EASL MASLD Guidelines Academy 2026

Module 2: A deeper dive into MASLD

Antwerp University Hospital, 08-09 October 2026

Organisers:

Sven Francque, MD, PhD, hepatology

Luisa Vonghia, MD, PhD, hepatology

Wilhelmus Kwanten, MD, PhD, hepatology

Local faculty:

Thomas Vanwolleghem, MD, PhD, hepatology

Wim Verlinden, MD, PhD, hepatology

Len Verbeke, MD, PhD, hepatology

Ann Driessen, MD, PhD, pathology

Joanne Verheij, MD, PhD, pathology

Bart Op de Beeck, MD, PhD, radiology

Lotte Schoenmakers, MBSc, PhD student

Zouhir Gadi, MBSc, PhD student

Haifeng Lu, MD, PhD student

Yang Lu, MD, PhD student

Eveline Dirinck, MD, PhD, endocrinology

Jonathan Mertens, MD, PhD student, endocrinology

Emeline Van Craenenbroeck, MD, PhD, cardiology

Wim Vanden Berghe, PhD, genetics and epigenetics

External faculty:

Amalia Gastaldelli (PhD, Pisa, Italy)

Bart Staels (PhaD, PhD, Lille, France)

Programme

Preparation

- Recommended literature will be provided
- Participants will have to fill out a 5-section questionnaire (250 words/section) on their current understanding of key issues in MASLD.

  Questions about the pathophysiology, MASLD as part of a systemic disease, and diagnostic features. This will help the faculty to adapt the content according to the level of knowledge and the gaps that can be identified.

- Participants will have to fill out a 20-item quiz before the start of the module.

  Will serve as a basis for a discussion at the end of the module and for a re-evaluation after the module.

In-person 2-day meeting

DAY 1

08.30-09.00 Arrival and Welcome coffee

09.00-09.05 Introduction (Sven Francque)

Part 1 Basic concepts of metabolism

09.05-09.25 The liver as a central organ of metabolism (Wilhelmus Kwanten)
Refresh knowledge about the central role of the liver in metabolism in physiological conditions, and how any chronic liver disease can have an influence on this, starting straight ahead with an understanding of how a diseased liver can impact metabolism (and hence to set the scene that MASLD is not just a consequence of the metabolic syndrome

09.25-09.50 Obesity, clinical obesity and dysfunctional adiposity (Eveline Dirinck)
Explain the newer concepts related to obesity beyond a BMI-based concept, and some general pathophysiological considerations regarding the role of dysfunctional adipose tissue (in general, not focused on the liver specifically); also explain how lean people can be metabolically unhealthy

09.50-10.05 Q&A
The Q&A sessions are meant to be highly interactive and will involve all faculty, with some prepared questions to structure the discussion

Part 2 MASLD Pathophysiology Part 1

10.05-10.25 Understanding the pathophysiology of MASLD: the global concept (Sven Francque)
Summarise the current understanding of the pathophysiology of MASLD/MASH as part of a systemic metabolic disease, with a complex multidirectional interaction between the liver and extrahepatic sites like the adipose tissue, the gut, the muscle and the cardiovascular system; highlight the complex interplay between all these players; also highlight the influence of genetic, epigenetic and environmental factors that contribute to patient heterogeneity; discuss the balance between damage and repair/defence that is important to understand disease progression/regression and patient heterogeneity

10.25-10.45 MASLD pathophysiology: understanding insulin resistance (Amalia Gastaldelli)

Focusing on insulin action and IR as a global and as an organ-specific feature, what the different types of IR are and how it can be measured, discuss how it plays a role in MASLD pathophysiology, and clearly highlight the difference and different roles of adipose tissue IR and liver IR; to coordinate with the next talk to avoid overlap

10.45-11.00 Coffee break

11.00-11.20 MASLD pathophysiology: the adipose tissue-liver axis (Luisa Vonghia)
Highlight the contribution of adipose tissue (the dysfunctional adipose tissue) to liver disease specifically, building further on the talk "Obesity, clinical obesity and dysfunctional adiposity"; briefly mention adipose tissue IR but coordinate with the previous talk on IR; discuss inflammation and adipokines, role of PPAR (mainly gamma) and FGF21 and other relevant pathways; also inflammatory mediators and immunological alterations

11.20-11.40 MASLD pathophysiology: the role of the liver vasculature (Wilhelmus Kwanten)
Discuss the preclinical and clinical findings across the disease spectrum, including the data suggesting a presinusoidal component, pharmacological modulation (to coordinate with "CVD drugs and the liver", as well as the recent PSVD data; and all potential implications (including diagnostics)

11.40-12.00 How to understand MASLD in special populations: Type 1 Diabetes and MASLD in lean individuals (Jonathan Mertens)
Discuss the current data on MASLD in people with type 1 diabetes; discuss some recent pathophysiological insights and the concept of dual diabetes, and highlight how the occurrence of MASLD may still be reflective of the pathophysiological mechanisms highlighted in the previous sections; also discuss MASLD in people without overweight and to what extent more subtle metabolic derangements can be present and when to think about specific genetic diseases that are part of the SLD umbrella but should not be classified as MASLD

12.00-12.20 Q&A

Part 3 The liver tissue: what the biopsy tells us

12.20-12.50 The liver lesions in MASLD: basics and beyond (Joanne Verheij)
Discuss in detail the lesions that can be seen on the liver biopsy and that are characteristic for MASLD, not just the cardinal features but also more detailed features that can make a differentiation between several disease stages and that might have prognostic importance; discuss the different main classification systems and their differences including the more recent more granular fibrosis staging as proposed by EPOS-FLIP and also highlight how Immunohistochemistry and new technologies can provide detailed information, discuss the potential positioning of the biopsy as a specialised tool in experienced hands in the current diagnostic armamentarium and how it should position in relation to a NIT-based approaches; include a brief section on the histological endpoints in clinical trials with their pros and cons

12.50-13.00 Introduction to workshop 1 (Ann Driessen)

13.00-14.00 Lunch

14.00-15.00 Workshop 1: the liver biopsy revisited (Ann Driessen, Joanne Verheij)
Several cases will be demonstrated, commented upon by the two experienced hepatopathologists and discussed with the group, focusing on first of all the standard reading and scoring, but also illustrating the importance of a more detailed assessment of specific features and showing some examples of less explored lesions and of some recent advances

Part 4 The role of NITs and imaging: a critical appraisal

15.00-15.15 An introductory case

15.15-15.40 NITs: from diagnosis to prognosis-pitfalls and gaps (Sven Francque)

Discuss the context of use of NIT's an the gradual shift from a diagnostic to a prognostic context of use, the limitations of NITs in terms of diagnostic accuracy and the intrinsic variability that has an impact on both cross=sectional diagnostic accuracy and their use in terms of disease monitoring and assessing treatment response; discuss how the prognostic context of use is important and how changes over time should be interpreted both in terms of diagnostic as well as prognostic use and indicate the existing knowledge gaps

15.40-16.00 Coffee break

16.00-16.25 The role of imaging beyond ultrasound (Bart op de Beeck)

Discuss the role of imaging modalities like CT, MRI, MRE, MR-PDFF and others, for features of steatohepatitis but also other features that can be relevant, e.g., iron; discuss limitations, variability and other relevant issues regarding accuracy, reproducibility and accessibility

16.25-16.40 Back to the case

16.40-17.00 Q&A

Part 5 MASLD Pathophysiology part 2

17.00-17.20 Linking the mode of action of drugs to disease pathophysiology: incretins (Amalia Gastaldelli)

Discuss in detail the mode of action of incretins in general, how they impact on the different aspects of metabolic syndrome, and how they can be potentially beneficial in MASLD by linking their mode of action to disease pathophysiology; do not discuss in detail the clinical trial results, as this is the topic of another module, this module should focus on getting deeper insights in pathophysiology and how this can be pharmacologically tackled; Clearly discuss and separate indirect and direct hepatic effects

17.20-17.40 Linking the mode of action of drugs to disease pathophysiology: Thyroid Hormone Receptor agonists, FGF and PPARs (Sven Francque)

Discuss in detail the mode of action of these drugs and how they can be potentially beneficial in MASLD by linking their mode of action to disease pathophysiology; do not discuss in detail the clinical trial results, as this is the topic of another module, this module should focus on getting deeper insights in pathophysiology and how this can be pharmacologically tackled; Clearly discuss and separate indirect and direct hepatic effects

17.40-18.00 Round table discussion (all)

18.00-23.00 Social activity and dinner

DAY 2

Part 6 Ultrasound, liver biopsy and liver stiffness assessment

08.30-08.55 Liver stiffness assessment: different techniques, thresholds and pitfalls (Wim Verlinden)

Discuss the relevant techniques (VCTE, 2D SWE…) and the factors that influence the values even in physiological conditions; discuss the techniques and how to perform them in order to have high quality results; discuss inter- and intra-observer and intra-individual variability and how this should influence interpretation; discuss the different thresholds according to the different techniques; discuss in general different machines/vendors and the upcoming point-of-care devices

08.55-09.15 Liver biopsy: different techniques, dos and don'ts (Thomas Vanwolleghem)

Explain the practicalities of the percutaneous and the transjugular liver biopsy, potential complications, precautions, indications and contraindications; discuss needle types, and yields in terms of quality of the biopsy for the different techniques and needle types and how this can be optimised

09.15-09.30 EUS-guided liver biopsy (Len Verbeke)

Explain briefly the technique, the needle type, number of passes, the quality metrics of the samples that can be obtained (length and width, number of fragments, total length…), safety in different stages of liver disease severity, potential differences between this left lobe sampling and the traditional right lobe sampling

09.30-10.30 Workshop 2: Ultrasound, liver biopsy and liver stiffness assessment (Luisa Vonghia, Lotte Schoenmakers, Wilhelmus Kwanten, Wim Verlinden, Len Verbeke, Zouhir Gadi)

In small groups at different stations the different techniques will be showcased, as much as possible with life demonstrations in patients, including a demonstration of contrast-enhanced ultrasound (Luisa Vonghia, Zouhir Gadi) as a way of studying liver vascularization and blood flow and, given the role of vascular alterations, how this can be linked to different features of disease severity

10.30-10.50 Coffee break

Part 7 The liver and the heart

10.50-11.15 Atherosclerotic cardiovascular disease and HFpEF in MASLD (Haifeng Lu, Wilhelmus Kwanten)

Discuss what is currently known about the link between MASLD and cardiovascular disease, not only focusing on atherosclerotic cardiovascular disease but including the data on heart failure, most notably HFpEF; discuss association versus data on the potential independent contribution of MASLD to cardiovascular disease; discuss also subclinical disease versus clinical disease and the available data on both

11.15-11.30 Current understanding of HFpEF and how this could be linked to MASLD (Emeline Van Craenenbroeck)

Provide some basic insights on the entity of HFpEF, its diagnosis and pathophysiology, and how, starting from the pathophysiology of both entities, MASLD could be linked to HFpEF, purely by association or potentially also with a causal relationship

11.30-12.00 Mechanisms behind the liver-heart axis (Bart Staels)

Discuss, mainly based on the preclinical evidence, how the liver in MASLD could impact on cardiovascular disease and most notably on atherosclerotic cardiovascular disease; discuss also what the pathways are that could be tackled to modulate that

12.00-12.20 CVD drugs and the liver (Sven Francque)

Discuss the evidence concerning drugs that are used to prevent or treat cardiovascular disease, like some of the antihypertensives, antiaggregant or

anticoagulant therapies, on the severity of liver disease and how they could be disease modifiers in MASLD and in cirrhosis

12.20-12.40 CVD assessment in MASLD (Lotte Schoenmakers, Emeline Van Craenenbroeck)
Discuss what a detailed cardiovascular assessment should look like in patients with MASLD, which investigations are useful to perform according to the disease stage, which tests of subclinical cardiovascular disease are available and could reasonably be incorporated in the work-up of patients with MASLD/ MASH

12.40-13.00 Q&A

13.00-14.00 Lunch

Part 8 MASLD pathophysiology part 3: MASLD and cholestasis

14.00-14.15 Cholestasis and cholate stasis on the liver biopsy: a feature of MASLD? (Ann Driessen)
Explain the 2 terms, explain what can be seen on a liver biopsy and then discuss more specifically what has been observed so far in MASLD

14.15-14.20 What can we learn from clinical data (Yang Lu, Bart Staels)
Give an overview of the current clinical data on bile acid homeostasis disturbances in MASLD, the clinical meaning of the alterations of the bile acid pool and the data supporting a link between elements of cholestasis and the severity and progression of MASLD

14.20-14.45 Druggable targets: FXR and PPAR (Bart Staels)
Briefly explain the mechanisms behind the potential link between alterations in bile acid metabolism and the different metabolic and inflammatory aspects as well as fibrogenesis in MASLD; then focus on FXR and PPAR pathways and their drugs, explain how these drugs could work in MASLD whilst they have been successfully used in cholestatic liver diseases

14.45-15.00 Q&A

Part 9 MASLD pathophysiology part 4: (epi)genetics and patient heterogeneity

15.00-15.20 Epigenetics (Wim Vanden Berghe)
After a brief introduction about the basics of epigenetics, discuss the current evidence of how epigenetic modifications contribute to disease progression and hence also to disease heterogeneity, and how this can be used for diagnosis and/or risk stratification and/or management

15.20-15.40 PNPLA3 (Luisa Vonghia)
Explain the physiology/pathophysiology linked to PNPLA3, what the impact is on disease progression, how it relates to metabolic risk factors to explain disease progression, how it can help with diagnosis/risk stratification; PNPLA3 as a modifier of treatment response in clinical trials and in routine practice; PNPLA3 as a target for therapy

15.40-16.00 Coffee break

16.00-16.20 The role of other polymorphisms (Wilco Kwanten)
Discuss the physiological and pathophysiological role of other polymorphisms than PNPLA3, the usefulness or absence here of of polygenic risk scores, influence on treatment response? Therapeutic targets?

16.20-16.40 From clusters to the individual patient: can we understand patient heterogeneity? (Sven Francque)
Explain how patient heterogeneity can be understood from a background of differences in susceptibility to metabolic risk factors and heterogeneity in terms of liver defence and repair mechanisms, discuss current data coming from cluster analysis and how to develop this further in an era aiming at precision medicine

16.40-17.00 Q&A

Part 10 The quiz revised

17.00-17.45 The quiz revised: right and wrong (all)

This should allow for a lively discussion on most of the topics that have been discussed; it is an opportunity to check whether all the items in the quiz have been satisfactorily addressed by the faculty and if there are remaining issues where the participants need additional input from the faculty and can provide feed-back on the content of what was discussed

17.45-18.00 Closing and farewell

Post-meeting

- Participants will have to add an update to each section of the 5 section questionnaire (250 words/section) on their current understanding of key issues in MASLD and indicate where the module has changed their views

  This will help evaluating the module and identify areas where the module can be improved for further editions (if any)

- Participants will have to fill out again the 20-item quiz that was taken before the start of the module

- General survey on the module (provided by EASL)