Diabetes Metabolism and the Heart

Diabetes, Stoffwechsel und Herz

CVOT SUMMIT 2026

CARDIOVASCULAR, KIDNEY AND METABOLIC OUTCOMES

Virtual CVOT Summit 2026, 19–20 November 2026

FINAL PROGRAMME AND ABSTRACTS

8.5 European CME credits (EACCME®)

The Virtual CVOT Summit 2026, 19 – 20 November 2026 has been accredited by the European Accreditation Council for Continuing Medical Education (EACCME®) with **8.5 European CME credits (ECMEC®s)**. Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity.

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Welcome



Oliver Schnell
(Munich, Germany)

Dear Colleagues,

Since its initiation in 2015, the CVOT Summit has served as an international platform for discussing the evolving landscape of cardiovascular outcome trials (CVOTs) and their implications for clinical practice. Over the years, the summit has become a leading interdisciplinary congress, bringing together cardiologists, nephrologists, endocrinologists, diabetologists, obesity and liver specialists as well as primary care physicians from around the world to exchange knowledge, share insights, and explore advances in patient care.

It is our great pleasure to invite you to the 12th Annual CVOT Summit, taking place on 19–20 November 2026. Continuing our established tradition, the congress will be broadcast live from Munich to a global virtual audience, enabling healthcare professionals worldwide to participate in this scientific exchange.

This year's programme will feature state-of-the-art lectures, expert-led panel discussions, and interactive sessions exploring the latest scientific evidence and clinical advances in the management of cardiovascular disease, heart failure, chronic kidney disease, obesity, liver disease, and diabetes. Particular emphasis will be placed on translating emerging research findings into practical strategies for improving patient outcomes.

We warmly invite you to join us for two days of scientific exchange, expert insights, and interdisciplinary dialogue.

Our sincere gratitude goes to all participants, speakers, and partners who have contributed to the continued success of the CVOT Summit. Their dedication, expertise, and scientific contributions have been instrumental in establishing the summit as an internationally recognised platform for scientific exchange and interdisciplinary collaboration.

We encourage you to register at your earliest convenience and look forward to welcoming you to the Virtual CVOT Summit 2026.

With kind regards,



Oliver Schnell
President Local Organising Committee

Local organising committee

President
Oliver Schnell

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DDG Deutsche Diabetes Gesellschaft

General information

The CVOT Summit 2026 is run as virtual event. It can be visited via **www.cvot.org**.

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This accreditation is kindly supported by Abbott, AstraZeneca, Bayer, Boehringer Ingelheim, Dexcom, Eli Lilly, Roche, Sanofi and Wörwag (10,500 €)

THURSDAY, 19 NOVEMBER 2026 VIRTUAL

15:00–15:10 CET Welcome and Introduction
Oliver Schnell (Munich, Germany)

15:10–16:15 CET Emerging Frontiers in Cardiovascular, Kidney, and Metabolic Care
Chairs: Francesco Giorgino (Bari, Italy), Alice Pik Shan Kong (Sha Tin, Hong Kong)

15:10–15:25 **From comorbidities to a single syndrome: A new understanding of cardiovascular, renal and metabolic diseases**
Neha Pagidipati (Durham, NC, USA)

15:25–15:45 **Gut Hormone Agonists 2026: Emerging Therapies and New Metabolic Opportunities**
Timo Müller (Munich, Germany)

15:45–16:00 **Advancing Cardiorenal Protection Through Aldosterone Synthase Inhibition**
Erika Jones (Cape Town, South Africa)

16:00–16:15 **Discussion**

16:15–17:15 CET New Guidelines 2026 – Transforming Clinical Care in Chronic Diseases
Chairs: Shelley Zieroth (Winnipeg, Canada), Pardeep Jhund (Glasgow, UK)

16:15–16:30 **Beyond Weight Loss: A New Framework for Pharmacological Obesity Management**
Andreea Ciudin (Barcelona, Spain)

16:30–16:45 **Breaking Down Silos: Advancing Integrated Cardiovascular, Kidney and Metabolic Care**
Per-Henrik Groop (Helsinki, Finland)

16:45–17:00 **The 2026 ESC Heart Failure Guidelines: Key Updates for Clinical Practice**
Theresa McDonagh (London, UK)

17:00–17:15 **Discussion**

17:15–17:30 **Break**

17:30–18:30 CET Policy Session: Transforming Cardiometabolic Health: Advancing Science, Integrated Care and Policy
Chair: Oliver Schnell (Munich, Germany)

17:30–17:35 **Welcome and Set the Scene**
Oliver Schnell (Munich, Germany)

17:35–17:45 **Obesity as a Cardiovascular Health Priority**
Matthias Blüher; Leipzig University (Leipzig, Germany)

17:45–17:55 **EU Safe Hearts Plan**
Marianne Takki; European Commission (Brussels, Belgium)

17:55–18:05 **From Risk Detection to Structured Care – "Modernizing Check-Up 35"**
Florian Kahles; University Hospital RWTH Aachen (Aachen, Germany)

18:05–18:25 **Panel Discussion: From Screening to Structured Care – Building an Interconnected Cardiometabolic Prevention Strategy**
Faculty and further Participants:
Winfried Klausnitzer; Heart Failure Germany (Nuremberg, Germany)

Sonja Mayer; Bavarian Chamber of Pharmacists (Gröbenzell, Germany)
Raffaele Napoli; University of Naples Federico II (Naples, Italy)
Bart Torbeyns; European Diabetes Forum (Brussels, Belgium)

18:25 – 18:30 **Closing Remarks**
Oliver Schnell (Munich, Germany)

18:30 – 19:30 CET Satellite Symposium: CVOTs in type 2 diabetes and obesity: past, present and future

Chair: Jonathan Rachman (UK)

supported by Eli Lilly and Company

18:30 – 18:35 **Welcome and introduction**
Jonathan Rachman (UK)

18:35 – 19:25 **CVOTs in type 2 diabetes and obesity: past, present and future**
John Wilding (UK)

19:25 – 19:30 **Closing**
Jonathan Rachman (UK)

19:30 – 20:15 CET Oral Presentations – Part 1

Chair: Pinar Topsever (Istanbul, Turkey)

19:30 – 20:15

OP 1: Beyond cardiovascular-kidney-metabolic syndrome: towards a comprehensive nexus of cardiometabolic multiple long-term conditions
Stephan Jacob (Villingen-Schwenningen, Germany), Andrew J. Krentz (London, UK)

OP 2: What Patients With Cardiovascular-Kidney-Metabolic Syndrome Ask: A Clustering of 25 Million Patient Portal Messages
Matthias Christian Lutz (Stanford, USA)

OP 3: Standard Cost-Utility Analyses Underestimate Value: A Multidomain Economic Model of Spotlight-AQ Digital Health in Diabetes Care
Katharine Barnard-Kelly (Southampton, UK)

OP 4: An AI-Based Knowledge Center for Cross-Disciplinary Retrieval of Cardio-Kidney-Metabolic Clinical Guidelines
Elena Tonchevska (Munich, Germany)

OP 5: An AI-Based Knowledge Center to Support Evidence-Based Management of Hypertension
Moritz Schroll (Munich, Germany)

20:15 CET Closing Day 1

Oliver Schnell (Munich, Germany)

FRIDAY, 20 NOVEMBER 2026 VIRTUAL

08:30–09:30 CET Type 1 Diabetes: From Disease Modification to Organ Protection

Chair: Helen M. Colhoun (Edinburgh, UK)

08:30–08:45 **Rethinking Type 1 Diabetes: Screen, Detect, Prevent**
Chantal Mathieu (Leuven, Belgium)

08:45–09:00 **Immunomodulation in Early-Stage Type 1 Diabetes – Practical Considerations**
Nikolas Hillenbrand (Frankfurt, Germany)

09:00–09:15 **FINE-ONE: A New Chapter in Kidney Protection for Type 1 Diabetes?**
Andreas L. Birkenfeld (Tübingen, Germany)

09:15–09:30 **Discussion**

09:30–10:30 CET Continuous Monitoring: Current Practice and Emerging Perspectives

Chair: Antonio Ceriello (Milan, Italy)

09:30–09:45 **Continuous Glucose Monitoring in Type 2 Diabetes and Basal Insulin: Implications for Cardiovascular Risk Reduction**
Jochen Seufert (Freiburg, Germany)

09:45–10:00 **Continuous Glucose Monitoring in Clinical Practice: Framework for Practical Implementation**
Julia Mader (Graz, Austria)

10:00–10:15 **Continuous Ketone Monitoring for People with Diabetes: Expert Recommendations on the Application of a New Technology**
Ketan Dhatariya (Norwich, UK)

10:15–10:30 **Discussion**

10:30–10:35 **Break**

10:35–11:05 CET Satellite Symposium: Beyond HbA1c: Preserving cardio-renal function in insulin-treated type 2 diabetes through continuous glucose monitoring

Chair: Roman Vangoitsenhoven (Leuven, Belgium)

supported by
Roche Diagnostics International Ltd

10:35 – 10:38: **Welcome and introduction**
Roman Vangoitsenhoven (Leuven, Belgium)

10:38 – 10:49: **Dysglycaemia as an independent cardio-renal risk factor**
Francesco Cosentino (Stockholm, Sweden)

10:49 – 11:00: **Standardized CGM metrics & red flags for cardio-renal risk reduction**
Roman Vangoitsenhoven (Leuven, Belgium)

11:00 – 11:05: **Panel discussion and Q&A**

11:05–11:15 **Break**

11:15 – 12:30 CET Chronic Kidney Disease Treatment in Motion: From Broad Protection to Precision Care

Chairs: Beatriz Fernández-Fernández (Madrid, Spain), Linong Ji (Beijing, China)

11:15 – 11:30 **Changing the Trajectory of Disease Across the Cardiorenal Continuum: the Role of SGLT2 Inhibition**
Christoph Wanner (Würzburg, Germany)

11:30 – 11:45 **The 2026 ESC Guidelines for the Management of CVD and CKD**
William Herrington (Oxford, UK)

11:45 – 12:00 **A New Therapeutic Option for Non-Diabetic CKD? Lessons from FIND-CKD**
Brandon Neuen (Sydney, NSW, Australia)

12:00 – 12:15 **Emerging Perspectives on Focal Segmental Glomerulosclerosis – A New Era of Targeted Care for Rare Kidney Disease?**
Daniel P. Gale (London, UK)

12:15 – 12:30 **Discussion**

12:30 – 13:15 CET Heart Failure Therapy in Transition: New Evidence, New Guidelines, New Treatment Strategies

Chairs: Naoki Sato (Kawaguchi, Japan), Per-Henrik Groop (Helsinki, Finland)

12:30 – 12:45 **Non-steroidal MRA in HFpEF: From Evidence to Guideline-Based Practice**
John McMurray (Glasgow, UK)

12:45 – 13:00 **Emerging Therapies in Heart Failure: What's Next in Pharmacological Care?**
Katharina Marx-Schütt (Aachen, Germany)

13:00 – 13:15 **Discussion**

13:15 – 13:45 **Break**

13:45 – 14:45 CET Rethinking Cardiovascular Risk Prevention

Chairs: Sigrun Halvorsen (Oslo, Norway), Zaneta Petrulioniene (Vilnius, Lithuania)

13:45 – 14:00 **Hard-to-Control Hypertension: From Treatment Gaps to New Therapeutic Strategies**
Michel Azizi (Paris, France)

14:00 – 14:15 **Lipoprotein(a): From Inherited Risk to Targeted Prevention**
Cristina Gavina (Porto, Portugal)

14:15 – 14:30 **From Risk Factors to Risk Networks: Rethinking Cardiovascular Prevention**
Eberhard Standl (Munich, Germany)

14:30 – 14:45 **Discussion**

14:45 – 16:00 CET Multidisciplinary Strategies in Obesity, Type-2-Diabetes, and Metabolic Liver Disease

Chairs: Yehuda Handelsman (Tarzana, CA, USA), Dror Dicker (Petah Tikva, Israel)

14:45 – 15:00 **Oral Incretins in T2D and Obesity: Evidence, Potential and Perspectives**

Sean Wharton (Ontario, Canada)

15:00 – 15:15 **Triple-Agonists: Redefining the Incretin Landscape in T2D and Obesity**
Virginia Bellido (Sevilla, Spain)

15:15 – 15:30 **The Liver in Metabolic Disease: New Perspectives on GLP-1–Based and Insulin-Sensitizing Approaches**
Wendy Spearman (Cape Town, South Africa)

15:30 – 15:45 **Metabolic Liver Health: Integrating Nutrition, Weight Management, and Complementary Hepatoprotective Strategies**
Jens Marquardt (Lübeck, Germany)

15:45 – 16:00 **Discussion**

16:00 – 16:45 CET Primary Care for Cardiovascular Risk Prevention: Reaching Patients, Connecting Care

Chairs: Pinar Topsever (Istanbul, Turkey), Philippe Richard Domeyer (Athens, Greece)

16:00 – 16:10 **Digital, Local, Integrated: New Models for Cardiorenal Prevention in Primary Care**
Ana María Cebrián Cuenca (Murcia, Spain)

16:10 – 16:20 **Reaching the Unreached: Primary Care and Risk Prevention in Rural Areas**
Banshi Saboo (Ahmedabad, India)

16:20 – 16:30 **Implementing the New ESC Heart Failure Guidelines: Practical Insights from a Heart Failure Nurse**
Ekaterini Lambrinou (Limassol, Cyprus)

16:30 – 16:45 **Discussion**

16:45 – 17:00 **Break**

17:00 – 18:00 CET Industry's Perspective – Panel Discussion

Oliver Schnell (Munich, Germany)

18:00 – 18:45 CET Oral Presentations – Part 2

Chair: Boris Mankovsky (Kyiv, Ukraine)

18:00 – 18:45 **OP 6: Effect of BMI on kidney-cardio outcomes with finerenone in a broad CKD population (INFINITY)**
Peter Rossing (Herlev, Denmark)

OP 7: Impact of finerenone on kidney-cardio outcomes by baseline HbA_{1c} levels in a broad chronic kidney disease population: A pooled analysis (INFINITY)
David Cherney (Toronto, Canada)

OP 8: Sarcopenia as a Metabolic and Inflammatory Phenotype in Patients with MASLD
Kateryna Pivtorak (Vinnytsya, Ukrain)

OP 9: Cardiac Conditions on Death Certificate Line 1(a) Without a Cardiovascular Underlying Cause: U.S. Trends, 1999–2020
Jacob Willes (Salt Lake City, USA)

18:45 – 19:00 CET Abstract Awards and Closing CVOT Summit 2026

Oliver Schnell (Munich, Germany)

Oral Presentations (OP)

OP 1

Beyond cardiovascular-kidney-metabolic syndrome: towards a comprehensive nexus of cardiometabolic multiple long-term conditions

Andrew J. Krentz, Stephan Jacob, Peter Libby, and Robert H. Eckel; On behalf of the Cardiometabolic Health Advancement through Multidisciplinary Partnerships (CHAMP) Alliance

London, UK

Background and Aims: The American Heart Association's cardiovascular-kidney-metabolic (CKM) construct usefully recognises relationships between cardiovascular, renal and metabolic disease. However, it does not capture the full extent of cardiometabolic multimorbidity.

Methods: We propose a broader nexus, encompassing metabolic dysfunction-associated steatotic liver disease (MASLD), neurodegenerative disease, psychiatric and reproductive endocrine disorders, obstructive sleep apnoea, frailty, sarcopenia and obesity-associated cancers. These are not separate comorbidities: they develop and interact across the life course through shared biological, environmental, and social determinants.

Results: Dysfunctional adipose tissue is a central upstream driver. Calorie excess, adipocyte dysfunction, hypoxia and inflammation promote ectopic fat deposition, damaging the liver, muscle, pancreas, kidneys and heart. Insulin resistance connects this adipose dysfunction to hyperglycaemia, dyslipidaemia, endothelial dysfunction and atherosclerosis. Chronic inflammation, microvascular injury, mitochondrial dysfunction and accelerated biological ageing amplify injury across organs. Feedback loops are often bidirectional: renal disease can worsen insulin resistance, MASLD drives atherogenic dyslipidaemia and cardiovascular disease impairs renal perfusion.

Prevention should target upstream causes. Mediterranean or DASH-style diets (dietary approaches to stop hypertension), broader food-based interventions, physical activity and attention to the gut microbiome can improve cardiometabolic and mental health outcomes. Pharmacotherapies reinforce the shared-pathway model: incretin-based medicines improve weight, glycaemia, lipids, liver fat and cardiovascular outcomes; SGLT-2 inhibitors and mineralocorticoid receptor antagonists benefit cardiac and renal disease; anti-inflammatory agents are under investigation.

Conclusion: The expanded nexus supports integrated, mechanism-based care, research spanning multiple outcomes, and health services organised beyond specialty silos. The CKM concept remains clinically valuable but should be viewed as a transitional framework within a wider model of chronic disease prevention and treatment.

OP 2

What Patients With Cardiovascular-Kidney-Metabolic Syndrome Ask: A Clustering of 25 Million Patient Portal Messages

Matthias Christian Lutz, Summer Ngo, Jimin Hwang, Sulaiman Somani, David J. Maron, Alexander T. Sandhu, Fatima Rodriguez, Paul Schmiedmayer

Stanford, USA

Background and Aims: Almost 90 % of US adults meet criteria for cardiovascular-kidney-metabolic (CKM) syndrome. Prevention is central to CKM care and rests largely on what patients understand and manage between visits. What patients need from their care teams during that time has not been characterised at scale. Portal messages record these needs as patients raise them, in their own words. We therefore characterised the needs expressed in messages written by patients across the CKM continuum.

Materials and Methods: In this retrospective study of Stanford Health Care patient portal messages from 2009 to 2026, messages were chained into multi-thread episodes within a 7-day linkage window, checked against an independent subject-line signal. A locally deployed open-weight large language model (Llama-3.3-70B) with a fixed two-step prompt then identified the questions, concerns and requests patients raised, restated each as a standalone question and paired it with the clinician's reply. A rule-based implementation of the 2026 AHA/ACC/ADA/ASN CKM guideline reconstructed each patient's stage annually from diagnoses, vital signs, laboratory values and medications, linking each episode to the stage at the time. Needs were embedded and grouped into fine-grained clusters, each classified for CKM relevance by prespecified criteria.

Results: In 25,040,005 patient messages, 603,327 patients (2,035,092 patient-years; median age 55 years [IQR 40 – 68]; 59.5 % female) expressed 19,591,683 needs. Of these, 12,335,676 (63.0 %) formed 115 clusters, stable across seeds (adjusted Rand index 0.91). The ten largest held 25.7 % of clustered needs: five concerned organising care, led by appointment scheduling (4.6 %), medication and pharmacy (3.0 %) and blood draws (2.9 %); three were common symptoms (respiratory, gastrointestinal, urinary); two concerned vaccination and glucose monitoring. Appointment scheduling was the largest cluster at every stage. Clusters about CKM itself held 11.1 % of clustered needs, rising from 3.6 % at stage 0 to 17.2 % at stage 4, yet remaining a minority at every stage.

Conclusion: Across the CKM continuum, most issues patients raise with their care teams lie beyond CKM itself, consistent with a syndrome whose criteria cover most US adults, while organising care stays prominent at every stage. Patient education and digital support for CKM will therefore need to address the practical work of reaching and navigating care alongside risk factor control.

OP 3

Standard Cost-utility Analyses Underestimate Value: A Multidomain Economic Model of Spotlight-AQ Digital Health in Diabetes Care

Katharine Barnard-Kelly, Eugene E Wright, James Gavin III, Aymeric Mahieu, Ethan Barnard, Caitlin Woombs, Tomas Walker, Paige Johnson, David Shelburne, Kathleen Punger, Ryan Charles Kelly, David Kerr, Daniel Cherñavvsky

Southampton, United Kingdom

Background and Aims: US payers primarily evaluate diabetes interventions

through claims-relevant outcomes, one-year budget impact, avoidable resource utilisation, care delivery efficiency, and operational value. Spotlight-AQ is a brief pre-visit digital biopsychosocial assessment and clinical workflow tool that identifies patient-reported priorities making consultations more personalised, effective, focused and actionable. The objective was to estimate the claims-relevant managed-care value of Spotlight-AQ using observed clinical, operational, and workforce data, and to model US payer scenarios including net monetary benefit of visit-time savings, burnout-related capacity value, and continuous glucose monitoring (CGM) value-realisation.

Materials and Methods: We conducted a secondary multidomain economic modelling analysis using published Spotlight-AQ trial data, a real-world Spotlight-AQ plus CGM pilot, health care professional burnout data, and external Medicaid CGM budget-impact estimates. The primary payer model used one-year, per-patient claims-relevant components: programme cost, visit-time value, conservative workforce-capacity value, and CGM value-realisation thresholds.

Results: The conventional model found Spotlight-AQ to be cost-effective, with incremental cost of approx. $16(USD). Spotlight-AQ reduced consultation duration by 20 % (mean 6.1 minutes in 15 and 30 min visits). Blended visit-time savings shifted net cost to -$4.62; adding conservative workforce value shifted net cost to -$9.62 and net monetary benefit to $409.62. In CGM scenarios, 1 % capture of externally modelled CGM savings increased net monetary benefit to $539.96, $430.28, and $418.79 in MDI, basal-insulin, and non-insulin populations, respectively.

Conclusion: Spotlight-AQ is economically attractive under a US managed-care framework. It is a low-cost workflow intervention with observed visit-efficiency benefits, conservative workforce-capacity value, and a plausible role in helping payers and risk-bearing providers realise more value from routine and CGM-supported care.

OP 4

An AI-Based Knowledge Center for Cross-Disciplinary Retrieval of Cardio-Kidney-Metabolic Clinical Guidelines

Elena Tonchevska, Tadej Battelino, Katharine Barnard-Kelly, Matthias Blüher, Helena N. Boll, Frank C. Brosius, Luca Busetto, Antonio Ceriello, Andreea Ciudin, Francesco Cosentino, James R. Gavin III, Francesco Giorgino, Jennifer Green, Linong Ji, Monika Kellerer, Sue Koob, Nebojsa Lalic, Nikolaus Marx, Barbara McGowan, Prashant Nedungadi, Helena W. Rodbard, Lars Rydén, Banshi Saboo, Wayne Huey-Herng Sheu, Eberhard Standl, Pinar Topsever, Frank Tacke, Christoph Wanner, Per Olav Vandvik, Oliver Schnell

Munich, Germany

Background and Aims: Clinical practice guidelines in the cardio-kidney-metabolic (CKM) field are increasing in number, scope, and complexity. Relevant recommendations are distributed across multiple disciplines and medical scientific societies. This can complicate the identification and consistent application of current guidance in patient care. With the AI-based Knowledge Center for Clinical Guidelines (https://ckm-guidelines.org), we aimed to develop a web-based question-answering system that enables healthcare professionals to retrieve recommendations across selected current CKM guidelines and trace them to the original source documents.

Materials and Methods: The knowledge base comprises selected current clinical guidelines covering cardiovascular disease, diabetes, chronic kidney disease, obesity, and metabolic liver disease. Users submit clinical questions in natural language. The system identifies relevant content within the included guidelines and generates responses with references to the corresponding source documents. Guideline selection and platform development were informed by clinical experts participating in the Taskforce to the Guideline Workshop.

Results: At the time of submission, the knowledge base comprised 68 clinical guidance documents, including clinical practice guidelines, consensus statements, practice guidance, position statements, and scientific statements, across five CKM domains. The platform allows users to query this content through a single interface. Depending on the clinical question, relevant recommendations can be retrieved from one or several source documents, with references to the original publications.

Conclusion: The AI-based Knowledge Center integrates selected multidisciplinary CKM guidelines within a source-referenced query system. This approach may complement established guideline resources and support guideline-informed patient care.

Funding information: Sciarc GmbH

OP 5

An AI-Based Knowledge Center to Support Evidence-Based Management of Hypertension

Moritz Schroll, Elena Tonchevska, Oliver Schnell

Munich, Germany

Background and Aims: Hypertension remains a major clinical challenge. Particularly when blood pressure is difficult to control, management requires consideration of multiple diagnostic and therapeutic approaches. Rapid advances in hypertension treatment, including emerging aldosterone-targeted therapies, have expanded the evidence base across studies and guidelines, making translation into clinical care even more complex. We therefore aimed to develop an AI-based Knowledge Center for Hypertension that provides healthcare professionals with centralised access to current scientific evidence and guidance on hypertension and its clinical management.

Materials and Methods: We assembled a knowledge base from relevant scientific studies, reviews, and current clinical guidelines on hypertension and developed an AI-based searching tool solely based on this verified content. Clinical queries are entered into the platform in natural language and matched with pertinent information from the incorporated sources. Based on the retrieved evidence, the system generates source-referenced answers that link back to the original publications.

Results: The AI-based Knowledge Center for Hypertension includes 118 scientific studies and reviews and 11 clinical guidelines at the time of submission. It covers current and emerging therapeutic approaches, including aldosterone-targeted strategies such as baxdrostat, as well

as evidence relevant to difficult-to-control hypertension. Current clinical guidelines are integrated alongside this evidence to support guideline-informed hypertension management. The platform can retrieve and combine information from these different source types within a single response. Each generated answer provides references and direct links to the original publications. Educational video previews and other complementary resources are integrated separately within the platform.

Conclusion: The AI-based Knowledge Center for Hypertension integrates scientific evidence and clinical guidelines in a single source-referenced question-answering platform. By facilitating access to relevant evidence across multiple sources, it may complement established information resources and support clinical decision-making in hypertension care.

Funding information: AstraZeneca has provided a grant towards this independent programme

OP 6

Effect of BMI on kidney–cardio outcomes with finerenone in a broad CKD population (INFINITY)

Peter Rossing, Carolyn SP Lam, Rajiv Agarwal, David ZI Cherney, Brendon L Neuen, Vlado Perkovic, Katherine Tuttle, Christoph Wanner, Pantelis Sarafidis, Luis M Ruilope, J David Smeijer, Meike Brinker, Yoriko De Sanctis, Hiddo JL Heerspink

Herlev, Denmark

Background and Aims: Adiposity is associated with higher circulating levels of aldosterone. Finerenone, a nonsteroidal mineralocorticoid receptor antagonist, reduces the risk of chronic kidney disease (CKD) progression, cardiovascular (CV) events and mortality in patients with CKD. We sought to evaluate whether the efficacy and safety of finerenone in CKD is modified by excess adiposity as measured by Body-Mass-Index (BMI).

Materials and Methods: This was an individual participant data meta-analysis (INFINITY) of three randomised, placebo-controlled trials in patients with CKD and type 2 diabetes (FIDELIO-DKD and FIGARO) and non-diabetic CKD (FIND-CKD). Patients were on optimised renin–angiotensin system inhibition and randomised 1:1 to finerenone or placebo. BMI categories were: non-elevated (BMI 18.5–<25 kg/m² [for non-Asian race] or 18.5–<23 [Asian race]); overweight (25–<30; 23–27.5); class I (30–<35; 27.5–<32.5); class II (35–<40; 32.5–<37.5); and class III (≥40; ≥37.5). Key outcomes were: composite kidney (kidney failure or sustained ≥57 % estimated glomerular filtration rate decrease from baseline); composite CV (CV death or heart failure hospitalisation); composite kidney-CV (combined kidney and CV composite events) and all-cause death. Safety outcomes included investigator-reported hyperkalaemia events.

Results: Of 14,574 total patients (12,990 [89.1 %] with diabetes), 1406 (9.6 %) had non-elevated baseline BMI, 4863 (33.4 %) were overweight, and 8211 (56.3 %) had class I–III obesity. Finerenone reduced composite kidney and kidney-CV outcome risk vs placebo (overall hazard ratio [HR] 0.76 [95 % confidence interval 0.68–0.86] and 0.77 [0.70–0.85], respectively) with no evidence of interaction with continuous baseline BMI ($p_{interaction}$ = 0.27 and 0.55, respectively). Finerenone also reduced CV composite outcome and all-cause death risk vs placebo across BMI categories (CV composite overall HR 0.80 [0.70–0.91], $p_{interaction}$ = 0.84; all-cause death overall HR 0.88 [0.79–0.99], $p_{interaction}$ = 0.58). The effect of finerenone on serious hyperkalaemia risk was not modified by BMI (overall incidence 1.1 % with finerenone vs 0.3 % with placebo; HR 3.75 [2.31–6.07], $p_{interaction}$ = 0.20).

Conclusion: In a broad population with CKD, the efficacy and safety of finerenone was consistent regardless of BMI.

This analysis was supported by Bayer AG, who funded the FIND-CKD, FIDELIO-DKD and FIGARO-DKD studies

OP 7

Impact of finerenone on kidney–cardio outcomes by baseline HbA_{1c} levels in a broad chronic kidney disease population: A pooled analysis (INFINITY)

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Background and Aims: In INFINITY, a pre-specified pooled analysis of three phase III trials in a broad population with chronic kidney disease (CKD), the non-steroidal mineralocorticoid receptor antagonist finerenone reduced the risk of CKD progression and cardiovascular (CV) outcomes. Here, we investigate the impact of baseline HbA_{1c} levels on the efficacy and safety of finerenone.

Materials and Methods: INFINITY comprised the FIGARO-DKD and FIDELIO-DKD (CKD and type 2 diabetes [T2D]) and FIND-CKD (non-diabetic CKD) trials. Patients were on optimised renin–angiotensin system inhibition and randomised 1:1 to finerenone or placebo. Patients were stratified by their baseline HbA_{1c} levels into four categories: normoglycaemia, pre-diabetes, and adequately or inadequately controlled T2D. Key outcomes were: composite kidney outcome (kidney failure or sustained ≥57 % decrease in eGFR from baseline); composite CV outcome (CV death or hospitalisation for heart failure); composite kidney-CV outcome, and all-cause death. Safety outcomes included hyperkalaemia events.

Results: Of the 14,551 patients with HbA_{1c} measurements at baseline, 958 had normoglycaemia, 625 had prediabetes and 12,968 had T2D (8109 adequately and 4859 inadequately controlled). Finerenone reduced the risk of the composite kidney and kidney-CV outcomes vs placebo (overall hazard ratio [HR] 0.76 and 0.77, respectively); this effect was consistent regardless of baseline HbA_{1c} levels ($p_{interaction}$ = 0.152 and 0.139, respectively). Finerenone also reduced the risk of the CV composite outcome and all-cause death vs placebo across baseline HbA_{1c} categories (overall HR 0.80 and 0.88; $p_{interaction}$ = 0.648 and 0.445, respectively). While the risk of serious hyperkalaemia was generally higher with finerenone vs placebo, overall incidence was low across HbA_{1c} categories (normoglycaemia: 0.6 % vs 0.9 %; prediabetes: 1.7 % vs 0.3 %; adequately controlled

T2D: 1.0 % vs 0.1 %; inadequately controlled T2D: 1.1 % vs 0.4 %).

Conclusion: In this broad CKD population, the efficacy and safety of finerenone was consistent across baseline HbA_{1c} levels.

This analysis was supported by Bayer AG, who funded the FIND-CKD, FIDELIO-DKD and FIGARO-DKD studies.

OP 8

Sarcopenia as a Metabolic and Inflammatory Phenotype in Patients with MASLD

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Background and Aims: Skeletal muscle acts as an endocrine organ and plays an important role in metabolic regulation. Sarcopenia is characterised by a reduction in muscle fiber number and size, impaired neuromuscular function, and myosteatosis. Fat infiltration of skeletal muscle contributes to insulin resistance, metabolic dysfunction, type 2 diabetes, and metabolic dysfunction-associated steatotic liver disease (MASLD). Sarcopenic obesity and progressive loss of muscle mass may further worsen metabolic status and increase cardiovascular risk. The aim of this study was to investigate the relationships between inflammation, insulin resistance, and sarcopenia in patients with MASLD.

Materials and Methods: The study included 256 patients with MASLD with normal weight, overweight, or obesity and 108 individuals without MASLD. Anthropometric parameters and body composition were assessed. Liver enzymes (AST, ALT, GGT), liver steatosis and fibrosis using elastography (FibroScan), and ECG findings were evaluated. The log adiponectin/leptin ratio was calculated. Cardiovascular risk was assessed using the SCORE scale for high-risk countries. Inflammatory markers (TNF-α, IL-1, IL-6, CRP, fibrinogen), endothelin-1, carotid intima-media thickness, atherosclerotic plaques and stenosis, and insulin resistance (HOMA-IR) were determined.

Results: Body weight was 1.3-fold higher in men and 1.5-fold higher in women with MASLD compared with healthy controls. BMI was 1.4-fold higher in both men and women with MASLD. Muscle mass was significantly lower in patients with MASLD than in healthy individuals ($p < 0.05$). Compared with healthy men, men with MASLD had 35.2 % higher body fat, while muscle and bone mass were 29.1 % and 32.0 % lower, respectively. In women with MASLD, body fat was 30.2 % higher, while muscle and bone mass were 17.4 % and 22.7 % lower, respectively. Patients with MASLD and sarcopenia demonstrated higher inflammatory activity and HOMA-IR and lower adiponectin levels than patients with preserved muscle mass. A strong inverse correlation was found between muscle mass and hsCRP levels in both men and women with MASLD ($r = -0.71$, $p < 0.001$).

Conclusion: Sarcopenia and MASLD share common pathogenic mechanisms, including insulin resistance, chronic low-grade inflammation, altered myokine and myostatin secretion, and reduced adiponectin levels. The association between reduced muscle mass and systemic inflammation highlights the importance of assessing sarcopenia in patients with MASLD.

OP 9

Cardiac Conditions on Death Certificate Line 1(a) Without a Cardiovascular Underlying Cause: U.S. Trends, 1999–2020

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Background and Aims: Death certificates may document a cardiac condition on Part I line 1(a) while the coded underlying cause falls outside cardiovascular disease. Characterising how often this occurs may identify differences in death certification patterns across demographic groups.

Materials and Methods: A serial cross-sectional analysis was conducted using the CDC WONDER Multiple Cause of Death database from 1999 through 2020. Cardiac-related deaths were defined as deaths with a cardiac condition documented on Part I line 1(a) of the death certificate. The primary outcome was the annual percentage of these deaths with a non-cardiovascular underlying cause, defined as an underlying cause outside ICD-10 codes I00–I99. Analyses were stratified by age, sex, and race, and grouped binomial logistic regression was used to evaluate temporal and demographic associations.

Results: Among 17,271,111 deaths with a cardiac condition documented on Part I line 1(a), 5,239,508 (30.34 %) had an underlying cause outside ICD-10 codes I00–I99. This proportion increased from 25.43 % in 1999 to 36.22 % in 2020, with increasing odds over time (OR per year, 1.02; 95 % CI, 1.02–1.02; $P < 0.001$). The proportion was highest among decedents younger than 35 years (56.19 %) and lowest among those aged 75 years or older (26.87 %); decedents younger than 35 years had 3.49 times the odds of a non-cardiovascular underlying cause compared with those aged 75 years or older (95 % CI, 3.46–3.53). Proportions were 30.94 % among females and 29.76 % among males. By race, proportions were 29.44 % among White, 34.55 % among Black, 39.59 % among Asian or Pacific Islander, and 38.90 % among American Indian or Alaska Native decedents. Temporal trends differed significantly by sex and race (interaction $P < 0.001$ for both).

Conclusion: Nearly one-third of deaths with a cardiac condition documented on Part I line 1(a) had a coded underlying cause outside cardiovascular disease, and this proportion increased over the study period. Differences were most pronounced by age, with smaller but significant variation by sex and race. These findings demonstrate substantial discordance between cardiac conditions documented on line 1(a) and the coded underlying cause and support further evaluation of death-certificate sequencing and coding practices when interpreting cardiovascular mortality trends.

Metabolic Risk and Treatment in Obesity and Liver Disease

P 01

Metabolic dysfunction-associated steatotic liver disease (MASLD) in daily clinical practice: who/how to screen and treat

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Metabolic dysfunction-associated steatotic liver disease (MASLD), and particularly its advanced form of steatohepatitis (MASH), are associated with increased cardiovascular and liver morbidity/mortality, as well as all-cause mortality. The prevalence of MASLD is continuously increasing worldwide (together with the global pandemic of "diabesity"). Therefore, apart from its prevention and early diagnosis, MASLD treatment is of paramount importance and has emerged as a major health issue with several drug therapies being developed.

Resmetirom and semaglutide are currently approved for use in adults with MASH and moderate-to-advanced fibrosis (stage F2 to F3) in both the USA and Europe. Several dual glucagon-like peptide-1 (GLP-1)/glucose-dependent insulinotropic polypeptide (GIP) (tirzepatide), GLP-1/glucagon (survodutide, mazdutide, pemvidutide, efinopegdutide) and GLP-1/amylin (cagrilintide-semaglutide, amycretin) receptor co-agonists and triple (GLP-1/GIP/glucagon) co-agonists (retatrutide, efocipegtrutide), as well as a GLP-1 receptor agonist/GIP receptor antagonist (maridebart cafraglutide) are being developed, primarily for obesity and type 2 diabetes. Ongoing phase 3 trials with these multi-agonists are assessing their effects on MASLD endpoints.

Drug combination therapies may be needed for MASH resolution in different patient groups. Apart from the injectable investigational GLP-1 based therapies, oral incretin mimetics may be valuable in daily practice, either as mono-agonists (e.g., oral semaglutide, orforglipron) or co-agonists (e.g., amycretin). Abnormal peri-/intra-organ fat accumulation, as well as the cardiovascular-kidney-liver-metabolic (CKLM) syndrome represent important clinical aspects to consider for the clinical use of incretin mono/multi-agonists. A therapeutic holistic approach (involving multidisciplinary care and individualisation of treatment strategy) remains the key player in our fight against dysfunctional adiposity and its related CKLM disorders.

P 02

Comparative analysis of cardio-metabolic parameters in women of fertile age depending on body weight

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Background and Aims: The complex of lipid and carbohydrate metabolism disorders, increased blood pressure (BP) and obesity, combined under the term "metabolic syndrome" (MS), is one of the predictors of cardiovascular events and type 2 diabetes. This problem is important in women of fertile age, as it can affect reproductive function. The progressive increase in the prevalence of obesity among young women necessitates the early diagnosis and correction of metabolic disorders. Components of MS can be present in the absence of obesity, potentially leading to disorders of hormonal health and fertility, which emphasises the importance of a comprehensive assessment of the metabolic profile.

Materials and Methods: A descriptive statistical analysis of the anthropometric and metabolic profiles of 67 women. Group I – 18–30 years old, with obesity (body mass index (BMI) > 30 kg/m²) – 20 women, without obesity (BMI ≤ 30 kg/m²) – 14 women. Group II – 30–45 years old, with obesity – 22 women, without obesity – 11 women.

Results: In women with obesity in Group I, the mean values were as follows: waist circumference (WC) was 103.75 cm, percentage of adipose tissue (AT) 47.69 %, triglycerides (TG) 1.43 mmol/l, high-density lipoproteins (HDL) 1.4 mmol/l, low-density lipoproteins (LDL) 2.78 mmol/l, total cholesterol 4.95 mmol/l, fasting blood sugar (FBS) 5.45 mmol/l, postprandial blood sugar (PPBS) 5.92 mmol/l, BP 127.8/81.45 mmHg. In women without obesity in Group I, the mean values were as follows: WC was 75.64 cm, AT 31.4 %, TG 0.87 mmol/l, HDL 1.59 mmol/l, LDL 3.4 mmol/l, total cholesterol 5.19 mmol/l, FBS 4.95 mmol/l, PPBS 5.67 mmol/l, BP 117.29/72.71 mmHg. In the second group with obesity, the average value of WC was 98.45 cm, AT 44.39 %, TG 1.72 mmol/l, HDL 1.45 mmol/l, LDL 3.9 mmol/l, total cholesterol 5.82 mmol/l, FBS 5.29 mmol/l, PPBS 6.14 mmol/l, BP 129.09/80 mmHg. In women without obesity in Group II, the mean values were as follows: WC was 71.18 cm, AT 30.23 %, TG 2.11 mmol/l, HDL 1.55 mmol/l, LDL 3.39 mmol/l, total cholesterol 6.0 mmol/l, FBS 5.14 mmol/l, PPBS 5.67 mmol/l, BP 122.27/75.45 mmHg.

Conclusion: Obesity is associated with higher levels of cholesterol and its fractions, glycaemia, and blood pressure. In women without obesity, according to BMI, an increase in lipid and carbohydrate profiles and a high percentage of adipose tissue were also observed. Waist circumference and adipose tissue content are more diagnostically significant criteria for visceral obesity and the associated metabolic syndrome than body mass index.

P 03

Short-Term Effects of Dapagliflozin on Glycaemic Control, Body Weight and FIB-4-Estimated Fibrosis Risk in Patients with and without Diabetes Mellitus: A Sex-Stratified Analysis in the Context of MASLD

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Background and Aims: Metabolic dysfunction-associated steatotic liver disease (MASLD) is closely associated with diabetes, obesity and cardiovascular risk. SGLT-2 inhibitors may provide metabolic benefits beyond glycaemic control, including weight reduction and improvement of non-invasive markers of hepatic fibrosis. The objective was to evaluate the 3-month effects of dapagliflozin on glycaemic control, body weight and

FIB-4 in patients with and without diabetes mellitus, and to assess sex-related differences and associations with left ventricular ejection fraction (LVEF).

Materials and Methods: A retrospective observational study included 31 patients: 16 with diabetes mellitus and 15 without diabetes. All patients received dapagliflozin and were evaluated at baseline and after three months. Fasting glucose, postprandial glucose, HbA_{1c}, BMI, body-weight reduction, FIB-4 and LVEF were assessed. Results were analysed according to diabetes status and sex.

Results: In patients with diabetes, fasting glucose decreased from 9.41 to 6.16 mmol/l and postprandial glucose from 11.19 to 7.06 mmol/l. HbA_{1c} decreased from 7.99 % to 7.23 %. In patients without diabetes, changes in glucose and HbA_{1c} were minimal. FIB-4 decreased from 2.24 to 1.74 in patients with diabetes and from 1.72 to 1.29 in those without diabetes. Mean weight loss was greater in patients with diabetes (2.31 kg) than in those without diabetes (1.19 kg). Baseline BMI, HbA_{1c} and FIB-4 were positively correlated with subsequent weight reduction ($r = 0.70$, $r = 0.68$ and $r = 0.67$, respectively; all $p < 0.001$). Baseline FIB-4 was also correlated with HbA_{1c} ($r = 0.46$, $p = 0.009$). Mean weight loss was 2.03 kg in men and 1.63 kg in women. No significant association was observed between LVEF and changes in FIB-4, HbA_{1c} or body weight.

Conclusion: Three months of dapagliflozin treatment was associated with improved glycaemic control, weight reduction and decreased FIB-4-estimated fibrosis risk, particularly in patients with diabetes. Higher baseline BMI, HbA_{1c} and FIB-4 were associated with greater weight loss. These findings suggest potential metabolic and hepatic benefits of dapagliflozin in patients with MASLD and cardiometabolic risk. Larger prospective studies are warranted.

P 04

Real-world treatment persistence among patients initiating semaglutide or tirzepatide in Sweden

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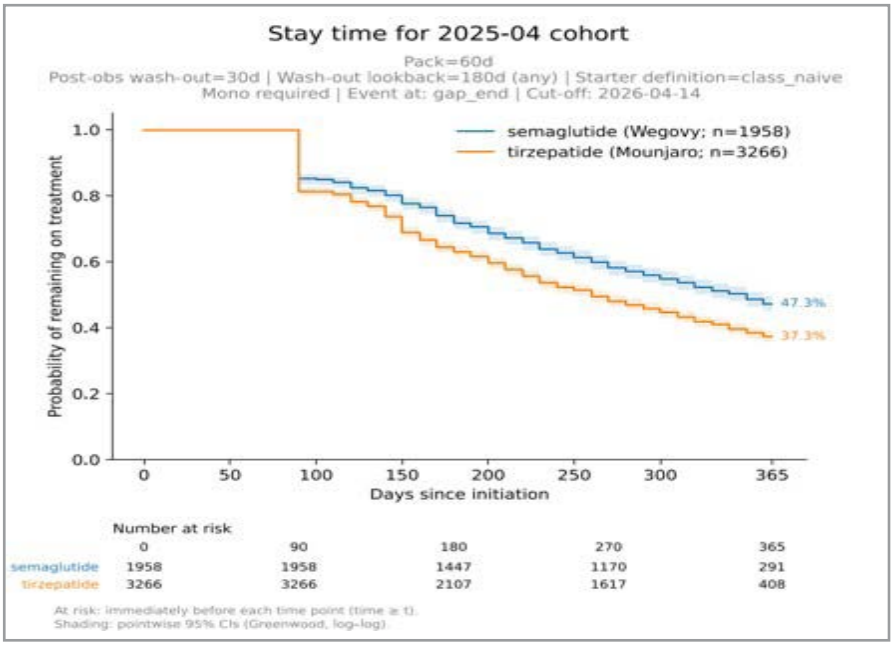


Fig. 1: Treatment persistence after initiation of semaglutide or tirzepatide.

Background and Aims: Long-term treatment persistence is important in the management of obesity as a chronic disease, but comparative real-world evidence remains limited. We aimed to describe and compare treatment persistence among patients initiating semaglutide or tirzepatide in Sweden.

Materials and Methods: We conducted a nationwide, retrospective register-based analysis using dispensing data from the Swedish Prescribed Drug Register, with follow-up available through April 2026. Data available to the study team were anonymized. Figure 1 is based on patients whose first recorded treatment in the captured product class was semaglutide or tirzepatide during April 2025. Patients were also required to have no dispensing of any captured product in the 180 days before initiation. Patients dispensing more than one captured product during follow-up were excluded. Patients were followed from first dispensing until discontinuation or administrative censoring. One dispensing was assumed to cover 60 days of treatment. Discontinuation was defined as a gap of at least 30 days after expected coverage ended, with the event assigned to the end of that gap. Treatment persistence was estimated using Kaplan-Meier methods. A Cox proportional hazards model was fitted as an unadjusted comparison of discontinuation hazard for semaglutide versus tirzepatide.

Results: The April 2025 cohort included 1,958 semaglutide initiators and 3,266 tirzepatide initiators. Estimated treatment persistence was higher for semaglutide than for tirzepatide throughout follow-up. At 180 days, 71.8% of semaglutide initiators remained on treatment compared with 63.1% of tirzepatide initiators. At 365 days, the corresponding estimates were 47.3% and 37.3%. Median time to treatment discontinuation was 350 days for semaglutide and 260 days for tirzepatide. Restricted mean survival time (RMST) through day 349 was 265 days for semaglutide and 242 days for tirzepatide.

The unadjusted Cox model estimated a hazard ratio for treatment discontinuation of 0.74 for semaglutide versus tirzepatide (95% CI 0.69-0.80; $p<0.001$).

Most semaglutide initiators started on 0.25 mg (79.0%), and the most common latest recorded dose was 2.4 mg (43.7%). Most tirzepatide initiators started on 2.5 mg (64.9%), with 5 mg also common (31.5%); the most common latest recorded dose was 5 mg (36.1%), followed by 15 mg (23.1%) and 10 mg (20.2%).

Conclusion: In Swedish real-world dispensing data, patients initiating semaglutide in April 2025 had higher estimated treatment persistence than patients initiating tirzepatide. As this was

an unadjusted, descriptive comparison without information on patient characteristics, these findings should not be interpreted as evidence of a causal difference in treatment persistence between the two medications.

Funding information: This study and the underlying analysis were funded by Novo Nordisk Scandinavia AB. Moa Lugner has served as a consultant to Novo Nordisk Scandinavia AB on activities unrelated to the present work.

P 05

Effects of Semaglutide on Metabolic, Inflammatory and Endothelial Biomarkers in Patients with MASH and F2–F3 Fibrosis

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Background and Aims: To evaluate the effects of semaglutide therapy on insulin resistance, adipokine profile, inflammatory mediators, endothelial dysfunction, and metabolic parameters in patients with metabolic dysfunction-associated steatohepatitis (MASH) and stage F2–F3 liver fibrosis.

Materials and Methods: Twelve patients with biopsy- or clinically confirmed MASH and F2–F3 liver fibrosis were included in the prospective observational study. Patients with F4 fibrosis/cirrhosis were excluded. Baseline assessment included anthropometric parameters, metabolic risk factors, glucose and lipid profile, AST, ALT, GGT, insulin, HOMA-IR, adiponectin, leptin, hsCRP, endothelin-1 (ET-1), and von Willebrand factor (vWF). The adiponectin-to-leptin ratio was calculated as log A/L. Non-invasive assessment of liver fibrosis was performed using transient elastography (FibroScan). After exclusion of contraindications, patients received semaglutide with dose escalation from 0.25 mg/week to 0.5, 1.0, 1.7, and 2.4 mg/week, according to tolerability. The therapy was combined with standard treatment of concomitant type 2 diabetes, arterial hypertension, and dyslipidaemia. Patients were reassessed after 72 weeks of treatment.

Results: At baseline, patients with MASH demonstrated a metabolic-inflammatory phenotype characterised by insulin resistance, adipokine imbalance, and markers of endothelial dysfunction. HOMA-IR showed a strong positive correlation with leptin ($r = 0.80$; $p = 0.00166$) and inverse correlations with adiponectin ($r = -0.66$; $p = 0.0033$) and the log A/L index ($r = -0.71$; $p < 0.001$). The semaglutide treatment period was associated with changes in body weight, metabolic parameters, insulin resistance, adipokine profile, inflammatory markers, and markers of endothelial dysfunction. Changes in ET-1, vWF, hsCRP, leptin, adiponectin, and HOMA-IR were evaluated as potential indicators of improvement in the systemic metabolic-inflammatory and vascular phenotype of MASH.

Conclusion: Patients with MASH F2–F3 demonstrate a metabolic-inflammatory phenotype characterised by insulin resistance, leptin/adiponectin imbalance, inflammation, and endothelial dysfunction. Semaglutide combined with lifestyle modification and cardiometabolic management may improve the metabolic and vascular components of MASH. Assessment of adipokines, insulin resistance, inflammatory and endothelial biomarkers may help characterise treatment response and identify patients who may benefit from GLP-1 receptor agonist therapy.

P 06

The Effect of Bariatric Surgery on Lipid Profile and Liver Function in Patients with Type 2 Diabetes

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Background and Aims: In patients with morbid obesity, with or without diabetes, bariatric surgery is more effective in improving metabolic disturbances than conventional medical therapy. Dyslipidaemia and nonalcoholic fatty liver disease are major drivers of cardiovascular risk in patients with type 2 diabetes, yet their long-term remission after bariatric surgery remains insufficiently studied. The objective of this study was to evaluate changes in lipid profile, liver function, and associated cardiovascular risk following bariatric surgery in patients with type 2 diabetes.

Materials and Methods: We examined 91 patients with type 2 diabetes and morbid obesity who underwent sleeve gastrectomy. Patients ranged in age from 23 to 67 years (mean 46.67 ± 8.60; 50 women, 41 men). Clinical and laboratory parameters were evaluated preoperatively and 2–4 years postoperatively.

Results: Postoperatively, a significant reduction in weight was observed (from 146.8 kg to 97.9 kg). The frequency of fatty liver disease decreased markedly (from 96.7 % [n = 88] to 19.78 % [n = 18], $p < 0.0001$), as did dyslipidaemia (from 72.53 % [n = 66] to 40.66 % [n = 37], $p < 0.0001$). Elevated liver function tests improved significantly: elevated ALT dropped from 27.47 % to 1.10 % ($p < 0.0001$), while elevated AST and GGT were no longer detected post-surgery. The prevalence of hypertension decreased from 82.42 % to 29.67 %. Regression analysis indicated that fatty liver remission was positively associated with preoperative weight loss of 5–10 %, increased physical activity, and diabetes remission, whereas it was negatively impacted by alcohol consumption, a sedentary lifestyle, thyroid dysfunction, a high-fat diet, and preoperative weight loss < 5 %.

Conclusion: In patients with type 2 diabetes and morbid obesity, bariatric surgery provides comprehensive metabolic normalisation beyond weight loss alone, substantially reducing dyslipidaemia, hepatic steatosis, and hypertension to lower overall cardiovascular risk.

P 07

Effects of Supplemental Dietary Lutein and Zeaxanthin on Lipid Peroxidations, Gene Expressions and Cardiometabolic Risk Factors on High-Fat-Diet Fed Rats

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Background and Aims: Cardiometabolic diseases are defined as a group of diseases presenting with insulin resistance and non-alcoholic fatty liver disease (NAFLD). Chronic inflammation leads to an increase

in reactive oxygen species (ROS), forming advanced glycation end products (AGEs). This study aimed to determine whether the daily dietary addition of lutein and zeaxanthin carotenoids reduces lipid peroxidation and lipid biomarkers and modulates hormone stimulation levels in obesity and hyperlipidemic model rats fed a high-fat diet.

Materials and Methods: The study was conducted on 60 male Wistar rats aged 7–8 weeks at GAÜNDAM. The study duration was a total of 14 weeks: obesity and hyperlipidaemic models were established using standard and high-fat diet feeding for 8 weeks, followed by 6 weeks of feeding with the addition of carotenoids to the diets, and the study was concluded at the end of 14 weeks. The standard rat diet was prepared to contain 10 % vegetable oil, the high-fat diet contained 45 % butter. The 60 animals were divided into six groups: CON (control): standard feed + 2.5 ml distilled water; CONL (control + lutein): standard feed + 1 mg/kg/day lutein extract; CONZ (control + zeaxanthin): standard feed + 50 mg/kg/day zeaxanthin extract; HFD (high-fat diet): high-fat feed + 2.5 ml distilled water; HFDL (high-fat diet + lutein): high-fat feed + 1 mg/kg/day lutein extract; and HFDZ (high-fat diet + zeaxanthin): high-fat feed + 50 mg/kg/day zeaxanthin extract.

The rats' body and liver organ weights, BMI and Lee index as anthropometric measurements, serum biochemical parameters (total cholesterol, triglycerides, HDL-C, LDL-C, TNF-α, IL-6, malondialdehyde [MDA], trimethylamine N-oxide [TMAO], and AGE), hormones (insulin, ghrelin, leptin), and liver histopathological imaging were performed.

Results: A statistically significant difference in the plasma AGE values was detected among the groups ($p = 0.011$). A statistically significant difference was detected in the plasma values of the ghrelin hormone of the rats between the groups ($p = 0.029$), and the plasma levels of the HFD group (1912.29 ± 164.22 ng/l) were found to be statistically significantly lower compared to the CONL group (3133.66 ± 222.36 ng/l). A statistically significant difference was detected among the histopathological scores of the rats, and the greatest level of improvement in liver damage was observed with lutein supplementation in the HFDL group ($p < 0.05$).

Conclusion: It was determined that lutein and zeaxanthin supplementation significantly reduced TNF-α levels, restored ghrelin hormone levels close to the control group, and especially prevented the elevation of serum AGE levels. In addition, lutein and zeaxanthin supplements were effective in improving liver damage caused by a high-fat diet, and histopathological scores showed a decrease compared to the high-fat diet group.

Cardiorenal and Vascular Comorbidity in Clinical Practice

P 08

Association of SGLT-2 Inhibitor Use with Vascular Outcomes in Patients with Diabetes Mellitus Undergoing Peripheral Revascularisation

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Background and Aims: SGLT2 inhibitors (SGLT2i) provide cardiovascular and renal benefits in type 2 diabetes; however, CANVAS reported an almost two-fold increase in lower-limb amputations with canagliflozin. We assessed SGLT2i use and major adverse cardiovascular events (MACE) and major adverse limb events (MALE) in patients with peripheral artery disease (PAD) undergoing lower-limb revascularisation (NCT07157475).

Materials and Methods: This observational study included patients with diabetes from three Polish vascular centres. Medical records were retrospectively reviewed; patients ≥ 1 year after revascularisation were contacted by telephone regarding MACE, MALE and SGLT2i use. SGLT2i users were compared with non-users. Normality was assessed by Shapiro-Wilk test; continuous variables by Student's t-test or Mann-Whitney U test and categorical variables by chi-square test.

Results: Before revascularisation, flozin-negative patients had lower body weight (76.5 ± 15.0 vs. 83.1 ± 13.0 kg, $p = 0.002$), higher BMI (25.7 ± 5.0 vs. 23.7 ± 3.7 kg/m², $p = 0.003$) and more former smokers (41.7 % vs. 15.2 %, $p < 0.001$). Flozin-positive patients were more often male (82.7 % vs. 67.3 %, $p = 0.02$) and had more myocardial infarctions (MI; 48.2 % vs. 21.2 %, $p < 0.001$), previous coronary revascularisation (51.9 % vs. 25.7 %, $p < 0.001$), heart failure (46.8 % vs. 24.1 %, $p = 0.001$), hypertension (97.5 % vs. 89.3 %, $p = 0.03$), hyperlipidaemia (72.8 % vs. 46.4 %, $p < 0.001$) and higher creatinine (1.10 vs. 0.97 mg/dl, $p = 0.009$). Endovascular procedures were more frequent in flozin-positive patients (76.5 % vs. 52.2 %), while open surgery was more frequent in flozin-negative patients (42.5 % vs. 21.0 %); hybrid procedures: 5.3 % vs. 2.5 % ($p = 0.003$). During follow-up, MALE was more frequent in flozin-positive patients (37.04 % vs. 15.04 %, $p = 0.00042$). At 3 months, patency deterioration (11.11 % vs. 1.77 %, $p = 0.0055$) and repeat intervention (9.88 % vs. 1.77 %, $p = 0.012$) were more frequent in flozin-positive patients, whereas MI occurred in 0 % vs. 5.31 % ($p = 0.035$). Foot amputation (25.93 % vs. 13.27 %, $p = 0.025$) and heart failure hospitalization (8.64 % vs. 0.88 %, $p = 0.007$) were more frequent in flozin-positive patients; wound infection (7.96 % vs. 1.23 %, $p = 0.037$) was more frequent in flozin-negative patients. Overall MACE, mortality, stroke and later outcomes did not differ significantly.

Conclusion: SGLT2i use was associated with higher MALE incidence and selected limb-related adverse outcomes, warranting confirmation in prospective studies.

P 09

Sex-Specific Association of Diabetic Peripheral Neuropathy with Heart Failure with Preserved Ejection Fraction in Type 2 Diabetes Mellitus

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Background and Aims: Heart failure (HF) and diabetic peripheral neuropathy (DPN) are common

complications of type 2 diabetes mellitus (T2DM). However, the relationship between these conditions and the potential influence of sex and age remain insufficiently studied. The aim was to evaluate the association between diabetic peripheral neuropathy and heart failure with preserved ejection fraction (HFpEF) in patients with T2DM.

Materials and Methods: A total of 340 patients with T2DM were included (median age 63.5 years; 221 men and 119 women). Median HbA_{1c} was 7.2 % [6.4 – 8.6], and median diabetes duration was 6.0 [2.0 – 12.0] years. HFpEF (EF > 50 %) was diagnosed based on clinical and echocardiographic data. DPN was diagnosed according to clinical symptoms and standardised assessments of tactile, pain, and temperature sensitivity, as well as Sudoscan and electroneuromyography. Statistical analysis included Fisher's exact test, odds ratios (OR) with 95 % confidence intervals, multiple logistic regression with interaction analysis, and the Mann-Whitney U test with Bonferroni correction.

Results: DPN was detected in 208 patients (61.2 %), while 132 patients (38.8 %) had no neuropathy. HF was present in 180 of 208 patients with DPN (86.5 %) and in 100 of 132 patients without DPN (75.8 %). A significant association between DPN and HF was observed in the overall cohort (OR = 2.06; 95 % CI: 1.17 – 3.61; p = 0.013). Patients with DPN had longer diabetes duration (8.0 vs 3.5 years; p < 0.001) and higher HbA_{1c} levels (7.55 % vs 6.9 %; p = 0.003). Sex-stratified analysis showed a significant association in men (OR = 3.05; 95 % CI: 1.55 – 6.00; p = 0.001) but not in women. In multivariable regression, female sex was an independent protective factor for DPN (aOR = 0.44; p < 0.001), whereas older age increased risk (aOR = 1.03 per year; p = 0.016).

Conclusion: DPN is significantly associated with HFpEF in patients with T2DM. This relationship demonstrates a pronounced sex-specific pattern, being present in men but not in women.

P 10

Renal Function in Type 2 Diabetes Mellitus: Comparison between INOCA and Obstructive Coronary Artery Disease

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Background and Aims: Type 2 diabetes mellitus (T2DM) is associated with a high risk of both ischemic heart disease and chronic kidney disease (CKD). In addition to obstructive coronary artery disease (CAD), increasing attention has been paid to ischaemia with non-obstructive coronary arteries (INOCA), particularly microvascular angina, which is considered a manifestation of systemic microvascular dysfunction. However, data on renal function according to different forms of ischemic heart disease in patients with T2DM remain limited. The aim was to compare renal function and markers of chronic kidney disease between patients with T2DM and INOCA and those with T2DM and obstructive CAD.

Materials and Methods: The study population consisted of 80 patients with type 2 diabetes mellitus, who were categorised into INOCA (n = 30) and obstructive coronary artery disease (n = 50) groups. Age, duration of T2DM, fasting glucose, glycated hemoglobin (HbA_{1c}), plasma creatinine, estimated glomerular filtration rate (eGFR, CKD-EPI), urine albumin-to-creatinine ratio (ACR), and plasma potassium levels were assessed. Data are presented as mean ± standard deviation. Between-group comparisons were performed using Student's t-test, and correlations were analysed using Pearson's method.

Results: Patients with obstructive CAD were older than those with INOCA (66.8 ± 8.3 vs 58.9 ± 11.3 years). Glycaemic control parameters did not differ significantly between groups (HbA_{1c}: 6.9 ± 1.4 % vs 6.7 ± 1.7 %). Renal impairment was more pronounced in the obstructive CAD group, with significantly lower eGFR (51.2 ± 15.8 vs 74.3 ± 16.8 ml/min/1.73 m^2; p < 0.001) and higher ACR (82.3 ± 115.8 vs 32.7 ± 34.3 mg/g; p = 0.028) compared with the INOCA group. An inverse association between eGFR and ACR was observed in both the INOCA and obstructive CAD groups, with a stronger correlation in patients with obstructive disease, indicating a consistent link between renal dysfunction and albuminuria across subgroups.

Conclusion: In patients with type 2 diabetes mellitus, obstructive coronary artery disease is associated with more severe renal dysfunction compared with INOCA, despite similar glycaemic control. The inverse relationship between eGFR and albuminuria highlights shared cardiorenal mechanisms underlying target organ damage in T2DM.

P 11

Peripartum Cardiomyopathy and Renal Dysfunction in Niger: Insights from a Resource-Limited Sub-Saharan African Setting

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Background and Aims: Peripartum cardiomyopathy (PPCM) is an idiopathic form of heart failure occurring toward the end of pregnancy or in the months following delivery. Renal dysfunction is a potentially under-recognised complication that may worsen prognosis through cardiorenal interactions. This study investigated the epidemiological, clinical, laboratory, and outcome characteristics of renal impairment among women hospitalised with PPCM at the National Hospital of Zinder, Niger.

Materials and Methods: We conducted a hospital-based cross-sectional study using retrospective data collected from January 2022 to December 2025. The study included 72 women hospitalised in the cardiology department with PPCM and confirmed renal impairment. Demographic, obstetric, clinical, echocardiographic, laboratory, and outcome data were extracted from medical records. Renal impairment severity was classified as moderate or severe. Bivariable analyses were performed to identify factors associated with severe renal impairment.

Results: Renal impairment occurred in 36 % of patients hospitalised with PPCM. The mean age was 27.19 years. Most patients lived in rural areas (52.8 %), had a low socioeconomic status (74.0 %), and were multiparous (72.2 %). Symptoms occurred during the postpartum period in 86.0 % of cases, while global heart failure was the predominant clinical presentation (83.0 %). Mean left ventricular ejection fraction was 31.5 %, mean serum creatinine was 191.30 µmol/l, and mean estimated glomerular filtration rate was 35 ml/min. Renal impairment was moderate in 60.0 % of cases and severe in 40.0 %. Severe renal impairment was significantly associated with hypertensive disorders of pregnancy (odds ratio [OR], 5.13; p = 0.003), left ventricular ejection fraction below 35 % (OR, 4.64; p = 0.037), and the occurrence of in-hospital complications (OR, 3.27; p = 0.018).

Conclusion: Renal impairment affected more than one-third of women hospitalised with PPCM and was associated with markers of severe cardiac dysfunction, hypertensive pregnancy disorders, and adverse clinical complications. Routine assessment of kidney function, early identification of high-risk women, and coordinated cardiology-nephrology-obstetric care may improve maternal outcomes in resource-limited settings.

P 12

Associations of Obesity and Metabolic Syndrome with Left Ventricular Hypertrophy in Maintenance Haemodialysis: A Cross-Sectional Study

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Background and Aims: Higher body mass index (BMI) has been associated with improved survival in haemodialysis, although it may not reflect adverse cardiac structural changes. This study examined the associations of obesity and metabolic syndrome with left ventricular hypertrophy (LVH) in patients receiving maintenance haemodialysis.

Materials and Methods: This single-centre cross-sectional study was conducted at the Departments of Medical Biochemistry and Nephrology and Kidney Transplantation, Ibn Rochd University Hospital, Annaba, Algeria. Adults undergoing maintenance haemodialysis with available LVH data were included. Obesity was defined as BMI ≥30 kg/m², and metabolic syndrome as the presence of at least three of five recorded metabolic components. Associations with LVH were assessed using logistic regression adjusted for age, sex, and smoking.

Results: Among 101 patients, 51 (50.5 %) had LVH, 14 (13.9 %) were obese, and 61 (60.4 %) had metabolic syndrome. LVH prevalence was 63.6 % in underweight, 47.4 % in normal-weight, 36.8 % in overweight, and 71.4 % in obese patients (p = 0.183), suggesting a descriptive U-shaped pattern. Obesity was associated with higher odds of LVH, although the estimate did not reach statistical significance (adjusted odds ratio [aOR] 2.94, 95 % confidence interval [CI] 0.83 – 10.43; p = 0.095). Metabolic syndrome was not independently associated with LVH (aOR 1.42, 95 % CI 0.57 – 3.53; p = 0.453). Patients with both obesity and metabolic syndrome had higher LVH prevalence than metabolically healthier non-obese patients (71.4 % versus 45.0 %; aOR 3.17, 95 % CI 0.78 – 12.89).

Conclusion: LVH burden was highest among obese haemodialysis patients, particularly those with metabolic syndrome, but associations were imprecise and not statistically significant.

Cardiac Remodelling and Clinical Management of Heart Disease

P 13

Clinical-metabolic and structural-functional portrait of a patient with diabetes mellitus and heart failure with preserved left ventricular ejection fraction.

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Background and Aims: Heart failure (HF) is a medical and social challenge not only for the Ukrainian medical community, but also for the world. Currently, there is insufficient data on the epidemiology and prognostic significance of HF and its subtypes. In Ukraine HF is the cause of all repeated hospitalisations in cardiology hospitals. Diabetes mellitus (DM) is a risk factor for cardiovascular events. HF and DM are independent risk factors that lead to a high mortality rate from cardiac events against the background of hyperglycaemia.

Materials and Methods: 47 patients with type 2 diabetes aged 40 to 60 years were examined at an endocrinology centre. The examination included anthropometric measurements, determination of glycosylated haemoglobin (HbA_{1c}), eGFR (CKD-EPI), NT-proBNP, galectin-3 and echocardiography.

Results: Among 47 patients with type 2 diabetes, 25 patients (53.2 %) had confirmed heart failure with preserved ejection fraction (HFpEF) and 22 (46.8 %) did not have HF. The mean age of the overall group was 51.45 ± 4.78 years; in the HFpEF group – 52.32 ± 4.55 years, in the group without HF – 50.45 ± 4.94 years. In patients with HFpEF, the median duration of DM was 10.0 [5.0; 13.0] years. The mean body weight was 102.10 ± 16.62 kg, BMI was 35.23 ± 6.09 kg/m², and waist circumference was 116.36 ± 13.42 cm. The waist-to-height ratio (WHtR) was 0.687 ± 0.081 on average. The mean HbA_{1c} level in the HFpEF group was 9.33 ± 1.94 %, the median was 8.9 [7.8; 10.5] %. Among patients with HFpEF, diabetic retinopathy was recorded in 14 patients (56.0 %), neuropathy in 24 (96.0 %), chronic kidney disease in 7 (28.0 %) and diabetic nephropathy in 7 (28.0 %). The mean eGFR was 84.88 ± 20.07 ml/min/1.73 m². In the HFpEF group, cardiovascular complaints were recorded in 24 patients (96.0 %); dyspnoea in 16 (64.0 %); paroxysmal nocturnal dyspnoea in 12.0 %; reduced exercise tolerance in 16 (64.0 %); palpitations in 16 (64.0 %); ankle oedema in 11 (44.0 %); fatigue/tiredness in 24 (96.0 %). The median NT-proBNP in patients with HFpEF was 250 [180; 300] pg/ml. The median galectin-3 was 12.1 [10.0; 16.2] ng/ml. The mean LVEF in the HFpEF group was 63.96 ± 6.04 %, LV mass index was 122.76 ± 28.13 g/m², LA dilatation was 22.41 ± 7.16 ml/m², E/e was 6.94 ± 1.95. The mean SCORE2-Diabetes in patients with HFpEF was 24.65 ± 9.95 %, median was 23.1 [19.2; 25.1] %.

Conclusion: The HFpEF group has a multicomponent profile: central obesity, insufficient glycaemic control, high prevalence of microvascular complications, and changes in HF biomarkers. It is this heterogeneity that creates the basis for further studies of HF in patients with diabetes.

P 14

Subclinical markers of myocardial remodeling and neurohumoral activation in patients with type 2 diabetes mellitus without heart failure

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Background and Aims: Diabetic cardiomyopathy is considered an important risk factor for heart failure. Early stages of diabetic cardiomyopathy develop asymptomatically making standard diagnostic methods insufficient for detecting subclinical myocardial damage. Assessment of extracellular volume (ECV) and global longitudinal strain (GLS) allows for the timely identification of early structural and functional remodeling. The aim was to evaluate parameters of myocardial structural and functional status such as ECV and left ventricular GLS in patients with type 2 diabetes mellitus (T2DM) without concomitant cardiovascular diseases and with preserved left ventricular ejection fraction (LVEF), and to investigate their association with N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels.

Materials and Methods: The study enrolled 48 patients with T2DM; the control group consisted of 39 virtually healthy individuals. Mean age of patients with T2DM was 55.7 ± 8.9 years with an increased BMI (mean BMI 34.1 ± 8.8 kg/m²). In the control group, the mean age was 58.6 ± 5.2 years and the mean BMI was 25.0 ± 2.2 kg/m². The ECV index was evaluated using cardiac magnetic resonance imaging (3T scanner). Segmental and global myocardial contractility were assessed by speckle-tracking echocardiography to determine GLS. Additionally, plasma levels of NT-proBNP were measured in all patients.

Results: LVEF did not differ statistically between the groups remaining within the normal range. The mean NT-proBNP value was significantly higher in patients with T2DM compared to healthy controls (150.6 ± 31.2 pg/ml vs. 70.8 ± 16.0 pg/ml, $p < 0.001$). The ECV index was significantly higher in the T2DM group compared to the control group (35.6 ± 5.1 % vs. 23.0 ± 2.9 %, $p < 0.001$), while the mean GLS value was substantially reduced in absolute terms (less negative) in patients with T2DM, indicating impairment of longitudinal contractility (-15.6 ± 2.4 % vs. -19.5 ± 1.5 %, $p < 0.001$). Correlation analysis demonstrated a statistically significant moderate direct correlation between plasma NT-proBNP levels and the ECV fraction ($r = 0.385$, $p = 0.007$), as well as an inverse correlation with the GLS strain parameter ($r = -0.361$, $p = 0.012$) in patients with T2DM.

Conclusion: In patients with T2DM without established cardiovascular disease and heart failure there is a significant increase in the ECV and a decrease in GLS which could indicate the development of diabetic cardiomyopathy and be the initial (preclinical) stage of heart failure with preserved ejection fraction (HFpEF).

P 15

Digoxin Discontinuation Versus Continuation in Patients with Heart Failure and Reduced Ejection Fraction: A Systematic Review and Meta-Analysis

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Background and Aims: Digoxin was once considered the first-line treatment for heart failure with reduced ejection fraction (HFrEF). However, it now remains an adjunctive therapy. Digoxin discontinuation has been a source of uncertainty regarding clinical outcomes, such as hospitalization and mortality. This systematic review and meta-analysis aims to compare the impact of discontinuing or continuing digoxin in patients with HFrEF.

Materials and Methods: We systematically searched PubMed, Embase, and the Cochrane Central Register of Controlled Trials from database inception through May 2026, for studies evaluating discontinuation or continuation of digoxin in patients with HFrEF. Outcomes included heart failure hospitalization, all-cause mortality, heart rate and body weight. Statistical analysis was performed using R statistical software. Dichotomous outcomes were pooled as risk ratios (RRs), and continuous outcomes as mean differences (MDs), with 95% confidence intervals (CIs), using the random-effects model. Heterogeneity was assessed with I^2 statistics. Leave-one-out sensitivity analyses were performed.

Results: We included five studies comprising a total of 4,149 patients. Of the included studies the four parallel trials comprised 4,133 participants; 2,511 patients (60.8%) were withdrawn from digoxin, whereas 1,622 patients (39.2%) continued digoxin. The mean age was 67.7± 12.1 years, 29.6% of participants were women, and the mean left ventricular ejection fraction was 29.8 ± 10.8%. There was no statistically significant difference between groups regarding heart failure hospitalization (RR, 1.19; 95% CI, 0.97–1.47; P=0.102; I^2=72.2%) and all-cause mortality (RR, 1.02; 95% CI, 0.96–1.08; P=0.512; I^2=0%). Continuation of digoxin was associated with a lower heart rate (MD, –5.90 beats/min; 95% CI, –10.04 to –1.77; P=0.005; I^2=58.5%) and lower body weight (MD, –1.34 kg; 95% CI, –2.07 to –0.61; P<0.001; I^2=5.8%). Sensitivity analyses indicated that the heart failure hospitalization estimate was influenced substantially by the observational study by Malik et al, which reduced heterogeneity from 72.2% to 21.8%.

Conclusion: Digoxin discontinuation in HFrEF patients was not associated with a statistically significant increase in all-cause mortality or hospitalizations for heart failure. Digoxin continuation was associated with lower resting heart rate and body weight. Given the limited and heterogeneous evidence, these findings should be interpreted cautiously, highlighting the need for adequately powered randomized trials to determine the safety of digoxin withdrawal in HFrEF patients.

P 16

Bisoprolol Across the Cardiovascular Continuum: An Expert Consensus on Contemporary Evidence and Clinical Applications

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Background and Aims: The objective was to assess the relevance of current evidence and develop consensus perspectives on the appropriate use of bisoprolol across different cardiovascular clinical scenarios.

Materials and Methods: The NEXT (Network for Excellence in Cardiovascular Therapeutics) expert group comprised 120 cardiology experts, representing approximately 2,400 cumulative expert-years of clinical experience. Following a structured review of contemporary evidence, the experts evaluated 10 predefined statements addressing the role of bisoprolol across the cardiovascular continuum. Each statement was assessed using a five-point Likert scale ranging from strongly disagree to strongly agree. Responses were converted into weighted mean scores, with a score >100 predefined as indicating consensus agreement.

Results: Consensus agreement was achieved for 9 of 10 statements, with weighted mean scores ranging from 101.9 to 116.2 among statements meeting the predefined threshold. Experts supported the continued relevance of bisoprolol as an evidence-based betablocker in contemporary cardiovascular practice (weighted mean [WM] 113.8) and its potential advantages in CYP2D6 poor metabolizers because of minimal CYP2D6 dependence (WM 113.9). Elevated heart rate (>80 bpm) was considered an important marker of sympathetic activity warranting consideration of β1-selective therapy beyond blood-pressure control (WM 115.4). Consensus also supported bisoprolol in secondary prevention after myocardial infarction (MI) in clinically stable patients with left ventricular ejection fraction (LVEF) ≥40 % (WM 111.9), consideration of betablocker therapy in post-MI patients with mildly reduced LVEF without clinical heart failure (WM 111.7), and continuation of long-term betablocker therapy following uncomplicated MI and LVEF ≥40 % when clinically appropriate (WM 109.6). Strong consensus was observed regarding bisoprolol as a key betablocker within guideline-directed medical therapy for heart failure with reduced ejection fraction (HFrEF) (WM 112.6) and the integration of betablocker therapy into guideline-directed medical therapy (GDMT) in HFrEF (WM 116.2). Evidence from the DELIVER trial also supported consideration/continuation of betablocker therapy in appropriate patients with heart failure with mildly reduced or preserved ejection fraction (HFmrEF/HFpEF) (WM 101.9). The statement addressing β1-selective blockade specifically for obesity-related hypertension and elevated sympathetic activity did not reach the predefined consensus threshold (WM 70.5). Overall agreement was significant (strongly agree 55 ± 24, 95 % CI 38 – 73; agree 46 ± 20, 95 % CI 31 – 60; p < 0.0001).

Conclusion: This expert consensus identified broad agreement regarding the contemporary role of bisoprolol across the cardiovascular continuum, encompassing heart-rate and sympathetic modulation, post-MI secondary prevention, and heart failure management. The findings reinforce the relevance of bisoprolol within evidence-based cardiovascular care while highlighting areas requiring further clinical evidence, particularly its role in obesity-related hypertension with elevated sympathetic activity.

P 17

External Aortic Cross-Clamping Versus Endo-Aortic Balloon Occlusion in Minimally Invasive Cardiac Surgery: A Systematic Review and Meta-Analysis of Patient Outcomes

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Background and Aims: Aortic occlusion during minimally invasive cardiac surgery (MICS) can be achieved using either external aortic cross-clamping (EACC) or endo-aortic balloon occlusion (EABO). Whether one strategy provides superior clinical outcomes remains uncertain.

Materials and Methods: PubMed, ScienceDirect, and ResearchGate were searched for studies published from the inception through 2023 that compared EACC with EABO in MICS. Overall mortality was designated as the primary outcome. Secondary outcomes included cardiopulmonary bypass (CPB) time, aortic cross-clamp time, conversion to sternotomy, aortic dissection, cerebrovascular events, and hospital length of stay.

Results: No significant difference in CPB time was identified between the two groups (MD, -8.04; 95 % CI, [-16.26, 0.18]; p = 0.06; I^2 = 88 %). Cross-clamp time was also comparable (MD, -5.46; 95 % CI, [-11.80, 0.89]; p = 0.09; I^2 = 88 %). Conversion to sternotomy occurred less frequently in the EACC group, although the difference was not significant (OR, 0.62; 95 % CI, [0.23, 1.68]; p = 0.35; I^2 = 71 %). Hospital length of stay did not differ significantly between EACC and EABO (MD, -0.16; 95 % CI, [-1.07, 0.76]; p = 0.74; I^2 = 96 %). A lower occurrence of aortic dissection was observed with EACC, but the difference was not considered significant (OR, 0.41; 95 % CI, [0.17, 1.00]; p = 0.05; I^2 = 0 %). A similar non-significant tendency favoring EACC was observed for cerebrovascular accident (CVA) (OR, 0.92; 95 % CI, [0.64, 1.31]; p = 0.64; I^2 = 50 %). Mortality was numerically lower in the EABO group, without a statistically significant difference (OR, 1.37; 95 % CI, [0.89, 2.11]; p = 0.15; I^2 = 0 %).

Conclusion: EACC showed a non-significant tendency toward a lower incidence of aortic dissection compared with EABO. No significant differences were identified in mortality, CPB time, aortic cross-clamp time, conversion to sternotomy, cerebrovascular events, or hospital length of stay.

P 18

Urgent Mitral Valve Replacement After Acute Ischemic Stroke in Infective Endocarditis

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Background and Aims: Infective endocarditis (IE) can cause severe complications such as systemic embolisation, heart failure, and multiorgan dysfunction. Neurological involvement, including ischemic stroke, occurs in up to 40 % of cases, complicating management.

Case Description: A 48-year-old man with severe mitral regurgitation (MR) from posterior leaflet prolapse, atrial fibrillation (AF), and diabetes mellitus presented with acute right hemiparesis, facial asymmetry. Brain CT showed an acute right basal ganglia infarct. Echocardiography revealed severe MR (Carpentier type II), flail PML, prolapsing AML, and a mobile vegetation (0.6 – 1.0 cm) on A2. Blood cultures grew ciprofloxacin-sensitive, multi-resistant coagulase-negative staphylococcus. He developed ARDS requiring intubation and was managed with targeted antibiotics, hemodynamic support, and rate control. AF transitioned to RVR, treated with digoxin, amiodarone, and bisoprolol. ICU management included norepinephrine, diuretics, fluid balance. A multidisciplinary team recommended urgent MVR due to severe MR, large vegetations, and high embolic risk. Warfarin was stopped five days preoperatively. Intraoperatively, large vegetations on both leaflets were excised, and a 31 mm bioprosthesis Mitral Magna Ease was implanted. Postoperative TEE confirmed good valve function. He recovered without major complications and was discharged on postoperative day 10.

Discussion: This case illustrates IE complicated by ischemic stroke, ARDS, and AF. Stroke in IE is often due to septic emboli from large, mobile mitral vegetations. Thrombolysis was withheld due to small-vessel stroke pattern, ongoing anticoagulation, and high haemorrhagic risk in IE, consistent with evidence showing increased post-thrombolytic intracerebral haemorrhage. Mechanical thrombectomy was not indicated as no large-vessel occlusion was present. ESC guidelines recommend urgent surgery for IE with heart failure, uncontrolled infection, or high embolic risk. Early surgery was performed safely within a week of ischemic stroke, eliminating the embolic source and restoring valve function.

Conclusion: Early, multidisciplinary intervention with urgent MVR in selected IE patients with ischemic stroke can prevent further embolic and septic complications, achieving favorable outcomes despite complex comorbidities. This case highlights the value of individualised, guideline-informed, and multidisciplinary decision-making in optimising outcomes for high-risk IE patients.

P 19

Switching from the metabolic to cardiac landscape

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Background and Aims: The patient's cardiovascular symptoms should always be taken seriously and the possibility of microvascular coronary artery disease should always be considered in a high risk individual.

Materials and Methods: A 56-year-old man was seen in the cardiology clinic of our hospital, in November, 2025, with a 4-week history of malaise, easy fatigability, intermittent exertional retrosternal discomfort (after taking 20 – 30 steps or two flights of stairs) and palpitations. He had hypertension, prediabetes, dyslipidaemia, metabolic dysfunction-associated steatotic liver disease (MASLD), chronic kidney disease (CKD-G, A1), migraine, cervical and lumbar spondylosis and was previously treated for iron and B12 deficiencies. He had chronic bloating, dyspepsia and bilateral discomfort in both thighs, especially on walking and led a sedentary lifestyle. No addictions, but had a skin allergy to contrast agents. Family history was positive for type 2 diabetes, hypertension, dyslipidaemia, osteoporosis and coronary heart disease. Medications included statin, beta-blocker, angiotensin receptor blocker, cholecalciferol, amitriptyline, sumatriptan, galcanezumab, proton pump inhibitor, chlorzoxazone and paracetamol.

Results: Laboratory findings were reported as normal except for elevated serum creatinine: 110 µmol/l [59 – 104] (eGFR 68 ml/min). Other values were LDL 2.14 mmol/l [< 2.58], triglycerides 2.38 mmol/l [up to 1.7], troponin T 4.82 ng/l [0 – 14], BNP 50.4 pg/ml [194 pg/ml (97.5th percentile)], HbA_{1c} 6.3 % [<5.7], Hb 14.7 g/dl [13 – 18] and D-dimer 0.27 µg/ml [0.22 – 0.45]. The remaining findings were unremarkable. Serial ECGs were normal, with no ischaemia. Exercise stress test was positive for ischaemia. Transthoracic echocardiography and 24-hour Holter monitoring were normal. Chest X-ray and CT were unremarkable. The patient underwent coronary angiography on 20 November 2025. It revealed ectasia of the proximal left anterior descending artery, with mild disease in the mid segment and moderate ostial disease of the first diagonal branch (D1). The coronary circulation was right-dominant. The remaining coronary arteries were normal. The patient was offered advice on a healthy lifestyle along with optimisation of medical therapy (aspirin, a calcium channel blocker, nitrates, an SGLT2 inhibitor and ranolazine were added). He continued to have episodic anginal symptoms, neck sweating and malaise despite complying with the prescribed medical therapy. Drug-eluting balloon angioplasty was performed in December 2025 for the moderate ostial lesion of D1. Subsequently, dynamic coronary microvascular testing was performed in April 2026 because the patient remained symptomatic. It revealed a combination of coronary spasm and endothelial and microvascular dysfunction. In August 2026, the patient underwent coronary sinus reducer insertion. At the time of reporting, he remained symptomatic.

Conclusion: In this era, a patient with suspected microvascular coronary artery disease can be properly endotyped, through coronary dynamic testing and subsequently, may be offered the specific cardiac medication and the insertion of a coronary sinus reducer.

P 20

Reversible Septal Hypertrophy in a Macrosomic Neonate of a Diabetic Mother: A Case Report

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Background and Aims: Hypertrophic cardiomyopathy (HCM) is a well-recognised cardiac complication in infants born to diabetic mothers, resulting from fetal hyperinsulinaemia and myocardial glycogen deposition. The condition is typically transient, but early identification and structured echocardiographic follow-up remain critical to prevent haemodynamic complications and guide clinical decision-making.

Case Presentation: We describe a macrosomic neonate born to a mother with long-standing diabetes mellitus since childhood, who was diagnosed with hypertrophic cardiomyopathy shortly after birth. Echocardiography demonstrated marked thickening predominantly involving the interventricular septum, raising initial concern for haemodynamic compromise, including possible left ventricular outflow tract obstruction. The infant was closely monitored clinically and haemodynamically in the immediate neonatal period. At one-month follow-up, the infant showed marked clinical improvement. Repeat echocardiographic assessment revealed partial regression of septal and myocardial hypertrophy, improved ventricular systolic function, and resolution of any outflow tract obstruction. Throughout the observation period, the infant remained haemodynamically stable and did not require pharmacological or surgical intervention, supporting a favourable, self-limited disease course. Serial echocardiography was central to diagnosis and monitoring in this case. Initial imaging confirmed septal-predominant hypertrophy characteristic of diabetic cardiomyopathy, while follow-up imaging documented measurable regression of wall thickness and normalisation of ventricular function, without outflow obstruction – findings consistent with the expected natural history of this transient condition when properly monitored.

Conclusion: Transient hypertrophic cardiomyopathy in infants of diabetic mothers, although generally self-limited, can represent a significant manifestation of adverse fetal metabolic programming and carries potential for serious complications if unrecognised. Optimal preconception and antenatal glycaemic control, combined with early postnatal echocardiographic screening and structured longitudinal follow-up, are essential to minimize morbidity and mortality. This case illustrates that prompt diagnosis and appropriate conservative management can result in excellent outcomes, whereas delayed recognition may lead to life-threatening haemodynamic consequences.

Inflammation, Hypoxia and Mitochondrial Function in Cardiometabolic Disease

P 21

Reduced mitochondrial DNA copy number is associated with prior myocardial infarction in patients with coronary artery disease and type 2 diabetes

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Background and Aims: Mitochondrial dysfunction contributes to cardiac remodeling in coronary artery disease (CAD) and type 2 diabetes (T2D), while ACE I/D polymorphism may influence cardiovascular phenotype. However, the role of mitochondrial DNA (mtDNA) copy number and its interaction with ACE genotype remain unclear.

Materials and Methods: Sixty-seven patients with CAD and T2D were enrolled and stratified by history of myocardial infarction (MI+ vs MI–). Telomere length and mtDNA copy number were measured in peripheral blood leukocytes using quantitative real-time PCR, with telomere length expressed as the T/S ratio according to Cawthon's method. ACE I/D polymorphism (II/ID/DD) was analysed in an additive genetic model. Between-group comparisons were performed using the Mann-Whitney U test. Hardy–Weinberg equilibrium and genotype distribution were assessed using the χ^2 test. Multivariable linear regression was used to identify independent predictors of mtDNA copy number after adjustment for age, diabetes duration, HbA_{1c}, and estimated glomerular filtration rate (eGFR). A two-sided $p < 0.05$ was considered statistically significant; p values between 0.05 and 0.10 were considered a trend.

Results: Patients with prior myocardial infarction were older, had longer diabetes duration, and lower eGFR than those without MI. HbA_{1c}, ACE genotype distribution, and telomere length did not differ significantly between groups. A trend toward lower mtDNA copy number was observed in the MI+ group (1.030 [0.826–1.322] vs 1.312 [0.967–1.614]; $p = 0.053$). In multivariable analysis, prior MI was independently associated with lower mtDNA copy number ($\beta = -0.323$; 95 % CI -0.606 to -0.040; $p = 0.025$). HbA_{1c} was also independently associated with lower mtDNA copy number ($\beta = -0.078$; $p = 0.010$). ACE I/D polymorphism showed neither an independent association with mtDNA copy number nor an interaction with MI status.

Conclusion: Although only a trend toward lower mtDNA copy number was observed in the unadjusted analysis, multivariable analysis demonstrated an independent association between prior MI and reduced mtDNA copy number. ACE I/D polymorphism did not influence mtDNA copy number or modify this association. These findings suggest a link between prior ischaemic injury and mitochondrial impairment in patients with CAD and T2D. Larger prospective studies are warranted.

P 22

Chronic low-grade inflammation and insulin resistance in internally displaced patients with type 2 diabetes and subclinical cardiac autonomic neuropathy

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Background and Aims: Wartime internal displacement represents a substantial

psychosocial stressor that may contribute to autonomic dysregulation, metabolic disturbances, and chronic low-grade inflammation (CLGI). These mechanisms may be particularly relevant in individuals with type 2 diabetes mellitus (T2DM) and cardiac autonomic neuropathy (CAN). The aim of this study was to compare markers of CLGI and insulin resistance (IR) in internally displaced and non-displaced patients with T2DM and subclinical CAN.

Materials and Methods: The study included 45 participants divided into three groups: 15 healthy controls (Group 1); 15 non-displaced patients with T2DM and subclinical CAN residing in Lviv and the surrounding region (Group 2); and 15 internally displaced patients with T2DM and subclinical CAN (Group 3). The groups were comparable in age, sex, and body mass index, while the two T2DM groups did not differ significantly in diabetes duration or glycated haemoglobin (HbA_{1c}) ($p > 0.05$). Subclinical CAN was identified based on cardiovascular autonomic reflex tests and short-term heart rate variability assessment. Serum concentrations of high-sensitivity C-reactive protein (hsCRP), immunoreactive insulin (IRI), tumour necrosis factor-α (TNF-α), interleukin (IL)-1β, IL-6, and IL-10 were measured using enzyme-linked immunosorbent assays. Insulin resistance was assessed using the homeostasis model assessment of insulin resistance (HOMA-IR), and the TNF-α/IL-10 ratio was calculated as an indicator of the pro-/anti-inflammatory balance. Continuous variables were analysed using non-parametric methods, with statistical significance set at $p < 0.05$.

Results: The most pronounced insulin resistance was observed in internally displaced patients with T2DM and subclinical CAN, with a HOMA-IR value of 5.2 ± 1.7 [2.8; 8.1], exceeding that observed in non-displaced patients with T2DM and subclinical CAN ($p < 0.05$). The internally displaced group also demonstrated the most pronounced elevation in hsCRP (9.9 ± 4.9 [2.1; 18.7] mg/l; $p < 0.05$). Increased concentrations of TNF-α, IL-6, and IL-10, together with an increased TNF-α/IL-10 ratio, were observed in patients with subclinical CAN. The highest TNF-α concentration and TNF-α/IL-10 ratio were observed in internally displaced patients with T2DM and subclinical CAN: 17.0 ± 4.9 [2.8; 21.6] pg/ml ($p < 0.001$) and 3.0 ± 2.9 [0.2; 9.9] ($p < 0.05$), respectively.

Conclusion: Among patients with T2DM and subclinical CAN, internally displaced individuals demonstrated greater insulin resistance and a more pronounced pro-inflammatory profile than non-displaced patients. The higher hsCRP and TNF-α concentrations together with an increased TNF-α/IL-10 ratio suggest a shift towards a pro-inflammatory state in internally displaced patients. These findings indicate an association between wartime internal displacement and additional metabolic and inflammatory burden in patients with T2DM complicated by subclinical CAN.

P 23

Effects of Semaglutide on the Integrated Stress Response, Mitochondrial Dysfunction and Apoptosis in Isoprenaline-Induced Cardiac Hypertrophy

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Background and Aims: Cardiac hypertrophy is an adaptive response to chronic cardiac stress that can progress to pathological remodeling and heart failure, in which the integrated stress response (ISR), mitochondrial dysfunction and apoptosis play important roles. GLP-1 receptor agonists have emerged as promising cardioprotective agents. This study investigated the effects of a GLP-1 receptor agonist, semaglutide (SG), on cardiac remodeling, the ISR, mitochondrial dynamics, and apoptosis in isoprenaline (ISO)-induced cardiac hypertrophy.

Materials and Methods: Male Sprague-Dawley rats were randomised into 4 groups: control, ISO, SG, and ISO + SG. Cardiac hypertrophy was induced by ISO (5 mg/kg/day, s.c.) for three weeks, while SG (120 µg/kg/day) was given during the last two weeks. Echocardiography was performed to assess cardiac structure and function before sacrifice. Body weight (BW), heart weight (HW), left ventricular weight (LVW) and blood glucose were measured. The expression of BNP, eIF2α, p-eIF2α, PERK, DRP1, MFN2, PINK1, SIRT3, BAX, BCL-2 and CHOP in the left ventricle was analysed by Western blotting.

Results: ISO induced marked cardiac hypertrophy, indicated by elevated HW/BW and LVW/BW ratios. SG did not alter these parameters but significantly reduced BW and blood glucose levels. Left ventricular posterior wall thickness in systole (LVPWs) and diastole (LVPWd) was increased in ISO-treated rats compared to controls. SG decreased LVPWd only in hypertrophic animals, with no effect in healthy rats. Ejection fraction (EF) and fractional shortening (FS) did not differ among groups. The hypertrophy marker BNP was increased in the ISO group, but not in the ISO + SG group. Among the ISR markers, PERK was enhanced in ISO-treated rats, while eIF2α and p-eIF2α expression levels were comparable across groups. No significant differences were observed in the expression of the mitochondrial fission protein DRP1, fusion protein MFN2, or mitophagy marker PINK1 among groups. The expression levels of SIRT3 and CHOP, linked to mitochondrial dysfunction or apoptosis, were increased in the ISO group, and SG treatment reversed this increase. The pro-apoptotic protein BAX and the anti-apoptotic protein BCL-2 were unchanged in the ISO group compared to controls, but BAX expression was reduced in the ISO + SG group.

Conclusion: These results suggest that SG does not affect functional and structural alterations in hypertrophic hearts; however, it can modulate molecular changes associated with mitochondrial function and apoptosis.

P 24

Intermittent hypoxia as a modulator of glutathione system activity in the heart of rats with type 2 diabetes

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Background and Aims: Oxidative stress (OS) is well-known to play a pathogenetic role in the development

of diabetes-induced cardiac diseases, including cardiomyopathy, heart failure, myocardial infarction, etc. A key component of antioxidant defense in the heart is the glutathione system, which maintains intracellular redox balance and protects the cell against oxidative injury. Periodic activation of reactive oxygen species (ROS) during intermittent hypoxia triggers a cascade of intracellular redox signaling, followed by the synthesis and activation of protective components, including antioxidants. Our previous research has shown that intermittent hypoxia upregulates antioxidant defense in the brain, liver and the cardiovascular system under different stress conditions. However, the role of intermittent hypoxia in the glutathione-induced antioxidant protection of cardiomyocytes under hyperglycaemia has not been fully investigated. The aim of this work was to evaluate the impact of mild intermittent hyperbaric hypoxia (IHH) on the glutathione pool state in the myocardium of rats with type 2 diabetes.

Materials and Methods: Experiments were performed on 6-month-old male Wistar rats fed a high-fat diet for four weeks; after two weeks of the experiment, 20 or 25 mg/kg of streptozotocin was administered. The development of insulin resistance was assessed using the insulin tolerance test. Blood glucose concentration (12 ± 1.5 mmol/l) in rats indicated type 2 diabetes (T2D). Rats were exposed to five sessions of 1 h hypoxia in a barochamber (3500 m) for three days. Oxidative stress biomarkers, including the content of thiobarbituric acid-reactive substances (TBARS), the level of reduced glutathione, as well as the activities of GSH-related enzymes (glutathione reductase (GR) and glutathione peroxidase (GPx)) were evaluated in the myocardium.

Results: It was shown that hyperglycaemia caused a marked increase in TBARS production, indicating intensified prooxidant processes and overproduction of ROS in the rat heart. We found decreased GSH content and GR activity in the diabetic rat heart compared with the control, although GPx activity increased. This may be linked to increased H_2O_2, a substrate for glutathione peroxidase, and to NADPH depletion, which is needed to maintain sufficient GSH levels in the glutathione reductase reaction. Adaptation to IHH reduced the TBARS content by 25 % ($p < 0.05$) and markedly increased the content of reduced glutathione by 87 % ($p < 0.05$) and the activity of GR by 56 % ($p < 0.05$), with a tendency to an increase in the activity of GPx in comparison with T2D rats.

Conclusion: Our findings indicate that IHH induces protective mechanisms against ROS-mediated oxidative stress damage in the heart of rats with hyperglycaemia, and this may be mediated through the modulation of glutathione system activity.

P 25

Mitochondrial mechanisms of cardioprotection in insulin resistance and the effects of hypoxic preconditioning in rats with myocardial hypertrophy

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Background and Aims: One of the main pathogenetic links of heart damage in metabolic diseases may be the lack of energy supply to the myocardium. One of the promising cardioprotective approaches is hypoxic preconditioning (HP). However, mitochondrial mechanisms under preconditioning conditions, especially in myocardial hypertrophy (MH), have so far been studied only fragmentarily in individual models. The purpose was to establish mitochondrial mechanisms of cardioprotection in rats with myocardial hypertrophy in the presence of insulin resistance and the effects of hypoxic preconditioning.

Materials and Methods: The study was conducted on sexually mature male Wistar rats and spontaneously hypertensive rats (SHR) with MH in compliance with the requirements of the European Convention for the Protection of Vertebrate Animals. Insulin resistance (IR) was induced by feeding the animals a high-fat diet for 14 days (58 % animal fat from the total caloric content of the diet). To reproduce HP, the rats were placed in a pressure chamber and "lifted" to an altitude of 5600 m (380 torr) for 3 h. A day later, myocardial tissue samples were taken from anaesthetised animals to study the functional activity of cardiomyocyte mitochondria by the Chance polarographic method and their structure by electron microscopic examination.

Results: In MH, there was a tendency to decrease the indices of oxidative phosphorylation (OP) and respiratory control by 39 % and 24.85 % ($p < 0.05$), respectively, during glutamate oxidation. IR contributed to a tendency to decrease in the indices of energy metabolism in both lines of rats. Preconditioned animals with MH showed a tendency to increase V3 and V4 during glutamate and succinate oxidation and a decrease during palmitoyl oxidation. Regarding the effect of HP in the presence of IR in SHR, there was a decrease in the OP of glutamate and succinate by 37.74 % and 39.17 % ($p < 0.05$), respectively, compared with the HP group. In morphometric studies, endothelial and sarcolemmal dysfunction developed in the myocardium of animals with IR of both lines, but a quantitative increase in mitochondria was observed, especially pronounced in SHR, along with preservation of their function and activation of intracellular connections with other organelles. HP led to an increase in mitochondrial dynamics in the myocardium. HP of animals with IR was characterised by activation of the myocardial mitochondrial apparatus and energy metabolism in Wistar rats and to a lesser extent in SHR. However, signs of myofibril damage were detected, more pronounced in the hypertrophied SHR myocardium.

Conclusion: Myocardial hypertrophy worsened the energy metabolism of the heart. Lipid loading contributed to an increase in the number of mitochondria and intensification of their work. Hypoxic preconditioning in this setting improved the ultrastructure of mitochondria, but there were signs of deterioration in their activity.

P 26

Hypoxic preconditioning improves myocardial mitochondrial ultrastructure in severe hypoxia and phosphoinositide-3-kinase blockade in rats

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Background and Aims: Hypoxic preconditioning (HP) has a cytoprotective effect through the activation of signaling pathways involved in compensatory and adaptive reactions. One of them is the IGF-1/PI3K/Akt signaling pathway, which is involved in the regulation of cell growth, proliferation, or death, and also affects cellular metabolism, glucose transport and gene expression. However, the possible effects of HP in severe hypoxia (SH) and the features of the protective pathway involving phosphoinositide-3-kinase (PI3K) in myocardial mitochondria (M) have been poorly studied. The aim was to investigate the effect of hypoxic preconditioning on the ultrastructure of rat myocardial mitochondria exposed to severe hypoxia and PI3K blockade.

Materials and Methods: The study was conducted on adult male Wistar rats in accordance with the requirements of the European Convention for the Protection of Vertebrate Animals. HP and SH were induced using a pressure chamber, "lifting" the animals to 5600 m for 3 h and after a day to 9000 m for 3 h, respectively. PI3K blockade was performed by administering wortmannin (Wort) at a dose of 2 µmol/l intravenously 30 min before SH. The ultrastructure of the myocardial M was examined by electron microscopy.

Results: The greatest increase in the number of M occurred under the influence of HP + SH, i. e. preconditioning was triggered, which promoted the activation of M morphogenesis. With SH and the addition of Wort to SH, this adaptation mechanism was not implemented. A significant increase in the number of damaged M was observed in all groups. The greatest increase in structurally altered M was observed in the group of animals exposed to SH with the addition of Wort. However, the number of damaged organelles occurred in both the SH group and the HP + SH group following administration of Wort. That is, the use of Wort under any hypoxic influence worsened the ultrastructural state of the myocardial mitochondrial apparatus. In the vast majority of the examined groups, there was an increase in the diameter of M. The exception was the HP + SH group. The diameter of M increased significantly more in the organelles of the subsarcolemmal subpopulation. In the intramyofibrillar subpopulation of M, the increase in diameter in all groups did not even reach 20 %. The use of Wort led to a significant increase in the diameter of subsarcolemmal M and indicated a possible path of necrotic organelle death.

Conclusion: Previous exposure to hypoxic preconditioning before severe hypoxia leads to a decrease in myocardial mitochondrial ultrastructural abnormalities and activates compensatory-adaptive cellular responses. The use of wortmannin leads to a deterioration of the condition. One of the mechanisms involved in the protective effects of hypoxic preconditioning is the IGF-1/PI3K/Akt-mediated signaling pathway.

Evidence, Comorbidity and Engagement in Cardiometabolic Care

P 27

DiaFight: a pilot study evaluating the acceptability and perceived impact of a gamified intervention in paediatric type 1 diabetes

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Background and Aims: Routine diabetes care may not fully address the individual psychosocial and self-management needs of children and adolescents with type 1 diabetes. This study combines Spotlight-AQ, an adaptive digital preconsultation assessment, with DiaFight, an educational card game intended to facilitate communication and engagement with diabetes self-management. The objective is to assess the feasibility, acceptability and perceived impact of the combined Spotlight-AQ and DiaFight intervention in routine paediatric diabetes care.

Materials and Methods: This prospective, single-centre, exploratory mixed-methods pilot study will enrol 30 children and adolescents aged 8 – 17 years, with type 1 diabetes of at least one year's duration, who use continuous glucose monitoring. Parents or legal guardians will also participate, and siblings aged 8 – 17 years may complete an optional questionnaire. During the three-month study, children and parents will complete their respective Spotlight-AQ assessments at baseline and at study end, and participants will be asked to play DiaFight at least twice weekly, with play documented in a game diary. The primary outcome is the acceptability and perceived impact of the combined intervention as reported by participants, families and healthcare professionals. Semi-structured interviews with a purposive subsample of at least 12 participants and their parents or legal guardians, as well as participating healthcare professionals, will undergo thematic and content analysis. Secondary exploratory outcomes include intervention-specific usability, acceptability and relevance ratings, DSTAR (Diabetes Strengths and Resilience measure) and child and parent PAID (Problem Areas In Diabetes) measures, HbA_{1c}, continuous glucose monitoring metrics, sensor use, and insulin-related measures. Quantitative analyses will be descriptive and hypothesis-generating.

Results: Outcome data are not yet available.

Conclusion: The study is designed to assess the feasibility, usability, acceptability and perceived impact of the combined intervention, explore barriers and facilitators to its implementation in routine paediatric diabetes care, and generate preliminary descriptive data on patient-reported outcomes and clinical and device-derived measures. The findings may support refinement of the intervention and the design of subsequent trials.

P 28

Glycohypoxia as a Cardiorenal-Metabolic Oxygen-Unloading Phenotype in Type 2 Diabetes: Mechanistic, Clinical and Haemodynamic Evidence

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Background and Aims: Glycohypoxia is an emerging pathophysiological framework in which chronic hyperglycaemia may increase haemoglobin oxygen affinity through HbA_{1c}-associated glycation, impair peripheral oxygen unloading, and generate functional tissue hypoxia despite preserved arterial oxygenation. We integrated sequential mechanistic, quantitative, clinical, and haemodynamic evidence to test whether this phenotype may contribute to cardiorenal-metabolic vascular burden in type 2 diabetes (T2DM).

Materials and Methods: The original framework linked haemoglobin glycation and left-shifted oxygen dissociation to oxidative stress, impaired hypoxic signaling, and diabetic organ injury. We then synthesised six human studies (n = 450) using random-effects meta-regression and modelled oxygen unloading at microvascular PO_2 of 20–40 mmHg. Exploratory validation was performed in 90 adults with T2DM stratified by HbA_{1c} (≤ 7.0 %, 7.1–8.9 %, ≥ 9.0 %; n = 30/group). A subsequent secondary analysis examined systolic/diastolic blood pressure across the same strata alongside P_{50}, 2,3-BPG, SpO_2-SaO_2 bias, lactate, PaO_2, and modelled oxygen-release indices.

Results: Meta-regression showed ΔP_{50} = -0.19 mmHg per 1 % HbA_{1c} (95 % CI -0.26 to -0.11; P < 0.001), predicting 0.5–1.3 % less oxygen unloading per 1 % HbA_{1c}. Clinically, P_{50} fell from 27.0 to 26.2 mmHg, 2,3-BPG from 4.8 to 4.2 µmol/g Hb, SpO_2-SaO_2 bias rose from +0.2 % to +1.8 %, and lactate from 1.2 to 1.9 mmol/l, while PaO_2 remained unchanged. Across the same HbA_{1c} strata, systolic pressure increased from 130.18 to 138.26 mmHg and diastolic pressure from 78.18 to 84.26 mmHg (both P < 0.001), corresponding to gradients of +2.69 and +2.03 mmHg per 1 % HbA_{1c}. Modelled oxygen-release impairment reached ~2–4 % in poorly controlled T2DM.

Conclusion: Worsening glycaemic burden was accompanied by parallel impairment of haemoglobin oxygen unloading and rising blood pressure without arterial hypoxaemia. These convergent findings support glycohypoxia as a measurable erythrocyte-vascular phenotype and a testable HbA_{1c}-oxygen-unloading-endothelial NO-vascular-pressure axis that may help connect hyperglycaemia with cardiovascular, renal, and microvascular complications. Prospective participant-level studies are required to establish causality.

P 29

The effect of therapeutic education programme on quality of life among type 2 diabetic patients

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Background and Aims: Type 2 diabetes mellitus is a chronic non-communicable disease with major impact on health in general and quality of life in particular. The aim of this study was to evaluate the effect of a therapeutic education programme on the quality of life among patients with type 2 diabetes.

Materials and Methods: Between May 2021 and June 2022, 320 outpatients were enrolled in a randomised controlled trial at the "National Fund for Social Security" Polyclinic in Sfax, Tunisia. The experimental group received the therapeutic education programme, whereas the control group received only standard care. For data collection, the Arabic version of the Diabetes Quality of Life (DQoL) questionnaire was employed for evaluating the quality of life at baseline and at the end line of the study after three months of the intervention.

Results: In total, 263 patients completed the intervention, 132 in the experimental group and 131 in the control group. In terms of the main baseline characteristics, the two groups were similar. At the end of the study, there was a significant difference in all domains of quality of life scores between the experimental and control groups: satisfaction (61.21 ± 9.65 versus 71.29 ± 6.23, p < 0.001), impact (42.25 ± 6.59 versus 49.50 ± 4.83, p < 0.001) and worries (49.20 ± 9.04 versus 58.09 ± 6.83, p < 0.001). Quality of life improved over time in the experimental group (61.87 ± 5.17 versus 52.63 ± 7.73, p < 0.001) and remained the same in the control group (61.35 ± 4.85 versus 61.21 ± 4.88, p = 0.644).

Conclusion: The benefits of implementing a diabetes education programme were observed across of all quality of life domains.

P 30

Impact of therapeutic education intervention and pillbox use on medication adherence and blood pressure control among uncontrolled high blood pressure patients in Tunisia

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Background and Aims: Despite growing awareness of hypertension, the global burden of the disease remains insufficiently addressed. Medication adherence, although well recognised, is often underestimated and is widely regarded as a key determinant of hypertension control. This study aimed to assess the impact of an intervention combining pillbox use and therapeutic education (TE) on medication adherence and blood pressure control among uncontrolled hypertensive patients in Tunisia.

Materials and Methods: A randomised controlled trial was conducted from May 2021 to December 2022 including a representative sample of 274 uncontrolled hypertensive patients taking ≥ 3 pills per day, followed at the National Fund for Social Security Sfax, Tunisia. Participants were randomly assigned to two groups. The first group received a TE intervention and a pillbox; the second group served as the control group. The intervention consisted of three 45-minute educational sessions. The data collection tool was a 2-section questionnaire including an information sheet and the Girerd medication adherence scale.

Results: In total, 274 patients were included. Overall, 85.0 % (n = 233) had no hypertension-related complications, while 88.0 % (n = 241) had at least one chronic comorbidity, mainly diabetes (65.3 %, n = 179) and dyslipidaemia (58.5 %, n = 141). Before the programme implementation, results showed that 11.7 % of the participants had a high level of medication adherence (n = 32). Post-intervention, medication adherence was significantly higher (32.1 % vs. 6.0 %, p < 0.001), as was blood pressure control (70.7 % vs. 19.4 %, p < 0.001) in the experimental versus control group, respectively.

Conclusion: Findings indicate that combining TE and pillbox use was effective in enhancing medication adherence levels and blood pressure control among uncontrolled hypertensive patients.

P 31

Is fasting a risky proposition?

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Background and Aims: The study aimed to observe the fasting trend and experience among the elderly home care patients with Type 2 diabetes.

Materials and Methods: This was an observational study, in which the clinical and laboratory data of the fasting, old aged individuals with Type 2 diabetes, was collected during the holy month of Ramadan (18th February – 18th March, 2026). All of them were Saudis, being followed up by the home care teams of our hospital. The patients' ages, genders, body mass indices, diagnoses, current medications, cognition, fasting complications (hypoglycaemia, dehydration, infection, thromboembolic event), vital signs, HbA_{1c} and eGFR (ml/min) were recorded by visiting them and through the hospital's electronic health records. The fasting risk for people with diabetes was estimated through the updated IDF-DAR calculator-2026. Their CKD (KDIGO) classes were also estimated. Their expected 10-year cardiovascular disease (CVD) risk scores (%) were calculated by using the AHA/ACC ASCVD 2013 calculator for people between 40 – 75 yrs and the ESC SCORE-2 OP calculator for those > 75 yrs.

Results: There were 35 fasting patients (64 %) with an age range of 58 – 89 yrs (mean 73 years) and mean body mass index of 28 kg/m^2. Type 2 diabetes was present in 63 %. Mean HbA_{1c} was 8.1 %. No fasting complication was noted. 18 % of patients with Type 2 diabetes had chronic kidney disease (mean eGFR 59 ml/min) with KDIGO classes I – IV. 36 % of patients with diabetes had either ischaemic heart disease or heart failure and 32 % were found to have a past history of stroke (cumulative ASCVD seen in 64 %). Out of the remaining 36 % without ASCVD, 75 % were > 75 yrs [mean CVD risk 45.5 %] and 25 % were between 40 – 75 yrs [mean CVD risk 18 %]. 23 % of them had cognitive impairment. Their Ramadan DAR fasting risk score (2026) ranged between 6.6 – 15.5 (mean 11).

Conclusion: 64 % of our elderly home care patients with type 2 diabetes safely fasted in Ramadan, despite having an ASCVD in the majority, > 45 % CVD risk among those without ASCVD and having a high fasting risk (mean 11).

P 32

Quality improvement project on optimising lipid control in patients with coronary artery disease in a district general hospital

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Background and Aims: Coronary artery disease (CAD) is a leading cause of morbidity and mortality in the United Kingdom. Lipid lowering therapy is effective in reducing major adverse cardiovascular events, with a 21 % relative risk reduction for every 1.0 mmol/l reduction in low density lipoprotein cholesterol (LDL-c) levels. This quality improvement project aims to assess lipid control in patients with CAD and optimise lipid-lowering therapy in line with the National Institute for Health and Care Excellence (NICE) guidelines.

Materials and Methods: 69 patients with CAD attending cardiology outpatient clinics in November 2024 were identified. Eleven patients were excluded due to the absence of a lipid profile within two years. Lipid profiles of the remaining 58 patients were reviewed. In accordance with NICE guidelines, a non-high-density lipoprotein cholesterol (non-HDL-C) target of 2.6 mmol/l or less was used as the target for optimising risk reduction (3). The patients who did not achieve target non-HDL-C were offered an intervention, either up-titration of existing statin therapy, addition of ezetimibe or initiation of inclisiran. Lipid profiles were repeated 2 – 3 months later.

Results: At baseline, 35 of the 58 patients (60 %) achieved the target non-HDL-C level. Among the 23 patients who did not achieve the target non-HDL-C level, four patients were not receiving lipid-lowering medication, ten were taking low-dose statins, six were taking maximum-dose statins and three were receiving combination therapy with ezetimibe. Patients were then contacted regarding treatment optimisation. Seven patients (30 %) proceeded with an intervention (Group 1) and nine patients (39 %) requested repeat blood tests prior to any intervention (Group 2). The remaining patients declined engagement. Two to three months later, three patients in Group 1 showed an improvement in non-HDL-C (mean 2.1 mmol/l), and therefore met the target. Three patients in Group 1 did not engage with repeat blood tests. One patient on a high intensity statin and inclisiran showed an improvement (non-HDL-C 3.2 mmol/l), but was still above target. Among the Group 2 patients, seven subsequently had non-HDL-C within target and therefore did not require an intervention, and two were not in range but declined further intervention.

Conclusion: We conclude that the lipid profiles of CAD patients are not tested annually in primary care, as seen in eleven patients in our initial cohort. Using the NICE target of non-HDL-C 2.6 mmol/l or less, only 60 % achieved the target on initial lipid lowering therapy. With optimised treatment, target non-HDL-C levels are achievable in most patients with CAD. In this study, patient engagement and concordance with medication were key obstacles in optimising lipid control. Hence, patient education and broader public health awareness are vital for improving effective secondary prevention of CAD.

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