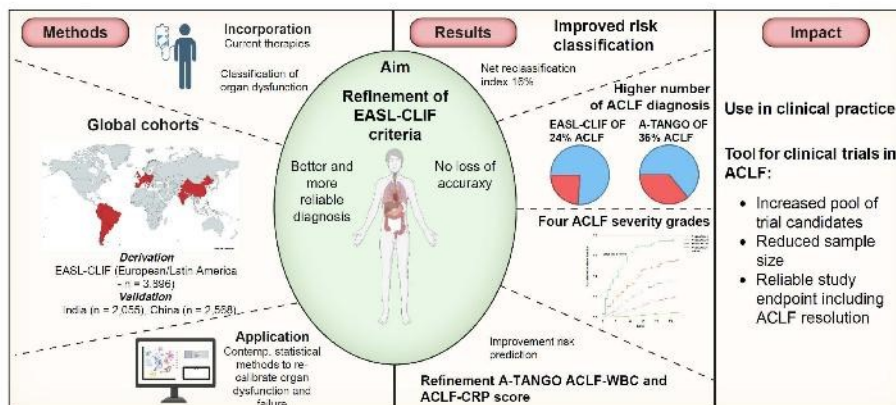
- ACLF criteria was developed in 2012. Many therapies and guidelines have changed since. :
- The individual OF scores are difficult to apply.
- ACLF Grade 1 is complex and difficult to use, with disparities in its diagnosis.
- Grade 3 is too broad with a risk of death ranging from 50-100%.
- CLIF-C ACLF score is difficult to use in G-CSF trials.
- Regulatory agencies require reproducible, quantitative and validated scoring systems if it is to be used for patient selection and end-point.

Engelmann C et al. J Hepatol 2026

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Refinement of the ACLF Criteria – A-TANGO Score



Engelmann C et al. J Hepatol 2026

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



A-TANGO OF Score

Organ System	Subscore 1	Subscore 2	Subscore 3
Liver	Bilirubin <6 mg/dl	Bilirubin ≥6 and <20 mg/dl	Bilirubin ≥20 mg/dl
Kidney	Creatinine <1.4 mg/dl	Creatinine ≥1.4 and <2 mg/dl or terlipressin	Creatinine ≥2 mg/dl or RRT or AKI 1b, 2, or 3
Brain	Grade 0–1	Grade 2	Grade 3–4 or mechanical ventilation
Coagulation	INR <1.6	INR ≥1.6 and <2.2	INR ≥2.2
Circulation	MAP ≥70 mmHg	MAP <70 mmHg	Vasopressor use
Respiration	PaO ₂ /FiO ₂ >350 or SpO ₂ /FiO ₂ >440	PaO ₂ /FiO ₂ >225 and ≤350 or SpO ₂ /FiO ₂ >325 and ≤440	PaO ₂ /FiO ₂ ≤225 or SpO ₂ /FiO ₂ ≤325 or mechanical ventilation

Engelmann C et al. J Hepatol 2026

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A-TANGO OF Score - Modifications

Organ System	Subscore 1	Subscore 2	Subscore 3
Liver	Bilirubin <6 mg/dl	Bilirubin ≥6 and <20 mg/dl	Bilirubin ≥20 mg/dl
Kidney	Creatinine <1.4 mg/dl	Creatinine ≥1.4 and <2 mg/dl or terlipressin	Creatinine ≥2 mg/dl or RRT or AKI 1b, 2, or 3
Brain	Grade 0–1	Grade 2	Grade 3–4 or mechanical ventilation
Coagulation	INR <1.6	INR ≥1.6 and <2.2	INR ≥2.2
Circulation	MAP ≥70 mmHg	MAP <70 mmHg	Vasopressor use
Respiration	PaO ₂ /FiO ₂ >350 or SpO ₂ /FiO ₂ >440	PaO ₂ /FiO ₂ >225 and ≤350 or SpO ₂ /FiO ₂ >325 and ≤440	PaO ₂ /FiO ₂ ≤225 or SpO ₂ /FiO ₂ ≤325 or mechanical ventilation

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A-TANGO OF Score – Diagnosing ACLF

Organ System	Subscore 1	Subscore 2	Subscore 3	
Liver	Bilirubin <6 mg/dl	Bilirubin ≥6 and <20 mg/dl	Bilirubin ≥20 mg/dl	Count as single organ failure for ACLF
Kidney	Creatinine <1.4 mg/dl	Creatinine ≥1.4 and <2 mg/dl or terlipressin	Creatinine ≥2 mg/dl or RRT or AKI 1b, 2, or 3	
Brain	Grade 0–1	Grade 2	Grade 3–4 or mechanical ventilation	
Coagulation	INR <1.6	INR ≥1.6 and <2.2	INR ≥2.2	Require add. organ dysfunction (Subscore 2)
Circulation	MAP ≥70 mmHg	MAP <70 mmHg	Vasopressor use	
Respiration	PaO ₂ /FiO ₂ >350 or SpO ₂ /FiO ₂ >440	PaO ₂ /FiO ₂ >225 and ≤350 or SpO ₂ /FiO ₂ >325 and ≤440	PaO ₂ /FiO ₂ ≤225 or SpO ₂ /FiO ₂ ≤325 or mechanical ventilation	

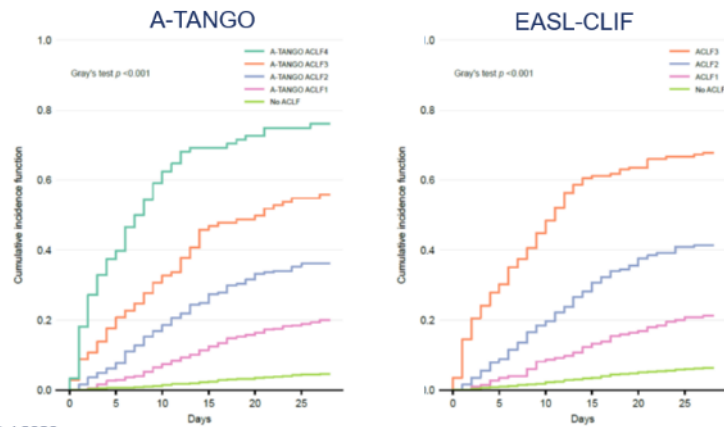
Engelmann C et al. J Hepatol 2026

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Mortality stratified by ACLF grade

28-day mortality – competing risk analysis



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A-TANGO Scores – Improved risk prediction

Table 5. Predictive discrimination ability of ACLF scores and MELD-Na score.

Score	28-day mortality, C-index (95% CI)	90-day mortality, C-index (95% CI)	Number of patients
Derivation cohort			
A-TANGO ACLF-CRP	0.738 (0.712–0.764)	0.697 (0.673–0.722)	1,072
A-TANGO ACLF-WBC	0.749 (0.724–0.774)	0.706 (0.683–0.729)	1,197
CLIF-C ACLF	0.756 (0.732–0.781)	0.714 (0.691–0.737)	1,196
A-TANGO OF	0.710 (0.683–0.737)	0.669 (0.644–0.693)	1,206
CLIF-C OF	0.716 (0.689–0.743)	0.674 (0.650–0.698)	1,205
MELD-Na	0.678 (0.651–0.705)	0.659 (0.635–0.683)	1,285

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ACLF Criteria — The need for harmonisation

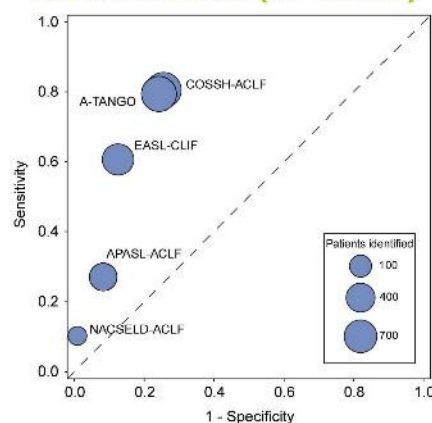
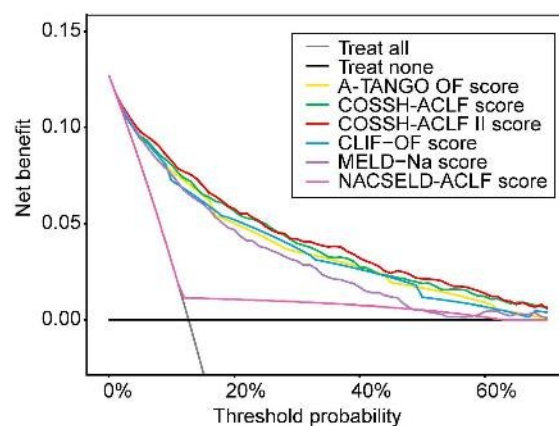


EASL-CLIF	COSSH	A-TANGO	APASL	NACSELD
<i>European Assoc. for the Study of the Liver — Chronic Liver Failure</i>	<i>Chinese Group on the Study of Severe Hepatitis B</i>	<i>A-TANGO Consortium</i>	<i>Asian Pacific Assoc. for the Study of the Liver</i>	<i>North American Consortium for End-Stage Liver Disease</i>
LIVER Bilirubin ≥ 12 mg/dL	LIVER Bilirubin ≥ 12 mg/dL	LIVER Bilirubin ≥ 20 mg/dL	Organ failures are not part of the APASL ACLF definition.	RENAL Need for RRT
KIDNEY Cr ≥ 2 mg/dL or RRT	KIDNEY Cr ≥ 2 mg/dL or RRT	KIDNEY Cr ≥ 2 mg/dL, RRT, or AKI 1b/2/3	Diagnosis rests on jaundice + coagulopathy + ascites/HE within 4 weeks, on a background of chronic liver disease.	BRAIN WH grade 3–4 HE
COAGULATION INR ≥ 2.5	COAGULATION INR ≥ 2.5	COAGULATION INR ≥ 2.2		RESPIRATION Need for non-invasive or mechanical ventilation
BRAIN WH grade 3–4 HE, or MV for HE	BRAIN WH grade 3–4 HE, or MV for HE	BRAIN WH grade 3–4 HE, or MV for cerebral failure		CIRCULATION Shock requiring vasopressor support
CIRCULATION Vasopressors	CIRCULATION Vasopressors	CIRCULATION Vasopressors		
RESPIRATION PaO ₂ /FiO ₂ ≤ 200 , SpO ₂ /FiO ₂ ≤ 214 , or MV	RESPIRATION PaO ₂ /FiO ₂ ≤ 200 , SpO ₂ /FiO ₂ ≤ 214 , or MV	RESPIRATION PaO ₂ /FiO ₂ ≤ 225 , SpO ₂ /FiO ₂ ≤ 325 , or MV		

WH = West Haven; HE = hepatic encephalopathy; MV = mechanical ventilation; RRT = renal replacement therapy; AKI = acute kidney injury; Cr = serum creatinine. This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Performance of the different models for ACLF and 28-day mortality in COSSH cohort from China (n=3370)



This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Consensus for harmonisation



SPECIAL ARTICLES

Defining Organ Failures in Patients With Cirrhosis: Consensus Statements



Jasmohan S. Bajaj,^{1,2} Debbie L. Shawcross,^{3,4} Ashok Choudhury,⁵ Constantine J. Karvellas,⁶ Jacqueline G. O'Leary,^{7,8} Jonel Trebicka,⁹ Anand V. Kulkarni,¹⁰ Sebastian Marciano,¹¹ Sumeet K. Asrani,¹² Madhumita Premkumar,¹³ Dominique Thabut,¹⁴ Qing Xie,¹⁵ Mark Sonderup,¹⁶ Simone I. Strasser,¹⁷ Alberto Queiroz Faria,¹⁸ Mario R. Alvares-da-Silva,¹⁹ Aldo Torre,²⁰ Jennifer C. Lai,²¹ Qin Ning,²² Nipun Verma,¹³ Sakkarin Chirapongsathorn,²³ Manuel Mendizabal,²⁴ Sombat Treeprasertsuk,²⁵ William Bernal,^{3,4} Lisa B. VanWagner,⁷ Rakhi Maiwall,⁵ Salvatore Piano,²⁶ Agustin Albillos,²⁷ Guadalupe Garcia-Tsao,^{28,29} Paolo Angeli,²⁶ K. Rajender Reddy,³⁰ Florence Wong,³¹ Richard Moreau,³² Shiv K. Sarin,⁵ and Patrick S. Kamath³³

Liver Centric Model
ACLF must have Liver failure defined by
Bilirubin >5mg/dl + INR >1.5

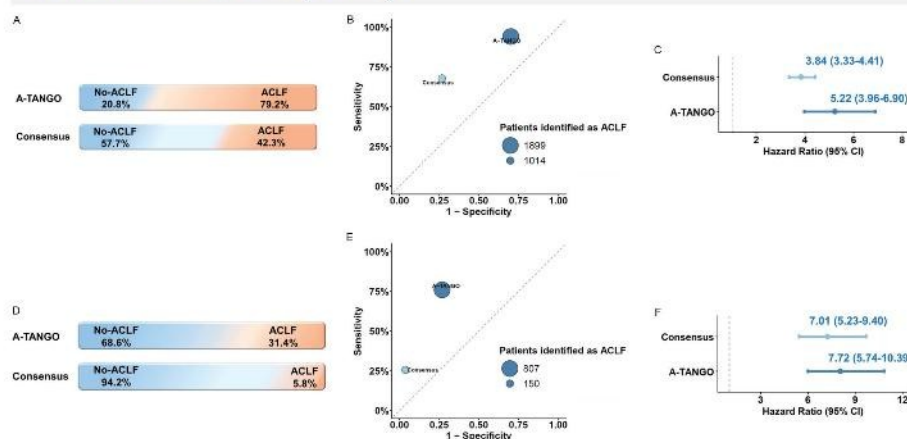
This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Consensus Definition vs ATANGO score



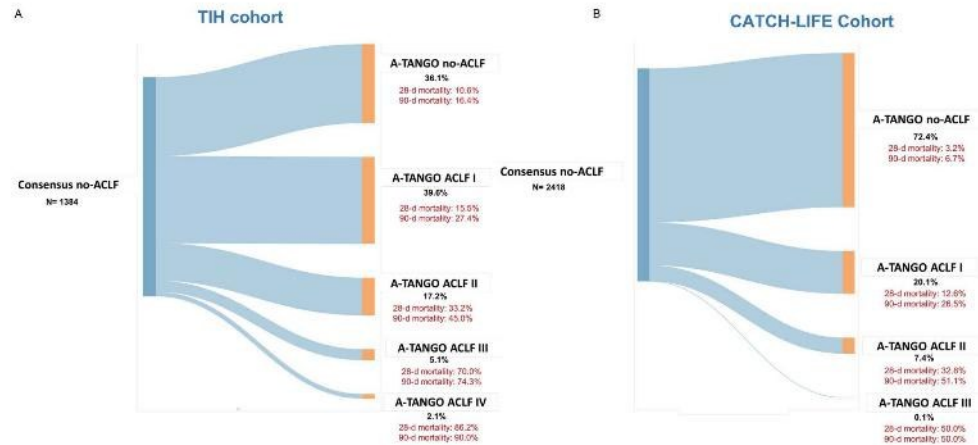
2,398 patients from the TIH cohort (India) and 2,568 from the CATCH-LIFE cohort (China)



This paper has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



The Consensus criteria underdiagnoses ACLF compared with A-TANGO



This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Revised proposal presented on 26/05/26



Outline of the Liver Centric Model

ACLF must have Liver failure defined by
Bilirubin >5mg/dl + INR >1.5

ACLF-1: Liver Failure + 1 EHOD

ACLF-2: Liver Failure + 1 EHOE

ACLF-3: Liver Failure + 2 EHOE

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



Discussion



- Explicit need for harmonization
- The process needs to be evidence based
- Has implications for regulatory agreement, drug development and clinical risk stratification
- Liver Transplantation is already prioritized using current classifications – need to make sure it is not disrupted
- First indication for the possible benefit of plasma exchange....

This project has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 945096.



10.2 Social Media Plan for Posts of all A-TANGO-related Activities at EASL Congress 2026

Wed, 27 May	Where	Who posts?	What	LinkedIn	BlueSky
	Poster area + e-poster	Nina	TOP-471-YI Annarein Kerbert (LUMC) The modified hepatic encephalopathy staging tool has significantly greater diagnostic consistency, prognostic performance and clinical impact compared with the West Haven criteria	 The modified hepatic encephalopathy staging tool (mHEST) demonstrates greater diagnostic consistency, prognostic performance and clinical impact compared with the West Haven criteria. #POSTER TOP-471-YI by Annarein Kerbert et al. presents the development and validation of mHEST as a more objective and reproducible tool for hepatic encephalopathy assessment. The study showed substantially improved inter-rater reliability, stronger association with ammonia levels, and better prognostic performance compared with the West Haven criteria, supporting its use in clinical	 The modified hepatic encephalopathy staging tool (mHEST) outperformed the West Haven criteria in diagnostic consistency and prognostic performance. #POSTER TOP-471-YI by Annarein Kerbert et al. #EASL2026 #HepaticEncephalopathy #Cirrhosis #Hepatology

				<p>trials and practice.</p> <ul style="list-style-type: none"> Presented at the #EASLCongress #EASL2026 #HepaticEncephalopathy #Cirrhosis #ClinicalTrials #Hepatology #LiverDisease #Biomarkers 	
	Poster area + e-poster	Nina	<p>WED-448-YI Olivia Greenham, UCL</p> <p>Development of a composite biomarker to predict risk of AKI and mortality following an acute decompensation of cirrhosis</p>	<p> Development of a composite biomarker to predict risk of AKI and mortality following acute decompensation of cirrhosis.</p> <p>#POSTER WED-448-YI by Olivia Greenham et al. explores the prognostic value of NGAL, ADMA and SDMA biomarkers in patients admitted with acute decompensation of cirrhosis.</p> <p>Higher composite biomarker scores were associated with increased risk of new AKI events and higher 3-month mortality, highlighting the potential role of novel biomarkers for early risk stratification and</p>	<p> Novel biomarker composites may help predict AKI and mortality following acute decompensation of cirrhosis.</p> <p>#POSTER WED-448-YI by Olivia Greenham et al. #EASL2026 #AKI #Cirrhosis #Biomarkers</p>

				<p>intervention.</p> <ul style="list-style-type: none">• Presented at the #EASLCongress #EASL2026 #Cirrhosis #AKI #Biomarkers #Hepatology #LiverDisease #ClinicalResearch	
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Thurs, 28 May	Where	Who posts?	What	LinkedIn	BlueSky
12:50 - 13:00) 12:45- 13.45	Bréchet - Hall 8.0	Sonja (foto), Nina (post)	Genetic cholestasis in adults: when to test? Thomas Berg (Univ Hospital Leipzig) Chair + Speaker Topic: Immune-mediated & Cholestatic Diseases Part of INDUSTRY Satellite Symposium Ipsen: Rare genetic liver diseases - Bridging the gap from genetic testing to patient management	Catching A-TANGO member @Thomas Berg in his session Genetic cholestasis in adults: when to test? - part of the topic immune- mediated & cholestatic diseases. #cholestasis #hepatology #EASL2026	Catching A-TANGO member @Thomas Berg in his session Genetic cholestasis in adults: when to test? - part of the topic immune- mediated & cholestatic diseases. #cholestasis #hepatology #EASL2026
10:00- 10:30	Track Hub 6 Cirrhosis & Complication s	Nina	Networking Session EF CLIF	see concentris post / reposted	

Fri, 29 May	Where	Who posts?	What	LinkedIn	BlueSky
08:30 - 09:50	Burroughs - Hall 8.0	Nina	Journal of Hepatology symposium: Symposium / General Hepatology Frank Tacke, Charité (Co-Chair) Incidence and implications of abstinence-induced recompensation in alcohol-related cirrhosis		
	Poster area + e-poster		FRI-401-YI Meiqian Hu, UCL Whole blood, microglial and astrocytic, transcriptomics and metabolomics reveals the association of type I interferon-associated immune-metabolic reprogramming in overt hepatic encephalopathy	🧠 Whole blood, microglial and astrocytic transcriptomics and metabolomics reveal type I interferon-associated immune-metabolic reprogramming in overt hepatic encephalopathy. #POSTER FRI-401-YI by Meiqian Hu et al. uses integrative multi-omics analyses to investigate immune-metabolic mechanisms underlying overt hepatic encephalopathy (OHE). The study identified a type I interferon-driven immune response coupled with metabolic reprogramming,	🧠 Type I interferon-driven immune-metabolic reprogramming may contribute to overt hepatic encephalopathy. #POSTER FRI-401-YI by Meiqian Hu et al. #EASL2026 #HepaticEncephalopathy #Cirrhosis #Metabolomics

				<p>suggesting a potential role for interferon signaling and tryptophan metabolism in neuroinflammation and glial dysfunction in OHE.</p> <p>📍 Presented at the #EASLCongress #EASL2026 #HepaticEncephalopathy #Cirrhosis #Neuroinflammation #Metabolomics #Hepatology #MultiOmics</p>	
	Poster area + e-poster		<p>FRI-413 Rajiv Jalan, UCL Modulation of the gut microbiome by Yaq-001 ameliorates fecal microbiome transplant (FMT) induced liver injury from cirrhotic specific pathogen-free mice to germ-free mice</p>	<p>🌱 Modulation of the gut microbiome by Yaq-001 ameliorates FMT-induced liver injury from cirrhotic mice to germ-free mice.</p> <p>#POSTER FRI-413 by Jinxia Liu et al. investigates how the microbiome therapeutic Yaq-001 modulates the gut-liver axis in experimental cirrhosis.</p> <p>The study found that Yaq-001 reshaped gut microbial communities, reduced liver injury and endotoxemia, and modulated host gene</p>	<p>🌱 Yaq-001 modulated the gut microbiome and reduced FMT-induced liver injury in experimental cirrhosis.</p> <p>#POSTER FRI-413 by Jinxia Liu et al.</p> <p>#EASL2026 #GutMicrobiome #Cirrhosis #Hepatology</p>

				<p>expression linked to metabolism and stress responses, supporting its potential as a microbiome-targeted therapy in liver disease.</p> <p>📍 Presented at the #EASLCongress #EASL2026 #GutMicrobiome #Cirrhosis #GutLiverAxis #Hepatology #MicrobiomeTherapeutics #LiverDisease</p>	
	Poster area + e-poster	Nina	<p>FRI-434 Rajiv Jalan, UCL</p> <p>Targeting disrupted immunometabolism-induced mitochondrial bioenergetic failure using YAQ006 and YAQ005 reduces mortality in ACLF</p>	<p>📄 Targeting disrupted immunometabolism-induced mitochondrial bioenergetic failure using YAQ006 and YAQ005 reduces mortality in ACLF.</p> <p>#POSTER FRI-434 by Supachaya Sriphoosanaphan et al. investigates combined ammonia scavenging and TLR4 inhibition as a therapeutic strategy in acute-on-chronic liver failure (ACLF).</p> <p>The study demonstrated that combined treatment with</p>	<p>⚡ Combined YAQ006 and YAQ005 treatment reduced hyperammonemia and improved survival in experimental ACLF.</p> <p>#POSTER FRI-434 by Supachaya Sriphoosanaphan et al.</p> <p>#EASL2026 #ACLF #Hyperammonemia #Hepatology</p>

				<p>YAQ006 and YAQ005 provided synergistic reductions in hyperammonemia and liver injury, achieving 100% coma-free survival in experimental ACLF models.</p> <p>• Presented at the #EASLCongress #EASL2026 #ACLF #Hyperammonemia #Immunometabolism #Hepatology #LiverDisease #TranslationalResearch</p>	
	Poster area + e-poster	Nina	<p>FRI-435 Rajiv Jalan, UCL</p> <p>Ammonia-induced mitochondrial bioenergetic failure is driven through a TLR4-dependent mechanism that specifically targets Complex IV</p>	<p> Ammonia-induced mitochondrial bioenergetic failure is driven through a TLR4-dependent mechanism that specifically targets Complex IV.</p> <p>#POSTER FRI-435 by Supachaya Sriphoosanaphan et al. investigates the molecular mechanisms linking hyperammonemia to mitochondrial dysfunction in liver disease.</p> <p>The study identified TLR4 as a key mediator of</p>	<p> TLR4-dependent mitochondrial dysfunction may drive ammonia-induced hepatocyte injury in liver disease.</p> <p>#POSTER FRI-435 by Supachaya Sriphoosanaphan et al.</p> <p>#EASL2026 #Hyperammonemia #ACLF #Hepatology</p>

				<p>ammonia-induced mitochondrial injury, showing that TLR4 inhibition restored Complex IV activity, reduced mitochondrial stress, and protected hepatocyte bioenergetics.</p> <p>• Presented at the #EASLCongress #EASL2026 #Hyperammonemia #ACLF #Mitochondria #Hepatology #LiverDisease #TranslationalResearch</p>	
09:30-10:00	Cirrhosis and Complications Track Hub	Nina	NETWORKING SESSION		
10:00-10:30	Cirrhosis and Complications Track Hub	Nina	NETWORKING SESSION: The A-TANGO Score: A novel tool for risk stratification and clinical endpoints in ACLF		
16:15-17:00	Track Hub 6 - Cirrhosis & Complications	Nina	<p>Poster tour: Cirrhosis and its complications - Experimental and pathophysiology</p> <p>Minneke Coenraad, LUMC (Chair)</p> <p>Basic Science / Cirrhosis & Complications</p>	Post also for A-TANGO, see Rajiv's student's poster (Meiqian Hu)	

Sat, 30 May	Where	Who posts?	What	LinkedIn	BlueSky
	Poster area + e-poster		SAT-352-YI Joscha Vonderlin/Kumar/Tacke/Engelmann (Charité), Loss of crypt-adjacent barrier-supportive macrophages promotes gut barrier failure and organ injury in alcohol-related hepatitis	<p>🔵 Loss of crypt-adjacent barrier-supportive macrophages promotes gut barrier failure and organ injury in alcohol-related hepatitis.</p> <p>#POSTER SAT-352-YI by Joscha Vonderlin et al. investigates how intestinal macrophage dysfunction contributes to gut barrier failure and organ injury in alcohol-related hepatitis.</p> <p>The study identified a barrier-supportive macrophage population whose loss under chronic metabolic stress was associated with impaired epithelial regeneration, increased gut permeability, and progression toward ACLF.</p> <p>🔴 Presented at the #EASLCongress #EASL2026 #ACLF #AlcoholRelatedHepatitis</p>	<p>🔵 Loss of barrier-supportive intestinal macrophages may promote gut barrier failure and organ injury in alcohol-related hepatitis.</p> <p>#POSTER SAT-352-YI by Joscha Vonderlin et al.</p> <p>#EASL2026 #ACLF #GutLiverAxis #Hepatology</p>

				#GutLiverAxis #Macrophages #Hepatology #LiverDisease	
	Poster area + e-poster	Nina	SAT-476 Meiqian Hu, UCL A-TANGO organ failure (OF) score performs better than EASL-CLIF, NACSELD and APASL scores for the diagnosis of acute-on-chronic liver failure (ACLF) in the independent COSSH cohort <ul style="list-style-type: none"> Data from 3,370 hospitalized cirrhosis patients with acute decompensation / 5 ACLF diagnostic models were compared, including A-TANGO, EASL-CLIF (European), COSSH-ACLF (Chinese), the APASL (Asia), and NACSELD (USA). Prognostic performance was assessed using net reclassification improvement (NRI), C-indices, and integrated discrimination improvement (IDI). 	<p>🌐 How do the world's major ACLF scoring systems compare in the same patient cohort?</p> <p>We analysed 3,370 hospitalized cirrhosis patients in the COSSH cohort and compared the following diagnostic models: A-TANGO Score vs EASL-CLIF (both European) vs COSSH-ACLF (Chinese) vs APASL (Asia) vs NACSELD (USA).</p> <p>Key findings:</p> <ul style="list-style-type: none"> ⚡ The A-TANGO and COSSH-ACLF Scores captured the largest number of ACLF patients ⚡ Both showed strong performance for predicting 28- and 90-day mortality ⚡ Other frameworks markedly underestimated ACLF diagnosis ⚡ Only 39 patients met criteria in ALL 5 	<p>🌐 Which ACLF score performs best globally?</p> <p>In 3,370 COSSH cohort patients, the A-TANGO score matched the COSSH -ACLF Score for ACLF diagnosis + short-term mortality prediction, while other models markedly underestimated ACLF.</p> <p>📍 Visit our poster to discuss harmonizing ACLF definitions.</p> <p>SAT-476 Meiqian Hu, UCL</p> <p>A-TANGO organ failure (OF) score performs better than EASL-CLIF, NACSELD and APASL scores for the diagnosis of acute-on-chronic liver failure (ACLF) in the independent COSSH cohort</p>

				<p>models — but mortality reached 93.3%</p> <p>The results support a potential path toward harmonizing ACLF definitions globally.</p> <p>♦ Stop by our poster to dive into the data and discuss what this means for ACLF diagnosis, prognostication, and future international collaboration.</p> <p>#ACLF #Hepatology #LiverFailure #Cirrhosis #ClinicalResearch</p>	
	Poster area + e-poster		<p>SAT-474 Jonas Schumacher / Janett Fischer / Florian van Bömmel / Thomas Berg (Univ Hospital Leipzig) Exploring rhabdomyolysis in critically ill decompensated cirrhotic patients - a retrospective analysis of a german transplant centre</p>	<p>🔗 Exploring rhabdomyolysis in critically ill patients with decompensated cirrhosis.</p> <p>#POSTER SAT-474 by Lukas Hofmann et al. investigates the incidence, causes, and clinical impact of rhabdomyolysis in critically ill patients with decompensated liver cirrhosis.</p> <p>The study found</p>	<p>🔗 Rhabdomyolysis may be an underestimated contributor to AKI and mortality in critically ill patients with decompensated cirrhosis.</p> <p>#POSTER SAT-474 by Lukas Hofmann et al.</p> <p>#EASL2026 #Cirrhosis #AKI #Hepatology</p>

				<p>that rhabdomyolysis, particularly statin-associated rhabdomyolysis, was linked to high rates of AKI and poor short- and long-term transplant-free survival, highlighting a potentially underestimated contributor to adverse outcomes in decompensated cirrhosis.</p> <p>• Presented at the #EASLCongress #EASL2026 #Cirrhosis #AKI #Rhabdomyolysis #ACLF #Hepatology #CriticalCare</p>	
	Poster area + e-poster		<p>SAT-475 Rajiv Jalan, UCL Serological responses COVID-19 vaccination: a potential biomarker to predict liver-related complications in patients with compensated advanced liver disease (cACLD)</p>	<p>📌 Serological responses to COVID-19 vaccination may predict liver-related complications in patients with compensated advanced chronic liver disease.</p> <p>#POSTER SAT-475 by Changze Hong et al. explores whether vaccine-induced immune responses can serve as biomarkers of cirrhosis-associated</p>	<p>📌 Impaired serological response after COVID-19 vaccination was associated with liver-related complications in cACLD.</p> <p>#POSTER SAT-475 by Changze Hong et al. #EASL2026 #cACLD #COVID19 #Hepatology</p>

				<p>immune dysfunction in cACLD.</p> <p>The study found that impaired serological response after COVID-19 vaccination was associated with increased liver-related complications, supporting vaccine response as a potential biomarker of immune dysfunction and future risk stratification in cACLD.</p> <p>• Presented at the #EASLCongress #EASL2026 #cACLD #COVID19 #ImmuneDysfunction #Hepatology #LiverDisease #Biomarkers</p>	
	Poster area + e-poster		<p>SAT-489 Sina Oesinghaus / Pohl/Tacke/Engelmann (Charité)</p> <p>Body composition is a predictor of treatment response and survival in hepatorenal syndrome (HRS-AKI)</p>	<p>🧠 Body composition is a predictor of treatment response and survival in hepatorenal syndrome (HRS-AKI).</p> <p>#POSTER SAT-489 by Sina Oesinghaus et al. investigates whether AI-based CT body</p>	<p>🧠 AI-based body composition analysis may help predict treatment response and survival in HRS-AKI.</p> <p>#POSTER SAT-489 by Sina Oesinghaus et al. #EASL2026</p>

				<p>composition analysis can predict treatment response and outcomes in patients with HRS-AKI.</p> <p>The study found that reduced muscle mass and altered muscle-to-fat distribution were independently associated with terlipressin non-response and poorer 90-day transplant-free survival, supporting body composition analysis as a potential tool for risk stratification in HRS-AKI.</p> <p>📌 Presented at the #EASLCongress #EASL2026 #HRS-AKI #Cirrhosis #Sarcopenia #Hepatology #LiverDisease #ArtificialIntelligence</p>	#HRS-AKI #Cirrhosis #Hepatology
	Poster area + e-poster		<p>SAT-510-YI Alex Zubko / Pohl/Tacke/Engelmann (Charité)</p> <p>Reduced thrombin generation capacity in decompensated cirrhosis and acute-on-chronic liver failure is associated with short-term mortality</p>	<p>🔴 Reduced thrombin generation capacity in decompensated cirrhosis and acute-on-chronic liver failure is associated with short-term mortality.</p>	<p>🔴 Reduced thrombin generation capacity was associated with short-term mortality in decompensated cirrhosis and ACLF.</p>

				<p>#POSTER SAT-510-YI by Alexander Zubko et al. evaluates thrombin generation assay (TGA) profiles in patients with acute decompensation and ACLF.</p> <p>The study found that profound alterations in thrombin generation were associated with organ failure and short-term mortality, supporting TGA as a promising global coagulation marker for risk stratification in acutely ill patients with cirrhosis.</p> <p>📍 Presented at the #EASLCongress #EASL2026 #ACLF #Cirrhosis #Coagulation #Hepatology #LiverDisease #Biomarkers</p>	<p>#POSTER SAT-510-YI by Alexander Zubko et al. #EASL2026 #ACLF #Cirrhosis #Hepatology</p>
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