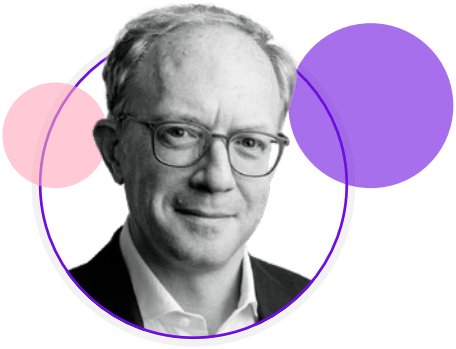
Congress Interviews

In these exclusive interviews from the European Association for the Study of the Live (EASL) Congress 2026, leading hepatology experts reflect on a meeting defined by scientific progress, multidisciplinary collaboration, and a growing focus on translating research into meaningful patient outcomes. They discuss advances across liver disease prevention, diagnosis, and treatment, including developments in precision medicine, MASLD, autoimmune and cholestatic liver diseases, and the role of lifestyle interventions in long-term care. Key themes include earlier disease detection, personalised treatment strategies, patient-centred care, and the importance of collaboration across specialties to address the evolving global burden of liver disease.

Featuring: Sven Francque, Shira Zelber-Sagi, and Sarwa Darwish Murad

Citation: EMJ Hepatol. 2026;14[1]:85-89. https://doi.org/10.33590/emjhepatol/696128BJ



Sven Francque

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Q1 There has been increasing discussion about whether steatotic liver diseases should be viewed as a spectrum rather than a single disease entity, particularly given metabolic, genetic, and alcohol-related overlap. Do you think this 'spectrum' model better reflects what we see in clinical practice?

Yes, absolutely. I think we should go back a little bit to the pathophysiology of the disease, because this is largely driven by extrahepatic factors. The dysfunctional adipose tissue, mainly, with the release of free fatty acids and a lot of inflammatory mediators, has a huge impact on the liver. The second level or layer of complexities is how well the liver is equipped with machinery to cope with that metabolic and inflammatory stress. So, at both levels, there is a huge heterogeneity among patients. We know that with the same caloric overload, some people will still be metabolically healthy despite living with obesity. Then there are others with minor degrees of obesity who will be very metabolically unhealthy. So, based on genetic, epigenetic, and environmental factors, you have a huge heterogeneity in the vulnerability of people to the deleterious consequences of caloric overload. But then, on top of that, you have that spectrum of liver vulnerability, and it's that complex constellation that makes the total population of patients with steatotic liver disease notoriously heterogeneous.

> **You have a huge heterogeneity in the vulnerability of people to the deleterious consequences of caloric overload**

Q2 Metabolic dysfunction-associated steatotic liver disease (MASLD) is increasingly recognised as a multisystem disease with significant metabolic and cardiovascular implications. How has this changed the way hepatologists approach patient care?

Traditionally, hepatologists were focused on the organ and very specific causes of liver damage, these being direct liver toxins like alcohol, viral hepatitis, and autoimmune hepatitis. We also are taking into account that some diseases have extrahepatic manifestations. But MASLD is something that is intrinsically correlated or related to dysfunctional adipose tissue. With the liver being such an important central organ to whole body metabolism, a chronically diseased liver also has an extrahepatic impact, most notably on the cardiovascular system. In MASLD, it's all so closely interconnected that we cannot just see it as a liver problem in isolation, because it's mainly driven by factors that are coming from outside the liver, and it has important consequences, not just on the liver, but also on the extrahepatic tissue.

To complete our increasing understanding of how this is really happening, we are forced to think outside the liver and take into account both managing the causes of MASLD and the consequences of MASLD. We have to step out of our silo of a purely liver-centric approach. It's also what we have seen with some of the pharmacological approaches: some of them lean heavily towards improving the metabolic milieu that drives the disease, and some of them have very little direct intrahepatic targets, and still, they hugely benefit the liver.

Q3 Despite advances in non-invasive testing, identifying patients at the highest risk of progression remains challenging. Where do you believe the biggest diagnostic gaps still exist?

The difficulty is that we need to gain insight into an organ that, beyond biopsy, is not easily accessible for direct assessment. And, as we discussed earlier, the patient population is highly heterogeneous. It is a challenge to get the precise idea about the severity of the disease, because what we can measure in the blood, or what we can measure in terms of imaging characteristics of the liver, is influenced by many factors, and we have been shaped by the liver biopsy to understand this disease and its severity, but of course, we cannot apply the liver biopsy to every potential patient at risk, and we can also not use it for the follow-up of the patient. So, we definitely need tools that help us better characterise the severity across the disease spectrum. We do have some tools that help us in a very rough classification of fibrosis, but fibrosis doesn't tell it all. When people get to more advanced stages of the disease, fibrosis is obviously still an important feature. But processes like the alterations of the vascular structure and function become increasingly important once the disease advances. Also, parenchymal extinction and bad regeneration become more and more important, and those aspects are still very difficult to capture with the tools that we currently have, or it's by very specialised tools that are available in the research context, but not for routine clinical practice. So, thinking in terms of disease, spectrum, and that tipping point where the risk of evolving towards

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the compensation and the point where regression of disease becomes less likely, even if you improve on the metabolic causes of the disease, identifying that tipping point in patients is still a big challenge. Because first of all, we don't know exactly where it is. As I said, we're very framed by thinking in terms of cirrhotic versus non-cirrhotic, but that's an oversimplification. So, there's a lack of knowledge and a lack of tools to identify disease.

Another challenge is that many patients initially present in primary care, where clinicians may not specialise in hepatology. How do you determine when these patients should be referred to a hepatologist?

That flow of patients and creating a system that really selects the ones that need to go to a more advanced step in the diagnostic and management framework is a huge challenge. Because the population is so heterogeneous, not every patient with liver steatosis needs to be seen by a highly specialised hepatologist. There needs to be a funnel. I think awareness and knowledge are, of course, one thing, but we still also need to improve the algorithms and the tools that we have to help primary care, but also secondary non-hepatology care to make an adequate diagnosis and have an adequate referral pattern.

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Q4 Given your involvement in clinical trials and consortia in NAFLD/MASLD, how do you see emerging pharmacological therapies being integrated into real-world practice over the next few years?

Well, there are a few challenges. First, there is the science and the results of the clinical trials, but then there's the implementation and how it is picked up in the community. That's a matter of awareness and education, but it's also something that has to do with policy change and reimbursement. So, at the level of regulators and payers, there's still a huge amount of work to be done in terms of education. Sometimes, even the scientific evidence that is generated is questioned. Because this is a very slowly evolving disease, its impact on patient outcomes often only becomes apparent over the long term. And even before they get to compensated liver disease, there's a huge impact on quality of life, although that's more difficult to quantify. But because it's so slowly evolving, and it's so closely interconnected with other conditions like obesity, it's very difficult to make people accept that this is something that needs a particular treatment, because the hard clinical evidence is very difficult to generate. If you really want to demonstrate that a drug has an impact on the long run in terms of mortality or clinical events, you need very long trials, in which you have many intercurrent events. So that's the kind of evidence that is very difficult to generate. As long as we adhere to the view that the added value of a drug must be demonstrated solely through clinical event outcomes, without accepting surrogate endpoints or real-world evidence, progress will remain challenging.

I can't even imagine what it must have been like for researchers conducting long-term research and then having COVID happen and completely changing the mortality rates in studies. How did they account for that when analysing the data?

Yes, that's extremely challenging. The problem is that we still do not have well-validated surrogate markers. We do not have the equivalent of HbA1c for MASLD, for example. There is a huge unmet need for a marker that can serve, in the short term, as a validated surrogate for long-term clinical benefit.

Q5 There is growing interest in combination therapies for steatotic liver disease. Do you believe combination treatment approaches will become the future standard of care?

Standard of care is probably a little bit too strong a term, although combination therapies certainly make sense. One of the reasons is that, based on the results we have seen so far, we have drugs that are efficacious, but the effect size does not appear to be close to 50%, let alone 100%. That is partly due to the way the endpoints have been defined, because there are benefits beyond those specific endpoints. However, it also reflects the complex pathophysiology of the disease. Targeting one particular aspect of the pathophysiology may provide meaningful benefit for some patients, but for others, it will not be sufficient. Therefore, the complexity of the disease, together with the fact that treatment outcomes are partly influenced by how success is defined, makes combination therapy a very plausible and attractive concept.

By taking that approach, especially in patients without advanced disease, a large proportion will probably improve, or at least disease progression will be halted. As such, they do not necessarily require combination therapy from the outset. In these patients, the first step should be to optimise all the cardiometabolic drivers and consequences of the disease and then assess the response. If, upon re-evaluation, the liver condition has not improved sufficiently, a second, more liver-targeted therapy could then be added. On the other hand, patients who already have advanced liver disease at presentation, or those whose cardiometabolic comorbidities are relatively well controlled, may be candidates for combination therapy from the start.

So, I think a more granular approach is needed than simply stating that combination therapies are the standard of care. At the very least, treatment needs to be individualised. We are not yet at the stage of truly personalised medicine in this disease, but a degree of treatment individualisation is certainly necessary.

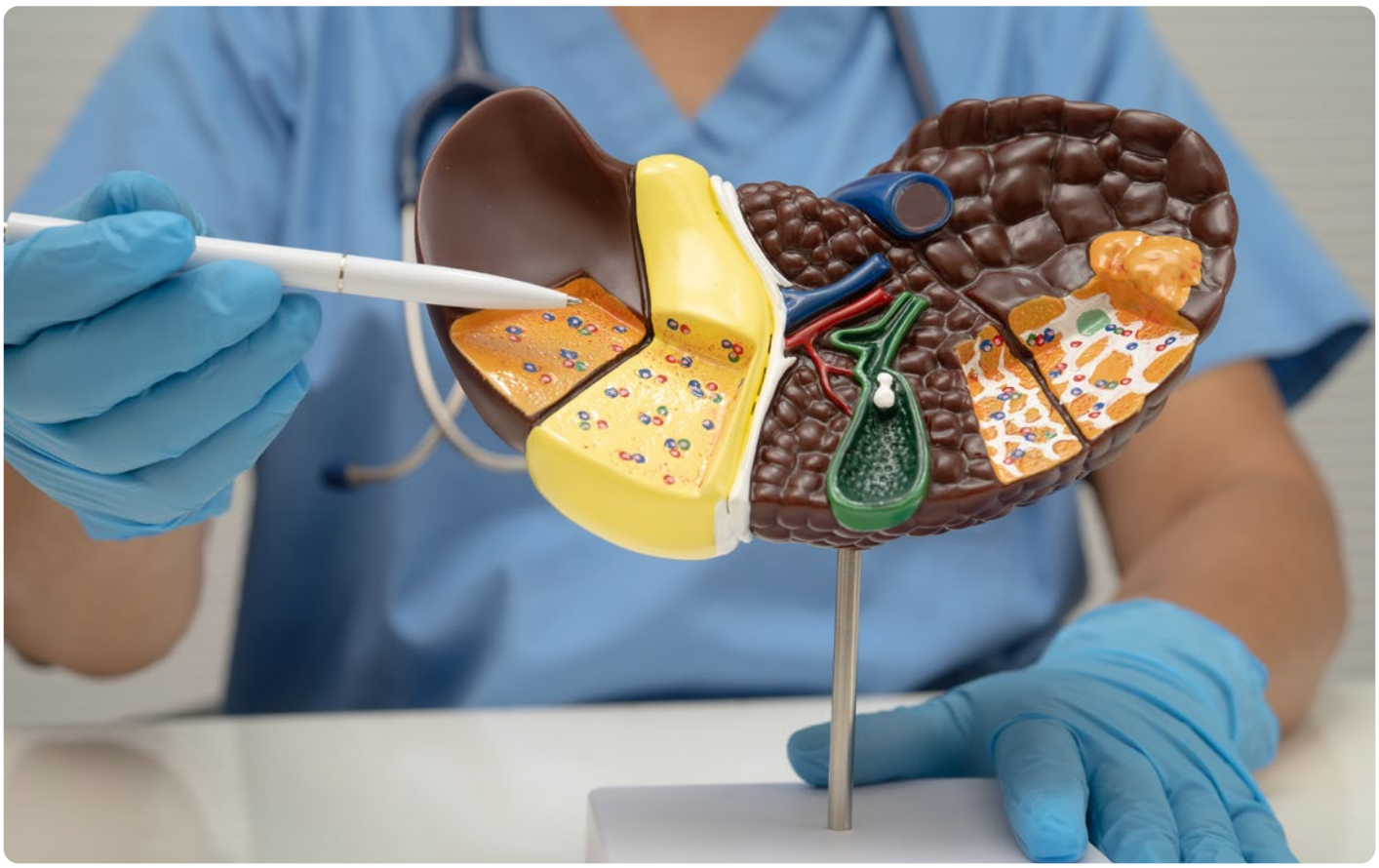
Q6 In your experience, what is the most common misconception clinicians still have about disease progression or risk stratification in MASLD?

It is my impression that, for many non-hepatologists and even some people in the hepatology space, there's still that conception that a little bit of liver steatosis or slightly elevated liver enzymes is not that dangerous. There is also that confusion with alcohol consumption. Alcohol and metabolic risk factors are frequently present together; it is easy to say drink a little less alcohol, lose a little weight, and that will solve the problem. The consequence of that attitude is that, even if most patients do not have advanced liver disease or have little risk of progressing to liver disease, it neglects the fraction of patients that do have advanced liver disease. At the very least, clinicians should keep in mind the possibility that this may be more than just a little liver fat. The basic attitude should be, I should investigate this with a multi-tier approach. I should not minimise it; I should investigate. Moreover, even if there is just liver fat, it means that the patient is metabolically unhealthy. While this may not necessarily represent severe liver disease, it is nonetheless a condition of metabolic ill health that requires further investigation and appropriate management.

Do you think some of these misconceptions stem from the common belief that the liver can regenerate? Could that create a false sense of reassurance, leading clinicians or patients to delay action and simply reassess the situation a few months later?

It's an interesting point; it's true that compared to others, the liver is quite a robust organ with a huge regenerative capacity. That's probably one of the reasons why the consequences of the disease, in most of the patients, tend to appear rather late in life, even though the disease itself often develops much earlier. It's important to emphasise that this is not the case for everybody. Although the liver is probably better equipped than many other organs and has more defence and regeneration capacity than many other organs, there's still a huge difference in heterogeneity between people.

The capacity of the liver to cope with metabolic inflammatory stress might be, on average, better than the kidney or the heart, but it's still not perfect. So, neglecting that and ignoring that is one of the main reasons why people, in the end, still develop these deleterious consequences that could be prevented if tackled earlier.



What we also tend to forget is that the risk of Type 2 diabetes substantially increases if you have liver steatosis and steatohepatitis. So, it's not just about the liver; it's also, again, the liver being so central to metabolism. We still tend to think a little too much of the liver being the victim of metabolic syndrome, but it's more than that. It's really a motor of vicious circles, and it's very central to the pathophysiology of the consequences of metabolic syndrome, not just the victim.

Q7 As EASL 2026 comes to a close, which developments in advanced liver disease management have stood out to you most this year, and how might they shape clinical practice over the next decade?

Well, one of the added values of this Congress is that you get the newest scientific progress presented, and there were some interesting things about, for example, the refinement of the use of tips or data on treatments based on the use of albumin, so that's the cutting edge in terms of scientific novelties. But, of course, many of them will not immediately transform clinical practice because we need to generate more evidence, and it needs to be reviewed. So, one of the added values of a Congress like this is that it's also the place where we discuss the newest consensuses and guidelines. We also had the results of the Baveno VIII update on the treatment of portal hypertension presented. This is really transformative because it is shaping clinical practice throughout the globe. Another thing was the preconference session on the definition of acute-on-chronic liver failure.

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These kinds of processes about guidelines, as well as consensus on definitions and nomenclature from a worldwide perspective rather than just an isolated European one, are very important because they have a direct impact on daily clinical practice and on research. They help to harmonise research efforts. In that regard, I think those were two important highlights of the Congress. Another highlight was also the consensus on porto-sinusoidal vascular disorder, an important cause of portal hypertension. Again, this was a worldwide effort, not just an EASL effort, for which the conclusions were presented at Baveno VIII a few weeks ago, and also during this Congress, and were also published to coincide with the Congress.