5TH ANNIVERSARY of Endocrinology, Diabetes & Metabolism Hong Kong
EDM HK Annual Meeting 29 – 30 October 2022
Hong Kong Convention and Exhibition Centre
Programme Book
In the treatment of patients with type 2 diabetes and established CV disease receiving standard of care,†‡§ CV death can strike at any time

**BATTLE CV DEATH NOW MORE THAN EVER**$ 

**JARDIANCE demonstrated 38% RRR in CV death**

- Established HbA1c efficacy
- Demonstrated safety profile
- Convenient, once-daily oral dosing

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**Jardiance** (empagliflozin)

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**THE ONLY BAD WITH CV INDICATION**

Jardiance is indicated in TZDM patients and established cardiovascular disease to reduce the risk of cardiovascular death.

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Dear Colleagues,

On behalf of the Organizing Committee, I welcome you all to the Annual Meeting of Endocrinology, Diabetes & Metabolism Hong Kong (EDM HK), jointly organized by the Division of Endocrinology, Diabetes and Metabolism, Department of Medicine, The University of Hong Kong, as well as KK Leung Diabetes Centre and Osteoporosis Centre of Queen Mary Hospital. We are honoured by your presence on this particular occasion, celebrating the 5th Anniversary of EDM HK.

This exciting and inspiring 2-day scientific programme comprises state-of-the-art lectures on various important endocrine disorders: diabetes, osteoporosis, thyroid and many others. Amidst the COVID-19 pandemic, the Symposium on ‘COVID-19 and Endocrinology’ aims to deliver up-to-date summaries of research in this rapidly evolving field. Furthermore, our first ‘EDM HK Cases of the Year’ features young fellows sharing interesting cases, which will shed light on our clinical practice.

We would like to express our sincere gratitude to all our sponsors, chairpersons and speakers for their continuous support and contributions to this Meeting. We hope that you will find it fruitful and rewarding.

Dr. David Lui
Chairman
Organizing Committee
EDM HK 2022
Chairman
Dr. David TW Lui

Members
Professor Karen SL Lam
Dr. WS Chow
Dr. TP Ip
Dr. Alan CH Lee
Dr. Johnny YC Chang
Dr. Lawrence CK Tang
Ms. Karen KC Wong
Ms. SK Leung
Ms. Tina WT Lau

Professor Kathryn CB Tan
Dr. YC Woo
Dr. Paul CH Lee
Dr. Eunice KH Leung
Dr. Charlene SL Woo

Ms. Amy SW Yee
Ms. Connie HN Loong
Ms. Michelle HY Lee
## ACCREDITATIONS

### CME

<table>
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<tr>
<th>Organization</th>
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<th>29 October</th>
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<td>Hong Kong College of Paediatricians</td>
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### CNE

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<td>Hospital Authority Hong Kong West Cluster</td>
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<td>時間</td>
<td>會議室 S226 - S227</td>
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</table>
| 10:00 – 10:40 | (一): 強筋健骨・飲食有方  
陳錦華中醫師  
黃杏雯營養師 |
| 10:40 – 11:05 | (二): 監測血糖全攻略  
李巧宜護士  
王家緻護士 |
| 11:05 – 11:30 | (三): 尋「藥」記  
蔡祥熙醫生  
伍超明醫生 |
| 11:30 – 11:45 | 問題環節                                                      |

Public lectures will be conducted in Cantonese
## 29 October 2022 (Saturday)

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<tr>
<th>Time</th>
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| **Lecture (1)** (Sponsored by Amgen)  
Chairperson: Dr. TP Ip | The importance of sequencing therapy after osteo-anabolic agents  
Professor Michael McClung (USA) |  |
| 13:00 – 13:40 |  |  |
| 13:40 – 13:45 | Q & A |  |
| 13:45 – 13:55 | Opening Ceremony |  |
| **Lecture (2)** (Sponsored by AstraZeneca)  
Chairperson: Professor Karen Lam | A roadmap for optimizing the care and outcomes of diabetes patient with kidney disease  
Professor Peter Rossing (Denmark) |  |
| 13:55 – 14:40 |  |  |
| 14:40 – 14:45 | Q & A |  |
| **Symposium (1A)**  
Chairpersons: Dr. WS Chow and Professor Brian Lang |  |  |
| 14:45 – 15:05 | Approach to thyroid nodules  
Dr. Matrix Fung (Hong Kong) | Classification of PitNET: updates in 2022  
Dr. Chariene Woo (Hong Kong) |
| 15:05 – 15:25 | Fatty liver disease: a diabetologist’s perspective  
Dr. CH Lee (Hong Kong) | Congenital adrenal hyperplasia: management and transition to adulthood  
Dr. Gloria Pang (Hong Kong) |
| 15:25 – 15:45 | The tales of atypical fractures  
Dr. YC Woo (Hong Kong) | Updates on the management of PCOS  
Dr. Raymond Li (Hong Kong) |
| 15:45 – 16:00 | Q & A | Q & A |
| 16:00 – 16:30 | Break |  |
| **Lecture (3)** (Sponsored by Eli Lilly)  
Chairperson: Dr. John Ma | Considerations when choosing between type 2 diabetes therapy: the role of once-weekly GLP-1 RA  
Professor Michael Cummings (UK) |  |
| 16:30 – 17:10 |  |  |
| 17:10 – 17:15 | Q & A |  |
| **Lecture (4)** (Sponsored by Novartis)  
Chairperson: Professor Kathryn Tan | Managing familial hypercholesterolemia: achieving optimal treatment targets  
Professor Frederick Raal (South Africa) |  |
| 17:15 – 17:50 |  |  |
| 17:50 – 17:55 | Q & A |  |
### SCIENTIFIC PROGRAMME

#### 30 October 2022 (Sunday)

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<td><strong>Plenary Lecture (1)</strong></td>
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<td>Chairperson: Dr. Alan Lee</td>
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</table>
| 09:00 – 09:35 | **Advances in the management of Graves’ disease**  
*Professor Marius Stan (USA)* |
| 09:35 – 09:40 | **Q & A**                         |
| 09:40 – 10:20 | **Lecture (5) (Sponsored by Boehringer Ingelheim)**  
Chairperson: Dr. YY Ho |
| 10:20 – 10:25 | **Q & A**                         |
| 10:25 – 10:55 | **Break**                         |

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<tr>
<th>Time</th>
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<tr>
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<td><strong>Symposium (2A)</strong></td>
<td><strong>Symposium (2B)</strong></td>
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<tr>
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<td>Chairpersons: Dr. Nicole Chau and Professor Alice Kong</td>
<td>Chairpersons: Dr. Emmy Lau and Dr. Jenny Leung</td>
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</table>
| 10:55 – 11:15 | COVID-19 and diabetes  
*Professor Andrea Luk (Hong Kong)* | Calcium: when it gets too high and too low  
*Dr. Joanne Lam (Hong Kong)* |
| 11:15 – 11:35 | COVID-19 and thyroid  
*Dr. David Lui (Hong Kong)* | Role of combination T4 and T3 replacement in the management of hypothyroid patients  
*Dr. Alan Lee (Hong Kong)* |
| 11:35 – 11:45 | Q & A | Q & A |

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<tr>
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| 11:45 – 12:25 | **Lecture (6) (Sponsored by Otsuka)**  
Chairperson: Dr. SC Tiu |
| 12:25 – 12:30 | **Q & A**                         |
| 12:30 – 13:30 | **Lunch Break**                   |

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| 13:30 – 14:10 | **Lecture (7) (Sponsored by Novo Nordisk)**  
Chairperson: Professor Rosie Young |
| 14:10 – 14:15 | **Q & A**                         |
### 30 October 2022 (Sunday)

| Time          | Room S221 | **Lecture (8) (Sponsored by Sanofi)**  
|---------------|-----------|---------------------------------------------|
| 14:15 – 14:50 |           | *Advancing therapy using fixed-ratio combination of basal insulin and GLP-1 RA in suboptimally controlled basal insulin-treated type 2 diabetes*  
|               |           | *Chairperson: Dr. Michele Yuen*  
| 14:50 – 14:55 |           | Q & A  
| 14:55 – 15:25 | Break     | **Lecture (9) (Sponsored by Bayer)**  
|               |           | *New approaches to delay CKD progression in diabetes: battling inflammation and fibrosis*  
|               |           | *Chairperson: Dr. Grace Kam*  
| 15:25 – 16:05 |           | Q & A  
| 16:05 – 16:10 |           | **Plenary Lecture (2)**  
|               |           | *Bone fragility in diabetes*  
|               |           | *Chairperson: Dr. David Lui*  
| 16:10 – 16:50 |           | **EDM HK Cases of the Year**  
|               |           | *Chairpersons: Dr. Doris Chan, Dr. Vincent Fok, Dr. Raymond Hue and Dr. CL Wong*  
| 16:50 – 17:05 |           | *Hypertensive urgency in a young man revealed an unexpected hereditary syndrome*  
|               |           | *Chairperson: Dr. Ingrid Mak (Hong Kong)*  
| 17:05 – 17:20 |           | *Atypical metatarsal fracture in a Chinese post-menopausal woman with osteoporosis on long-term denosumab*  
|               |           | *Chairperson: Mr. Andy Kan (Hong Kong)*  
| 17:20 – 17:35 |           | *Cushing's syndrome secondary to pro-opiomelanocortin (POMC) secretion from a pancreatic yolk sac tumour in an adult*  
|               |           | *Chairperson: Dr. Johnny Chang (Hong Kong)*  
| 17:35 – 17:50 |           | *Primary pigmented nodular adrenocortical Disease (PPNAD) - sequential or bilateral adrenalectomy?*  
|               |           | *Chairperson: Dr. KY Wong (Hong Kong)*  
| 17:50 – 17:55 |           | Closing Remarks |
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<td>Amgen Hong Kong Limited</td>
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LIST OF OVERSEAS SPEAKERS

**Professor Michael Cummings**
Honorary Professor  
Department of Diabetes & Endocrinology  
Portsmouth Hospitals NHS Trust  
Queen Alexandra Hospital  
UK

**Professor David Matthews**
Emeritus Professor of Diabetes Medicine  
Department of Medicine  
University of Oxford  
UK

**Professor Jennifer Green**
Associate Professor  
Department of Medicine  
Duke University  
USA

**Professor Per-Henrik Groop**
Chairman  
Department of Internal Medicine  
University of Helsinki  
Finland

**Professor Francois Raal**
Head  
Division of Endocrinology & Metabolism  
University of the Witwatersrand  
South Africa

**Professor Marius Stan**
Consultant  
Department of Internal Medicine  
Mayo Clinic  
USA

**Professor Serge Ferrari**
Chairman  
Department of Medicine  
Geneva University Hospital  
Switzerland

**Professor Michael McClung**
Founding Director  
Oregon Osteoporosis Center  
USA

**Professor Michael Matthew**
Emeritus Professor of Diabetes Medicine  
Department of Medicine  
University of Oxford  
UK

**Professor Peter Rossing**
Head of Complications Research  
Steno Diabetes Center Copenhagen  
Denmark

**Professor Joseph Verbalis**
Chief  
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Georgetown University  
USA
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Associate Consultant
Department of Medicine & Geriatrics
Pok Oi Hospital

Dr. Nicole Chau
Associate Consultant
Department of Medicine & Geriatrics
Princess Margaret Hospital

Dr. CH Choi
Deputy Chief of Service
(Manpower & Training)
Department of Medicine
Queen Elizabeth Hospital

Dr. WS Chow
Consultant
Department of Medicine
Queen Mary Hospital

Dr. Vincent Fok
Associate Consultant
Department of Medicine & Geriatrics
Caritas Medical Centre

Dr. Matrix Fung
Endocrine Surgeon
Division of Endocrine Surgery
The University of Hong Kong

Dr. YY Ho
Consultant
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Tuen Mun Hospital

Dr. Raymond Hue
Associate Consultant
Department of Medicine
Pamela Youde Nethersole Eastern Hospital

Dr. TP Ip
Consultant
Department of Medicine
Tung Wah Hospital

Dr. Grace Kam
Consultant
Department of Medicine & Geriatrics
United Christian Hospital

Professor Alice Kong
Professor
Department of Medicine & Therapeutics
The Chinese University of Hong Kong

Dr. Joanne Lam
Honorary Clinical Assistant Professor
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The University of Hong Kong

Professor Karen Lam
Chair Professor
Department of Medicine
The University of Hong Kong

Professor Brian Lang
Clinical Professor
Department of Surgery
The University of Hong Kong

Dr. Emmy Lau
Consultant
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Pamela Youde Nethersole Eastern Hospital

Dr. Alan Lee
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Dr. CH Lee
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Kwong Wah Hospital

Ms. Michelle Lee
Advanced Practice Nurse
Department of Medicine
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Dr. Jenny Leung
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Ruttonjee & Tang Shiu Kin Hospitals
Dr. Raymond Li  
Clinical Associate Professor  
Department of Obstetrics & Gynaecology  
The University of Hong Kong

Dr. David Lui  
Clinical Assistant Professor  
Department of Medicine  
The University of Hong Kong

Professor Andrea Luk  
Professor  
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The Chinese University of Hong Kong

Dr. John Ma  
Specialist in Endocrinology, Diabetes & Metabolism  
Private Practice

Dr. Ingrid Mak  
Associate Consultant  
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Queen Elizabeth Hospital

Dr. Jason Ng  
Associate Consultant  
Department of Medicine  
Queen Elizabeth Hospital

Dr. Gloria Pang  
Associate Consultant  
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Hong Kong Children’s Hospital

Professor Kathryn Tan  
Clinical Professor  
Department of Medicine  
The University of Hong Kong

Dr. SC Tiu  
Honorary Consultant  
Department of Medicine  
Queen Elizabeth Hospital

Dr. Joanna Tung  
Associate Consultant  
Department of Paediatrics & Adolescent Medicine  
Hong Kong Children’s Hospital

Ms. Carman Wong  
Registered Dietitian in Canada  
The Jockey Club School of Public Health & Primary Care  
The Chinese University of Hong Kong

Dr. CL Wong  
Specialist in Endocrinology & Diabetes  
Private Practice

Ms. Karen Wong  
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Department of Medicine  
Queen Mary Hospital

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Resident  
Department of Medicine  
Queen Mary Hospital

Dr. YC Woo  
Consultant  
Department of Medicine  
Queen Mary Hospital

Professor Rosie Young  
Emeritus Professor  
Department of Medicine  
The University of Hong Kong

Dr. Michele Yuen  
Honorary Clinical Assistant Professor  
Department of Medicine  
The University of Hong Kong
The importance of sequencing therapy after osteo-anabolic agents

Professor Michael McClung

Founding Director
Oregon Osteoporosis Center
USA

Osteoporosis is a chronic illness characterized by low bone mass and deterioration of bone microarchitecture that weakens the skeleton and predisposes to fractures. None of our available therapies cures osteoporosis, and the skeletal benefits of treatment dissipate when treatment is stopped. Consequently, long-term treatment is required and often involves the use of multiple drugs in various sequences to optimize treatment response. Osteoanabolic, or bone-building drugs restore bone structure as well as bone mass. Teriparatide, a synthetic fragment of parathyroid hormone (PTH), activates remodeling-based bone formation and also stimulates bone resorption. Romosozumab, an anti-sclerostin antibody, stimulates both modeling- and remodeling-based bone formation and reduces osteoclastic bone resorption. Both agents have been shown to be more effective than bisphosphonates in increasing bone mineral density (BMD) and reducing fracture risk in patients at high risk of fracture. These studies led to the approval of teriparatide and romosozumab as treatments for women with postmenopausal osteoporosis at high risk of fracture.

Safety concerns with teriparatide include hypercalcemia and orthostatic hypotension, most commonly after the first dose. Romosozumab is associated with mild injection site reactions and a risk of serious adverse cardiovascular (CV) events compared to alendronate but not to placebo. While the explanation for this disparity in CV risk is still unknown, those findings led to the warning about the potential risk of CV outcomes with romosozumab and the recommendation that romosozumab not be used in patients at high CV risk.

Because of the waning of the anabolic effects of these treatments with continued use, courses of osteoanabolic therapy are from 12 - 24 months, after which transition to an anti-remodeling drug, either a bisphosphonate or denosumab, is required to maintain or improve BMD. In addition, the fracture protection effects accomplished with a course of romosozumab therapy persist for at least two years after transition to the anti-remodeling drug. The increase in BMD and the reduction in fracture risk is greater with 12 months of romosozumab followed by 12 months of denosumab compared to 24 months of denosumab therapy. At this time, there are no data about the effects of switching from romosozumab to teriparatide, to either a bisphosphonate or to denosumab is required to preserve the BMD and fracture protection benefits of the osteoanabolic agent.

The decision to switch from an osteoanabolic agent to either a bisphosphonate or to denosumab is not informed by data from randomized trials. As a result, that decision has to be made on the basis of clinical considerations including the presence of contraindications to either treatment and the patient’s preference. Appreciating that on-treatment hip BMD correlates with current fracture risk and that the increase in BMD after osteoanabolic therapies appears to be greater with denosumab compared to alendronate, one might choose denosumab as the follow-on therapy for patients whose hip BMD was still in the osteoporosis range. Another consideration is that the increase in BMD with a second course of osteoanabolic therapy is larger in a patient taking alendronate than in those taking denosumab. Thus, if a second course of bone-building therapy is contemplated, therapy with alendronate for 12 months rather than denosumab might be the choice after first course of anabolic therapy.

The BMD response to osteoanabolic agents is greater in treatment-naïve patients than in those treated with any osteoporosis medication. These findings emphasize the importance of using drugs in the optimal sequence and have led several recent society guidelines to recommend that osteoanabolic therapies should be the initial therapy for patients at very high risk of fracture.
A roadmap for optimizing the care and outcomes of diabetes patient with kidney disease

**Professor Peter Rossing**  
*Head of Complications Research  
Steno Diabetes Center Copenhagen  
Denmark*

Diabetes is the most common cause of kidney failure in the Western world. Chronic kidney disease (CKD) in diabetes is a condition characterized by a gradual increase in urinary albumin excretion, blood pressure levels and cardiovascular risk, and declining glomerular filtration rate (GFR), which can progress to kidney failure. Chronic kidney disease is common among patients with diabetes, and it develops in approximately 30% of the patients with type 1 diabetes (T1D) and 50% of those with type 2 diabetes (T2D), but in many this is diagnosed late because of lack of symptoms. Patients with diabetes should be screened for CKD annually but this is often not done. Screening should include both albuminuria measurements and estimates of GFR. Multiple factors are associated with CKD in diabetes, and patients with diabetes often require multiple therapies aimed at prevention of progressive CKD and its associated co-morbidities and mortality. Management of cardiorenal risk factors, including lifestyle modifications (diet, exercise, and stop smoking), glucose, blood pressure and lipid control, use of agents blocking the renin angiotensin aldosterone system and use of SGLT2 inhibitors in patients with T2D and other agents with proven renal or cardiovascular benefit are the cornerstones of therapy. RAS inhibition has been standard of care for 20 years but is still not always implemented. New options is SGLT2 inhibition, initially introduced to lower glucose, but now dapagliflozin is indicated for CKD treatment in type 2 diabetes, based on DAPA-CKD study and DECLARE. Early intervention is important to optimize benefit, and this is now recommended in many current guidelines including the ADA 2022, EASD – ADA 2022, and the KDIGO 2022 guideline on management of diabetes and CKD.
Approach to thyroid nodules

Dr. Matrix Fung

Endocrine Surgeon
Division of Endocrine Surgery
The University of Hong Kong

Thyroid nodules are common, with a reported prevalence of more than 50% in autopsy studies. Majority of thyroid nodules are benign. All patients with clinically detectable thyroid nodules should be evaluated with thyroid function tests and thyroid ultrasonography (USG). Standardized evaluation protocols have been established to estimate the risk of malignancy based on sonographic features and size of nodules, and hence determine the need of fine needle aspiration biopsy (FNA) and further management. The Bethesda System for Reporting Thyroid Cytopathology is the current international standard for thyroid FNA reports. The Bethesda system could estimate the risk of malignancy and hence guide management. Surgery is the main treatment for malignant thyroid nodules. For benign nodules, conventional management options range from observation to surgery. Recently, there are huge interests and advances in ablation strategies for thyroid nodules, such as radiofrequency ablation, microwave ablation or high-intensity focused ultrasound. Ablative treatment have the advantage of minimal to no scars, yet careful case selection is crucial to achieve satisfactory outcomes. Management of thyroid nodules should be individualized, taking into account the expectation of the patient and the expertise available.
Fatty liver disease: a diabetologist’s perspective

Dr. CH Lee

Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

The global prevalence of fatty liver disease (FLD) is rising along with the epidemics of diabesity. Over 70% of individuals with type 2 diabetes (T2D) have fatty liver. The relationship between fatty liver and T2D is mutually detrimental. Metabolic dysfunction-associated fatty liver disease (MAFLD) is a new entity recently proposed by a panel of international experts. Theoretically, all patients with T2D and FLD belongs to the MAFLD population. This talk will provide an overview of the latest evidence that support FLD as an emerging diabetic complication of increasing importance, and to present the current recommendations, focusing on the assessment and therapeutic strategies, on the management of FLD among T2D patients.
The tales of atypical fractures

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Atypical fracture of the femur (AFF) has been reported as a complication of long-term bone turnover suppression since 2005. Initially, they are linked to long-term bisphosphonate therapy and subsequently have also been reported following denosumab therapy. Fear of this side effect remains one of the hurdles physicians face while persuading patients to receive osteoporosis treatment. While the relative risk of AFF with bisphosphonate therapy is increased, the absolute risk remains very low, ranging from 3.2 to 50 cases per 100,000 person-years. It is known that anti-resorptive therapy needs to be stopped if an AFF is identified. However, fracture prevention in osteoporotic patients after sustaining AFF remains challenging.

Will the lessons from the tales of atypical fractures in the past 17 years highlight us in anti-osteoporosis management strategies?
Classification of PitNET: updates in 2022

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Pituitary neuroendocrine tumour (PitNET) is one of the top three commonest brain tumours. Its classification has evolved over time, from the 2004 classification based on clinical phenotype, to the 2017 advocation on a lineage-restricted classification. The role of transcription factors SF1, TPIT, Pit1 in the differentiation of distinct adenohypophyseal lineage was highlighted in the 2017 classification as follows: gonadotroph tumours (SF1 positive), corticotroph tumours (TPIT positive), null cell tumours (transcription factor and hormone negative) as well as lactotroph, somatotroph and thyrotroph tumours characterized by Pit1 positivity and respective prolactin, growth hormone and thyroid-stimulating hormone positivity.

In 2022, the International Agency for Research on Cancer published the fifth edition of the World Health Organization (WHO) Endocrine Organ Tumour Classification, which further consolidated the role of transcription factors in the classification of PitNET. Entities included SF1-lineage, TPIT-lineage, Pit1-lineage PitNET and PitNET without distinct lineages.

The significance of such classification of PitNET is highlighted by increasing evidence revealing differences in tumour behaviour of individual PitNET. SF1-lineage PitNET are usually indolent, with higher complete resection rates and less tumour progression or recurrence; whilst TPIT-lineage PitNET (especially silent corticotroph adenoma) runs an aggressive course with propensity for invasion and recurrence. Looking ahead, precise identification of tumour subtypes may aid future research on potential drug targets, a personalized approach to early adjuvant therapy and individualized radiological surveillance strategies.
Congenital adrenal hyperplasia: management and transition to adulthood

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Congenital adrenal hyperplasia (CAH) is a group of inborn errors of steroid metabolism, the commonest form of which is 21 hydroxylase deficiency (21OHD). Cortisol production from the adrenal cortex is inadequate, leading to hyper secretion of corticotropin and adrenocorticotropic hormone from the hypothalamus and pituitary gland, resulting in adrenal hyperplasia with structural disruption of the adrenal cortex and medulla. By-products of this hyperactive axis results in overproduction of progestins and androstenedione, which is converted to testosterone and dihydrotestosterone and results in post natal androgen excess. Around 75% of patients who present in the neonatal period would also have hypoaldosteronism which together with hypocortisolism can lead to life threatening hyponatraemic dehydration and shock.

Combating issues of classic CAH, including gender ambiguity, salt wasting crises, lifelong requirement of medications, and discussions of urogenital surgeries require full on parental engagement since the neonatal stage. Dedication from all parties to ensure adherence to a carefully adjusted treatment regime is important for the physical and mental well being for the young patient. As the child progresses into adolescence, the focus of management shifts from optimising growth and sexual maturity to managing long term complications, fertility and family planning. This talk will explore the management issues as the child with CAH progresses into early adulthood, and highlights importance of transitional care in which the young adult is equipped to take on major responsibility for his/her condition.
Updates on the management of PCOS

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Polycystic ovary syndrome (PCOS) is a common reproductive endocrine disorder. Currently, it is diagnosed by the presence of two out of the following three criteria: oligo- and/or anovulation, clinical and/or biochemical hyperandrogenism, and ultrasound features of polycystic ovaries, with the exclusion of other aetiologies.

Physical and health implications of PCOS including menstrual irregularity, anovulatory subfertility, hyperandrogenic symptoms and metabolic disturbances like hypertension, diabetes mellitus, hyperlipidaemia and obesity. Obese patients should be advised to reduce weight, which can improve spontaneous or induced ovulation and hence fertility, reduce obstetric risks, as well as improve other metabolic profiles in general. Weight reduction should be achieved by diet and exercise. In the long term, regular monitoring of blood pressure, body weight, blood sugar (by an oral glucose tolerance test) and lipids is advised.

Chronic anovulation is associated with an increased risk of endometrial hyperplasia and cancer due to unopposed oestrogen exposure. Combined oral contraceptive (COC) pills can provide good cycle control, protects the endometrium, provides contraception if there is no fertility wish, and lowers free androgen (by enhancing SHBG synthesis) hence ameliorating hyperandrogenic symptoms. Alternatively, periodic progestogen treatment can be used to induce withdrawal bleeding in case of amenorrhoea for more than 2-3 months.

Acne and hirsutism can be ameliorated by cosmetic measures, dermatological therapy or COC pill; occasionally a more potent anti-androgen may be required. Treatment of hirsutism may take 6 months or more to show appreciable effects.

Letrozole or clomiphene citrate can be used as the first-line therapy for ovulation induction, with the former being more effective. Monitoring by ultrasound is advisable, at least in the first treatment cycle, with dose adjustment when needed. The optimal dose can be maintained for at least 6 ovulatory cycles. Multiple pregnancies have been reported in 8-10% of clomiphene treatment cycles. Metformin alone is less effective as first-line fertility treatment, but may serve as co-treatment with clomiphene for those who are obese or clomiphene-resistant. Laparoscopic ovarian drilling or gonadotrophin induction can be a second-line treatment for clomiphene resistance. In vitro fertilisation can be reserved for those who failed ovulation induction as above, or for those who have other concurrent indications for it.
Considerations when choosing between type 2 diabetes therapy: the role of once-weekly GLP-1 RA

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UK

Type 2 diabetes is associated with the triad of insulin resistance, weight gain and hyperglycaemia that increases microvascular and to a lesser extent cardiovascular (CV) risk. Newer therapies have needed to address these issues since traditional glucose lowering therapies did not reduce CV risk and many were associated with weight gain.

GLP-1 RAs offer a newer alternative approach that can simultaneously impact upon weight loss and reduce CV risk alongside their glucose lowering properties with low intrinsic risk of hypoglycaemia. GLP-1 RAs with proven cardiovascular benefits is now recommended for type 2 diabetes patients with established atherosclerotic cardiovascular disease or with indicators of high risk for cardiovascular disease (target organ damage or multiple risk factors) by the latest European Association for the Study of Diabetes (EASD) guidelines treatment algorithm. The recent REWIND study has shown for the first time that a GLP1-RA can reduce CV risk in primary prevention as well as individuals with established CV disease.

Compared to daily injections, once weekly GLP-1 RA such as dulaglutide is an appealing treatment options owing to their reduced dosing frequency and ease of use, which might help improve treatment adherence and persistence. This presentation will provide an overview of clinical trial evidence and real world data regarding the role and practical use of GLP-1 RA for type 2 diabetes management.
Managing familial hypercholesterolemia: achieving optimal treatment targets

Professor Frederick Raal
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South Africa

Severe familial hypercholesterolemia (FH) remains a difficult condition to treat. As a result of markedly elevated LDL-cholesterol levels from birth, subjects with severe FH suffer from accelerated, premature atherosclerotic cardiovascular disease often resulting in premature death. However, over the past three decades there have been remarkable advances in treatment for this condition. Lipid lowering therapies which act mainly by upregulating LDL receptor function, such as high intensity statin, and ezetimibe form the backbone of treatment but this combination is not sufficient to attain LDL-cholesterol targets in the majority of FH subjects. The addition of PCSK9-inhibitor therapy has revolutionized the treatment of severe FH. Monoclonal antibodies directed against PCSK9 are effective and have been shown to reduce the cardiovascular event rate in large outcome studies, but this therapy needs to be administered every two weeks or monthly. Inclisiran, a small interfering double stranded RNA (siRNA) harnesses the natural process of RNA interference and inhibits the production of PCSK9 by hepatocytes “(turns off the tap)” and because of its long duration of action only needs to be administered subcutaneously 6 monthly. Overcoming the challenges of severe FH has been a long and difficult journey, but with the treatment options now available, the future for severe FH looks bright.
Advances in the management of Graves’ disease

Professor Marius Stan

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The management of Graves disease (GD) is undergoing a reevaluation. We continue to use long established therapies - radioactive iodine (RAI), surgery and antithyroid drugs (ATD) but we are looking differently at their outcomes and the long term implications of each therapy.

We’re seeing an increase in use of ATD as primary therapy over the last 10 years, in parallel with a decrease in the use of RAI. At the same time we’re seeing an increased use of long-term ATD (beyond 24 months of therapy) if the initial therapy has not been followed by remission. Both these trends are very likely related to concerns about quality of life in patients with hypothyroidism, as well as concerns about the potential risks associated with RAI. The utilisation of surgery has remained stable over the years.

We are also seeing major advances in regard to the major complication associated with Graves disease, thyroid eye disease (TED) or Graves orbitopathy (GO). Teprotumumab has become an effective therapy for patients with active and moderately-severe disease combined with significant proptosis. The place of teprotumumab in the algorithm of TED management is currently being defined by a number of professional societies.

The success of teprotumumab therapy along with advances in the technical ability to create monoclonal antibodies against various targets, is spurring a number of pharmaceutical companies to pursue alternative therapies for GD and TED. It is very likely that our future approach against these entities will be much more specific towards their actual pathophysiology. Hopefully these approaches will also be able to avoid long term hypothyroidism as a consequence of GD therapy.
The cardiorenal side of SGLT2 inhibitors: exploring advances from type 2 diabetes to heart failure

Professor Jennifer Green  
Associate Professor  
Department of Medicine  
Duke University  
USA

Heart failure is a widespread condition affecting 60 million people worldwide and expected to increase as the population ages. There is currently a high unmet need in the treatment of heart failure, as approximately half of all those diagnosed are expected to die within five years. Diabetes, cardiovascular disease, and chronic kidney disease (CKD) are often intercorrelated, suggesting the importance of cardio-renal-metabolic approach in managing these patients. Sodium-glucose cotransporter 2 (SGLT2) inhibitors, are oral anti-hyperglycemic agents that have shown cardiorenal benefits in patients with type 2 diabetes mellitus (T2DM), and results are replicated in more recent trials in heart failure population.

In EMPA-REG OUTCOME trial, empagliflozin demonstrated significant benefits in 3-point MACE, cardiovascular (CV) death, hospitalization for heart failure and all-cause mortality. In addition to the CV benefit, empagliflozin also reduced the risk of developing incident or worsening nephropathy.

In two trials targeting on patient with heart failure with reduced ejection fraction (EMPEROR-Reduced) and those with preserved ejection fraction (EMPEROR-Preserved), empagliflozin demonstrated significant risk reduction in the primary endpoints of hospitalization for heart failure or CV death versus standard of care, regardless of diabetic status or left ventricular ejection fraction. Added that, both trials showed a slower decline in kidney function in patients on top of standard of care.

The largest and broadest SGLT2 inhibitor trial in CKD to-date, EMPA-KIDNEY, has been recommended to stop early due to the evidence that empagliflozin is more effective than the placebo in reducing risk of primary endpoint.

In this lecture, clinical evidence along the cardio-renal-metabolism axis, and recent international guideline recommendations on the use of SGLT2 inhibitors in optimal heart failure treatment will be discussed.
COVID-19 and diabetes

Professor Andrea Luk

Professor
Department of Medicine & Therapeutics
The Chinese University of Hong Kong

Coronavirus disease 2019 (COVID-19) caused by severe respiratory syndrome coronavirus 2 (SARS-CoV-2) has evolved into a global catastrophe affecting over 610 million people with a death toll of more than 6 million. People with diabetes are more vulnerable to severe complications and have a two-fold excess risk of death from acute COVID-19, with the hazards being higher in type 1 than type 2 diabetes, in younger than older age groups, when compared with people without diabetes. There is growing recognition that COVID-19 has health manifestations beyond the respiratory system. Past exposure to SARS-CoV-2 may worsen metabolic control in people with diabetes or cause dysregulation of glucose metabolism in those without diabetes. Notably, a rise in diabetic ketoacidosis during the pandemic raised concerns that SARS-CoV-2 may induce diabetes. Large epidemiological studies reported an increase in burden of incident diabetes which persisted for up to 12 months after the initial viral exposure. Some proposed direct effects of viral infection on insulin function, insulin secretion and autoimmunity targeting pancreatic beta-cell islets, although these associations have not been fully substantiated. Changes in eating habits and physical behaviour as well as psychological stress may also add to the risk of developing diabetes. Lastly, interruptions of health services delivery and delay in seeking medical care due to concerns about contagion will adversely affect disease control in people with chronic conditions including diabetes, with long-term sequelae. The full health impact of COVID-19 on our population is yet to be determined, and ongoing monitoring and review are required as new strains emerge, as herd immunity and vaccination coverage builds, and as our societal behaviour moves to a new state.
COVID-19 and thyroid

Dr. David Lui  
*Clinical Assistant Professor*  
*Department of Medicine*  
*The University of Hong Kong*

Now entering the third year of the COVID-19 pandemic, more than 600 million people worldwide have been infected by COVID-19, resulting in more than 6.5 million deaths. COVID-19 is associated with both pulmonary and extra-pulmonary manifestations. Case reports of autoimmune thyroid disorders and subacute thyroiditis following COVID-19 infection have suggested the potential of SARS-CoV-2 to cause thyroid dysfunction.

Local data showed that abnormal thyroid function occurred in around 15% of COVID-19 patients, with the commonest pattern being non-thyroidal illness syndrome, which in turn carries prognostic significance in COVID-19. As the number of COVID-19 survivors is growing, long COVID is an emerging public health issue. While thyroid function and autoimmunity do not appear to play a significant role in manifestations of long COVID, interferon beta-1b, which has been used in COVID-19 treatment, is associated with modest changes in thyroid autoimmunity. Based on all the existing evidence, recommendations regarding thyroid evaluation post-acute COVID-19 will be discussed.

COVID-19 vaccination has demonstrated efficacy in protecting against COVID-19-related adverse outcomes. Cases of thyroid dysfunction following COVID-19 vaccination have raised concerns, especially among people with thyroid disorders. Nonetheless, case reports do not establish causality. Using local population-based registry and cohort studies, we have evaluated the thyroid-specific outcomes among COVID-19 vaccine recipients with or without known thyroid dysfunction, providing reassuring data to support COVID-19 vaccination.
Calcium: when it gets too high and too low

Dr. Joanne Lam
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Department of Medicine
The University of Hong Kong

Calcium is required for the proper functioning of muscle contraction, nerve conduction, hormone release, and blood coagulation. Calcium metabolism is regulated by concentrations of circulating PTH, vitamin D, and, to a lesser extent, calcitonin. Disorders of calcium metabolism are frequently encountered in clinical practice. In this talk, the clinical manifestations, etiology, diagnostic approach and management for hypercalcemia and hypocalcemia will be presented.
Role of combination T4 and T3 replacement in the management of hypothyroid patients

Dr. Alan Lee  
Associate Consultant  
Department of Medicine  
Queen Mary Hospital

Levothyroxine monotherapy (LT4), titrated to maintain thyroid stimulating hormone (TSH) within an euthyroid reference range, represents the standard treatment of primary hypothyroidism due to various aetiologies. For most patients this well-established approach is successful in resolving symptoms of hypothyroidism, and in preserving long-term outcomes and quality of life. However, a significant minority are persistently symptomatic despite normalization of TSH levels. This usually results in poor quality of life and creates significant tension in doctor-patient relationship.

Several postulations have been proposed to explain patient dissatisfaction with LT4 monotherapy. Normal TSH level may not guarantee normal serum free triiodothyronine (fT3) level or euthyroid states in all target tissues (e.g. cholesterol, energy expenditure). The optimal set point for thyroid hormone homeostasis can be highly individualized, therefore population-based laboratory reference ranges may not adequately guide thyroid hormone replacement. Certain polymorphisms in type 2 deiodinase (DIO2) were associated with reduced DIO2 activity, which may explain impaired peripheral T4 to T3 conversion in susceptible patients.

LT4/T3 combination has been the main alternative approach when hypothyroid patients are not satisfied with LT4 monotherapy. Overall, individual randomized controlled trials and recent meta-analyses failed to demonstrate clear or consistent benefits from adding T3 to LT4 therapy. Nonetheless, considering the limitations in these trials, potential benefits of LT4/T3 combination in selected patients cannot be excluded. This presentation will summarize the controversy and updated evidence of LT4/T3 combination, as well as outline the practical approach on the “when and how” of LT4/T3 combination.
Best practices for management of hyponatremia and SIAD

**Professor Joseph Verbalis**

*Chief*

*Division of Endocrinology & Metabolism*

*Georgetown University*

*USA*

Treatment of the hyponatremic patient with the syndrome of inappropriate antidiuresis (SIAD) presents a clinical challenge, particularly in the presence of comorbidities. In contrast to patients with congestive heart failure (CHF), the hyponatremic SIAD patient is clinically euolemic, without excess sodium retention. This allows therapeutic options that are not feasible in patients with hypervolemia, such as CHF. Currently available treatment options for SIAD include fluid restriction, administration of hypertonic saline, loop diuretics with NaCl tablets, demeclocycline, mineralocorticoids, urea, and vasopressin receptor antagonists. However, all of these treatments have limitations and some may exacerbate underlying comorbid conditions. Deciding among them requires knowledge of these limitations, as well as careful monitoring of the rate of correction of the serum sodium concentration to prevent the osmotic demyelination syndrome (ODS) from overly rapid correction of hyponatremia.

To define current best practices for managing hyponatremia and SIADH, the objectives of this presentation are:

1. To appreciate the differential diagnosis of hyponatremic disorders, and particularly the criteria for diagnosing SIAD;

2. To understand brain adaptation to hyponatremia via the process of brain volume regulation, and the implications of this process for hyponatremic symptoms and ODS following correction of hyponatremia;

3. To review and update current guidelines for therapy of SIAD, particularly the appropriate use of hypertonic saline, AVP receptor antagonists (vaptans), and urea, how these differ depending on the etiology of the disorder, the duration of the disorder, and the presence of neurological symptoms;

4. To highlight new developments in hyponatremia, and particularly exercise-associated hyponatremia and emerging data on falls, fractures and hyponatremia-induced osteoporosis.
Use of oral GLP-1 RA in diabetes management

Professor David Matthews
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UK

The discovery of the incretin axis marked a fundamental change in the way that Type 2 Diabetes (T2DM) can be treated. With continuous infusion GLP-1 has been known, for the last twenty years, to control glycaemic excursions in an explicit glucose-dependent manner. But the half-life was a few minutes. Continuous and dedicated research yielded both homologues (exenatide) and analogues (liraglutide) of the protein, with half-lives of many hours, that could be injected. These were approved for medical use in 2005 and 2009 respectively. Further research demonstrated that the duration of action could be extended to allow once weekly administration and so semaglutide, with a half-life of about seven days, was approved in 2017.

But semaglutide is a protein and so is given by weekly injection. If it is taken by mouth, like all proteins, it is converted to amino acids in the gut. A tiny amount will penetrate the stomach wall giving a bioavailability of <0.01% - a non-starter for clinical use. But using an absorption enhancer to bring the bioavailability up to 1%, with the industrial capacity to manufacture 100 times more than would be needed by injection, and giving the semaglutide daily to allow for fluctuations in absorption, meant that oral semaglutide became a reality in 2019.

So in clinical use we now have an oral agent that is truly glucose-dependent in its action to control glycaemia, and which has many additional beneficial effects. Oral semaglutide reduces weight (and can indeed be used in non-diabetic obesity), encourages reduced food intake, decreases lipogenesis, increases insulin sensitivity, decreases cardiovascular disease, and decreases inflammation.

This lecture will address the details of what we now understand about oral semaglutide, and how and when it can be used in clinical practice. Oral semaglutide can be regarded as one of the sentinel breakthrough in therapeutics for Type 2 diabetes of the 21st century.
Advancing therapy using fixed-ratio combination of basal insulin and GLP-1 RA in suboptimally controlled basal insulin-treated type 2 diabetes

Dr. Ingrid Mak  
Associate Consultant  
Department of Medicine  
Queen Elizabeth Hospital

The large heterogeneity and disease complexity of Type 2 Diabetes (T2D) have created a significant interest in developing new drug treatments that address various biological mechanisms involved in its pathophysiology, and prompted a push towards more personalized use of these medications. Despite the increasing use of more modern oral drugs like DPP4i and SGLT2i nowadays, there are still a number of patients with significant hyperglycaemia requiring insulin therapy.

For patients on basal insulin with inadequate disease control, treatment intensification using combination therapy should be considered. The addition of pre-prandial insulin (as bolus or a component of premixed insulin) has been traditionally adopted as the next step. However, this approach of ≥ 2 insulin injections causes much inconvenience to patients ultimately leading to clinical inertia and non-adherence, as well as undesirable side effects from high dose insulin therapy including hypoglycaemia and weight gain.

The ADA guideline recommends consideration of early combination therapy using basal insulin with a GLP1-RA in some patients at treatment initiation to extend the time to treatment failure. For those already using basal insulin, combination therapy with a GLP-1RA is recommended for greater efficacy and durability of treatment effect. Such approach could theoretically correct multiple defects in the pathophysiology of T2D and preserve pancreatic β-cell functions. Additionally, this combination might mitigate some of the side effects of insulin (hypoglycaemia and weight gain) and GLP-1RAs (gastrointestinal upset). Two different once-daily, fixed ratio combination (FRC) products containing basal insulin plus a GLP-1RA are available: insulin glargine U100 plus lixisenatide (Soliqua/ iGlarLixi) and insulin degludec plus liraglutide (Xultophy/ iDegLira). These FRC therapies are easier to administer than multiple injections and complex insulin regimens, thereby improving drug compliance and convenience.

SoliMix trial compared the efficacy and safety of once-daily iGlarLixi with twice-daily premixed BIAsp 30 in T2D patients suboptimally controlled with basal insulin plus 1-2 oral anti-diabetic drugs [Diabetes Care 2021;44:2361-2370]. At 26 weeks, iGlarLixi was both non-inferior and superior for HbA1c reduction versus BIAsp 30 (P < 0.001), and iGlarLixi was also superior to BIAsp 30 for body weight change (mean difference -1.9 kg). Lower incidence of hypoglycaemia was also observed in the iGlarLixi group. DUAL VIII study compared the durability of iDegLira versus insulin glargine (iGlar) U100 in insulin-naïve patients inadequately controlled with oral anti-diabetic drugs [Lancet Diabetes Endocrinol. 2019;7(8):596-605]. Over 104 weeks, fewer patients in the iDegLira group met criteria for intensification (37% vs 66%, HR 0.45) compared to the iGlar U100 group. The median time to treatment intensification was beyond 2 years for iDegLira and around 1 year for iGlar U100. At 104 weeks, there were also significantly less weight gain, more reduction in fasting plasma glucose, and lower rate of hypoglycaemia in the iDegLira group. These trials demonstrated that the FRCs of basal insulin and GLP-1 RA could potentially produce superior and more durable glucose lowering effects in both basal insulin-treated and insulin-naïve patients, with the additional benefits of body weight reduction and fewer hypoglycaemic episodes.

In this lecture, Dr. Mak will further elaborate on this promising approach of advancing therapy from an optimal dose of basal insulin to one of these FRC agents, using some real case scenarios for illustration. With the advancement in T2D drug treatments, most patients should be able to achieve optimal control as long as they are cooperative in terms of lifestyle, drug choice and adherence.
New approaches to delay CKD progression in diabetes: battling inflammation and fibrosis

Professor Per-Henrik Groop
Chairman
Department of Internal Medicine
University of Helsinki
Finland

Diabetes is a high global disease burden. Approximately 40% of diabetic patients have chronic kidney disease (CKD). Comorbid CKD further increases the risks for cardiovascular morbidity and mortality compared with diabetes alone. Despite recent advances in the treatment, patients with CKD and type 2 diabetes are at high residual risk of cardiorenal events.

CKD progression in type 2 diabetes is driven by the combined effects of metabolic, haemodynamic and inflammatory and fibrotic factors. Yet, the current therapeutic armamentarium to prevent CKD progression in type 2 diabetes is limited to the control of blood pressure and glucose levels. Targeting inflammation and fibrosis mediated by mineralocorticoid receptor overactivation is one of the potential therapeutic approaches beyond traditional treatments focusing on primarily metabolic and haemodynamic factors.

Recent clinical studies have shown that non-steroidal mineralocorticoid receptor antagonist (MRA) reduces the risk for kidney and cardiovascular events in patients with diabetic kidney disease. Non-steroidal MRA differs from steroidal MRA in the structure and pharmacological properties. For instance, the non-steroidal MRA finerenone has demonstrated a lower incidence of hyperkalaemia compared with the steroidal MRA spironolactone. In this context, inflammation and fibrosis driven by mineralocorticoid receptor can present a promising treatment target in the management of diabetic kidney disease.
Bone fragility in diabetes

Professor Serge Ferrari
Chairman
Department of Medicine
Geneva University Hospital
Switzerland

Diabetes is associated with an increased risk of fractures, yet the alterations and pathophysiological mechanisms of bone fragility in this condition remain poorly understood. Epidemiological studies have indicated that longer duration of disease, poor glycemic control and microvascular complications, as well as insulin use increase fracture risk. Recent case-control studies and meta-analyses further indicate that fracture risk increases with HbA1c above 8%, respectively decreases with HbA1c below 8% in patients receiving metformin, but this relationship is abrogated in insulin users, which is explained by an increased incidence of hypoglycemia and falls among the latter.

Bone turnover markers, including CTx, P1NP and osteocalcin (OC), which are usually not elevated in diabetes, do not seem to predict fracture risk in this condition. Regarding the structural bases of bone fragility in diabetes, there is much controversy about the actual alterations that may explain increased fracture risk. Several cohort studies using high-resolution pQCT have found a rather increased trabecular bone volume, although this may partly be explained by an artifact due to cortical trabecularisation. In contrast, cortical volumetric density and thickness appear to be decreased, and porosity increased, among diabetics, leading to an overall decline of estimated bone strength (by FEA). Nevertheless the importance and nature of a potentially increased cortical porosity in diabetes remains uncertain, as is the relationship between structural alterations and fracture risk. Eventually, changes in the material properties of bone, such as accumulation of AGEs in the bone matrix and altered collagen cross-linking thereby, remains a possibility, further substantiated by a recent study on hip bone samples from diabetics, but large-scale evaluation of this parameter in diabetics cohorts is still missing. Regarding treatment, so far the only available data are sub-group analyses in limited diabetic subsets from osteoporosis trials, suggesting similar effects of anti-resorptives and anabolics as in non-diabetics. However no study has evaluated the efficacy and safety of osteoporosis drugs in diabetic bone disease.

In conclusions, diabetes is increasingly recognize as a major risk factor for fragility fractures. However the evaluation of fracture risk in diabetics remains a challenge, as is the treatment of bone fragility in this condition.
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(BELOW-GROUP CHANGE/YEAR IN MEAN eGFR (CHRONIC SLOPE): 1.9 mL/min/1.73 m2 (FORXIGA/placebo)2

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**RIVA-DM** study was a cohort analysis within the US Optum® De-Identified EHR database between 2010 and 2019. It included patients with NVAF and diabetes: 32,078 patients on Xarelto® and 83,971 patients on warfarin. Patients had follow-up data for an average of 2.9 years. The primary efficacy and safety outcomes were incidence rates of developing the composite of SSE/vascular death or major/CRNM bleeding resulting in hospitalization.

CRNM=clinically relevant non-major; CV=cardiovascular; ICH=intracranial hemorrhage; MALE=major adverse limb events; NVAF=non-valvular atrial fibrillation; SSE=stroke/systemic embolism.

**References:**
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- Better adherence shown in real-world studies vs. other GLP-1 RAs

* Trulicity 0.75 mg demonstrated statistically superior HbA1c reduction in patients with type 2 diabetes in 4 phase III clinical trials vs. metformin, sitagliptin, exenatide 80 μg, exenatide 2.0 mg, and placebo. Trulicity 1.5 mg demonstrated noninferior HbA1c reduction vs. exenatide 2.0 mg as well as noninferiority vs. insulin glargine in patients with type 2 diabetes and CVD.

† Trulicity 0.75 mg significantly reduced the risk of MACE (composite of non-fatal MI, non-fatal stroke, or CV death) vs. placebo by 12% on top of standard of care. CV benefit was consistent across subgroups of patients with and without established CVD.

álido, diastolic BP, and BP. 29. real-world studies, 30. percent of patients with T2DM were switching to once-weekly Trulicity relative to once-weekly exenatide. 31. An occurrence of pancreatitis, which was reported in patients with diabetes mellitus, was considered an adverse event.

References:

Trulicity Avoided Prescribing Information:
Indications: Trulicity is indicated for the treatment of adults with insufficiently controlled type 2 diabetes mellitus as an adjunct to diet and exercise. 1. As monotherapy when metformin is considered inappropriate due to intolerance or contraindications. 2. In addition to other medical products for the treatment of diabetes. Dosage: Adult Monotherapy: 0.75 mg once weekly. Add-on therapy, 1.5 mg once weekly. Efficacy and safety in patients aged 75 years and older have been established. Trulicity is associated with an increased risk of hypoglycemia. Limitations in patients with congestive heart failure.

Adverse Drug Reactions: Abdominal distension, abdominal pain, acute pancreatitis, constipation, decreased appetite, diarrhea, dyspepsia, flatulence, fatigue, first-degree atrioventricular block, flu-like, gastric ulcer, nausea, musculoskeletal pain, musculoskeletal strain, musculoskeletal symptoms, musculoskeletal weakness, nasopharyngitis, upper respiratory tract infection. (USPC210C2019) Full prescribing information is available upon request.
Start your patient with **TRESIBA®**: Ultra-long duration of action

- Successful reductions in HbA1c
- Significantly lower risk of hypoglycaemia versus glargine U100
- Flexibility in day-to-day dosing time when needed
- Significantly lower day-to-day variability in glucose-lowering effect vs glargine U100 and U300
- Approved for a broad range of patients

Further information is available from:
Novo Nordisk Hong Kong Ltd.
Unit 923A-928, 9/F Trade Square, 681 Cheung Sha Wan Road, Kowloon, Hong Kong
Tel: +852 3725 1300 Fax: +852 2386 0800 www.novonordisk.com

TRESIBA®
insulin degludec [rDNA origin] injection
**SAMSCA®** is effective at raising serum **Na**⁺ in **HF** patients over 30 days

Pooled analysis of SALT-1 and SALT-2, mean change from baseline vs. placebo (P<0.0001)*

- **Day 4**: 3.5 vs. 0.5 mEq/L
- **Day 30**: 6.6 vs. 2.4 mEq/L

**SAMSCA®** has a significant effect on fluid balance in **HF** patients

Mean net fluid balance at day 1 in patients with baseline serum **Na**⁺ <135mEq/L (p=0.0027)*

**SAMSCA® -1860mL** vs. **Placebo -787mL**

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*Results from pooled analysis of SALT-1 and SALT-2 in congestive heart failure subgroup. SALT-1 and SALT-2 were two phase 3 randomized, double-blind trials in which patients with chronic or intermittent **HF** were randomized to **SAMSCA** (1860 mg) or placebo (787 mg) daily. **SAMSCA** was started at 15 mg daily, then daily dose was increased to 30 mg daily or 60 mg daily, as determined by the individual subject serum sodium response. The mean primary and placebo for 3 patients were the change in the average daily serum sodium concentration from baseline to day 4 and the change from baseline to day 30.

2. SAMSCA® (vildagliptin)/Hong Kong Prescribing Information revised Mar 2019.

**HF** Heart Failure; **Na**⁺ Sodium

**Abbreviated Prescribing Information**

**SAMSCA** (vildagliptin) 31.5 mg, 6.3 mg, and 3.2 mg: INDICATIONS: treatment of clinically significant hyperglycemia and euvolemic hypotension (serum sodium <125 mEq/L) or less marked hypotension that is symptomatic and has resulted in fluid restriction. Laboratory studies during heart failure and treatment of Euvolemic Hypotension (14 of 380 patients) showed the following changes in serum sodium: **12** decreased 10 mEq/L or more, **21** increased 10 mEq/L or more, **4** increased 20 mEq/L or more, and **1** decreased 20 mEq/L or more. **SAMSCA** therapy may reduce the serum sodium level. Laboratory monitoring of serum sodium concentration is recommended with **SAMSCA** therapy. Laboratory monitoring of serum sodium concentration should be performed during the first month of therapy. If serum sodium decreases significantly, treatment with **SAMSCA** may be discontinued or continued with a reduced dose.

**Contraindications**: Patients with a history of hypokalemia, in whom potassium levels are low or have been low, are at increased risk of developing hyperkalemia. Hyperkalemia may add to the risk of hypotension and bradycardia. Patients with heart failure who are at risk of hyperkalemia should be monitored closely during therapy with **SAMSCA**. Patients with known or suspected hypokalemia or hypochloremia should be treated with potassium chloride or other electrolyte replacement before starting **SAMSCA** therapy. **SAMSCA** contains vildagliptin, a thiazolidinedione derivative, and thiazolidinediones may increase serum sodium levels. **SAMSCA** therapy may be associated with a decrease in the mean serum sodium level.
2 DOSES A YEAR*  
FOR EFFECTIVE AND SUSTAINED LDL-C REDUCTION†

In ONR-10 (N=1,561), LEQVIO® demonstrated LDL-C reduction in patients with established ASCVD.†

In ONR-10 clinical trial, LEQVIO® demonstrated LDL-C reduction in ASCVD patients:‡

*52% reduction in LDL-C from baseline in the LEQVIO group (51%) at month 12 compared to the placebo group (3%).

†Patients in both study arms were on a maximally tolerated statin.‡

Patients in both study arms were on a maximally tolerated statin.‡

Study design: ONR-10 was a multicenter, double-blind, randomized, placebo-controlled 12-month clinical trial. Patients with established ASCVD were taking a maximally tolerated dose of statin with or without other lipid-modifying therapy and maintained at LDL-C reduction. The ONR-10 trial, in addition to patients with ASCVD, included adults who were ASCVD risk equivalent (i.e., type 2 diabetes, familial hypercholesterolemia, or a 10-year risk of a cardiovascular event of 10% or as assessed through Framingham Risk Score for Type 2 Diabetes in Asian or African populations). A total of 1,561 patients were randomized to receive LEQVIO or placebo. Following a 12-week placebo run-in period, patients were randomized 1:1 to receive LEQVIO (150 mg)/placebo administered subcutaneously every 2 months or placebo administered subcutaneously every 4 weeks, for a total of 12 months. The primary outcome measure was percentage change from baseline in non-HDL cholesterol at month 12. The ONR-10 trial was conducted 2007-2014.

References:

**Note:** The ONR-10 trial is a randomized clinical trial comparing the effects of LEQVIO® to placebo in patients with established ASCVD. It was conducted between 2007 and 2011, and involved 1,561 patients who were randomized to receive LEQVIO® or placebo. The primary outcome measure was the percentage change from baseline in non-HDL cholesterol at month 12. The trial was designed to evaluate the effectiveness of LEQVIO® in reducing LDL-C levels in these patients.

**Note to readers:** This information is intended for healthcare professionals and should be used in conjunction with local guidelines and clinical expertise. It is not intended as a substitute for individual medical advice. Novartis Pharmaceuticals is the manufacturer of LEQVIO® (inclusiran).
For patients living with heart failure, Time is essential.

So is starting with ENTRESTO®.

Make ENTRESTO your first choice in place of an ACEI/ARB to help patients stay out of the hospital, live longer, and feel better right from the start.1,4,12

Help your patients stay out of the hospital, live longer, and feel better, so they have more time for what matters.

*In place of an ACEI orARB
ACEI—angiotensin-converting enzyme inhibitor; ARB—angiotensin receptor blocker; HF—heart failure; MOA—mechanism of action; ESC—European Society of Cardiology; AHA—American Heart Association; ACC—American College of Cardiology; HFSA—Heart Failure Society of America


ENTRESTO® is endorsed by ESC 2021 HF Guideline
"The Essential HF Intervention"

Change the heart.
Change heart failure 1,3,3
Reverse cardiac remodeling, improve cardiac structure and function, and target HF via a unique dual MOA that inhibits nephrin and RAAS

Provide the HF treatment superior to ACEI in all stages of the HFrEF patient journey 2-5,6
Your first choice in the hospital or outpatient setting, whether patients are newly diagnosed or have worsening symptoms

Make a lasting difference patients can feel 2,5,11
Help your patients stay out of the hospital, live longer, and feel better, so they have more time for what matters

Another Preferred Therapy

ENTRESTO® is endorsed by AHAMCC/HFSA 2022 HF Guideline

For stage C HFrEf patients

Sacubitril/valsartan

Novartis Pharmaceuticals (HK) Ltd
79/F, Gil Tower, One Bay East, 83 He Fung Road, Kwun Tong, K.H.
Tel: 2982 0222 Fax: 2957 0274

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Change the heart.
Change heart failure 1,3,3
Reverse cardiac remodeling, improve cardiac structure and function, and target HF via a unique dual MOA that inhibits nephrin and RAAS

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CHOOSE SOLIQUA™ FOR THE POWER TO GET TO TARGET

SoliMix supports the use of SOLIQUA™ as a favourable alternative to premix insulin when intensifying from BI + OADs. The first HEAD-TO-HEAD, randomised controlled trial comparing SOLIQUA™ to premix insulin.

SOLIQUA™ achieved SUPERIOR HbA1c REDUCTION and WEIGHT CHANGE with LESS HYPOGLYCAEMIA vs premix insulin.

**SUPERIOR HbA1c REDUCTION**

Relative reduction by SOLIQUA™ vs premix

-18.2% (97.5% CI: -0.2% (-0.4, -0.1)%; p<0.001)

**SUPERIOR WEIGHT CHANGE**

LS mean difference (95% CI): -1.86 kg

**LESS HYPOGLYCAEMIA**

Nearly 3x more patients at goal

Nearly 3x achieved HbA1c <7% without hypoglycaemia and weight gain

(19.4% vs 7%, respectively; p<0.001)

Odds ratio (95% CI): 3.40 (2.19, 5.28)

SOLIQUA™ is indicated for the treatment of adults with insufficiently controlled type 2 diabetes mellitus to improve glycemic control as an adjunct to diet and exercise in addition to metformin with or without SGLT-2 inhibitors.

**SOLIQUA™** achieved SUPERIOR HbA1c REDUCTION and WEIGHT CHANGE with LESS HYPOGLYCAEMIA vs premix insulin.

LS mean difference (95% CI): -1.86 kg

**LEVEL-2 HYPOGLYCAEMIA**

-55%

Odds ratio (95% CI): 0.45 (0.28, 0.73)

Precautions: Elderly: Soliqua can be used in elderly patients. Progressive deterioration of renal function may lead to a steady decrease in insulin requirements. Renal impairment. Not recommended in severe renal impairment and end-stage renal disease. Frequent glucose monitoring and dose adjustment may be necessary in patients with mild to moderate renal impairment. Hypoglycaemia may occur if dose is higher than recommended. Avoid patients with recent procedures for hypoglycaemia while driving and using machines. Discontinue Soliqua if pancreatitis is suspected. Insulin biosimilars may not adequately concomitantly. Insulin analogs may mask the effects of insulin. Patients with type 1 diabetes mellitus. Treatment of diabetic ketoacidosis.


Sanofi Hong Kong Limited

CONTOUR®PLUS ELITE is an easy-to-use system that supports diabetes management providing clear, accurate readings you can trust.¹ ²

- **Readings you can trust** to be highly accurate¹ and support blood glucose management.
- **Easy to understand** blood sugar results with the smartLIGHT™ feature.²
- **Avoid re-lancing** with 60-second Second-Chance® sampling.³
- **Map your journey** with the CONTOUR®DIABETES app.

When it comes to diabetes management, **Trust CONTOUR®**.

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美好生活 一觸可及
Ultra Plus Flex®
穩豪智優型血糖機

颜色指示功能
ColoursSure™
TECHNOLOGY

○ 新一代金屬基試紙，減少干擾
○ 高準確性 試紙英國製造
○ 藍芽傳輸測試結果
○ 兼容『智抗糖』行動應用程式，儲存和追蹤測試結果無難度

符合國際標準 EN ISO15197:2015
免調碼 5秒測試
個人化血糖範圍限制值

永久保養

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SURPRISINGLY CLEVER

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The Accu-Chek Guide system exceeds industry standards with tighter accuracy.

Strip ejector button
Strip removal is quick and clean.

Clever SmartPack vial
Spill-resistant vial—easier to slide out one strip a time.

Smartly stored data
Wirelessly sends results to the mySugr app.
ACKNOWLEDGEMENTS