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Scientific Meetings

12TH GLOBAL SUMMIT ON

DIABETES & ENDOCRINOLOGY

JULY 26-28, 2025

AMSTERDAM, NETHERLANDS

Venue: Holiday Inn Express Arena Towers
Address: Hoogoorddreef 66b, Amsterdam-Zuidoost,
Amsterdam, 1101 BE, Netherlands

Scientific Program

Day 1

Scientific Program

July 26, 2025 | Amsterdam, Netherlands

08:30–08:45: Registrations

08:45–09:00: Opening Ceremony

Keynote Presentations

09:00–09:40

Title: What should be the therapeutic target in the treatment of Type-2 Diabetes

Ravi Muppirala

Jeeva Therapeutics, USA

09:40–10:20

Title:Missing links in the aetiology and progression of Type 2 Diabetes

G. Naresh Kumar

Jeeva Therapeutics, USA

Session Introduction

Session Chair: Ravi Muppirala, Jeeva Therapeutics, USA

Session Co-Chair: G. Naresh Kumar,Jeeva Therapeutics, USA

Oral Presentations

10:20–10:45

Title:Exercise-induced changes in postural stability among patients with diabetes

Milena Raffi

University of Bologna, Italy

Group Photo | Coffee Break 10:45-11:00

11:00–11:25

Title: Incretin Drugs for Diabetes Management

Rania Alnounou

London Consulting Medical Center, United Arab Emirates

11:25:11:50	**Title: Early Screening of Type 1 Diabetes** **Lauri Deane** Time in Range Diabetes Education, USA
11:50:12:15	**Title: Profiling of circulating microRNA in different subtypes of Type 2 Diabetes in the Emirati Population** **Fatima Gamil Mohammed Sulaiman** Mohammed Bin Rashid University United Arab Emirates
12:15–12:40	**Title: Subclinical hypothyroidism: To treat or not to treat?** **Hanaa Tarek El-Zawawy** American Hospital Dubai, United Arab Emirates

Lunch Break 12:40-13:40

13:40–14:05	**Title: The amyloid oligomer modulator anle138b has disease modifying effects in a human IAPP transgenic mouse model of type 2 diabetes mellitus (hIAPP Ob/Ob mice)** **Mohammed Mreef H Albariqi** King Abdulaziz City for Science and Technology, Saudi Arabia
14:05–14:30	**Title: Knowledge, Attitudes, and Practices of Sports Instructors Regarding Type 1 Diabetes: A Comparative Study Between Public and Private Sectors in Menzel Bourguiba, Tunisia** **Emna Hariz** Kit Institute, Netherlands

14:30–15:30 Poster Presentations

P001	**Title: Challenges and Strategies in Type1Polyendocrinopathy Associated with Inaugural Type 1 Diabete": About a case** **Benfiala Mouna** Central Army Hospital, Algeria
P002	**Title: MIRNA expression profile in women with Gestational Diabetes** **Duaa Ahmed Elhaga** Sidra Medicine, Doha, Qatar

Group Photo | Coffee Break 15:30-15:40

Panel Discussion & Certificate Falicitation

Day –1 Ends

Day 2

July 27, 2025 | Amsterdam, Netherlands

Scientific Program

Oral Presentations

09:00–09:20	**Title: Unlocking Okara's Potential: A Plant-Based Approach to Managing Diabetes** **Nazir Ahmad** Abasyn University Islamabad Campus, Pakistan
09:20–09:40	**Title: Non-pharmacological and Pharmacolgical approaches to Metabolic dysfunction-associated Steatotic Liver Disease (MASLD): An Overview** **Mariya Tabassum** Abdul Malek Ukil Medical College, Noakhali, Bangladesh
09:40–10:00	**Title: Subclinical hypothyroidism: To treat or not to treat?** **Hanaa Tarek El-Zawawy** American Hospital Dubai, United Arab Emirates
10:00–10:20	**Title: Therapeutic ketosis and the broad field of applications for the ketogenic diet: Ketone ester applications & clinical updates** **Raffaele Pilla** St. John of God Hospital, Italy
10:20–10:40	**Title: Functional Food for Type 2 Diabetes Mellitus: Okara Noodles** **Kaisun Nesa Lesa** Khulna University, Bangladesh
10:40–11:00	**Title: Clinical Significance of Second to Fourth Digit Ratio in Infertile Males and Its Correlation with Semen Analysis and Testosterone Levels** **Saurabh Gupta** Pacific Medical College and Hospital, India

Panel Discussion

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HYBRID EVENT

KEYNOTE PRESENTATIONS

DAY 1

Ravi Muppirala
Jeeva Therapeutics, USA

What should be the therapeutic target in the treatment of Type-2 Diabetes?

Currently, Hyperglycemia is the chief therapeutic tar- get in Type-2 Diabetes (T2D). Some of the therapeutic approaches such as insulin and SGLT2 inhibitors focus mainly on reducing hyperglycemia. Some argu- ments are presented here to discover if it is the correct therapeutic target or if hypeinsulinemia should be con- sidered along with it. Previously we argued that why the preferred method to maintain euglycemia in T2D must be simulating the physiological pulsed endoge- nous insulin secretion; it is still valid.

It is known, that most types of peripherally adminis- tered insulin (PAI) have drawbacks; viz. being non-native, lacking c-peptide, not mimicking physiological insulin secretion oscillations (ISO) that reduce insulin receptor saturation, contributing to weight gain (often as visceral fat). Also, PAI is far less effective in countering hyperglycemia, as it doesn't mimic pancreatic release of insulin in terms of differential exposure - viz. higher levels to the liver and 30% to muscle and adi- pose tissues. Thus regulation of glycemic control by PAI results in increased obesity coupled with trigger- ing endothelial dysfunction. Also, PAI does not mimic insulin pulsatile release by islets. PAI administered for mere glycemic control results in higher insulin exposure to adipose and skeletal muscle, thereby inducing dysfunction not only in these organs but also in endothelium. Hence, it may be more beneficial to steer away from PAI.

The question of therapeutic target becomes important far before hyperglycemia establishes itself as a hallmark symptom in the advanced stages of type-2. Ma- jor issues involved in this metabolic disorders such as T2D is the time scale of 20-30 years from initiation, detection, early stages, full blown stages and advanced stages of the disease. Interestingly, early stages of hy- perinsulinemia is common to many metabolic diseases viz T2D, NAFLD and CVD. It involves increased biomass of pancreatic β cells with hypertrophy, increased proinsulin/insulin (PI/I ratio) ratio and amy- loid plaques around the islets. The progression of this stage into specific metabolic disorder is unclear. Con- trarily, later stages of T2D is associated with loss of pancreatic β cells coupled with insulin resistance in liver, muscle and adipose tissues in conjunction with exaggerated endothelial dysfunction resulting in CVD. Hence, the question of stage specific therapeutic tar- get(s) become important far before hyperglycemia es- tablishes itself as a hallmark symptom in the advanced stages of T2D.

Regulation of intestinal epithelial barrier permeability is crucial at all stages of the disease. Similarly, hyperinsulinemia manifests even in very early stages of the disease involving ER stress. Additionally, relieving β- cell exhaustion by periodic inhibition of insulin secre- tion could decrease PI/I ratio and amyloid plaque for- mation. Excess visceral fat formation and subsequent insulin resistance is known to be mediated by inflam- mation involving free fatty acids and ceramides. Ex- cess PAI is known to elicit side effects by decreasing autophagy, decreasing antioxidant enzyme synthesis and activating eNOS leading to Cardiovascular mor- bidity. In the early stages these are more pronounced at specific tissue regions and progressively become more systemic.

Although recent research demonstrates the merits of portally administered insulin, access to the portal vein is not practical. While DPP4-I/GLP-1, even with con- comitant glitazones, are good therapeutic approaches, they remain insufficient to achieve tight glycemic con- trol. Additionally, they fail to mimic the ISO and feed- ing correlated hepatic portal secretion. This is reflect- ed in healthy individuals, GLP-1 released from ente- rochromaffin cells interacts with vagus nerve to delay gastric emptying only 50% acts on hepatocytes and 20% on pancreas due its degradation in transport by endothelial DPP4. Thus GLP-1 analogs administered exogenously cannot mimic physiological insulin secre- tion.

Meanwhile it may be beneficial to revisit insulin secre- tagogues (IS) and modify traditional therapy, while improving approaches to endogenous secretion and concomitantly minimizing side effects. Dosage of sul- fonylureas should be reduced to maintain a very basal secretion. This should be co-administered with a short acting IS (e.g. meglitinides) prior to feeding. This ap- proach can be titrated to mimic native secretion. Dys- functional aspects of ISO from β-cell should be given critical consideration in the therapy, which are likely to alleviate autocrine, paracrine and other signaling dependent secretion of glucagon, amylin and somato- statin. Reducing inflammation and enhancing NADPH in pancreatic β cells make IS more effective.

Thus, hyperglycemia may not be the right target. Mimicking physiological pulsatile insulin release, blocking temporarily/ intermittently insulin release while maintaining intestinal epithelial barrier could be more effective in the management of T2D. Therapeutic formulations to achieve these prop- erties in a continuous and chronic delivery are critical.

Biography:

Ravi Muppirala is a Biophysicst who has held academic appointments at T.I.F.R., Carnegie-Mellon, Syracuse University and University of Michigan. His expertise and interests span bio-molecular structure-dynamics, origins of primitive cells and type-2 diabetes.

G. Naresh Kumar
Jeeva Therapeutics, USA

Missing links in the aetiology and progression of Type 2 Diabetes

Type 2 diabetes (T2D) is caused by genetic and envi- ronmental factors such as diet, inactive lifestyle, chronic stress, disturbed circadian rhythms, etc. How- ever, most of these factors are also implicated for other chronic disorders such as non-alcoholic fatty liver dis- ease (NAFLD), obesity and cardiovascular disease. Metabolic syndrome with abnormal biochemical and physiological parameters leads to these metabolic dis- orders. Leaky gut, hyperinsulinemia, hyperglycemia, insulin resistance, hyperglyceridemia, hypercholes- terolemia and chronic tissue inflammation are associ- ated with metabolic dysfunction of liver, adipose tissue (subcutaneous and visceral), skeletal muscle, vascular endothelium, intestine and pancreas. The extent and nature of tissue specific dysfunction varies from the onset of metabolic syndrome to specific disease state. Hyperisulinemia is implicated for the onset of meta- bolic syndrome which further progresses to postpran- dial hyperglycemia, and hyperglyceridiemia. Hyperin- sulinemia itself is induced by increased ceramides and free fatty acids in lymph due to mesenteric lymphatic damage from chronic leaky gut. However, the contri- bution of islet inflammation, pancreatic β cell endo- plasmic reticulum stress and decrease in hepatic in- sulin clearance is not clear.

Hyperinsulinemia appears to be mediated by mesen- teric inflammation in response to the leakage of chy- lomicrons from damaged lymphatics. This leads to post prandial hyperglycemia with decrease in skeletal muscle glucose uptake resulting in healthy obesity. At this stage, proinflammatory cytokines like ceramides and free fatty acids are elevated in lymph but not in blood. These initial stages further change to hyper- triglyceridemia and increase in fasting glucose levels associated with proinflammatory ceramides and free fatty acids presence even in blood. However, the na- ture of metabolic disorder exhibited in an individual is not clear. Accumulation of fat in liver leads to NAFLD, fasting hyperglycemia to pre-diabetes and increased oxidized Low-density Lipoproteins (oxLDL) to formation of atherosclerotic plaques.

In case of T2D, lymph node dysfunction causes insulin resistance in pancreas, liver, skeletal muscle, adipose tissue and endothelial cells. However, there is no clari- ty on the contribution of insulin resistance of these organs responsible for the progression from healthy

obesity (Subcutaneous adipose tissue) Unhealthy Obesity (Visceral adipose tissue) prediabetesT2D microvascular complications macrovascu- lar complications. Many clinical parameters are corre- lated with the metabolic dysfunction such as HbA1C, glucose intolerance, free fatty acids, ce- ramides, LDL and HDL.

Diabetic complications are correlated with HbA1C levels > 8.0 in which fasting glucose levels are > 10 mM and these conditions favor advanced glycation end product (AGE) mediated endothelial and extra cellular matrix damage. In a vicious cycle, AGE prod- ucts increase oxidative damage involving endothelial Nitric oxide synthase and NADPH oxidase. Increased reactive oxygen species react with LDL into oxidized form

and damage endothelial cells. Monocytes enter intima at the damaged sites and become foam cells which accumulate oxLDL triggering high levels secre- tion of IL6, TNFα and IL1β. At these sites endothelial cells undergo dedifferentiation into macrophages and osteocytes in atherosclerotic plaques. Rupture of ma- ture atherosclerosis plaques progressively lead to mi- cro- and macro-vascular complications. The role of chylomicrons and de novo lipogenesis towards Lipo- toxicity in organs and Visceral adipose inflammation may provide an insight to these processes. Interesting- ly, chylomicrons in normal individuals get converted into remnants within 30 min while very low density lipoproteins are present for a few hours for becoming remnants.

Above mentioned processes describe common patho- physiological processes but individual pathogenicity depends on the pronounced component. Moreover, gender dependent pathophysiological parameters to specific metabolic disorder are unclear. In cases of Polycystic ovarian syndrome, estrogen dysfunction leads to severe insulin resistance and T2D. Similarly, hypothyroidism leads to obesity and thyroid receptor agonist Resmetirom is used for the treatment of NAFLD. However, only 10% and 5% of T2D patients have clinical and subclinical hypothyroidism, respec- tively. While hypothyroidism is prevalent in women, there is no significant gender based difference in T2D incidence. A challenging issue is to explain how indi- vidual progression of T2D happens in individuals without micro- or macro-vascular complications or Hypertension.

Biography:

G. Naresh Kumar is Retired Professor, Department of Biochemistry/, The Maharaja Sayajirao University of Baroda, Gujarat, India. His expertise and interests span Designer Probiotics, Metabolic Engineering of Bacteria, Metabolic Syndrome, Origins of primitive cells and Type-2 diabetes. The research dealt with exploiting probi- otic Escherichia coli strains for delivering antioxidants to ameliorate toxic effects of heavy metals, arsenite, ethanol, carbon tetrachloride, dimethyl hydrazine. E. coli has been modified to confer beneficial effects of delaying ageing, improving iron absorption and decreasing deleterious effects of dietary high fructose and sucrose.

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SPEAKER PRESENTATIONS

DAY 1

Milena Raffi
University of Bologna, Italy

Exercise-induced changes in postural stability among patients with diabetes

Exercise plays a vital role in both preventing and managing diabetes. One of its key benefits is improving postural control by enhancing the processing of various sensory input, critical components for maintaining balance and stability. During self-motion, we are exposed to optic flow patterns that provide information about heading direction. These patterns have a direct effect on body sway.

The study aims to evaluate the effects of an 18-month exercise program on balance control and postural muscle activation in a cohort of uncompensated diabetic patients (HbA1c>52 mmol/mol).

EMG and stabilometric data were recorded in 12 diabetic people who followed an 18 months exercise program (age 59,1±10,2; 33% oral medication; 56% insulin therapy) and in 12 diabetic people with sedentary habits (age 59,4±12,1; 71% oral medication; 14% insulin therapy) at baseline, 6, 12 and 18 months. During recordings, the subjects stood on two force platforms while viewing optic flow stimuli projected on a screen. EMG data were recorded bilaterally from the tibialis anterior and soleus. Data acquisition included 2 trials per stimulus and two baseline trials (upright stance in the dark). EMG and center of pressure (COP) data have been analyzed separately by repeated measures ANOVA.

Significant effects and interactions have been found in both COP directions for time, group, stimuli, laterality and sex ($p<0.05$). EMG data revealed a significant interaction between stimuli and muscle ($p=0.008$).

Results suggest that diabetic people who exercise regularly have a better body balance than sedentary counterparts. This augmented control improved across the time of exercise.

Key words: Optic flow, physical exercise, training, diabetes, muscle

Biography:

Milena Raffi is associate professor of Human Physiology at University of Bologna, Italy. She has published more than 50 papers in peer-review journals with IF and has been serving as an editorial board member of repute. The research activity focuses on various aspects of cognitive functions: role of visual perception on postural and motor control, functional characterization of eye movement neurons, cellular mechanisms involved in the analysis of visual perception, role of spatial attention and oculomotor functions. She is Principal Investigator of several national and international projects.

Rania Alnounou
London Consulting Medical Center, United Arab Emirates

Incretin drugs for diabetes management

Aim of the study (objectives)

To critically evaluate healthcare practice based on a literature background, and to suggest real practice improvement.

Background: The prevalence of type 2 diabetes, is increasing at an alarming rate, with only half of patients achieving the recommended HbA1c target. This study tends to focus on incretin based treatment for type 2 diabetes, and their utility in clinical practice specifically in private clinical practice in Abu Dhabi,

Incretin drug group, stimulates insulin secretion and inhibit glucagon production, they also improve beta-cell health, slow gastric emptying, promote early satiety and reduce food intake (Nyenwe EA 2011)

This study is a clinical audit supported by literature review that shows new drugs for diabetes management and despite being expensive, still do not reach the target of diabetes control. A lot of gaps in our clinical practice, especially primary care level need to be modified to achieve holistic management.

Participants: I consided around 150 patients with type 2 DM who are taking Incretin drugs with

or without other diabetic medication. The drugs which be followed are: GLP-1RA:

Liraglutide (victoza)

Dulaglutide (trulicity)

Exenatide (byetta, bydureon) DPP-4 Inhibitors

Sitagliptin (Januvia, janumet)

Linagliptin (tradjenta,jentadueto)

Vildagliptin (Galvus,Galvusmet)

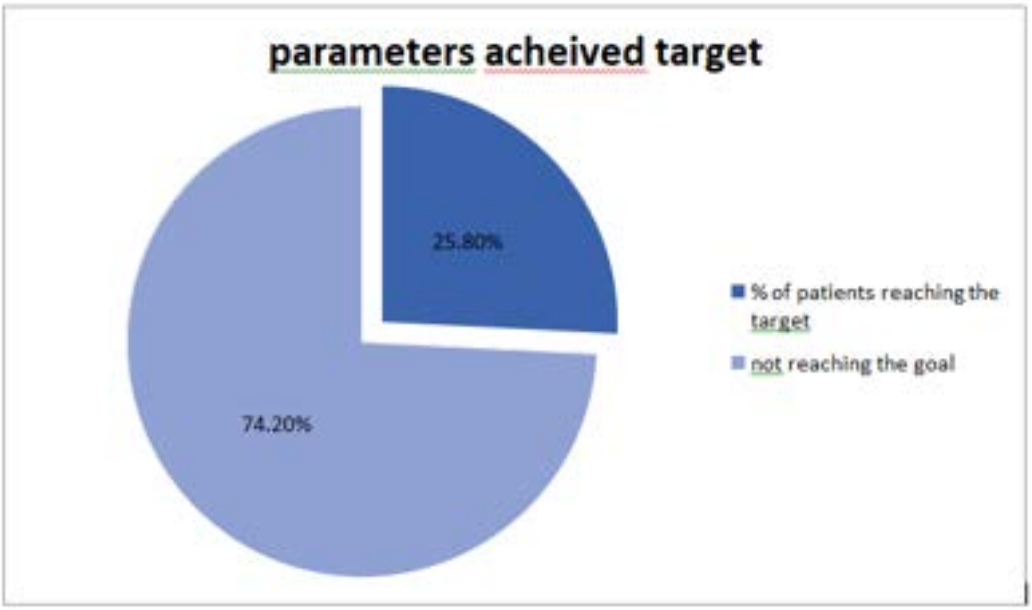
Variables: I followed five parameters, HBA1c, BG, BP, BW, and eGFR.

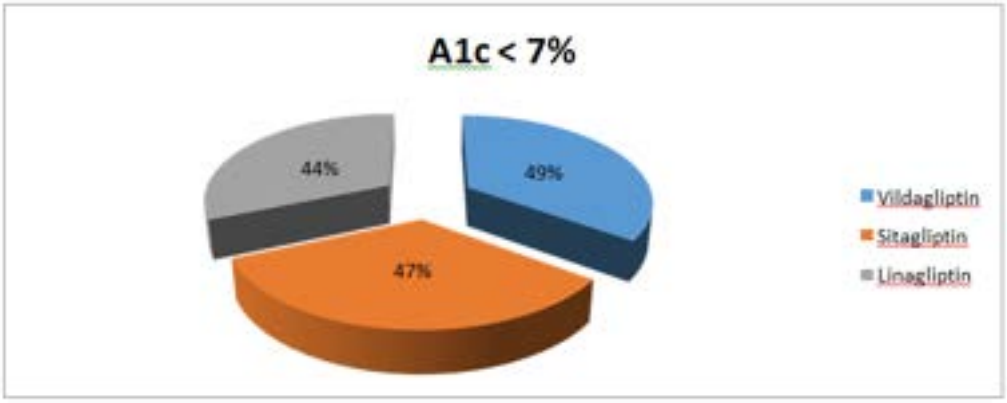
Settings: the audit conducted in the UAE, Abu Dhabi, Al Noor hospital, Al Bateen and musaffah

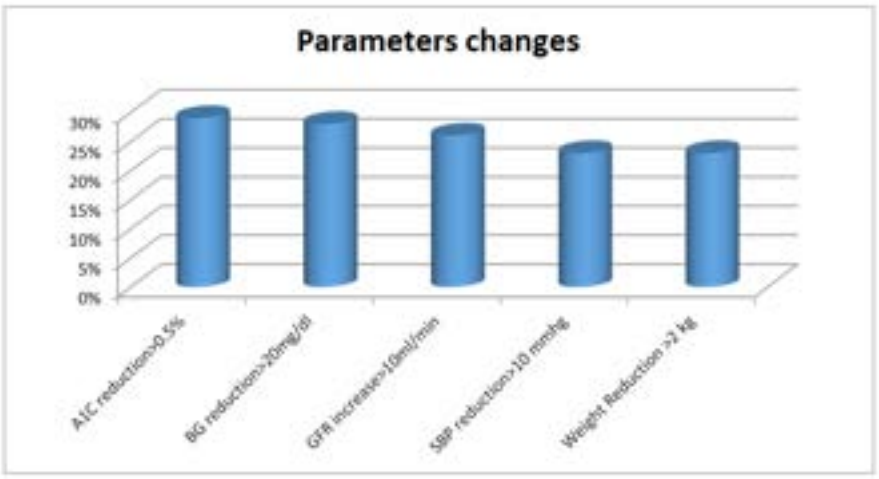
branches, for 3-6 months between September and December 2016.

Results:

Criterion	Number sampled	achievement	standard
- Main criterion: HbA1c< 7% (NICE)	97 patients	49%	70%
- Main criterion: HbA1c<8%(AL NOOR)	97 patients	74%	95%
- A1c reduction by 0.5%-1% after 3-6 months of treatment	82 patients	29%	70%
- BG reduction > 20mg/dl (1.1 mmol/l) after 3-6 months of treatment	83patients	28%	80%
- GFR increase > 10 ml/min after 3- 6 months of treatment	78patients	26%	70%
- SBP reduction> 10mmhg after 3-6 months of treatment	88 patients	23%	70%
- Weight reduction > 2 kg after 3-6 months of treatment	84 patients	23%	90%
- Parameters achieved the overall target after 3-6 months of treatment	97 patients	25.8%	90%







Suggestions are summarized below:

Development of a system which would enable easier identification of uncontrolled DM.

Development of a system which would facilitate communication between all related team particularly regarding cardiology, ophthalmology, podiatric, and dietitian, coordinated team of health professionals is required.

Development of structured education program with education classes for patients with diabetes.

Identification criteria for routine case examination of health visits using check-list (which examines a number of aspects of patients, including structured education, eye test, labtest, foot test, cardiology consultation, echocardiogram, biomarkers (ie, NT-proBNP) etc).

Establish diabetic clinic with multidisciplinary team. Meanwhile there was a suggestion about making diabetic day every week to follow all patients with diabetes.

Development of telecare intervention via web-based system or mobile devices, to be in continuous contact with HCPs.

Establish a new medication program, in our local pharmacy to give extra help and advice about any new medication prescribed for the first time to treat long term condition like diabetes.

Lauri Deane
Time in Range Diabetes Education, USA

Early Screening of Type 1 Diabetes

Type 1 diabetes is a progressive autoimmune disease characterized by the immune system attacking and destroying insulin producing beta cells in the pancreas. The progression occurs in 3 stages. Stage 1 and 2 are the pre-symptomatic stages and can be identified by the presence of ilet antibodies. Early stages 1 and 2 can occur months to years before the onset of symptoms and progression to stage 3 (clinical) type 1 diabetes. Selecting high risk individuals and diagnosing early autoimmune diabetes allows for monitoring of hyperglycemia and need for insulin, prevention of ketoacidosis (DKA) and subsequent complications, as well as potentially delaying progression to stage 3 through emerging immunotherapies.

Biography:

Lauri Deane is a Board-Certified Advanced Practice Registered Nurse (APRN, FNP-C), Certified Diabetes Care and Education Specialist (CDCES) and is Board Certified in Advanced Diabetes Management (BC-ADM). With 14+ years' experience in nursing, she has spent the last 8 years focusing on advancements in diabetes management and treatments. Lauri is an expert in the use of diabetes technologies such as insulin pumps and continuous glucose monitors. Lauri is a professional member of the American Diabetes Association (ADA) and actively participates in interest groups regarding autoimmune diabetes. Previous experience includes academic medicine with the University of Miami Diabetes Research Institute. Lauri is an advocate for her patients and families living with type 1 diabetes and is passionate about educating health care providers on early detection. Lauri's dedication to this specialization is rivaled only by her commitment to patient education, empowerment and support.

Fatima Gamil Mohammed Sulaiman
Mohammed Bin Rashid University, United Arab Emirates

Profiling of circulating microRNA in different subtypes of Type 2 Diabetes in the Emirati Population

Introduction: Type 2 diabetes (T2D) is a heterogeneous disease influenced by both genetic and environmental factors. Recent studies suggest that T2D subtypes may exhibit distinct gene expression profiles. In this study, we aimed to identify T2D cluster-specific miRNA expression signatures for the previously reported five clinical subtypes that characterize the underlying pathophysiology of long-standing T2D: severe insulin-resistant diabetes (SIRD), severe insulin-deficient diabetes (SIDD), mild age-related diabetes (MARD), mild obesity-related diabetes (MOD), and mild early-onset diabetes (MEOD). Methods: We analyzed the circulating microRNAs (miRNAs) in 45 subjects representing the five T2D clusters and 7 non-T2D healthy controls by single-end small RNA sequencing. Results: Bioinformatic analyses identified a total of 430 known circulating miRNAs and 13 previously unreported novel miRNAs. Of these, 71 were upregulated and 37 were downregulated in either controls or individual clusters. Each T2D subtype was associated with a specific dysregulated miRNA profile, distinct from that of healthy controls. Specifically, 3 upregulated miRNAs were unique to SIRD, 1 to MARD, 9 to MOD, and 18 to MEOD. Among the downregulated miRNAs, 11 were specific to SIRD, 9 to SIDD, 2 to MARD, and 1 to MEOD. Conclusion: Our study confirms the heterogeneity of T2D, represented by distinguishable subtypes both clinically and epigenetically and highlights the potential of miRNAs as markers for distinguishing the pathophysiology of T2D subtypes.

Biography:

Fatima Sulaiman is a PhD student at the College of Medicine, Mohammed Bin Rashid University of Medicine and Health Sciences, Dubai. She was educated in the UAE and the UK. She obtained her Master's degree in Molecular Medicine at the University of Aberdeen, United Kingdom (2019-2020). She is currently a fourth-year PhD student at MBRU and has extensive research experience in the field of human genetics and COVID-19. She is currently involved in the study deconstructing the heterogeneity of type 2 diabetes using clinical, genomic, and circulating extracellular RNA biomarkers in Emirati patients.

Hanaa Tarek El-Zawaw
American Hospital Dubai, United Arab Emirates

Subclinical hypothyroidism: To treat or not to treat?

Subclinical hypothyroidism (SCH) represents a challenge in clinical practice. SCH is a form of mild hypothyroidism, meanwhile, it poses multiple risks with many adverse clinical consequences.

SCH treatment has been a matter of debate long ago and significant controversy still exists as to whether to treat or not to treat.

The treatment of SCH has been the subject of an enormous amount of work with many published studies. The American Thyroid Association guidelines settled some instances where the treatment of SCH is to be considered.

The treatment of SCH should be individualized given the patient's profile and risk assessment.

Biography:

Hanaa Tarek El-Zawawy is an Endocrinology Consultant at American Hospital Dubai and an Assistant Professor of Endocrinology at Alexandria University Faculty of Medicine where she had her doctorate degree in Endocrinology in May 2015 at the age of 31 years.

El-Zawawy is a member of the Egyptian Society of Endocrinology and Obesity as well as the European Society of Endocrinology. She participates in many national & international conferences and workshops every year.

She authors 18 peer-reviewed publications. She was cited 147 times, H-index=7. Also, she is an active reviewer in many international journals and receives a yearly award from the publisher “WILEY” for her contributions.

Emna Hariz
Kit Institute, Netherlands

Knowledge, Attitudes, and Practices of Sports Instructors Regarding Type 1 Diabetes : A Comparative Study between Public and Private Sectors in Menzel Bourguiba, Tunisia

Introduction: Physical education is crucial for all students, particularly those with type 1 diabetes (T1D). It plays an integral role in their overall management. However, without adequate precautions, it exposes diabetic individuals to serious metabolic risks such as ketosis and hypoglycemia. A well-prepared school environment, especially the sports instructor, is essential to ensure safe physical activity. The objective of this study was to compare the opinions, attitudes, and levels of knowledge of sports instructors regarding T1D in the public versus private sectors.

Materials and Methods: A cross-sectional study was conducted, involving 45 sports instructors from Menzel Bourguiba: 32 from the public sector and 15 from the private sector. Their knowledge, attitudes, and practices regarding T1D were analyzed.

Results: Among respondents, 64.5% of public sector instructors and 13.3% of private sector instructors reported having or currently working with children or adolescents with T1D, showing a statistically significant difference (p=10^-3). The average knowledge score on T1D was 4.1/10 for the public sector and 3.4/10 for the private sector. Those scoring ≥5 were 34.4% and 13.3% in the public and private sectors, respectively, with no significant difference between groups. The lowest response rates in both sectors concerned the threshold defining hypoglycemia, precautions before and during physical activity (p=10^-3), and the roles of different insulin types (p=10^-3). Univariate analysis revealed a correlation between knowledge scores and years of experience but no association with the sector. Most public sector instructors highlighted the lack of necessary equipment, such as emergency kits and infirmaries, to manage diabetic students during emergencies.

Conclusion: It is essential to create a suitable environment for young diabetics and conduct seminars to enhance the knowledge of sports instructors and teachers about T1D in our health area. This will improve the safety of children and adolescents with T1D during physical activities.

Keywords: Type 1 Diabetes, Physical Activity, Public Sector, Private Sector, Sports Instructor

Highest education degree: Doctor of Medicine (MD)

Biography:

Emna Hariz is a medical doctor and a resident in Preventive Medicine, pursuing a Master of Public Health and Health Equity at KIT Institute in Amsterdam. Her research focuses on social determinants of health, adverse childhood experiences, and infectious disease prevention. She has conducted studies on childhood trauma, suicide risk, and healthcare-associated infections. She has authored multiple scientific publications and presented them at international conferences. Dr. Hariz has extensive experience in epidemiology, community health, and medical education. She actively participates in public health initiatives, including vaccination campaigns and health promotion activities, with a commitment to health equity and vulnerable populations

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POSTER PRESENTATIONS

DAY 1

Benfiala Mouna
Central Army Hospital, Algeria

Challenges and Stratégies in Type 1 Polyendocrinopathy Associated with Inaugural Type 1 Diabetes" : About a case

Introduction: Autoimmune polyendocrinopathy type 1 (APECED is a rare condition characterized by the coexistence of multiple autoimmune endocrine disorders. These include type 1 diabetes, which occurs in approximately 60–70% of cases (1), adrenal insufficiency, hypoparathyroidism, and chronic mucocutaneous candidiasis.This complex association poses significant challenges for clinicians in terms of diagnosis and management due to its clinical variability and impact on patients' overall health.

Case Report: We report the case of a 27-year-old female patient followed in the endocrinology department since April 2015 for type 1 autoimmune polyendocrinopathy. Her condition included chronic mucocutaneous candidiasis, hypoparathyroidism, Biermer's anemia, autoimmune hypothyroidism, and adrenal insufficiency. She was on Levothyrox ,Fludrocortisone,Calcium, Alfacalcidiol, Hydrocortisone and vitamin B12.Following a major hyperglycemic episode with severe spontaneous diabetic ketoacidosis, the diagnosis of diabetes mellitus was made.The acute condition was managed with self-pulsating syringe insulin therapy,rehydration and electrolyte correction. After clinical improvement, the patient was transferred to the endocrinology department for further evaluation and therapeutic education.Clinical evaluation revealed a normoweight patient reporting cardinal symptoms of polyuria, polydipsia, and a 3 kg weight loss.Physical examination showed mucocutaneous candidiasis on the inner thighs, hyperpigmentation and asthenia, with no signs of hypocalcemia or over/under-dosing of Levothyrox or Hydrocortisone.Biologically, fasting blood glucose was 0.91 g/L, HbA1c was 12.4%. Mycological examination confirmed mucocutaneous candidiasis.The patient was started on an optimized insulin therapy regimen (total dose: 0.44 U/kg/day, basal dose: 0.12 U/kg/day),achieving satisfactory glycemic control. She also received dietary education, local antifungal treatment and appropriate psychological support.The diagnosis of type 1 diabetes was established based on the autoimmune context (APECED), the patient's young age, overt cardinal symptoms, presentation with diabetic ketoacidosis, and insulin dependency.

Conclusion: The management of patients with type 1 autoimmune polyendocrinopathy requires a multidisciplinary,proactive and personalized approach due to the complexity of the disease. Continuous monitoring is crucial to detect any autoimmune disorders associated on time.

Keys words: Addison's disease; Autoimmune Polyglandular Syndrome type 1 (APS-1); Autoimmune-poly-endocrine-candidiasis-ectodermal-dystrophy (APECED); Chronic hypoparathyroidism; Chronic mucocutaneous candidiasis.

Duaa Ahmed Elhag
Sidra Medicine, Qatar

MIRNA expression profile in women with Gestational Diabetes

Introduction: MicroRNAs (miRNAs) are small non-coding RNAs that play critical roles in regulating host gene expression. Recent studies have indicated a role of miRNAs in the pathogenesis of gestational diabetes mellitus (GDM), a common pregnancy-related disorder characterized by impaired glucose metabolism. Aberrant expression of miRNAs has been observed in the placenta and/or maternal blood of GDM patients, suggesting their potential use as biomarkers for early diagnosis and prognosis. Additionally, several miRNAs have been shown to modulate key signaling pathways involved in glucose homeostasis, insulin sensitivity, and inflammation, providing insights into the pathophysiology of GDM.

MATERIALS AND METHODS: 12 Plasma samples were obtained from pregnant women at delivery time point (3 samples from GDM mother group, 3 samples from mother control group, 3 samples from GDM cord blood group, 3 samples from cord blood control group). MiRNA was extracted from 200 Ml plasma samples using the standard protocol from Qiagen Kit. Total RNA concentration was quantified using qubit and samples with concentration above 50ng/ml were selected for the c-DNA synthesis experiment. MiRNA samples were reverse transcribed using the miRCURY Locked Nucleic Acid (LNA™) Universal Reverse Transcription (RT) kit. Sensitive and specific miRNA profiling was performed using LNA-enhanced, SYBR® Green-based miRCURY LNA miRNA miRNome PCR Panels. Real-time PCR was performed using triplicate RT reactions per sample group on miRCURY LNA miRNome Human Panels I and II (730 miRNAs in total). The amplification was performed in a LightCycler® 480 Real-Time PCR System (Roche) in 384-well plates. The initial data analysis was performed using the Roche LightCycler software, for determination of the raw Cp (or Ct depends on PCR machine) by the 2nd derivative method, then raw Cp values were uploaded in Qiagen gene globe software for further analysis. Data from miRNAs with Cp values less than 40 were included. Normalized expression is shown as fold changes in GDM mother blood and GDM cordblood compared to non GDM control samples. Reference genes were selected using geoNorm algorthim. Enrichment analysis was done separately using the MITURNET platform.

Results: Four MIRNA were highly expressed in women with GDM at delivery time point compared to controls including hsa-miR-486-5p, hsa-miR-15b-5p, hsa-miR-30a-5p, hsa-miR-181a-5p. Also, Twenty-two MIRNA were highly expressed in GDM cord blood in which 2 of them were overlapped with the GDM mother group.

Discussion and Conclusion: GDM showed to be associated with pathways that are linked with diabetes complications and inflammatory response such AGE-RAGE signaling pathway in addition to insulin and cytokines signaling pathways. Also, the differentially expressed MIRNA showed to control different genes that may play a role in the pathogenesis of GDM. This highlights the importance of MIRNA based studies to assess the MIRNA signature in women with GDM.

Keywords: Gestational diabetes, Micro RNA, AGE-RAGE signaling pathway

Biography:

Duaa Elhag is a biochemist and a molecular biologist. She gained her master's degree in biochemistry and molecular biology from Al-Neelian University in Khartoum, Sudan in 2017. During her master's thesis, she investigated the role of haptoglobin genetic polymorphism in the severity of sickle cell anemia in Sudanese kids. In 2018 she joined Sidra Medicine in Doha, Qatar as a research specialist. Her current research focuses on identifying biomarkers for different pregnancy complications and she has many publications in this field mainly in gestational diabetes and preterm birth.

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Nazir Ahmad
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Unlocking Okara's Potential: A Plant-Based Approach to Managing Diabetes

Hyperglycemia resulting from the loss of β-cells and/or insulin resistance is a hallmark of diabetes mellitus, a metabolic disease. With a high content of dietary fiber (50%) and protein (25%), fat (10%), vitamins, and phytochemical components like isoflavones and soyasaponins, okara, a soybean byproduct, has garnered special attention for its health benefits as a source of fiber-rich foods that can help people with diabetes. In order to provide a comprehensive overview of the health advantages of okara for the treatment of diabetes mellitus, this review gathered data. To conduct this review, electronic databases were searched to find pertinent literature published between 2010 and 2023. In order to improve satiety and lower blood glucose levels, okara can reduce body weight and postpone the digestion and absorption of carbs. In conclusion, eating okara, a functional food element, helps manage diabetes successfully.s

Biography:

NAZIR AHMAD is currently working as an Assistant Professor at the Department of Pharmacy, Abasyn University Islamabad Campus, Pakistan. He has completed his PhD at the age of 30 years from Faculty of Pharmacy, Universitas Gadjah Mada, Indonesia under GMIF fully funded scholarship (session 2021-24) and MPhil Pharmacology from the Faculty of Pharmaceutical Sciences, Government College University Faisalabad, Pakistan under Master Level PEEF Scholarship (session 2017-19). His major interests are Diabetes Mellitus, Neuropharmacology, and Natural Products. He has five years work experience as pharmacist. He is the Scientist at the Research and Development, Well Grow Nutraceuticals Faisalabad, Pakistan. He has published more than 10 papers in reputed journals and has been serving as a reviewer of repute.

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Non-pharmacological and Pharmacolgical approaches to Metabolic dysfunction-associated Steatotic Liver Disease (MASLD): An Overview

Metabolic dysfunction-associated steatotic liver disease (MASLD) closely associates with obesity and type 2 Diabetes. Lifestyle interventions, aiming at substantial weight loss, are cornerstones of MASLD, treatment by improving the histological outcomes. Originally developed as antidiabetic drugs, Incretin Mimetics and SGLT2 Inhibitors also reduce steatosis and fibrosis. Certain Incretin agonists effectively improve histological features of MASLD. On the other hand, despite moderate weight gain, one PPARγ agonist was found to improve MASLD with certain benefit on fibrosis in the RCT. We here discuss liver-related outcomes, induced by different MASLD treatment options and their association with weight loss. As such, we have compared results from clinical trials on drugs acting via weight loss (Incretin Mimetics, SGLT2 Inhibitors) with those exerting no weight loss (Pioglitazone). Furthermore, other drugs in development, which directly target hepatic lipid metabolism (lipogenesis inhibitors, FGF21 analogs), have also been addressed.

Keywords: Metabolic dysfunction-associated steatotic liver disease (MASLD), Metabolic dysfunction-associated steatohepatitis (MASH), Drug treatment, Weight loss, Obesity.

INTRODUCTION: Recently, based on an international consensus, the term NAFLD was replaced by "Metabolic-dysfunction associated steatotic liver disease" (MASLD) [1]. MASLD requires the presence of steatosis and at least one cardiometabolic risk factor (overweight/obesity, hyperglycemia, hypertension, hypertriglyceridemia, or low high density lipoprotein cholesterol), in the absence of alcohol consumption. MASLD closely relates to obesity, insulin resistance and type 2 Diabetes, with which it shares many pathogenic features [2]. MASLD has been recognized as an important risk factor for several other diseases including hepatocellular carcinoma (HCC), extra-hepatic malignancies and chronic kidney disease [3]. Currently, MASLD affects about 25% of the world's adult population, placing a tremendous burden on healthcare sectors [4].Approximately 10–20 % of people with steatosis progress to "Metabolic dysfunction-associated steatohepatitis" (MASH) [5]. The prevalence of MASH in the general population is projected to rise by 40% within 2030 in Europe [5]. Nowadays, MASLD has evolved as one of the main reasons of liver transplantation in the US and Europe [6,7].

Obesity, Insulin Resistance and MASLD: In people with obesity, MASLD prevalence is estimated to be about 75% [8]. The first step in MASLD pathogenesis is adipose tissue dysfunction. With overweight and obesity, adipocytes need to cope with excessive nutrient delivery and storage, which is achieved by cellular hypertrophy. This adipocyte hypertrophy results in tissue hypoxia and mechanical stress, which trigger immune cell activation and invasion [9]. Consequently, insulin resistance develops, paralleled by altered adipokine (i.e. adiponectin, leptin etc.) secretions and impaired mitochondrial functions, further promoting adipose tissue inflammation [10,11]. Visceral adipose tissue (VAT) compartments also contribute to the development and progression of MASLD [12]. People with Asian ethnicity show higher VAT accumulation

at a given BMI compared to people with Caucasian backgrounds, which may explain the higher prevalence of MASLD in Asian population [21,22]. VAT is associated with higher lipolysis, greater insulin resistance and increased release of pro-inflammatory and pro-fibrogenic mediators [13]. In both obese and lean individuals, VAT mass correlated not only with insulin resistance of liver and adipose tissues but also with liver lipid content and the degree of liver fibrosis [14].In accordance, the presence of MASLD predicts both the transition from MHO to MUO and future cardiometabolic risk [13,16]. The rate of hepatic de novo lipogenesis is much greater in MASLD, which further contributes to intrahepatic lipid accumulation [15]. With increasing hepatic lipid accumulation, there is generation of toxic lipid intermediates (diacylglycerols, ceramides) and altered hepatocellular mitochondrial respiration rates. These together drive hepatic insulin resistance, hepatocellular inflammation and oxidative stress - all of these fueling hepatic pro-fibrotic pathways [17,18]. In addition, the presence of hyperglycemia [19] and altered bile acid secretion patterns also drive MASLD [20].

Non-pharmacological treatment of MASLD : Lifestyle interventions

Caloric restrictions can rapidly reduce steatosis and hepatic insulin resistance [22–24]. Histological improvement of different MASLD components depended on the amount of weight loss. Whereas about 5–7 % of weight loss already lead to a reduction in liver lipid content in 65 % of people with MASLD, MASH resolution (defined as the absence of hepatocellular ballooning) was achieved in 64 % by a 7–10 % decrease in body weight [25-28]. Reduction in >10% of body weight resulted in a 100 % rate of steatosis improvement, a 90 % rate of MASH resolution and even reversed existing fibrosis by at least one stage in 81 % of people [27]. Healthy dietary habit is also very relevant, as Mediterranean diets showed beneficial effects on liver lipid content [29,30]. These diets rely on plant-based foods. Although their total fat content amounts to 30–40 % of daily energy intake, the distinct fat composition with a higher monounsaturated-to-saturated fatty acid ratio likely contributes to lower liver lipid accumulation [31-33]. Other components, such as low intake of red meat and high intake of antioxidant polyphenols, may also mediate the beneficial effects of Mediterranean diets [30,34]. International guidelines {AASLD (American Association for the Study of Liver Diseases), EASL (European Association for the Study of the Liver), ESPEN (European Society for Clinical Nutrition and Metabolism)} also recommend Mediterranean diets for people with MASLD [35–37]. A recent met analysis indicated beneficial effects of gradual weight loss as compared to rapid weight loss in regards to fat mass and basal metabolic rate [38]. For any lifestyle concept, it is essential to achieve loss of fat mass (FM) while maintaining lean body mass (LBM). Loss of LBM impedes sustainability of weight loss by causing low basal metabolic rate and slowing of metabolism, which may result in regain of fat mass [39]. Physical exercise improves liver lipid content [40,41]. The combination of diet and exercise are more successful than each intervention alone [42]. As such, current guidelines recommend combating MASLD based on both lifestyle modifications, including a healthy diet and regular exercise [6].

Pharmacological treatment of MASLD : Drugs with weight loss-dependent effects for MASLD treatment

There is increasing evidence that pharmacologically-induced weight loss can also reduce liver lipid content. Besides weight loss, other drug-elicited effects may contribute in parallel to the recovery of liver homeostasis.

1.Incretin mimetics

GLP-1 receptor agonists

In general, all GLP-1RAs or “Glucagon-like peptide-1 receptor agonists” (short-acting e.g. Exenatide and long-acting e.g. Liraglutide, Semaglutide, Albiglutide) can induce weight loss by central GLP-1 receptor activation in specific regions in the hypothalamus, promoting satiety and decreasing appetite [43,44].

Subsequent reduced caloric intake is regarded as the major mechanism of GLP-1RA-mediated weight loss. The average HbA1c reduction with GLP-1RA in MASLD studies was estimated to be 0.5 % in individuals with and without type 2 Diabetes [45]. Importantly, GLP-1RA reduce the risk of "major adverse cardiac events" (MACE) improve kidney function in people with type 2 diabetes [46]. The primary mediator of the observed improvements in MASH by GLP-1RA treatment is presumably weight loss. Two RCTs with Liraglutide and Semaglutide in mixed collectives of people with and without type 2 diabetes provided evidence for higher rates of histological MASH resolution without worsening of fibrosis compared to placebo. MASH resolution was mainly driven by improvements in steatosis and ballooning for Liraglutide [47], whereas Semaglutide also reduced inflammation [48]. Both treatments were accompanied by a weight loss of 5.5 % and 12.5 % as well as a HbA1c reduction of 0.5 % and 1.2 %, respectively. In people with compensated cirrhosis with and without type 2 diabetes, Semaglutide showed a weight loss of 8.8 % and marked reduction in liver lipid content [49]. Thus, taking into account beneficial liver-related effects as well as cardiovascular and renal benefits and the favorable safety profile, GLP-1RA should be considered for MASLD treatment, especially in obesity and type 2 Diabetes [50]. In a small trial in people with biopsy-confirmed MASH without type 2 diabetes, Liraglutide decreased adipose tissue lipolysis as well as leptin levels paralleled by increasing circulating adiponectin [23]. Of note, although Gliptins also inhibit GLP-1-cleaving dipeptidyl peptidase 4 (DPP-4), no clinically relevant effects on liver lipid content, inflammation or fibrosis have been observed with these group of drugs.

Dual and Triple agonists: RCTs investigating the effects of Tirzepatide (dual agonist) on liver histology in people with MASH are ongoing. After 52 weeks of treatment with the highest dose of Tirzepatide, about 69 % and 43 % of all participants achieved a weight loss of 10% and 15%, respectively, suggesting histologic improvement beyond hepatic steatosis [51,52]. Phase 2 trials investigating the effects of Cotadutide and Survodutide (dual agonists) on histological components of MASH are also ongoing [53]. First results from RCTs with Retatrutide (triple agonist) indicate unprecedented weight loss (24.2% body weight reduction in the obese population) as well as marked reductions in hepatic lipid content, with a subgroup analysis showing MASLD resolution in >85 % of people [54,55].

2.SGLT Inhibitors: In recent RCTs in cohorts with type 2 diabetes and MASLD, SGLT2I treatment led to a 2–4 % decrease in body weight [56]. Utilizing imaging methods, Canagliflozin failed to induce a statistically significant reduction in liver lipid content [57], whereas both Dapagliflozin and Empagliflozin were able to reduce liver lipid content in people with type 2 Diabetes, with and without MASLD [56]. Of note, the amount of weight loss determined the amount of liver lipid reduction for both Empagliflozin [56,58] and Canagliflozin [57] in the respective RCTs. In another RCT, Empagliflozin led to a placebo corrected 2.3-fold greater reduction in liver lipid content and a 36 % increase in plasma adiponectin levels in people with type 2 Diabetes [58]. In a small-scale pilot study, Empagliflozin reduced steatosis, ballooning and fibrosis after 24 weeks of treatment in people with type 2 Diabetes and MASH when compared to a pretreatment group [59]. A recent 72-week RCT with Ipragliflozin including participants with type 2 diabetes and MASLD, reported higher rates of MASH resolution and fibrosis regression with Ipragliflozin, accompanied by a BMI reduction of 1.06 kg/m2 and decrease in HbA1c of 0.4% [60].

3.Metformin: Metformin is associated with modest but consistent decrease in body weight (averaging 2% after 1 year) [61,62]. But there is no solid evidence from liver imaging or histology in regards to a beneficial effect of metformin on MASLD components in clinical studies [63,64]. Currently there is only one small scale placebo-controlled study with Metformin. In this study, no differences were detected in histological scores of MASH components and fibrosis between both the groups. Also increased adiponectin levels have been reported with metformin treatment [65].

Drugs for MASLD treatment with weight-loss-independent metabolic effects

Peroxisome proliferator-activated receptor (PPAR) agonists:

Due to their pivotal role in hepatic inflammation and fibrogenesis, distinct PPAR variants (α, δ, γ) have been identified as potential pharmacological targets to combat MASLD [66].

PPARα agonists: PPARα agonist action on lipid metabolism is driven by stimulation of hepatic fatty acid transport, lipolysis and peroxisomal as well as mitochondrial β-oxidation [67]. However, pharmacologic targeting of PPARα does not suffice to reduce liver lipid content to a clinically relevant extent [68,69].

PPARγ agonists: PPARγ is highly expressed in white adipose tissues (WAT) and controls non-esterified fatty acid (NEFA) uptake, lipogenesis as well as reduces adipocyte tissue inflammation [66]. In MASH, treatment with Pioglitazone, the most prescribed PPARγ agonistic drug over the last few decades, was repeatedly associated with improvements in liver histology, despite a net weight gain of 2–4 % of body weight [70,71]. A meta-analysis of 8 RCTs in people with biopsy-proven MASH concluded that Pioglitazone was not only associated with the improvement of MASH, but also reversal of fibrosis [72]. However, the risk-benefit ratio of Pioglitazone remains debatable due to the increased risk of hospitalization from heart failure due to fluid retention [73].

Dual/pan-PPAR agonists: The dual α/δ PPAR agonist Elafibranor failed to prove superiority to placebo in a 72-week phase 3 study regarding the primary endpoint MASH resolution without worsening of fibrosis (19 vs. 15 % resolution rate) and also for fibrosis improvement (25 vs 22 %) [74]. Lanifibranor, a pan-PPAR agonist, proved effective for MASLD treatment in a 24-week Phase 2b RCT in people with MASH, but displayed similar side effects like Pioglitazone [75].

Modulators of the mitochondrial pyruvate carrier: MSDC-0602 K, a PPARγ-sparing Pioglitazone derivative, was developed to target mitochondrial pyruvate carriers (MPC) 1 and 2 [76-78]. However, a phase 2b RCT in people with biopsy-confirmed MASH and fibrosis failed to meet the predefined histological endpoints [79].

Fibroblast growth factor 21 (FGF21) analogues: Improvements in dyslipidemia and MASLD have been repeatedly observed in clinical trials with FGF21 analogues [80,81]. Recently published results from phase 2 studies with histological endpoints suggested fibrosis regression in people with MASH-induced cirrhosis with Efruxifermin but also Pegozafermin treatment [81,82], which position FGF21 analogues as a potential future pharmacological treatment option for advanced metabolic liver disease.

Lipogenesis inhibitors: Acetyl-CoA carboxylase (ACC), converts acetyl-CoA to malonyl-CoA and is a rate-limiting step in DNL. During a 12-week RCT in people with MASLD, the ACC inhibitor Firsocostat led to a moderate reduction in steatosis without changes in body weight or glycemia, but adversely caused an increase in plasma triglyceride levels [83]. Another ACC inhibitor, Clesacostat, showed an even more pronounced reduction of liver lipid content in a 16-week RCT but also led to greater increases in plasma triglyceride levels [84]. Aramchol, an inhibitor of stearoyl coenzyme A desaturase 1 (rate-limiting enzyme in the biosynthesis of monounsaturated fatty acids), demonstrated only a numerical benefit over placebo regarding steatosis improvement in a 52-week phase 2 RCT in people with biopsy-confirmed MASH [85]. In contrast, for MASH resolution and fibrosis regression, the drug was superior to placebo.

Combination of drugs: On one hand, drug combinations may increase response rates and effectiveness of treatment, which has so far been limited with monotherapies [86]. A RCT on the combination of GLP-1RA and SGLT2I in people with type 2 Diabetes, showed additive effects on body weight, glycemic status as well as on MASLD progression [87]. The combination of Semaglutide with drugs having weight loss-independent MASH-relieving effects is currently tested in a trial [88]. Semaglutide and Firsocostat or Cilofexor were more effective in reducing steatosis compared to semaglutide alone (8 vs. 10–11 % absolute reduction in liver fat

determined by magnetic resonance imaging) despite similar weight loss [88].

Limitations of the clinical trials: It is already established that the above described MASLD treatment options have reduced other metabolic comorbidities. But evidence on their effectively reducing the number of liver-related events, are still not enough [89]. As fibrosis develops slowly over many years and also reverses slowly [90], this bears the risk that current clinical trials, with most of them including an intervention phase of 12 to 72 months, may not adequately detect the changes in prognosis [91]. This issue has recently been addressed by longer-term follow-up studies, not only for lifestyle interventions but also for drug treatments. Although MASH resolution correlates with fibrosis regression, assessment of MASH components may be flawed by inter-reader variability [92].

Conclusion: Currently, lifestyle modifications aiming at weight loss remain the basis for MASLD treatment due to their favorable effects on metabolic health, although the outcomes may vary from person to person. Metabolic drugs inducing weight loss, especially Incretin mimetics, are valuable tools for achieving MASH resolution but still lack enough evidence of fibrosis regression. Very recently, effective improvement of the different histological MASLD components has been achieved in the clinical trials of drugs which act independently of changes in body weight (PPAR agonists, FGF21 analogues, lipogenesis inhibitors). Apart from MASH resolution and fibrosis regression, the long-term success of MASLD treatment strategies needs complementary evaluation of endpoints in the future studies.

Hanaa Tarek El-Zawawy
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Subclinical hypothyroidism: To treat or not to treat?

Subclinical hypothyroidism (SCH) represents a challenge in clinical practice. SCH is a form of mild hypothyroidism, meanwhile, it poses multiple risks with many adverse clinical consequences.

SCH treatment has been a matter of debate long ago and significant controversy still exists as to whether to treat or not to treat.

The treatment of SCH has been the subject of an enormous amount of work with many published studies. The American Thyroid Association guidelines settled some instances where the treatment of SCH is to be considered.

The treatment of SCH should be individualized given the patient's profile and risk assessment.

Biography:

Hanaa Tarek El-Zawawy is an Endocrinology Consultant at American Hospital Dubai and an Assistant Professor of Endocrinology at Alexandria University Faculty of Medicine where she had her doctorate degree in Endocrinology in May 2015 at the age of 31 years.

El-Zawawy is a member of the Egyptian Society of Endocrinology and Obesity as well as the European Society of Endocrinology. She participates in many national & international conferences and workshops every year.

She authors 18 peer-reviewed publications. She was cited 147 times, H-index=7. Also, she is an active reviewer in many international journals and receives a yearly award from the publisher “WILEY” for her contributions.

Raffaele Pilla
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Therapeutic ketosis and the broad field of applications for the ketogenic diet: Ketone ester applications & clinical updates

It has been recently shown that nutritional ketosis is effective against seizure disorders and various acute/chronic neurological disorders. Physiologically, glucose is the primary metabolic fuel for cells. However, many neurodegenerative disorders have been associated with impaired glucose transport/metabolism and with mitochondrial dysfunction, such as Alzheimer's/Parkinson's disease, general seizure disorders, and traumatic brain injury. Ketone bodies and tricarboxylic acid cycle intermediates represent alternative fuels for the brain and can bypass the rate- limiting steps associated with impaired neuronal glucose metabolism. Therefore, therapeutic ketosis can be considered as a metabolic therapy by providing alternative energy substrates. It has been estimated that the brain derives over 60% of its total energy from ketones when glucose availability is limited. In fact, after prolonged periods of fasting or ketogenic diet (KD), the body utilizes energy obtained from free fatty acids (FFAs) released from adipose tissue. Because the brain is unable to derive significant energy from FFAs, hepatic ketogenesis converts FFAs into ketone bodies-hydroxybutyrate (BHB) and acetoacetate (AcAc)-while a percentage of AcAc spontaneously decarboxylates to acetone. Large quantities of ketone bodies accumulate in the blood through this mechanism. This represents a state of normal physiological ketosis and can be therapeutic. Ketone bodies are transported across the blood-brain barrier by monocarboxylic acid transporters to fuel brain function. Starvation or nutritional ketosis is an essential survival mechanism that ensures metabolic flexibility during prolonged fasting or lack of carbohydrate ingestion. Therapeutic ketosis leads to metabolic adaptations that may improve brain metabolism, restore mitochondrial ATP production, decrease reactive oxygen species production, reduce inflammation, and increase neurotrophic factors' function. It has been shown that KD mimics the effects of fasting and the lack of glucose/insulin signaling, promoting a metabolic shift towards fatty acid utilization. In this work, the author reports a number of successful case reports treated through metabolic ketosis.

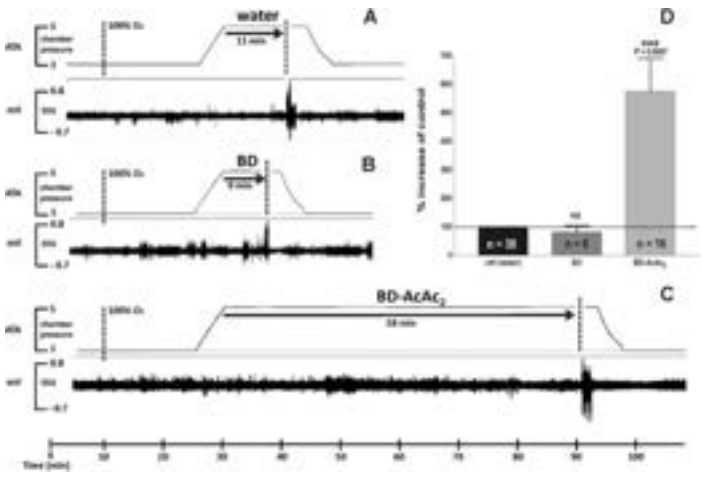

Figure 1: Ketone Ester significantly increased resistance against Central Nervous System Oxygen Toxicity seizures (D'Agostino D.P. et al., 2013 Am J Physiol Regul Integr Comp Physiol. 304(10):R829- 36).

Biography:

Raffaele Pilla, Pharm.D., Ph.D., Doctor Europaeus, received his Master's degree in Pharmacy at G. d'Annunzio University in Chieti-Pescara, Italy in 2005, where he also served internships at the Cell Physiology Laboratory and Molecular Biology Laboratory. Prior, he was an Erasmus Student at Faculté de Pharmacie de Reims in Reims, France. He received his Doctor Europaeus in 2010 from Pitié-Salpétrière Institute in Paris, France. Also in 2010, he received his Ph.D. in Biochemistry,

Physiology, and Pathology of Muscle at G. d'Annunzio University in Chieti-Pescara, Italy. He was hired as a Postdoctoral Scholar in the Department of Pharmacology and Physiology at the University of South Florida in Tampa, on two research grants funded by the Office of Naval Research (US Navy) and Divers' Alert Network. He has written and lectured widely worldwide. He has been involved in ongoing research at the University of South Florida with the use of ketone

Saurabh Gupta
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Clinical Significance of Second to Fourth Digit Ratio in Infertile Males and Its Correlation with Semen Analysis and Testosterone Levels

Infertility and subfertility are synonymous with defective sperm production. Oligospermia and hypogonadism are more prevalent in Indian infertile men than the western counterparts. Although not indicated in every infertile male patient it adds to the diagnosis and helps in directed management. This is a novel study targeting the finger length and digit ratio in second or index (2D) and fourth or ring (4D) finger of both the hands in a group of 51 infertile males with oligospermia. Hormonal assessment including serum total testosterone (T) was also done in them which was later correlated with both the 2D:4D ratio and severity of oligospermia. Results indicated significant differences in digit ratio of right vs left hands in both the cohorts and also amongst the patients with low and normal T Patients with low sperm counts and low T levels had significantly low ratio in right compared to left hand. Decline in semen quality including sperm counts and progressive motility was seen in patients with low testosterone. Thus, measurement of digit length and ratio is a cost effective and time saving method for indirect estimation of androgenisation of male patients but need further validation by large scale studies.

Biography:

Dr. Saurabh Gupta has completed his MBBS at the age of 25 years from BJ Medical College, Gujarat University and MD Medicine from Safdarjung Hospital, Delhi in 2016. He pursued his interest in Endocrinology thereafter and attained Subspeciality Certification from MRCP in 2020. He is the currently Associate Professor and Head Endocrinology department in Pacific Medical University, Udaipur, Rajasthan, India.. He has published many papers in reputed journals and has been serving as expert reviewer in reputed journals.

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