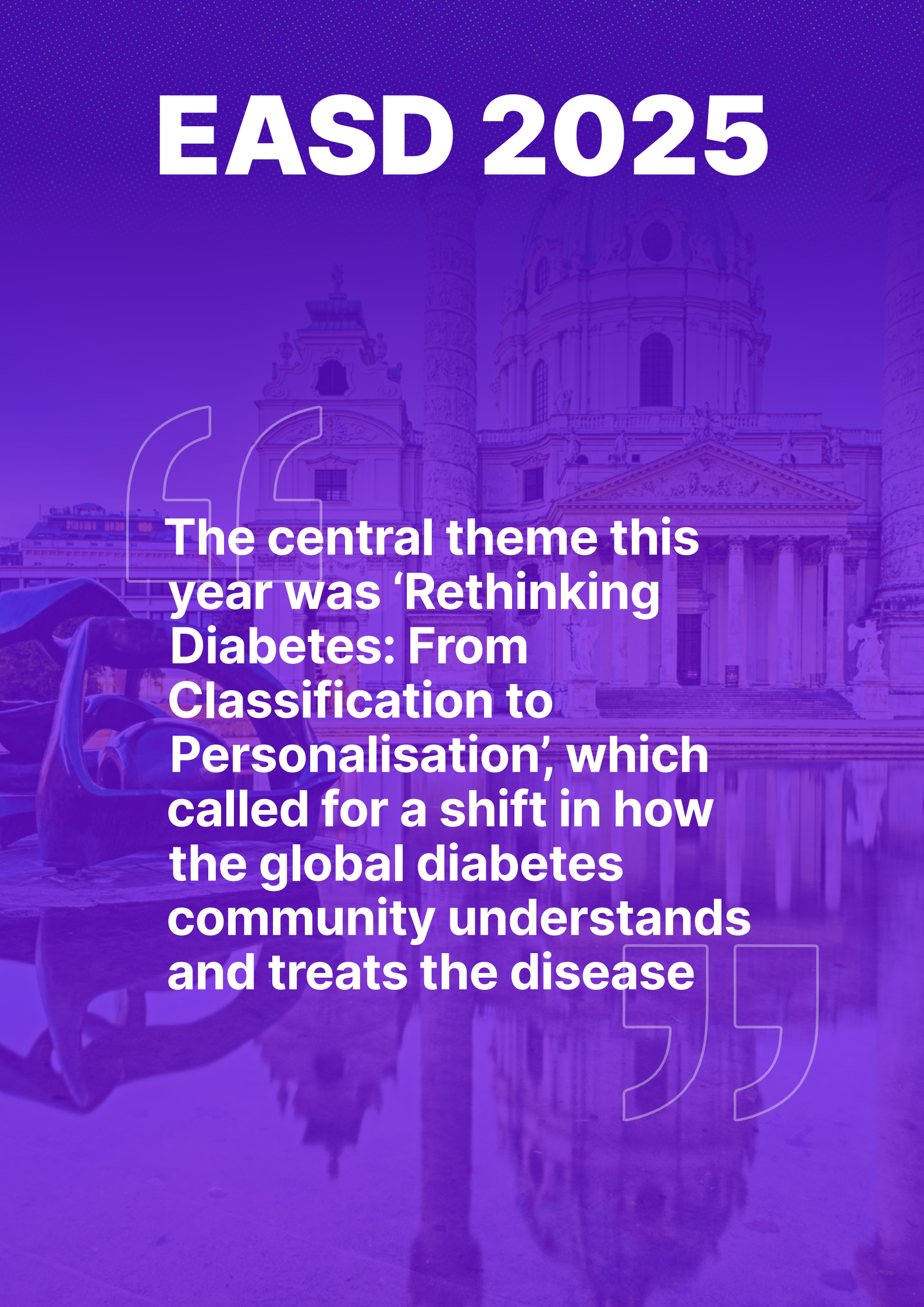
EASD 2025



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Congress Review

Review of the European Association for the Study of Diabetes (EASD) Annual Meeting 2025

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THE 61ST ANNUAL Meeting of the European Association for the Study of Diabetes (EASD) took place in the historic and elegant city of Vienna, Austria, from 15th–19th September. Against the backdrop of grand imperial architecture and a thriving scientific community, the Annual Meeting united researchers and clinicians in an atmosphere of discovery and collaboration.

The central theme this year was 'Rethinking Diabetes: From Classification to Personalisation', which called for a shift in how the global diabetes community understands and treats the disease. The opening ceremony set the tone with a warm welcome to this year's attendees by the EASD President, Chantal Mathieu, who reflected on "how many people with diabetes do not fit neatly into a single box," and called for a move from "types to traits" and categorisation to individualisation, in line with the EASD theme. She emphasised that true progress lies in embracing the heterogeneity of diabetes and tailoring care to each patient's unique disease profile, a vision that aligns with the growing momentum in precision medicine.

Mirroring this philosophy, the scientific programme featured 1,354 abstracts, a record number that resulted in close to 2,000 oral and short oral presentations, with topics ranging from novel therapies and disease-modifying treatments to implementation science and digital health innovation.

The ceremony continued with the introduction of new session formats by Programme Committee Chair Tina Vilsboll, which was met with great enthusiasm by the audience. Alongside the popular Rising Star Symposia, this year's Meeting featured interactive Lab Talks, giving attendees a look into leading laboratories, and Early Bird Symposia, which showcased the latest breaking clinical trials.

A notable highlight was the presentation of EASD's first-ever clinical guideline focusing on diabetes distress, a topic identified as an unmet need by both clinicians and people living with diabetes.

EASD also reaffirmed its dedication to nurturing the next generation of researchers through the Early Career Academy, led by Patrick Schrauwen, Chair of the EASD Early Career Academy Committee, and through global partnerships spearheaded by Francesco Giorgino, Chair of the EASD Global Council.

In her closing remarks, Mathieu expressed gratitude to the Executive Board, the Düsseldorf secretariat, and particularly to long-serving team member Mary Hatter, who retired after decades of service to EASD. With humour and humility, she concluded "it has been an honour to be your president," before offering a final wish for good health, peace, and a joyful reunion at EASD 2026 in Milan, Italy.

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Physical Activity Predicts Heart Risk and Survival in Newly Diagnosed Type 2 Diabetes

PHYSICAL activity is a well-established component of managing Type 2 diabetes (T2D), with benefits including improved glycaemic control, better insulin sensitivity, and healthier body composition. While its protective cardiovascular effects in the general population are widely recognised, less is known about its prognostic value in people newly diagnosed with T2D. A new large-scale Danish study presented at EASD 2025 has found that higher levels of self-reported physical activity are independently associated with a significantly lower risk of cardiovascular events and all-cause mortality, even after adjusting for conventional cardiovascular risk factors.[1]

The prospective cohort study analysed data from 11,355 adults enrolled in the Danish Centre for Strategic Research in Type 2 Diabetes (DD2) cohort between 2010–2023. All participants had been diagnosed with T2D within the previous 2 years and had no history of cardiovascular disease at baseline. Physical activity levels were self-reported using the Saltin-Grimby scale and categorised as sedentary, light, or moderate-to-vigorous. Participants were followed for a median of 8.4 years, with primary outcomes being major adverse cardiovascular events (MACE) and all-cause mortality. Those with prior cardiovascular disease were excluded.

Among the participants, 18.0% were sedentary, 62.5% engaged in light activity, and 19.5% in moderate-to-vigorous physical activity. During follow-up, 1,149 MACE and 1,048 deaths occurred. After adjusting for age, sex, lifestyle factors, and central obesity, light activity was associated with a 23% reduced risk of MACE (hazard ratio [HR]: 0.77; 95% CI: 0.68–0.95) and a 27% lower risk of all-cause mortality (HR: 0.78; 95% CI: 0.65–0.94). For moderate-to-vigorous physical activity, the reductions were 28% for MACE (HR: 0.70; 95% CI: 0.57–0.87) and 33% for all-cause mortality (HR: 0.69; 95% CI: 0.54–0.87). These associations remained significant even after adjusting for low-density lipoprotein cholesterol, HbA1c, blood pressure, kidney function, and albuminuria.

The findings highlight self-reported physical activity as a robust, independent predictor of cardiovascular outcomes and survival in people with newly diagnosed T2D. While causality cannot be established from observational data, and self-reporting may introduce measurement bias, the persistent associations suggest real-world relevance. Clinicians should consider physical activity levels not only as a modifiable risk factor, but also as a useful prognostic marker in early diabetes care. Simple assessments of activity may help identify high-risk patients who could benefit most from targeted interventions.

> **Higher levels of self-reported physical activity are independently associated with a significantly lower risk of cardiovascular events and all-cause mortality**

Study Maps Type 1 Diabetes Risk to Rural Childhood Environments in Sweden

SWEDEN has one of the highest rates of Type 1 diabetes (T1D) globally, yet its geographical distribution remains uneven. This variation has long hinted at the role of environmental risk factors, particularly in early life. While previous research has examined where patients were born or diagnosed, new research presented at EASD 2025 took a more detailed approach, tracking residential history from birth to diagnosis to map high- and low-risk areas over time. A key finding was that areas with the highest T1D incidence were rural, while Sweden's largest cities had significantly lower rates.[2]

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up to

↑2.7

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↓0.1

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Researchers identified all patients aged 0–30 diagnosed with T1D in Sweden between 2005–2022 using the Swedish National Diabetes Register. This cohort included 21,774 individuals, 57.7% of whom were male, and the mean age at diagnosis was 13.6 years. Address histories from birth to diagnosis were obtained from Statistics Sweden. Using spatio-temporal scan statistics, the team identified clusters of unusually high or low incidence, both at the time of diagnosis and during the first 5 years of life. Land use characteristics within these clusters were also analysed.

Four high-risk clusters were identified based on residence at diagnosis, all in central Sweden, with relative risks (RR) ranging 1.3–1.8. Notably, no high-risk clusters were found in Sweden's major cities. In contrast, low-risk clusters were concentrated in the largest urban areas, with RRs between 0.5–0.8. When analysing residence during the first 5 years of life, the picture became even clearer: 11 high-risk and 15 low-risk clusters were identified. The northern regions had the highest RRs (up to 2.7), while the lowest risk (RRs as low as 0.1) was again seen in urban centres. Land cover analysis showed that high-risk areas were dominated by forests and agriculture, while low-risk clusters were largely urban and open land.

This study strongly supports the hypothesis that environmental exposures in rural areas during early childhood increase the risk of developing T1D. While it cannot determine causality or identify specific environmental triggers, it provides a robust framework for future investigations. Limitations include a lack of direct environmental measurements and potential confounding by socioeconomic factors. Clinically, these findings highlight the importance of environmental context in early-life diabetes prevention strategies and warrant further attention in both research and public health planning.

Sex-Specific Heart Risk Gaps Found Between Type 1 and Type 2 Diabetes

CARDIOVASCULAR disease (CVD) remains the leading cause of death worldwide, with people living with Type 1 (T1D) or Type 2 diabetes (T2D) facing significantly elevated risk. While extensive research has been done on the cardiovascular implications of both diabetes types, direct comparisons of risk between T1D and T2D within each sex have been lacking. A new Swedish cohort study presented at EASD 2025 addresses this gap, revealing important sex-specific differences in cardiovascular outcomes. Notably, younger men with T2D are at higher cardiovascular risk than those with T1D, while women with T1D consistently face greater risk than those with T2D across all age groups.[3]

Researchers analysed data from over 400,000 adults with diabetes (38,351 with T1D and 365,675 with T2D), aged 18–84 years, using records from the Swedish National Diabetes Register. Individuals were followed for 5 years (2016–2020) to assess incidence of myocardial infarction (MI), heart failure, stroke, cardiovascular mortality, and all-cause mortality. Cox proportional hazards models were used to estimate risk, adjusting for age and diabetes type, with further adjustments for established cardiovascular and socioeconomic risk factors.

In men under 50 years of age, T2D was associated with significantly higher risk compared to T1D for all CVD (hazard ratio [HR]: 1.51), MI (HR: 2.40), and heart failure (HR: 2.16). Conversely, men over 70 years of age with T2D had lower MI risk (HR: 0.74). Overall, males with T2D had lower cardiovascular and all-cause mortality than those with T1D (HR: 0.84 and 0.90, respectively). For women, those with T2D had consistently lower risks across all outcomes compared to those with T1D. For instance, in the 50–59-year age group, women with T2D had reduced risk of all CVD (HR: 0.75) and MI (HR: 0.59). Sex-stratified analyses confirmed female sex to be protective in both diabetes types, although this protective effect was less pronounced in T1D.

These findings have important clinical implications. They suggest that younger males with T2D warrant more aggressive cardiovascular risk assessment and intervention, while women with T1D may benefit from more intensive management than current practice provides. A key limitation is that the observational nature of the study cannot establish causality, and residual confounding may remain. Nonetheless, this large-scale, sex-specific analysis highlights the need to tailor cardiovascular risk strategies not only to diabetes type, but also to sex and age.

> **Younger men with T2D are at higher cardiovascular risk than those with T1D, while women with T1D consistently face greater risk than those with T2D across all age groups**

Hepatitis B Immunity Linked to Lower Diabetes Risk

NEW research presented at EASD 2025 suggests that immunity to hepatitis B virus (HBV) may lower the risk of developing diabetes, highlighting an unexpected metabolic benefit of HBV vaccination.[4]

The study utilised data from de-identified electronic medical records within the TriNetX (Cambridge, Massachusetts, USA) Research Platform, focusing on adults with documented hepatitis B surface antibody (HBsAb) levels but no prior history of HBV infection or diabetes. Diabetes was defined based on clinical diagnosis, diabetes medication use, or HbA1c ≥6.5%. Propensity score matching was performed to balance demographic factors and comorbidities between groups with and without HBV immunity.

Results showed that individuals with HBV immunity had a 15% lower risk of developing diabetes compared to unimmunised individuals (hazard ratio [HR]: 0.85; 95% CI: 0.84–0.87). A dose-response relationship was observed, with higher antibody levels corresponding to greater reductions in diabetes risk. Participants with HBsAb levels ≥100 mIU/mL had a 19% lower risk (HR: 0.81; 95% CI: 0.80–0.83), while those with levels ≥1,000 mIU/mL experienced a 43% lower risk (HR: 0.57; 95% CI: 0.54–0.60) compared with those with antibody levels <10 mIU/mL.

Age-stratified analyses revealed consistent benefits across all age groups. Immunised individuals aged 18–44 years, 45–64 years, and ≥65 years demonstrated 20% (HR: 0.80; 95% CI: 0.78–0.82), 11% (HR: 0.89; 95% CI: 0.87–0.92), and 12% (HR: 0.88; 95% CI: 0.84–0.91) lower diabetes risks, respectively, compared with their unimmunised counterparts.

These findings suggest that HBV immunity may have protective metabolic effects independent of viral infection status. The observed association between HBV antibody levels and diabetes risk reduction supports the potential of HBV vaccination as a dual-benefit public health measure, offering both viral protection and possible metabolic advantages.

Further investigation is warranted to clarify the underlying mechanisms linking HBV immunity to improved glucose regulation and to evaluate whether enhanced HBV vaccination strategies could contribute to diabetes prevention efforts, particularly in regions with high prevalence of both conditions.

Sleep Apnoea Treatment Improves Survival in Type 2 Diabetes

A LARGE Swedish study, presented at EASD 2025, has found that people with Type 2 diabetes (T2D) who were prescribed continuous positive airway pressure (CPAP) therapy for obstructive sleep apnoea (OSA) had significantly lower long-term mortality than those who were not. The findings suggest that treating OSA may be an important, yet under-recognised component of diabetes management.[5]

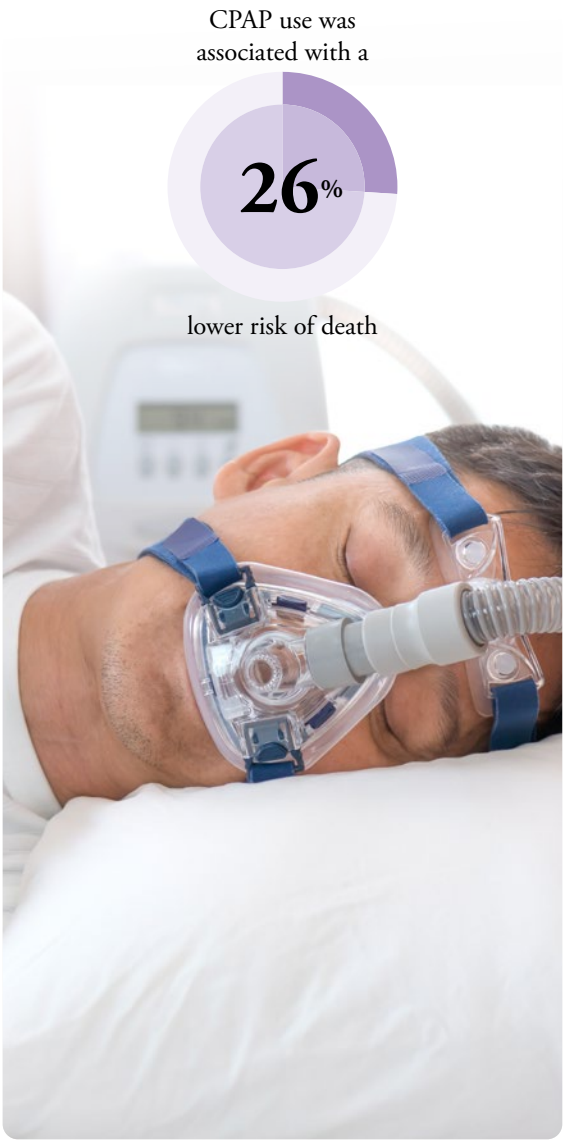
OSA frequently coexists with T2D, affecting 50–80% of patients. Despite its association with increased cardiovascular risk and mortality, OSA often remains undiagnosed and is not currently considered a modifiable risk factor in diabetes care. While CPAP is known to improve sleep quality and daytime symptoms, its long-term effects on survival in patients with T2D have been unclear due to limited follow-up in previous studies.

Using data from five national Swedish registers, researchers followed 750,299 adults with T2D over a period of 14-year. Of these, 12,388 individuals had confirmed OSA and had been prescribed CPAP, while 737,911 individuals, whose OSA status was unknown, had never received CPAP. The CPAP-treated group tended to be younger (mean: 58 years versus 65 years) and had a higher average BMI (34.7 kg/m² versus 30.6 kg/m²).

During follow-up, there were 212,336 deaths in the non-CPAP group and 764 in the CPAP group. After adjusting for multiple factors, including age, sex, cardiovascular history, BMI, smoking, and other clinical variables, CPAP use was associated with a 26% lower risk of death, corresponding to a hazard ratio of 0.74 (95% CI: 0.68–0.82; p<0.001).

> **These results provide compelling evidence that long-term CPAP use may improve survival in people with both T2D and OSA**

These results provide compelling evidence that long-term CPAP use may improve survival in people with both T2D and OSA. However, further research using causal inference methods is needed to confirm whether the observed association reflects a direct protective effect of CPAP therapy.



Oral Baricitinib Preserves Insulin Production in Recent-Onset Type 1 Diabetes

A NEW analysis from the BANDIT trial has reinforced the therapeutic potential of baricitinib in preserving β-cell function in individuals recently diagnosed with Type 1 diabetes (T1D). Building on earlier findings that demonstrated clinical benefits after 48 weeks of treatment, the latest data presented at EASD 2025 reveal that these effects begin to diminish once the drug is discontinued. Crucially, this is one of the few studies to demonstrate significant C-peptide preservation with an oral agent in T1D, a key marker of ongoing insulin production.[6]

In the double-blind RCT, 91 participants aged 10–30 years who were within 100 days of T1D diagnosis were enrolled. All had detectable C-peptide and at least one islet autoantibody. They were randomly assigned in a 2:1 ratio to receive either oral baricitinib 4 mg daily or placebo for 48 weeks. C-peptide responses were assessed using mixed-meal stimulation at baseline, during treatment (Weeks 12, 24, and 48), and at follow-up visits at Weeks 72 and 96 after treatment cessation.

At the primary endpoint of 48 weeks, baricitinib-treated participants had significantly higher median stimulated C-peptide levels compared to placebo (0.65 versus 0.43 nmol/L/min; p=0.001). However, by 96 weeks, after stopping treatment, the difference narrowed and was no longer statistically significant (0.37 versus 0.26; p=0.336). Similarly, initial improvements in glucose time-in-range and insulin requirements waned during the off-drug period. While 75% of baricitinib recipients met the quantitative response score threshold at 48 weeks versus 55% in the placebo group (p=0.0154), this advantage was lost post-treatment. No baseline factors predicted sustained response, and there were no new safety concerns during follow-up.

These findings support the sustained, but not permanent, benefit of baricitinib in early T1D, highlighting the potential need for longer-term treatment to maintain clinical improvements. The oral administration and favourable safety profile offer a compelling case for baricitinib's use in routine care. However, the waning effects post-cessation limit the current findings and suggest further trials are needed to explore extended or maintenance dosing strategies, as well as the drug's role in earlier stages of the disease.

> **The oral administration and favourable safety profile offer a compelling case for baricitinib's use in routine care**

Study Confirms Strong Two-Way Link Between Depression and Diabetes in Older Adults

A LARGE-SCALE European study presented at EASD 2025 has confirmed a robust, two-way link between depressive symptoms and diabetes in older adults, with findings that hold true regardless of the country or most sociodemographic factors. Drawing on data from three major ageing cohort studies, the research explored whether factors like healthcare quality, income inequality, and physical activity influenced the strength of the relationship. A key finding was that depressive symptoms increased the risk of diabetes, and diabetes likewise predicted the development of depressive symptoms, independent of where participants lived.[7]

Researchers combined data from over 77,000 individuals aged 50 years and above from the English Longitudinal Study on Ageing, the Irish Longitudinal Study on Ageing, and the Survey on Health, Ageing and Retirement in Europe. They used a survival analysis approach to examine two pathways: from depression to diabetes and from diabetes to depression. Country-level factors included healthcare quality and socioeconomic indicators, while individual-level variables covered age, gender, BMI, smoking, and physical activity.

The study found that elevated depressive symptoms were associated with a 15% increased risk of developing diabetes (hazard ratio [HR]: 1.15; 95% CI: 1.11–1.20), while having diabetes increased the likelihood of developing depressive symptoms by 48% (HR: 1.48; 95% CI: 1.37–1.61). All sociodemographic factors significantly predicted either diabetes or depression individually ($p<0.01$), but none, except BMI, altered the strength of the relationship between the two conditions. Specifically, individuals with diabetes and a higher BMI had a slightly increased risk of developing depressive symptoms (HR: 1.02; 95% CI: 1.01–1.04; $p=0.006$). No substantial variation in the strength of the associations was found across different European countries.

The findings suggest that the bidirectional relationship between depressive symptoms and diabetes remains largely stable across diverse European populations and healthcare systems. For clinical practice, this highlights the importance of mental health screening in diabetes care and, conversely, metabolic risk monitoring in patients with depression. While the study was comprehensive, limitations include reliance on self-reported measures and the observational design, which precludes conclusions about causality. Nonetheless, the consistent association across countries and demographic factors highlights the need for integrated approaches in managing both physical and mental health in ageing populations.

Having diabetes increased the likelihood of developing depressive symptoms by

Semaglutide Shows Promise in Protecting Against Diabetic Retinopathy

A RECENT Greek study presented at EASD 2025 has suggested that semaglutide, a glucagon-like peptide-1 (GLP-1) receptor agonist, provides strong protection against diabetic retinopathy, the leading cause of blindness among working-age adults.[8]

The study explored the impact of GLP-1 drugs, which are commonly used to treat Type 2 diabetes and obesity. These drugs mimic the action of the GLP-1 hormone, which plays a role in insulin production, digestion, appetite regulation, and increased feelings of fullness. In addition, the study highlighted the potential antioxidant and anti-inflammatory properties of GLP-1 drugs, which may be crucial in protecting the eyes from diabetic retinopathy.

The study involved lab-based tests using human retinal endothelial cells exposed to high glucose levels and oxidative stress, mimicking diabetic conditions. Semaglutide was applied at various concentrations to the retinal cells for 24 hours, followed by a series of tests. The results showed that retinal cells treated with semaglutide were up to twice as likely to survive compared to untreated cells. Additionally, three key markers of oxidative stress, apoptosis (cell death), mitochondrial superoxide production, and accumulation of advanced glycation end-products, were significantly reduced in the treated cells. Apoptosis decreased from around 50% in untreated cells to just 10% in semaglutide-treated cells, while mitochondrial superoxide production fell from about 90% to just 10%. Further analysis revealed that semaglutide upregulated genes involved in antioxidant production, providing more evidence that the drug could repair cellular damage caused by diabetes-like conditions.

> **The results showed that retinal cells treated with semaglutide were up to twice as likely to survive compared to untreated cells**

These promising results suggest that GLP-1 receptor agonists, such as semaglutide, have powerful antioxidant effects that could protect retinal cells from damage and possibly repair existing harm. This is particularly important given the high prevalence of diabetic retinopathy in people with diabetes, affecting up to 90% of individuals with Type 1 diabetes and 50–60% with Type 2 diabetes. Clinical trials are needed to confirm these findings in human patients and to determine whether GLP-1 receptor agonists can slow or even halt the progression of diabetic retinopathy. If proven effective, these drugs could play a vital role in clinical practice by slowing or halting the progression of diabetic retinopathy, ultimately reducing the number of people affected by vision-threatening stages of the condition.

Smoking Strongly Linked to Insulin-Resistant Type 2 Diabetes

RECENT research has suggested that Type 2 diabetes (T2D) is not a single disease, but instead comprises four distinct subtypes: two severe (severe insulin-resistant diabetes [SIRD] and severe insulin-deficient diabetes) and two milder forms (mild obesity-related diabetes and mild age-related diabetes). Understanding how lifestyle factors such as smoking affect the risk of each subtype could support more targeted prevention efforts. In a large-scale study combining Swedish and Norwegian data presented at EASD 2025, researchers found that smoking significantly increased the risk of all T2D subtypes, with the strongest association observed for SIRD.[9]

Data were drawn from two large population-based studies: the Swedish case-control study ESTRID (2010–2024) and the Norwegian cohort study HUNT (1984–2008). Together, these included 3,325 incident T2D cases and 3,897 controls, with 873,349 person-years of follow-up. Participants were classified into one of the four T2D subtypes. Genetic risk scores for overall T2D, insulin resistance (IR), and insulin secretion were calculated to explore gene-environment interactions. Associations between smoking and T2D subtype risk were analysed using pooled relative risk (RR) estimates. A two-sample Mendelian randomisation (MR) approach was applied to support causal inference.

Compared with never smokers, ever smokers were at more than twice the risk of developing SIRD (RR: 2.15; 95% CI: 1.64–2.82). The association was weaker but still significant for mild obesity-related diabetes (RR: 1.29; 95% CI: 1.06–1.57), mild age-related diabetes (RR: 1.27; 95% CI: 1.12–1.44), and borderline for severe insulin-deficient diabetes (RR: 1.20; 95% CI: 0.98–1.47). Heavy smokers (≥15 pack-years) were at substantially higher risk across all subtypes, with the strongest effect again observed for SIRD (RR: 2.35; 95% CI: 1.72–3.23). Population-attributable risk estimates indicated that smoking accounted for over one-third of SIRD cases (35.3%). Additive interaction was found between heavy smoking and genetic susceptibility to T2D and insulin deficiency, but not IR. MR analyses confirmed the observed associations.

These findings strengthen the evidence that smoking is a major modifiable risk factor for all forms of T2D, particularly SIRD, which is driven by IR. Importantly, individuals with a genetic predisposition to T2D or impaired insulin secretion appear to be more vulnerable to the harmful metabolic effects of smoking. While the observational nature of parts of the study limits causal certainty, the use of MR helps to mitigate this. The results support the integration of smoking cessation advice into diabetes prevention strategies, with particular attention to those at high genetic risk.

Who Stops Semaglutide? Large Study Reveals Predictors of Discontinuation

THE INCREASING use of semaglutide for weight loss (SEMA-WL) has raised concerns about real-world treatment persistence, particularly in patients without diabetes. While clinical trials suggest high efficacy, less is known about how long people continue taking the medication in routine clinical settings. A recent study presented at EASD 2025 used nationwide registry data from Denmark to assess discontinuation rates and identify which patient groups are more likely to stop treatment. Strikingly, one in two adults stopped SEMA-WL within the first year of use.[10]

Using linked Danish health registries, researchers identified 77,310 adults without diabetes who initiated SEMA-WL between 1st December 2022–1st October 2023. Individuals were considered to have discontinued treatment if there was a gap of more than 60 days between prescriptions. Poisson regression was used to calculate age- and sex-adjusted risk ratios (RR) for various demographic and clinical factors potentially associated with treatment discontinuation.

Of the 77,310 people included (median age: 50 years; 71% women), 52% discontinued semaglutide within 12 months. Discontinuation occurred rapidly for some: 18% had stopped within 3 months, 31% within 6 months, and 42% within 9 months. Younger adults aged 18–30 years were significantly more likely to stop treatment compared with those aged 45–60 years (RR: 1.48; 95% CI: 1.45–1.51). Male users were also more likely to discontinue use (RR: 1.12; 95% CI: 1.11–1.14). Other predictors of early discontinuation included prior use of psychiatric (RR: 1.12) or gastrointestinal medications (RR: 1.09), cardiovascular disease (RR: 1.11), higher comorbidity (Charlson index 3+; RR: 1.09), and residence in lower-income municipalities (RR: 1.14).

> **One in two adults stopped SEMA-WL within the first year of use**

These findings suggest that, in real-world settings, a significant proportion of patients stop SEMA-WL relatively soon after starting, highlighting important limitations in long-term adherence. For clinical practice, this emphasises the need to assess not only eligibility for treatment but also the likelihood of persistence, particularly among younger adults, men, and those with complex health or socioeconomic challenges. Limitations of the study include lack of data on reasons for discontinuation (e.g., side effects, cost, or perceived benefit), as well as the reliance on prescription data, which may not reflect actual medication use. Nonetheless, these insights are crucial for shaping patient expectations and informing follow-up strategies in obesity care.

Younger adults aged **18–30 years** were significantly more likely to stop treatment compared with those aged **45–60 years**

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