An Overview of the 2026 EASD Annual Meeting with Focus on the Cardiometabolic Impact of Adipose Tissue Dysfunction

Author:	*André C. Carpentier[1] 1. Department of Medicine, Division of Endocrinology, Faculté de médecine et des sciences de la santé, Université de Sherbrooke, Centre de recherche du Centre hospitalier universitaire de Sherbrooke, Canada *Correspondence to Andre.Carpentier@USherbrooke.ca

Conflicts of interest:	Carpentier has declared grants or contracts from Eli Lilly and Novo Nordisk, paid to institution; received consulting fees from Eli Lilly, Novo Nordisk, and Merck; served on a Data Safety Monitoring or Advisory Board for ICAN and the COLCOT T2D trial; and acted in a leadership role for Fondation du CHUS and Génome Québec.
Keywords:	Adipose tissue, cardiometabolic complications, diabetes, insulin resistance, MASLD, obesity.
Citation:	EMJ Diabet. 2026;14[1]:23-26. https://doi.org/10.33590/emjdiabet/2O5Q0H37

THE 2026 European Association for the Study of Diabetes (EASD) Scientific Meeting was very rich in presentations relating to adipose tissue structure and function as determinants of cardiometabolic outcomes. A great emphasis was put on MASLD, a cardiometabolic condition that often implicates dysregulated adipose tissue, fatty acid metabolic, immune-inflammatory, and endocrine functions. The number of relevant presentations was so large and so often performed simultaneously that it is impossible to summarise them all in the present article. Here are the highlights that reflect my selection of live session attendance during the meeting.

INSULIN RESISTANCE AND ADIPOSE TISSUES

The oral presentation session titled 'Insulin resistance and diabetes across the lifespan' that I had the pleasure to co-chair with Kalliopi Pafili from the German Diabetes Research Center in Dusseldorf, Germany, included two presentations that pointed towards the importance of obesity in diabetes and its complications. Tessa Crume, Colorado School of Public Health, Aurora, Colorado, USA, presented the trends of Type 1 and Type 2 diabetes incidence over a 21-year period in Colorado, USA. An acceleration of both Type 1 and Type 2 diabetes incidence was observed since 2014. The discussion pointed towards the rise in obesity prevalence as the cause for this acceleration of diabetes incidence, including Type 1 diabetes.

Jacques Murray Leech, University of Exeter, UK, showed the results from a study in 524 individuals from the UK Biobank carrying one missense variant of *INSR* conferring insulin resistance with a paradoxically favourable metabolic profile, including lower triglycerides and low-density lipoprotein cholesterol levels, while BMI was similar. There was no increase in diabetes, all-cause mortality, cardiovascular diseases, or malignancies in carriers of INSR variants. Many of the findings were replicated in two independent cohorts. This study

CC BY-NC 4.0 Licence • Copyright © 2026 EMJ

demonstrates that pure insulin resistance without the typical adiposopathy may not be an important contributor to Type 2 diabetes and its complications.

Finally, Wilhelmina Leppähaara, University of Turku, Finland, reported that hepatic insulin resistance, assessed by insulin-mediated suppression of endogenous glucose production, and adipose tissue insulin resistance, assessed by insulin-mediated 18-fluoro-deoxyglucose uptake with positron emission tomography (18FDG-PET) during a euglycemic hyperinsulinemic clamp, but not skeletal muscle insulin resistance, also assessed by 18FDG-PET during the insulin clamp, significantly drove whole-body insulin resistance associated with ageing in 503 individuals studied.

NEW FRONTIERS IN THE TREATMENT OF OBESITY

In the session titled 'Challenges in future obesity management', the first speaker, Martin Heni, Eberhard Karls University of Tübingen, Germany, showed unpublished data from a very large cohort study that specific organ ectopic fat excess (in the liver, pancreas, muscles, and in visceral adipose tissues) is associated specifically with different risks of diseases (breast cancer, major atherosclerotic cardiovascular events, atrial fibrillation, osteoarthritis, liver disease, and renal failure). Brain insulin signalling measured from nasal insulin administration is altered in visceral obesity and contributes to metabolic heterogeneity.

Timo Müller, Helmholtz Munich, Oberschleißheim, Germany, the second speaker, argued that multiple hormones are required for the regulation of food intake and energy metabolism. He and his colleagues have designed a pharmacological treatment strategy to use glucagon-like peptide-1 (GLP-1) +/- glucose-dependent insulinotropic polypeptide (GIP) agonists to co-deliver a triple peroxisome proliferator-activated receptor (PPAR) agonist to cells that express incretin receptors. He demonstrated formidable weight loss and metabolic improvement using GLP-1, GIP, and triple PPAR agonist treatment in mice that are not replicated with GLP-1 and triple PPAR agonist.[1] This shows that GIP

> **Updated guidance also emphasises that a reduction of liver fat fraction equal to or greater than 30% was shown to predict the resolution of MASH**



receptor expression targets the nuclear PPAR receptor agonists to the right cells (for example, adipocytes) to exert metabolic benefits. Müller further showed that GIP receptor plus GCG receptor co-agonism is very effective at inducing metabolic benefits but without the gastrointestinal and cardiac (increased heart rate and diastolic blood pressure) effects seen with GLP-1 agonism in rodents and non-human primates.

Carel le Roux, University College Dublin, Ireland, the third speaker, demonstrated that after bariatric surgery, as after any pharmacological intervention, there is highly variable weight loss between patients. Main predictors of weight loss include living with diabetes (lower weight loss), the type of surgical procedure used, and the weight loss experienced by a first-degree relative with the same intervention, while the immediate environment (same household) does not influence weight loss. Furthermore, the Innovative Medicines Initiative Stratification of Obesity Phenotypes to Optimize Future Obesity Therapy (SOPHIA) project shows that applying multiomic approaches does not outperform the current clinical prediction tool for weight loss after bariatric surgery. le Roux finally stressed that the current emphasis on percent weight loss does not predict cardiovascular benefits as well as the achievement of a waist-to-height ratio of less than 0.5. There is currently uncertainty about the exact treatment target of obesity to optimise treatment benefits.

WHEN DIABETES MEETS THE LIVER

The 'Diabetologia symposium: when diabetes meets the liver: MASLD in focus' featured Sami Qadri, University of Helsinki, Finland; Amalia Gastaldelli, CNR Area della Ricerca di PISA, Italy; and Cyrielle Caussy, Lyon 1 Université Claude Bernard, Lyon, France, and focused on the 2026 updated European Association for the Study of the Liver (EASL)-EASD-European Association for the Study of Obesity (EASO) guidance on the use of resmetirom and semaglutide as metabolic dysfunction-associated steatohepatitis (MASH)-targeted therapies.[2] While there is some controversy about the capacity of the non-invasive testing strategy recommended to identify those patients with significant liver fibrosis for treatment,[3] there is consensus that patients living with diabetes and those displaying several features of the metabolic syndrome are at increased risk of advanced liver disease. Furthermore, liver biopsy is not necessary to initiate therapy in those with non-invasive diagnosis indicative of F2–F3 liver fibrosis. While both resmetirom and semaglutide displayed similar efficacy to improve MASH and liver fibrosis,



CC BY-NC 4.0 Licence • Copyright © 2026 EMJ

the new EASL-EASD-EASO guidance suggests that the presence of conditions such as insufficiently controlled Type 2 diabetes, obesity-related comorbidities, cardiovascular disease, or chronic kidney disease should lead to the preferential use of semaglutide, while the absence of overweight/obesity, the presence or high risk of sarcopenia, and treatment with a GLP-1 receptor agonist already present should favour the use of resmetirom. This updated guidance also emphasises that a reduction of liver fat fraction equal to or greater than 30% was shown to predict the resolution of MASH.

> **Pure insulin resistance without the typical adiposopathy may not be an important contributor to Type 2 diabetes and its complications**

ADIPOSE TISSUE STRUCTURE AND FUNCTION IN CARDIOMETABOLIC DISEASES

A session on human adipose tissue featured Assaf Rudich, Ben-Gurion University of the Negev, Beer-Sheva, Israel, as the first speaker, who reported yet-unpublished single-nucleus gene expression analyses in human visceral adipose tissue, demonstrating that all cell types express the insulin receptor and signalling pathway genes to significant levels. *Ex vivo* insulin stimulation also induces a transcriptional response in all cell types that is significantly influenced by whole-body insulin resistance as assessed by Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) and Adipose Tissue Insulin Resistance Index (ADIPO-IR).

The second speaker, Ferdinand von Mayenn, ETH Zürich, Switzerland, showed that diet-induced obesity in mice induces a durable epigenetic signature in adipose tissues. *In vivo* semaglutide treatment does not reset this epigenetic memory of obesity in these animals but induces an additional semaglutide-specific epigenetic signature.

Karine Clement, Sorbonne Université, France, the third speaker of the session, discussed the increase in adipose tissue collagen and macrophage content after bariatric surgery and the inverse relation between adipose tissue collagen content and weight loss after surgery. She also showed the inverse relation between the amount of PDGFRα+/CD9+ progenitor cells and post-surgical metabolic improvement, and demonstrated that therapeutic targeting of these precursors improves the metabolic outcome.[4] She further stressed that collagen accumulation in adipose tissue results from reduced removal rather than increased deposition, implying that impaired remodelling is key to adipose tissue function. She finished her talk with a call to characterise the structure and function of adipose tissues to better stratify patient outcomes.[4]

CONCLUDING REMARK

Thus, much of the novel evidence presented during the 2026 EASD meeting points towards the critical role of abnormal adipose tissue mass, structure and function in diabetes and other cardiometabolic comorbidities. Furthermore, there is growing emphasis on the importance of adipose tissue function as opposed to mass/volume measurement, for better stratification of cardiometabolic risk.

References

1. Liskiewicz D et al. GLP-1R-GIPR-PPARα/γ/δ quintuple agonism corrects obesity and diabetes in mice. Nature. 2026;653(8115):776-85.
2. EASL; EASD; EASO. EASL-EASD-EASO guidance for the use of resmetirom and semaglutide as MASH-targeted therapy. J Hepatol. 2026;DOI:10.1016/j.jhep.2026.08.028.
3. Qadri SF. Screening and diagnosis of liver disease in diabetes: a guide and evidence-based assessment. Diabetologia. 2026;69(11):3048-72.
4. Rebière C et al. Combined pharmacological targeting of CD9+ progenitors alleviates obesity-induced adipose tissue fibrosis and metabolic impairment. Cell Rep Med. 2025;6(7):102197.