5TH ANNIVERSARY of

Endocrinology, Diabetes & Metabolism Hong Kong

EDM HK

Annual Meeting

29 – 30 October 2022

Hong Kong Convention and Exhibition Centre

Programme Book

HKU Med School of Clinical Medicine
Department of Medicine
香港大學內科學系

瑪麗醫院
QUEEN MARY HOSPITAL

K. K. LEUNG DIABETES CENTRE
瑪麗醫院
Queen Mary Hospital
梁銶琚糖尿病中心

STEOPOROSIS CENTRE 骨質疏鬆中心

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§

CV Indication Approved

JARDIANCE demonstrated 38% RRR in CV death[1,2]

- Established HbA1c efficacy[2]
- Demonstrated safety profile[1,2]
- Convenient, once-daily oral dosing[2]

ADA & EASD recognize JARDIANCE as the SGLT2 inhibitor with stronger evidence of CV benefits[3#]

Jardiance® (empagliflozin)

CV: cardiovascular; RRR: relative risk reduction; ADA: American Diabetes Association; EASD: European Association for the Study of Diabetes; CVD: cardiovascular disease; OAD: oral antidiabetic drug; T2DM: type 2 diabetes mellitus

Reference: 1. Zinman B, et al. N Engl J Med. 2015;373(22):2117-2118. **2.** Jardiance Hong Kong Prescribing Information. **3.** Davies MJ, D'Alessio DA, Fradkin J,et al. Management of hyperglycaemia in type 2 diabetes, 2018. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia. 2018.

† JARDIANCE demonstrated RRR in CV death in adult patients with insufficiently controlled type 2 diabetes (baseline HbA1c 7-10%) and established CV disease (coronary artery disease, peripheral artery disease, or a history of myocardial infarction or stroke).[1]

‡ Standard of care included CV medications and glucose-lowering agents given at the discretion of physicians.[1]

§ Empagliflozin versus placebo on top of standard of care.[1]

Management of hyperglycemia in type 2 diabetes, 2018. A consensus report by the ADA and EASD stated that among patients with established CVD, there is likely cardiovascular benefit, with the evidence of benefit modestly stronger for empagliflozin than canagliflozin[3]

JARDIANCE® Abbreviated Prescribing Information (aPI-JARD-02)

Presentation: Empagliflozin. Film-coated tablets 10 mg; 25 mg. **Indications: 10 mg and 25 mg:** Indicated in the treatment of type 2 diabetes mellitus to improve glycaemic control in adults as: monotherapy when diet and exercise alone do not provide adequate glycaemic control in patients for whom use of metformin is considered inappropriate due to intolerance; and as add-on combination therapy with other glucose-lowering medicinal products including insulin, when these, together with diet and exercise, do not provide adequate glycaemic control. Indicated in patients with type 2 diabetes mellitus and established cardiovascular disease to reduce the risk of cardiovascular death. **10 mg:** Jardiance is indicated in adults for the treatment of symptomatic chronic heart failure. **Dosage and administration: Type 2 diabetes mellitus:** 10 mg once daily. In patients tolerating 10 mg once daily and requiring additional glycaemic control, the dose can be increased to 25 mg once daily. Can be taken with or without food. No dose adjustment is required for patients with eGFR ≥ 30 mL/min/1.73m² or with hepatic impairment, or for elderly patients. **Heart Failure:** 10 mg once daily. Can be taken with or without food. In HF patients with or without T2DM, 10 mg may be initiated or continued down to an eGFR of 20 ml/min/1.73m² or CrCl of 20 ml/min. **Contraindication:** Hypersensitivity to empagliflozin or any of the excipients. For the treatment of Type 2 diabetes, JARDIANCE should not be used in patients with severe renal impairment (eGFR <30 mL/min/1.73m²), end-stage renal disease and patients on dialysis, as glycaemic efficacy depends on renal function. **Special warnings and precautions:** Should not be used in patients with type 1 diabetes or for treatment of ketoacidosis. Discontinue immediately when ketoacidosis is suspected or diagnosed. Treatment should be interrupted in patients who are hospitalised for major surgical procedures or acute serious medical illnesses, and may be restarted once the patient's condition has stabilised. For type 2 diabetes mellitus, should not be used in patients with severe renal impairment (eGFR <30 mL/min/1.73m²), end-stage renal disease and patients on dialysis. For HF, not recommended for use when eGFR < 20 mL/min/1.73m². Discontinue in cases of recurrent UTI. Due to a risk of modest decrease in blood pressure, caution should be exercised in patients with known cardiovascular disease, patients on diuretics, patients with history of hypotension or patients aged 75 years and older. Monitoring of volume status and electrolytes is recommended. Regularly examine the feet and counsel patients on routine preventative footcare. Caution is advised in patients at increased risk of genital infections. Avoid use during pregnancy and breast-feeding. Safety and effectiveness in children under 18 years of age have not been established. Initiation is not recommended in patients aged 85 years and older. Urine will test positive for glucose while patients are taking JARDIANCE. **Interactions:** Risk of dehydration and hypotension may increase when used in combination with thiazide and loop diuretics. Lower dose of insulin or an insulin secretagogue may be required to reduce the risk of hypoglycemia when used in combination with JARDIANCE. **Adverse reactions:** Hypoglycaemia (depends on type of background therapy of patients); Urinary tract infection, vaginal moniliasis, vulvovaginitis, balanitis and other genital infection; Increased urination, dysuria; Pruritus; Volume depletion; Thirst; Glomerular filtration rate decreased, blood creatinine increased, haematocrit increased, serum lipids increased. Post-marketing experience: Ketoacidosis, complicated urinary tract infections, necrotising fasciitis of the perineum (Fournier's gangrene), allergic skin reaction, angioedema. **Storage condition:** Please refer to outer packaging for special precautions for storage. **Note:** Before prescribing, please consult full prescribing information.

THE ONLY OAD WITH CV INDICATION

Jardiance is indicated in T2DM patients and established cardiovascular disease to reduce the risk of cardiovascular death[2]

Boehringer Ingelheim

Boehringer Ingelheim (HK) Ltd.
Suites 1504-9, Great Eagle Centre, 23 Harbour Road, Wanchai, Hong Kong
Tel: (852) 2596 0033 Fax: (852) 2827 0162 www.boehringer-ingelheim.com.hk

PC-HK-100806 (Jun 2022)

TABLE OF CONTENTS

WELCOME MESSAGE

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

ORGANIZING COMMITTEE

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. Chariene SL Woo

Ms. Amy SW Yee

Ms. Connie HN Loong

Ms. Michelle HY Lee

ACCREDITATIONS

CME				
Organization	**Max. for whole function**	**29 October**	**30 October**	**Group - category**
Hong Kong College of Community Medicine	TBA	TBA	TBA	PP-PP
The Hong Kong College of Family Physicians	10	5	5	OEA-5.02
The Hong Kong College of Obstetricians and Gynaecologists	5	5	5	PP-PN
The College of Ophthalmologists of Hong Kong	13.5	6.5	7	CME-PP
Hong Kong College of Orthopaedic Surgeons	8	5	5	PP-B
Hong Kong College of Paediatricians	12	6	6	A-PP
The Hong Kong College of Pathologists	14	5	9	CME-PP
Hong Kong College of Physicians	12	6	6	PP-PP
Hong Kong College of Radiologists	17	8	9	B-PP
The College of Surgeons of Hong Kong	7	2	5	CME-PP
The Medical Council of Hong Kong	10	5	5	CME-PASSIVE

CNE		
Organization	**29 October**	**30 October**
Hospital Authority Hong Kong West Cluster	4	6.5

2022年10月29日 (星期六)

時間	會議室 S226 - S227
主持：呂德威醫生及胡裕初醫生	
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)

Time	Room S221	Room S226 – S227
	Lecture (1) (Sponsored by Amgen) *Chairperson: Dr. TP Ip*	
13:00 – 13:40	**The importance of sequencing therapy after osteo-anabolic agents** *Professor Michael McClung (USA)*	
13:40 – 13:45	**Q & A**	
13:45 – 13:55	**Opening Ceremony**	
	Lecture (2) (Sponsored by AstraZeneca) *Chairperson: Professor Karen Lam*	
13:55 – 14:40	**A roadmap for optimizing the care and outcomes of diabetes patient with kidney disease** *Professor Peter Rossing (Denmark)*	
14:40 – 14:45	**Q & A**	
Time	**Room S221**	**Room S226 – S227**
	Symposium (1A) *Chairpersons: Dr. WS Chow and Professor Brian Lang*	**Symposium (1B)** *Chairpersons: Dr. KF Lee and Dr. Joanna Tung*
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*	
16:30 – 17:10	**Considerations when choosing between type 2 diabetes therapy: the role of once-weekly GLP-1 RA** *Professor Michael Cummings (UK)*	
17:10 – 17:15	**Q & A**	
	Lecture (4) (Sponsored by Novartis) *Chairperson: Professor Kathryn Tan*	
17:15 – 17:50	**Managing familial hypercholesterolemia: achieving optimal treatment targets** *Professor Frederick Raal (South Africa)*	
17:50 – 17:55	**Q & A**	

30 October 2022 (Sunday)

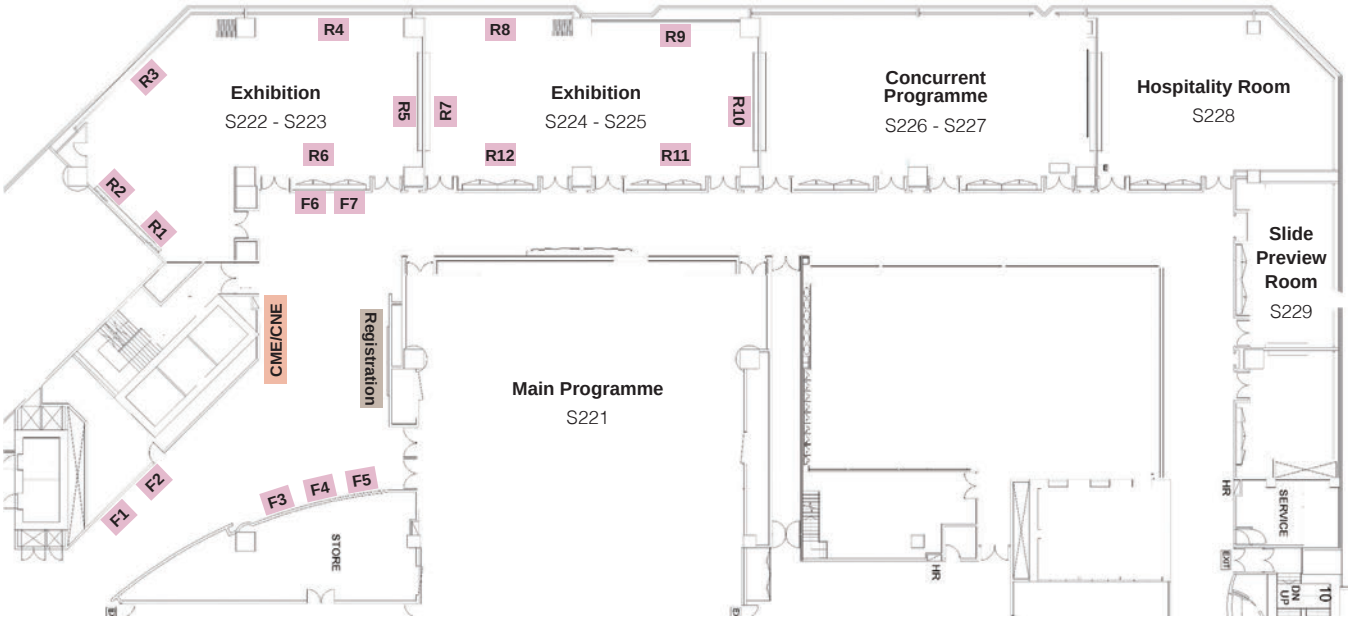
Time	Room S221	Room S226 – S227
	Plenary Lecture (1) *Chairperson: Dr. Alan Lee*	
09:00 – 09:35	**Advances in the management of Graves' disease** *Professor Marius Stan (USA)*	
09:35 – 09:40	**Q & A**	
	Lecture (5) (Sponsored by Boehringer Ingelheim) *Chairperson: Dr. YY Ho*	
09:40 – 10:20	**The cardiorenal side of SGLT2 inhibitors: exploring advances from type 2 diabetes to heart failure** *Professor Jennifer Green (USA)*	
10:20 – 10:25	**Q & A**	
10:25 – 10:55	**Break**	
Time	**Room S221**	**Room S226 – S227**
	Symposium (2A) *Chairpersons: Dr. Nicole Chau and Professor Alice Kong*	Symposium (2B) *Chairpersons: Dr. Emmy Lau and Dr. Jenny Leung*
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**
	Lecture (6) (Sponsored by Otsuka) *Chairperson: Dr. SC Tiu*	
11:45 – 12:25	**Best practices for management of hyponatremia and SIAD** *Professor Joseph Verbalis (USA)*	
12:25 – 12:30	**Q & A**	
12:30 – 13:30	**Lunch Break**	
	Lecture (7) (Sponsored by Novo Nordisk) *Chairperson: Professor Rosie Young*	
13:30 – 14:10	**Use of oral GLP-1 RA in diabetes management** *Professor David Matthews (UK)*	
14:10 – 14:15	**Q & A**	

30 October 2022 (Sunday)

Time	Room S221
	Lecture (8) (Sponsored by Sanofi) *Chairperson: Dr. Michele Yuen*
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** *Dr. Ingrid Mak (Hong Kong)*
14:50 – 14:55	**Q & A**
14:55 – 15:25	**Break**
	Lecture (9) (Sponsored by Bayer) *Chairperson: Dr. Grace Kam*
15:25 – 16:05	**New approaches to delay CKD progression in diabetes: battling inflammation and fibrosis** *Professor Per-Henrik Groop (Finland)*
16:05 – 16:10	**Q & A**
	Plenary Lecture (2) *Chairperson: Dr. David Lui*
16:10 – 16:50	**Bone fragility in diabetes** *Professor Serge Ferrari (Switzerland)*
	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** *Dr. Ingrid Mak (Hong Kong)*
17:05 – 17:20	**Atypical metatarsal fracture in a Chinese post-menopausal woman with osteoporosis on long-term denosumab** *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** *Dr. Johnny Chang (Hong Kong)*
17:35 – 17:50	**Primary pigmented nodular adrenocortical Disease (PPNAD) - sequential or bilateral adrenalectomy?** *Dr. KY Wong (Hong Kong)*
17:50 – 17:55	**Closing Remarks**

FLOOR PLAN

S220, Level 2, Phase 1 (Old Wing), Hong Kong Convention and Exhibition Centre



No. Exhibition Booth | Registration Counter | CME/CNE

LIST OF EXHIBITORS

Organization	Booth Number
Abbott Laboratories Limited	R10
Amgen Hong Kong Limited	F7
AstraZeneca Hong Kong Limited	F5
Bayer HealthCare Limited	F4
Boehringer Ingelheim (Hong Kong) Ltd.	F1
Celltrion Healthcare Hong Kong Limited	R9
Chong Lap (H.K.) Co., Ltd.	R1
Eli Lilly Asia, Inc.	F2
Ipsen Pharma (HK)	R11
JCare Medical Technology Co., Ltd.	R3
Medtronic Hong Kong Medical Limited	R7
Merck Pharmaceutical (Hong Kong) Ltd	R8
Novartis Pharmaceuticals HK Ltd.	R6
Novo Nordisk Hong Kong Limited	F6
Otsuka Pharmaceutical (H.K.) Ltd.	F3
Roche Diagnostics (Hong Kong) Limited	R4
Sanofi Hong Kong Limited	R2
Servier Hong Kong Limited	R12
ZP Therapeutics	R5

LIST OF OVERSEAS SPEAKERS

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

Professor Serge Ferrari
Chairman
Department of Medicine
Geneva University Hospital
Switzerland

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

Professor Michael McClung
Founding Director
Oregon Osteoporosis Center
USA

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

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

Professor Marius Stan
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Mayo Clinic
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Professor Joseph Verbalis
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Dr. Chris Chan
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Professor Karen Lam
Chair Professor
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Dr. Michele Yuen
Honorary Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

LECTURE (1)
(SPONSORED BY AMGEN)

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.

LECTURE (2)
(SPONSORED BY ASTRAZENECA)

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

Dr. YC Woo
Consultant
Department of Medicine
Queen Mary Hospital

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

Dr. Chariene Woo

Resident
Department of Medicine
Queen Mary Hospital

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

Dr. Gloria Pang

Associate Consultant
Department of Paediatrics & Adolescent Medicine
Hong Kong Children's Hospital

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

Dr. Raymond Li

Clinical Associate Professor
Department of Obstetrics & Gynaecology
The University of Hong Kong

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.

LECTURE (3)
(SPONSORED BY ELI LILLY)

Considerations when choosing between type 2 diabetes therapy: the role of once-weekly GLP-1 RA

Professor Michael Cummings

Honorary Professor
Department of Diabetes & Endocrinology
Portsmouth Hospitals NHS Trust Queen Alexandra Hospital
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.

LECTURE (4)
(SPONSORED BY NOVARTIS)

Managing familial hypercholesterolemia: achieving optimal treatment targets

Professor Frederick Raal

Head
Division of Endocrinology & Metabolism
University of the Witwatersrand
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

Consultant
Department of Internal Medicine
Mayo Clinic
USA

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 beed 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 asscіated with RAI. The utilisation of surgery has remained stable ver 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 succes 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 aleternative 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.

LECTURE (5)
(SPONSORED BY BOEHRINGER INGELHEIM)

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

Honorary Clinical Assistant Professor
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.

LECTURE (6)
(SPONSORED BY OTSUKA)

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 euvolemic, 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.

LECTURE (7)
(SPONSORED BY NOVO NORDISK)

Use of oral GLP-1 RA in diabetes management

Professor David Matthews

Emeritus Professor of Diabetes Medicine
Department of Medicine
University of Oxford
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.

LECTURE (8)
(SPONSORED BY SANOFI)

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.

LECTURE (9)
(SPONSORED BY BAYER)

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.

SUPPORTING ORGANIZATIONS

香港糖尿科護士協會
Association of Hong Kong Diabetes Nurses

香港糖尿病會
Diabetes Hongkong

Hong Kong Obesity Society
香港肥胖學會

HKSEMR
Hong Kong Society of Endocrinology, Metabolism and Reproduction

香港醫院藥劑師學會
The Society of Hospital Pharmacists of Hong Kong

香港復康會
The Hong Kong Society for Rehabilitation

香港兒童內分泌科學會
HKSPEM
The Hong Kong Society of Paediatric Endocrinology and Metabolism

OSHK
香港骨質疏鬆學會

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↓31% All-cause mortality vs placebo
(HR 0.69; 95% CI, 0.53, 0.88; p=0.004)[2]

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† ≥50% sustained decline in eGFR.
‡ There were comparable rates of the individual component of CV death vs placebo (3.0% vs 3.7%; HR 0.81; 95% CI, 0.58, 1.12).
§ Primary composite endpoint of ≥50% sustained decline in eGFR, reaching ESKD, and renal or CV death. ESKD is defined as the need for maintenance dialysis for at least 28 days and renal transplantation or sustained eGFR <15 mL/min/1.73m² for at least 28 days.
¶ Baseline eGFR categories: <45 mL/min/1.73m² and ≥45 mL/min/1.73m².
¶ Observed only in T2D patients.
** CKD stage groups: Stage 4 and Stage 2/3.
†† Diabetic nephropathy, glomerulonephritides, ischaemic or hypertensive CKD, or CKD of other or unknown cause.
‡‡ In patients with severe hepatic impairment, a starting dose of 5 mg is recommended. If well tolerated, the dose may be increased to 10 mg.
§§ In DAPA-CKD, patients may continue on FORXIGA 10 mg once daily if eGFR falls below 25 mL/min/1.73m².
¶¶ Due to limited experience, it is not recommended to initiate treatment with dapagliflozin in patients with GFR <25 mL/min.

AKI, acute kidney injury; CI, confidence interval; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HF, heart failure; hHF, hospitalization for heart failure; HR, hazard ratio; SAE, serious adverse event; SGLT2i, sodium-glucose co-transporter-2 inhibitor; T2D, type 2 diabetes; UACR, urine albumin-creatinine ratio.

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Abbreviated Prescribing Information (API)
FORXIGA (dapagliflozin)
Composition: Dapagliflozin propanediol monohydrate film coated tablet, 5 mg or 10 mg. **Therapeutic Indications:** For the treatment of insufficiently controlled type 2 diabetes mellitus in adults as an adjunct to diet and exercise, either as monotherapy when metformin is considered inappropriate due to intolerance, or in addition to other medicinal products for the treatment of type 2 diabetes. For the treatment of symptomatic chronic heart failure with reduced ejection fraction. For the treatment of chronic kidney disease. **Dosage and Administration:** Type 2 diabetes mellitus: Recommended dose is 10 mg to be taken orally once daily at any time of day with or without food. Tablets are to be swallowed whole. Heart Failure: Recommended dose is 10 mg to be taken orally once daily. Chronic Kidney Disease: Recommended dose is 10mg to be taken orally once daily. In patients with severe hepatic impairment, a starting dose of 5 mg is recommended. **Contraindications:** Hypersensitivity to the active substance or to any of its excipients. **Warnings and Precautions:** Renal function, risk of volume depletion and/or hypotension should be taken into account in patients. Dosage of insulin and sulphonylurea (SU) may need to be readjusted to reduce the risk of hypoglycaemia. May add to the diuretic effect of thiazide and loop diuretics and may increase the risk of dehydration and hypotension. Use with caution in patients with increased risk of diabetic ketoacidosis, on anti-hypertensive therapy with a history of hypotension; elderly (≥ 65 years). Treatment should be temporarily interrupted when volume depleted; when treating pyelonephritis or urosepsis; in patients who are hospitalized for major surgical procedures or acute serious medical illnesses, until ketone values are normal. Should not be initiated in patients with type 1 diabetes; hereditary problems of galactose intolerance, total lactase deficiency, or glucose-galactose malabsorption. Additional glucose lowering treatment should be considered for glycaemic control improvement.If GFR is persistently below 45 ml/min for the treatment of diabetes; no dose adjustment is required based on renal function for the treatment of heart failure and chronic kidney disease. Due to limited experience, it is not recommended to initiate treatment with dapagliflozin in patients with GFR < 25 mL/min. Discontinue if suspected or diagnosed diabetic ketoacidosis; if Fournier's gangrene is suspected; when pregnancy is detected; while breast-feeding. Limited or no data in cardiac failure NYHA class IV; pregnancy; and paediatric population. **Adverse Reactions:** Very common: hypoglycaemia when used with SU or insulin. Common: vulvovaginitis, balanitis and related genital infections, urinary tract infection, dizziness, rash, back pain, dysuria, polyuria, dyslipidaemia, decreased creatinine renal clearance (during initial treatment), and increased haematocrit. Uncommon: Fungal infection, volume depletion, thirst, constipation, dry mouth, nocturia, vulvovaginal and genital pruritus, increased blood creatinine (during initial treatment), increased blood urea, and decreased weight. Rare: diabetic ketoacidosis (when used in type 2 diabetes). Very rare: necrotising fasciitis of the perineum (Fournier's gangrene), angioedema. Not known: acute kidney injury. **Drug interaction:** Coadministration with rifampicin may reduce dapagliflozin systemic exposure; coadministration with mefenamic acid may increase dapagliflozin systemic exposure. Monitoring glycaemic control with 1,5-AG assay is not recommended in patients taking SGLT2 inhibitors. **Storage:** Store below 30 °C. **Local prescribing information is available upon request. API.HK.FOR.1221**

Intended for Healthcare professionals only.
Please visit contactsmedical.astrazeneca.com, for (1) enquiring Medical Information (MI), (2) reporting Individual Case Safety Report (ICSR) and/or (3) reporting Product Quality Complaint (PQC) to AstraZeneca Hong Kong Limited.

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HF T2D CKD
Bring Protection to Life

HK-6632 17/2/2022 4IC_2172C

EVENITY®
(romosozumab) injection

prolia®
(denosumab)

Every patient has a different starting point

MEET HER THERE

and help make her bones stronger

For your patients with **very low T-score** (e.g. less than -3.0) or with other serious risk factors, start with the sequence of **EVENITY®** followed by **PROLIA®** to help build and protect her bone.[1]

For your partients **with history of fragility fracture or low T-score** (e.g. less than -2.5) with other risk factors, start with **PROLIA®** to help strengthen her bone.[2,3]

* The risk of hip fracture was lowered by 38% (41 of 2046 patients [2.0%] vs. 66 of 2047 patients [3.2%], P = 0.02) in the romosozumab-to-alendronate group than in the alendronate-to-alendronate group in ARCH Study.[1]

† Indicators of very high fracture risk in patients with low bone density would include advanced age, frailty, glucocorticoids, very low T-scores, or increased fall risk. Patients who have been diagnosed with osteoporosis but are not at very high fracture risk are defined as high risk.[1]

ARCH=Active-Controlled Fracture Study in Postmenopausal Women with Osteoporosis at High Risk; BMD=Bone mineral density.

References: 1. Evenity (romosozumab) Hong Kong prescribing information. March 2020. **2.** Prolia (denosumab) Hong Kong prescribing information. Aug 2020. **3.** Camacho PM, et al. Endocr Pract. 2020;26(Suppl 1).

Prolia® (Denosumab) Abbreviated Prescribing Information
Prolia® (denosumab) Solution for Injection in Pre-filled Syringe 60 mg/mL

INDICATIONS Prolia is indicated for: i) treatment of postmenopausal women with osteoporosis at high risk for fracture, defined as a history of osteoporotic fracture, or multiple risk factors for fracture; or patients who have failed or are intolerant to other available osteoporosis therapy; ii) treatment to increase bone mass in men with osteoporosis at high risk for fracture, defined as a history of osteoporotic fracture, or multiple risk factors for fracture; or patients who have failed or are intolerant to other available osteoporosis therapy; iii) treatment of glucocorticoid-induced osteoporosis in men and women at high risk of fracture who are either initiating or continuing systemic glucocorticoids in a daily dosage equivalent to 7.5 mg or greater of prednisone and expected to remain on glucocorticoids for at least 6 months. High risk of fracture is defined as a history of osteoporotic fracture, multiple risk factors for fracture, or patients who have failed or are intolerant to other available osteoporosis therapy; iv) treatment to increase bone mass in men at high risk for fracture receiving androgen deprivation therapy for nonmetastatic prostate cancer. In these patients Prolia also reduced the incidence of vertebral fractures; v) treatment to increase bone mass in women at high risk for fracture receiving adjuvant aromatase inhibitor therapy for breast cancer. **DOSAGE AND ADMINISTRATION** The recommended dose of Prolia is 60 mg administered as a single subcutaneous injection once every 6 months. Administer Prolia via subcutaneous injection in the upper arm, the upper thigh, or the abdomen. All patients should receive calcium 1000 mg daily and at least 400 IU vitamin D daily. **CONTRAINDICATIONS** Hypocalcemia and pregnancy, as well as hypersensitivity to any component of the product. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE** Hypersensitivity: Clinically significant hypersensitivity including anaphylaxis has been reported with Prolia. Symptoms have included hypotension, dyspnea, throat tightness, facial and upper airway edema, pruritis, and urticaria. Hypocalcemia and Mineral Metabolism: Hypocalcemia may be exacerbated by the use of Prolia. Pre-existing hypocalcemia must be corrected prior to initiating therapy with Prolia. Hypocalcemia following Prolia administration is a significant risk in patients with severe renal impairment (creatinine clearance < 30 mL/min) or receiving dialysis. Concomitant use of calcimimetic drugs may worsen hypocalcemia risk and serum calcium should be closely monitored. Adequately supplement all patients with calcium and vitamin D. Osteonecrosis of the Jaw (ONJ): ONJ has been reported in patients receiving Prolia. The start of treatment or of a new course of treatment should be delayed in patients with unhealed open soft tissue lesions in the mouth. A dental examination with preventive dentistry and an individual benefit-risk assessment is recommended prior to treatment with Prolia in patients with concomitant risk factors. All patients should be encouraged to maintain good oral hygiene, undergo routine dental check-ups, and immediately report any oral symptoms such as dental mobility, pain or swelling, or non-healing of sores or discharge during treatment with Prolia. While on treatment, invasive dental procedures should be performed with caution and avoided in close proximity to Prolia treatment. Atypical Subtrochanteric and Diaphyseal Femoral Fractures: Atypical low-energy or low trauma fractures of the shaft have been reported in patients receiving Prolia. Patients should be advised to report new or unusual thigh, hip, or groin pain. Multiple Vertebral Fractures (MVF) Following Discontinuation of Prolia Treatment: Following discontinuation of Prolia treatment, fracture risk increases, including the risk of multiple vertebral fractures. If Prolia treatment is discontinued, patients should be transitioned to an alternative antiresorptive therapy. Serious Infections: Serious infections leading to hospitalization were reported in clinical trial. Advise patients to seek prompt medical attention if they develop signs or symptoms of severe infection, including cellulitis. Dermatologic Adverse Reactions: Dermatitis, eczema, and rashes. Most of these events were not specific to the injection site. Consider discontinuing Prolia if severe symptoms develop. Musculoskeletal Pain: Severe and occasionally incapacitating bone, joint, and/or muscle pain. Consider discontinuing use if severe symptoms develop. Suppression of Bone Turnover: In clinical trials treatment with Prolia resulted in significant suppression of bone remodeling as evidenced by markers of bone turnover and bone histomorphometry. Osteonecrosis of the external auditory canal: Osteonecrosis of the external auditory canal has been reported with denosumab. Possible risk factors include steroid use and chemotherapy and/or local risk factors such as infection or trauma. **PREGNANCY AND LACTATION** Pregnancy: Contraindicated. Breast-feeding: No information regarding the presence of denosumab in human milk, the effects on the breastfed infant, or the effects on milk production **PEDIATRIC, GERIATRIC AND RENAL IMPAIRMENT** Pediatric: Prolia is not recommended in pediatric patients younger than age 4 years. Geriatric: No overall differences in safety or efficacy were observed in clinical studies between elderly patients and younger patients, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out. Renal Impairment: No dose adjustment is necessary in patients with renal impairment. **UNDESIRABLE EFFECTS** The most common adverse reactions reported with Prolia in patients with postmenopausal osteoporosis are back pain, pain in extremity, musculoskeletal pain, hypercholesterolemia, and cystitis. The most common adverse reactions reported with Prolia in men with osteoporosis are back pain, arthralgia, and nasopharyngitis. The most common adverse reactions reported with Prolia in patients with glucocorticoid-induced osteoporosis are back pain, hypertension, bronchitis, and headache. The most common (per patient incidence ≥ 10%) adverse reactions reported with Prolia in patients with bone loss receiving androgen deprivation therapy for prostate cancer or adjuvant aromatase inhibitor therapy for breast cancer are arthralgia and back pain. Pain in extremity and musculoskeletal pain have also been reported in clinical trials. The most common adverse reactions leading to discontinuation of Prolia in patients with postmenopausal osteoporosis are back pain and constipation. **OVERDOSE** There is no experience with overdosage with Prolia.

Abbreviated Prescribing Information Version: HKPROPI02
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EVENITY® (Romosozumab) Abbreviated Prescribing Information
EVENITY® Solution for Injection in Prefilled Syringe 105 mg/1.17 ml

INDICATIONS EVENITY is indicated in treatment of severe osteoporosis in postmenopausal women at high risk of fracture. **DOSAGE AND ADMINISTRATION** The recommended dose is 210 mg romosozumab (administered as two subcutaneous injections of 105 mg each) once monthly for 12 months. Patients should be adequately supplemented with calcium and vitamin D before and during treatment. Following completion of romosozumab therapy, transition to antiresorptive therapy is recommended in order to extend the benefit achieved with romosozumab beyond 12 months. Missed doses: If the romosozumab dose is missed, administer as soon as it can be feasible. Thereafter, the next romosozumab dose should not be given earlier than one month after the last dose. Elderly: No dose adjustment is necessary in elderly patients. Renal impairment: No dose adjustment is required in patients with renal impairment. Serum calcium should be monitored in patients with severe renal impairment or receiving dialysis. Hepatic impairment: No clinical trials have been conducted to evaluate the effect of hepatic impairment. Paediatric population: The safety and efficacy of romosozumab in paediatric patients (age <18 years) have not yet been established. No data are available. Method of administration: Subcutaneous use. To administer the 210 mg dose, 2 subcutaneous injections of romosozumab should be given into the abdomen, thigh, or upper arm. The second injection should be given immediately after the first one but at a different injection site. Administration should be performed by an individual who has been trained in injection techniques. **CONTRAINDICATIONS** Hypersensitivity to the active substance(s) or to any of the excipients. Hypocalcaemia. History of myocardial infarction or stroke. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE** Myocardial infarction and stroke: In randomised controlled studies, an increase in serious cardiovascular events (myocardial infarction and stroke) has been observed in romosozumab treated patients compared to controls. When determining whether to use romosozumab for an individual patient, consideration should be given to her fracture risk over the next year and her cardiovascular risk based on risk factors (e.g. established cardiovascular disease, hypertension, hyperlipidaemia, diabetes mellitus, smoking, severe renal impairment, age). Romosozumab should only be used if the prescriber and patient agree that the benefit outweighs the risk. If a patient experiences a myocardial infarction or stroke during therapy, treatment with romosozumab should be discontinued. Hypocalcaemia: Transient hypocalcaemia has been observed in patients receiving romosozumab. Hypocalcaemia should be corrected prior to initiating therapy with romosozumab and patients should be monitored for signs and symptoms of hypocalcaemia. If any patient presents with suspected symptoms of hypocalcaemia during treatment, calcium levels should be measured. Patients with severe renal impairment (estimated glomerular filtration rate [eGFR] 15 to 29 ml/min/1.73 m2) or receiving dialysis are at greater risk of developing hypocalcaemia and the safety data for these patients is limited. Calcium levels should be monitored in these patients. Hypersensitivity: Clinically significant hypersensitivity reactions, including angioedema, erythema multiforme, and urticaria occurred in the romosozumab group in clinical trials. If an anaphylactic or other clinically significant allergic reaction occurs, appropriate therapy should be initiated and use of romosozumab should be discontinued. Osteonecrosis of the jaw: Osteonecrosis of the jaw (ONJ), has been reported rarely in patients receiving romosozumab. All patients should be encouraged to maintain good oral hygiene, receive routine dental check-ups, and immediately report any oral symptoms such as dental mobility, pain or swelling or non-healing of sores or discharge during treatment with romosozumab. Patients who are suspected of having or who develop ONJ while on romosozumab should receive care by a dentist or an oral surgeon with expertise in ONJ. Discontinuation of romosozumab therapy should be considered until the condition resolves and contributing risk factors are mitigated where possible. Atypical femoral fractures: Atypical low-energy or low trauma fracture of the femoral shaft, which can occur spontaneously, has been reported rarely in patients receiving romosozumab. Any patient who presents with new or unusual thigh, hip, or groin pain should be suspected of having an atypical fracture and should be evaluated to rule out an incomplete femur fracture. Patient presenting with an atypical femur fracture should also be assessed for symptoms and signs of fracture in the contralateral limb. Interruption of romosozumab therapy should be considered, based on an individual benefit-risk assessment. Sodium content: This medicinal product contains less than 1 mmol sodium (23 mg) per dose, that is to say essentially sodium-free. **INTERACTIONS** No drug interaction studies have been performed with romosozumab. No pharmacokinetic drug interactions are expected with romosozumab. **PREGNANCY AND LACTATION** Pregnancy: Romosozumab is not indicated for use in women of child-bearing potential or in pregnant women. There are no data from the use of romosozumab in pregnant women. A risk for malformations of developing digits in the human foetus is low following romosozumab exposure due to the timing of digit formation in the first trimester in humans, a period when placental transfer of immunoglobulins is limited. Breast-feeding: Romosozumab is not indicated for use in breast-feeding women. No data are available on excretion of romosozumab in human milk. Human IgGs are known to be excreted in breast milk during the first few days after birth, which is decreasing to low concentrations soon afterwards; consequently, a risk to the breast-fed infant cannot be excluded during this short period. Fertility: No data are available on the effect of romosozumab on human fertility. Animal studies in female and male rats did not show any effects on fertility endpoints. **ADVERSE REACTIONS** The most common adverse reactions were nasopharyngitis (15.6%) and arthralgia (12.4%). Hypersensitivity-related reactions occurred in 6.7% of patients treated with romosozumab. Hypocalcaemia was reported uncommonly (0.4% of patients treated with romosozumab). In randomised controlled studies, an increase in serious cardiovascular events (myocardial infarction and stroke) has been observed in romosozumab treated patients compared to controls. Adverse reactions are presented in order of decreasing seriousness by System Organ Class: Infections and infestations: Nasopharyngitis, Sinusitis; Immune system disorders: Hypersensitivity, Rash, Dermatitis, Urticaria, Angioedema, Erythema multiforme; Metabolism and nutrition disorders: Hypocalcaemia; Nervous system disorders: Headache, Stroke; Eye disorders: Cataract; Cardiac disorders: Myocardial infarction; Musculoskeletal and connective tissue disorders: Arthralgia, Neck pain, Muscle spasms; General disorders and administration site conditions: Injection site reactions **OVERDOSE** There is no experience with overdose in clinical trials.

Abbreviated Prescribing Information Version No.: HKEVEPI01
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HK-08946-EVE-2022-Sep Date of Approval: 21 Sep 2022

Xarelto® rivaroxaban

BAYER

RIVA-DM : New Real-world Findings Confirm the Efficacy and Safety of Xarelto® in Patients with NVAF and Diabetes[1-3]

REDUCED RISKS WITH XARELTO® vs WARFARIN

Composite Outcome[1]

↓9%

SSE/CV death

Diabetes-related Complications

↓19%

Need for dialysis or renal transplant[2]

↓15%

MALE[2]

↓15%

Any type of diabetic retinopathy[3]

Bleeding Events[1]

↓20%

Major bleeding

↓28%

ICH

RIVA-DM study was a cohort analysis within the US Optum® De-Identified EHR dataset between 2010 to 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: 1. Coleman CI, et al. Thromboembolism, bleeding and vascular death in nonvalvular atrial fibrillation patients with type 2 diabetes receiving rivaroxaban or warfarin. Cardiovasc Diabetol 2021;20:52. **2.** Costa OS, et al. Kidney, limb and ophthalmic complications and death in patients with nonvalvular atrial fibrillation and type 2 diabetes prescribed rivaroxaban or warfarin: an electronic health record analysis. EHRA Congress. 23–25 April 2021. **3.** Costa OS, et al. Ophthalmic complications in patients with nonvalvular atrial fibrillation and type 2 diabetes prescribed rivaroxaban or warfarin. EHRA Congress. 23–25 April 2021.

Xarelto 10 mg / 15 mg / 20 mg film-coated tablets
Abbreviated Prescribing Information (Please refer to the full prescribing information before prescribing)
Composition: Active ingredient: 10 mg / 15 mg / 20 mg rivaroxaban. Excipients: Microcrystalline cellulose, croscarmellose sodium, lactose monohydrate, hypromellose, sodium laurilsulfate, magnesium stearate, macrogol 3350, titanium dioxide (E171), iron oxide red (E172). **Indication and Posology:** Prevention of stroke and systemic embolism in adult patients with non-valvular atrial fibrillation (NVAF) with one or more risk factors, such as congestive heart failure, hypertension, age ≥ 75 years, diabetes mellitus, prior stroke or transient ischaemic attack: Recommended dose is 20 mg once daily (recommended maximum dose). Treatment of deep vein thrombosis (DVT) and pulmonary embolism (PE) and prevention of recurrent DVT and PE in adults: The recommended dose for the initial treatment of acute DVT or PE is 15 mg twice daily for the first three weeks followed by 20 mg once daily for the continued treatment and prevention of recurrent DVT and PE. When extended prevention of recurrent DVT and PE is indicated (following completion of at least 6 months therapy for DVT or PE), the recommended dose is 10 mg once daily. A dose of 20 mg once daily should be considered in patients with high risk. Prevention of venous thromboembolism (VTE) in adult patients undergoing elective hip or knee replacement surgery: The recommended dose is 10 mg once daily. The initial dose should be taken 6 to 10 hours after surgery, provided that haemostasis has been established. For patients undergoing major hip surgery, a treatment duration of 5 weeks is recommended. For patients undergoing major knee surgery, a treatment duration of 2 weeks is recommended. **Patients with NVAF who undergo percutaneous coronary intervention (PCI) with stent placement:** There is limited experience of a reduced dose of 15 mg Xarelto once daily (or 10 mg Xarelto once daily for patients with moderate renal impairment [creatinine clearance 30 – 49 ml/min]) in addition to a P2Y12 inhibitor for a maximum of 12 months in patients with non-valvular atrial fibrillation who require oral anticoagulation and undergo PCI with stent placement. **Renal impairment:** No dose adjustment is necessary in patients with mild renal impairment (creatinine clearance 50 - 80 ml/min). In patients with moderate (creatinine clearance 30 - 49 ml/min) or severe (creatinine clearance 15 - 29 ml/min) renal impairment the following dosage recommendations apply: For the prevention of stroke and systemic embolism in patients with non-valvular atrial fibrillation, the recommended dose is 15 mg once daily. For the treatment of DVT, treatment of PE and prevention of recurrent DVT and PE: 15 mg twice daily for the first 3 weeks. Thereafter, the recommended dose is 20 mg once daily. When the recommended dose is 10 mg once daily, no dose adjustment from the recommended dose is necessary. Limited clinical data for patients with severe renal impairment (creatinine clearance 15 - 29 ml/min) indicate that rivaroxaban plasma concentrations are significantly increased; therefore, Xarelto is to be used with caution in these patients. Use is not recommended in patients with creatinine clearance < 15 ml/min. **Contraindications:** Hypersensitivity to the active substance or any of the excipients; active clinically significant bleeding; lesion or condition if considered a significant risk for major bleeding; concomitant treatment with any other anticoagulants except under specific circumstances of switching anticoagulant therapy or when unfractionated heparin is given at doses necessary to maintain an open central venous or arterial catheter; hepatic disease associated with coagulopathy and clinically relevant bleeding risk including cirrhotic patients with Child Pugh B and C; pregnancy and breast feeding. **Warnings and Precautions:** Clinical surveillance in line with anticoagulation practice is recommended throughout the treatment period. Not recommended: in patients receiving concomitant systemic treatment with strong concurrent CYP3A4- and P-gp-inhibitors, i.e. azole antimycotics or HIV protease inhibitors; in patients with increased bleeding risk; in patients with severe renal impairment (creatinine clearance < 15 ml/min); in the treatment of acute pulmonary embolism; due to lack of data: in patients below 18 years of age, in patients with prosthetic heart valves, in patients concomitantly treated with dronedarone, in NVAF-PCI patients with a history of stroke/transient ischemic attack. Use with caution: please refer to the full prescribing information. Xarelto contains lactose. **Undesirable effects:** Common: anaemia, dizziness, headache, eye haemorrhage, hypotension, haematoma, epistaxis, haemoptysis, gingival bleeding, gastrointestinal tract haemorrhage, gastrointestinal and abdominal pains, dyspepsia, nausea, constipation, diarrhoea, vomiting, pruritus, rash, ecchymosis, cutaneous and subcutaneous haemorrhage, pain in extremity, urogenital tract haemorrhage, fever, renal impairment, peripheral oedema, decreased general strength and energy, increase in transaminases, post-procedural haemorrhage, contusion, wound secretion. Other undesirable effects (uncommon, rare, frequency not known): please refer to the full prescribing information.

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PP-XAR-HK-0219-1 09/2021 4IC_1673

SPARK THE MOTIVATION FOR CHANGE

trulicity 易週糖
dulaglutide injection once weekly
0.75 mg/0.5 mL, 1.5 mg/0.5 mL

Help your T2DM patients to start and stay on once-weekly Trulicity

Choose Trulicity as 1st Injectable with All-round Benefits

Powerful HbA1c reduction[1,*]

Proven CV benefit in patients with or without established CVD[2,3,†]

Simple once-weekly dosing in a ready-to-use pen with hidden needle[1,4,5]

Better adherence shown in real-world studies vs. other GLP-1 RAs[6,7,‡]

* Trulicity 1.5 mg demonstrated statistically superior HbA1c reduction in patients with type 2 diabetes in 8 phase III clinical trials vs metformin, sitagliptin, exenatide BID, insulin glargine, and/or placebo. Trulicity 1.5 mg demonstrated noninferior HbA1c reduction vs liraglutide 1.8 mg, as well as noninferiority vs insulin glargine in patients with type 2 diabetes and CKD[1].

† Trulicity 1.5 mg significantly reduced the risk of MACE-3 (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[2,3].

‡ In real-world studies, ~40% more patients with T2DM were adherent to once-weekly Trulicity relative to once-weekly semaglutide[6]; Moreover, persistence with Trulicity was the highest among GLP-1 RAs including twice-daily exenatide, once-weekly exenatide, liraglutide and lixisenatide[7].

BID=twice daily; CKD=chronic kidney disease; CV=cardiovascular; CVD=cardiovascular disease; GLP-1 RA=glucagon-like peptide-1 receptor agonists; HbA1C=haemoglobin A1c; MACE=major adverse cardiovascular event; MI=myocardial infarction; T2DM=type 2 diabetes mellitus.

References: 1. Trulicity Hong Kong Prescribing Information. 2. Gerstein HC et al. Lancet. 2019;394:121-130. 3. Gerstein HC et al. Diabetes Obes Metab. 2018;20:42-49. 4. Trulicity 0.75 mg Instructions for Use. 5. Trulicity 1.5 mg Instructions for Use. 6. Mody R et al. Diabetes Obes Metab. 2021;23:106-115. 7. Divino V et al. Diabetes Ther. 2019;10:1067-1088.

Trulicity Abbreviated Prescribing Information.

Indication: 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 medicinal products for the treatment of diabetes. **Dosage:** Adult Monotherapy: 0.75 mg once weekly. Add-on therapy: 1.5 mg once weekly. Elderly ≥75 years old: Initially 0.75 mg once weekly. Renal impairment: No dosage adjustment is required in patients with mild, moderate or severe renal impairment (eGFR <90 to ≥15 mL/min/1.73m2). **Administration:** To be injected subcutaneously in the abdomen, thigh or upper arm. It should not be administered intravenously or intramuscularly. The dose can be administered at any time of day, with or without meals. **Contraindications:** Hypersensitivity to dulaglutide or any of its excipients. **Special Precautions:** Do not use in patients with type 1 diabetes mellitus or for the treatment of diabetic ketoacidosis. Do not administer IV. Acute pancreatitis. Hypoglycaemia. Limited experience in patients with congestive heart failure. **Adverse Drug Reactions:** Abdominal distention, abdominal pain, acute pancreatitis, constipation, decreased appetite, dehydration, diarrhoea, dyspepsia, eructation, fatigue, first-degree atrioventricular block, flatulence, gastroesophageal reflux disease, hypoglycaemia, injection site reactions, nausea, sinus tachycardia, vomiting. EUSPC21OCT2019. **Full prescribing information is available upon request.**

PP-DG-HK-0219 09/2021 4iC_1934

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Increasing FREE WATER CLEARANCE with SAMSCA®

SAMSCA® is effective at raising serum Na^+ in HF patients over 30 days[1]

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

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

SAMSCA® has a significant effect on fluid balance in HF patients[1]

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

SAMSCA® -1860mL vs. Placebo -787mL

Indication[2]

SAMSCA® is indicated for the treatment of clinically significant hypervolemic and euvolemic hyponatremia (serum sodium <125mEq/L or less marked hyponatremia that is symptomatic and has resisted correction with fluid restriction), including patients with heart failure and Syndrome of Inappropriate Antidiuretic Hormone (SIADH).

*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 hyponatremia (<135 mEq/L) in a euvolemic or hypervolemic state were randomized to SAMSCA® (n=225) or placebo (n=223). SAMSCA® was started at 15 mg daily, then daily or less frequent titration to 30 mg daily or 60 mg daily as dictated by the individual subject serum sodium response. The two primary end points for all patients were the change in the average daily area under the curve for the serum sodium concentration from baseline to day 4 and the change from baseline to day 30.[1]

References: **1. Integrated Summary of Efficacy of Tolvaptan for the Indication of Hyponatremia (2007). Otsuka Pharmaceutical Development & Commercialization, Inc.**
2. SAMSCA® (tolvaptan) Hong Kong Prescribing Information revised Mar 2019.

HF: Heart failure; Na^+: Sodium

Abbreviated Prescribing Information

SAMSCA (tolvaptan) 15 mg & 30 mg oral tablets. INDICATION: treatment of clinically significant hypervolemic and euvolemic hyponatremia [serum sodium <125 mEq/L or less marked hyponatremia that is symptomatic and has resisted correction with fluid restriction], including patients with heart failure and Syndrome of Inappropriate Antidiuretic Hormone (SIADH). DOSAGE: Patients should be in a hospital for initiation and re-initiation of therapy to evaluate the therapeutic response. Too rapid correction of hyponatremia (e.g., >12 mEq/L/24 hours) can cause osmotic demyelination. Recommended starting dose: 15 mg once daily. Dosage may be increased at intervals ≥ 24 hr to 30 mg once daily, and to a maximum of 60 mg once daily. Limit use to 30 days to minimize the risk of liver injury. Avoid fluid restriction during the first 24 hours of therapy. CONTRAINDICATION: Autosomal Dominant Polycystic Kidney Disease; Urgent need to raise serum sodium acutely; Inability of the patient to sense or appropriately respond to thirst; Hypovolemic hyponatremia; Concomitant use of strong CYP 3A inhibitors e.g. clarithromycin, ketoconazole, itraconazole; Anuric patients; Hypersensitivity. SPECIFIC POPULATIONS: Only used during pregnancy if potential benefits justify the risk to the fetus. Avoid use in patients with underlying liver disease. Not recommended for patients with CrCl <10 mL/min. WARNINGS AND PRECAUTIONS: Avoid coadministration with moderate CYP 3A inhibitors. Too rapid correction of serum sodium can cause serious neurologic sequelae. Liver injury & discontinue treatment when patients develop symptoms indicative of liver injury. Dehydration and Hypovolemia. Co-administration with hypertonic saline not recommended. Avoid co-administration with CYP 3A inducers. Samsca may be increased when co-administrated with P-gp inhibitors. Monitor sign of hyperkalemia and cautious when co-administrated with drugs that increase serum potassium. ADVERSE REACTIONS: Thirst, dry mouth, asthenia, constipation, pollakiuria or polyuria, & hyperglycemia, pyrexia & anorexia. DRUG INTERACTIONS: CYP 3A inhibitors, grapefruit Juice, P-gp Inhibitors, rifampin and other CYP 3A Inducers, concomitant use increases digoxin AUC/Cmax. For details, please refer to the full prescribing information which is available upon request (HK REVISED: 03/2019).

HKOP-SAM-202202-001

Samsca (tolvaptan)

Otsuka Otsuka Pharmaceutical (H.K.) Ltd.
21/F, East Exchange Tower, 38 Leighton Road, Causeway Bay, Hong Kong. Tel: 2881 6299 Fax: 2577 5206

LEQVIO®
(inclisiran) injection 284 mg/1.5 mL

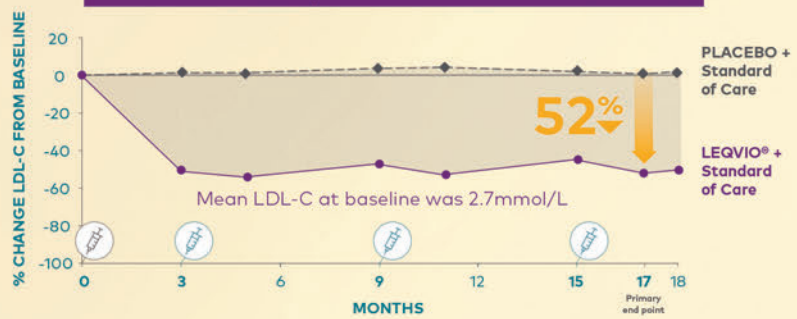
2 DOSES A YEAR*

FOR EFFECTIVE AND SUSTAINED LDL-C REDUCTION[1†]

FDA APPROVED[2]

EMA APPROVED[3]

In ORION-10 (N=1,561), LEQVIO® demonstrated LDL-C reduction in patients with established **ASCVD**[1,4]



Patients in both study arms were on a maximally tolerated statin.[1,4]

In ORION-10 clinical trial, LEQVIO® demonstrated LDL-C reduction in ASCVD patients:[4]

52% EFFECTIVE LDL-C REDUCTION

Between-group difference of -52.3% (95% CI: -55.7%, -48.8%; P<0.001) refers to the difference between the placebo group (1.0%) and the LEQVIO® group (-51.3%) at month 17.

*LEQVIO® is dosed initially, again at 3 months, and then once every 6 months.[1]
†LDL-C reduction was maintained during each 6-month dosing interval.[1]

Study design: ORION-10 was a multicenter, double-blind, randomized, placebo-controlled 18-month clinical trials. Patients with established ASCVD were taking a maximally tolerated dose of statin with or without other lipid-modifying therapy and required additional LDL-C reduction. The ORION-11 trial, in addition to patients with ASCVD, included adults who were ASCVD risk equivalent (type 2 diabetes, familial hypercholesterolemia, or a 10-year risk of a cardiovascular event of >20% as assessed by the Framingham Risk Score for Cardiovascular Disease or equivalent).

Abbreviations: ASCVD, atherosclerotic cardiovascular disease; CI, confidence interval; LDL-C, low-density lipoprotein cholesterol

References: 1. Leqvio. Hong Kong Prescribing Information. Novartis Pharmaceuticals. 2021. **2.** U.S. Food & Drug Administration. FDA approves add-on therapy to lower cholesterol among certain high-risk adults. https://www.fda.gov/drugs/news-events-human-drugs/fda-approves-add-therapy-lower-cholesterol-among-certain-high-risk-adults. Published Dec 2021. Accessed on 12 Apr 2022. **3.** European Medicine Agency. https://www.ema.europa.eu/en/medicines/human/EPAR/leqvio. Accessed on 22 Mar 2022. **4.** Ray KK, Wright RS, Kallend D, et al; ORION-10 and ORION-11 Investigators. Two phase 3 trials of inclisiran in patients with elevated LDL cholesterol. N Engl J Med. 2020;382(16):1507-1519.

Leqvio® Important note: Before prescribing, consult full prescribing information. **Presentation: Solution for injection:** Each pre-filled syringe contains 1.5 mL of solution containing 284 mg inclisiran (equivalent to 300 mg inclisiran sodium). **Indications:** Leqvio is indicated in adults with primary hypercholesterolaemia (heterozygous familial and nonfamilial) or mixed dyslipidaemia, as an adjunct to diet: • in combination with a statin or statin with other lipid lowering therapies in patients unable to reach LDLC goals with the maximum tolerated dose of a statin, or • alone or in combination with other lipid lowering therapies in patients who are statin intolerant, or for whom a statin is contraindicated. **Dosage and administration:** *Recommended dose:* 284 mg inclisiran administered as a single subcutaneous injection: initially, again at 3 months, followed by every 6 months. *Missed dose:* • If a planned dose is missed by less than 3 months, inclisiran should be administered and dosing continued according to the patient's original schedule. • If a planned dose is missed by more than 3 months, a new dosing schedule should be started – inclisiran should be administered initially, again at 3 months, followed by every 6 months. *Treatment Transition from PCSK9 Inhibitor Monoclonal Antibody:* Inclisiran can be administered immediately after the last dose of a monoclonal antibody PCSK9 inhibitor. To maintain LDL C lowering it is recommended that inclisiran is administered within 2 weeks after the last dose of a monoclonal antibody PCSK9 inhibitor. **Special populations:** *Renal impairment:* No dose adjustments are necessary for patients with mild, moderate or severe renal impairment or patients with end stage renal disease. There is limited experience with inclisiran in patients with severe renal impairment. Inclisiran should be used with caution in these patients. *Hepatic impairment:* No dose adjustments are necessary for patients with mild (Child Pugh class A) or moderate (Child Pugh class B) hepatic impairment. No data are available in patients with severe hepatic impairment (Child Pugh class C). Inclisiran should be used with caution in patients with severe hepatic impairment. *Pediatric patients (below 18 years):* The safety and efficacy of inclisiran have not been established. *Geriatric patients (65 years of age or above):* No dose adjustment is necessary. **Method of administration:** Intended for administration by a healthcare professional. For subcutaneous injection into the abdomen, alternative injection sites include the upper arm or thigh. Injections should not be given into areas of active skin disease or injury such as sunburns, skin rashes, inflammation or skin infections. Leqvio should be inspected visually for particulate matter prior to administration. Each pre-filled syringe is for single use only. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and precautions:** ***Haemodialysis:*** Considering that inclisiran is eliminated renally, haemodialysis should not be performed for at least 72 hours after inclisiran dosing. **Pregnancy, lactation, females and males of reproductive potential** ***Pregnancy:*** There are no or limited amount of data from the use of inclisiran in pregnant women. Animal studies do not indicate direct or indirect harmful effects with respect to reproductive toxicity. As a precautionary measure, it is preferable to avoid the use of inclisiran during pregnancy. ***Lactation:*** It is unknown whether inclisiran is excreted in human milk. Available pharmacodynamic/toxicological data in animals have shown excretion of inclisiran in milk. A risk to newborns/infants cannot be excluded. A decision must be made whether to discontinue breast feeding or to discontinue/abstain from inclisiran therapy, taking into account the benefit of breast feeding for the child and the benefit of therapy for the woman. ***Infertility:*** No human data. No effects on animal fertility. **Adverse drug reactions:** ***Common (≥1 to <10%):*** Adverse events at the injection site (includes injection site reaction, injection site pain, injection site erythema, and injection site rash). **Interactions:** Not a substrate, inhibitor or inducer of CYP450 enzymes or common drug transporters. Not expected to have clinically significant interactions with other medications. Drug-drug interaction assessments demonstrated a lack of clinically meaningful interactions with either atorvastatin, rosuvastatin or other statins. **Packs:** Solution in pre-filled syringe: 1's **Legal classification:** P1S1S3 Last revision: Sep 2021 Ref: EU Dec 2020

HK220412049?

NOVARTIS | Reimagining Medicine

Licensed from Alnylam Pharmaceuticals, Inc.

Novartis Pharmaceuticals (HK) Ltd.
7/F, Citi Tower, One Bay East, 83 Hoi Bun Road, Kwun Tong, Kowloon, Hong Kong
Telephone: (852) 2882 5222 | Fax: (852) 2577 0274

Change the heart. Change heart failure [1,4,7,9]

Reverse cardiac remodelling, improve cardiac structure and function, and target HF via a unique dual MOA that inhibits neprilysin and RAAS

Provide the HF treatment superior to ACEi in all stages of the HFrEF patient journey [4-6,8]

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

*In place of an ACEi or ARB **ACEi**=angiotensin-converting enzyme inhibitor; **ARB**=angiotensin II 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

REFERENCES. **1.** McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J.* 2021;00:1-128. **2.** Claggett B, Packer M, McMurray JJV, et al; for the PARADIGM-HF Investigators. Estimating the long-term treatment benefits of sacubitril–valsartan. *N Engl J Med.* 2015;373(23):2289-2290. **3.** Lewis EF, Claggett BL, McMurray JJV, et al. Health-related quality of life outcomes in PARADIGM-HF. *Circ Heart Fail.* 2017;10(8):e003430. **4.** ENTRESTO Summary of product characteristics. European Medicines Agency website. http://www.ema.europa.eu. Accessed 2018. **5.** Solomon SD, Claggett B, Packer M, et al. Efficacy of sacubitril/valsartan relative to a prior decompensation: The PARADIGM-HF trial. *JACC Heart Fail.* 2016;4(10):816-822. **6.** Velazquez EJ, Morrow DA, DeVore AD, et al; for the PIONEER-HF Investigators. Angiotensin–neprilysin inhibition in acute decompensated heart failure. *N Engl J Med.* 2019;380(6):539-548. **7.** Desai AS, Solomon SD, Shah AM, et al; for the EVALUATE-HF Investigators. Effect of sacubitril-valsartan vs enalapril on aortic stiffness in patients with heart failure and reduced ejection fraction: a randomized clinical trial. *JAMA.* 2019;322(11):1077-1084. **8.** Wachter R, Senni M, Belohlavek J, et al; on behalf of the TRANSITION Investigators. Initiation of sacubitril/valsartan in haemodynamically stabilised heart failure patients in hospital or early after discharge: primary results of the randomised TRANSITION study. *Eur J Heart Fail.* 2019;21(8):998-1007. **9.** Januzzi JL Jr, Prescott MF, Butler J, et al; for the PROVE-HF Investigators. Association of change in N-terminal pro–b-type natriuretic peptide following initiation of sacubitril-valsartan treatment with cardiac structure and function in patients with heart failure with reduced ejection fraction. *JAMA.* 2019;322(11):1085-1095. **10.** Ramani GV, Uber PA, Mehra MR. Chronic heart failure: contemporary diagnosis and management. *Mayo Clin Proc.* 2010;85(2):180-195. **11.** Chandra A, Lewis EF, Claggett BL, et al. Effects of sacubitril/valsartan on physical and social activity limitations in patients with heart failure: a secondary analysis of the PARADIGM-HF trial. *JAMA Cardiol.* 2018;3(6):498-505. **12.** Heidenreich P, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulations. 2022;101161CIR0000000000001062.

Entresto®
sacubitril/valsartan

The Essential HF Intervention

ENTRESTO tablets **Important note:** Before prescribing, consult full prescribing information. **Presentation: ENTRESTO 50 mg film-coated tablets** Each film-coated tablet contains 24.3 mg sacubitril and 25.7 mg valsartan (as sacubitril valsartan sodium salt complex). **ENTRESTO 100 mg film-coated tablets** Each film-coated tablet contains 48.6 mg sacubitril and 51.4 mg valsartan (as sacubitril valsartan sodium salt complex). **ENTRESTO 200 mg film-coated tablets** Each film-coated tablet contains 97.2 mg sacubitril and 102.8 mg valsartan (as sacubitril valsartan sodium salt complex). **Indications:** Treatment of symptomatic chronic heart failure (NYHA class II-IV) in adult patients with reduced ejection fraction to reduce the risk of cardiovascular death and hospitalization due to heart failure. **Dosage and administration: Adults:** •The recommended starting dose of ENTRESTO is 100 mg twice daily. The dose should be doubled at 2-4 weeks to the target dose of one tablet of 200 mg twice daily, as tolerated by the patient. •A starting dose of 50 mg twice daily is recommended for patients not currently taking an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin II receptor blocker (ARB), and should be considered for patients previously taking low doses of these agents. **•Geriatric patients:** The dose should be in line with the renal function. **•Pediatric patients:** ENTRESTO has not been studied. Use of ENTRESTO is not recommended. **•Renal impairment:** No dose adjustment is required in patients with mild renal impairment (Estimated Glomerular Filtration Rate [eGFR] 60-90 ml/min/1.73 m^2); A starting dose of 50 mg twice daily is recommended in patients with moderate renal impairment(eGFR 30-60 ml/min/1.73 m^2); A starting dose of 50 mg twice daily and caution is recommended in patients with severe renal impairment (eGFR <30 ml/min/1.73 m^2). Not recommended for patients with end-stage renal disease. **•Hepatic impairment:** No dose adjustment is required in patients with mild hepatic impairment (Child-Pugh A classification). A starting dose of 50 mg twice daily is recommended in patients with moderate hepatic impairment (Child-Pugh B classification) or with AST/ALT values more than twice the upper limit of the normal range. In patients with severe hepatic impairment use of ENTRESTO is not recommended. **•Method of administration** For oral use. May be administered with or without food. **Contraindications:** •Hypersensitivity to the active substance, sacubitril, valsartan, or to any of the excipients. •Concomitant use with ACE inhibitors. ENTRESTO must not be administered until 36 hours after discontinuing ACE inhibitor therapy. •Known history of angioedema related to previous ACE inhibitor or ARB therapy. •Concomitant use with aliskiren in patients with diabetes mellitus or in patients with renal impairment (eGFR <60 ml/min/1.73 m^2). • Second and third trimester of pregnancy. •Hereditary or idiopathic angioedema •Severe hepatic impairment, biliary cirrhosis and cholestasis **Warnings and precautions: •Dual blockade of the Renin-Angiotensin-Aldosterone System (RAAS):** ENTRESTO must not be administered with an ACE inhibitor due to the risk of angioedema. ENTRESTO must not be initiated until 36 hours after taking the last dose of ACE inhibitor therapy. If treatment with ENTRESTO is stopped, ACE inhibitor therapy must not be initiated until 36 hours after the last dose of ENTRESTO. • The combination of ENTRESTO with direct renin inhibitors such as aliskiren is not recommended. The combination of ENTRESTO with aliskiren-containing products is contraindicated in patients with diabetes mellitus or in patients with renal impairment (eGFR <60 ml/min/1.73 m^2). • ENTRESTO contains valsartan, and therefore should not be co-administered with another ARB containing product. **•Hypotension:** If hypotension occurs, temporary down-titration or discontinuation of ENTRESTO is recommended. Dose adjustment of diuretics, concomitant antihypertensive drugs, and treatment of other causes of hypotension (e.g. hypovolemia) should be considered. Sodium and/or volume depletion should be corrected before starting treatment with ENTRESTO. **•Impaired renal function:** •Evaluation of patients with heart failure should always include assessment of renal function. Down titration of ENTRESTO should be considered in patients who develop a clinically significant decrease in renal function. Caution should be exercised when administering ENTRESTO in patients with severe renal impairment (eGFR <30 ml/min/1.73 m^2). **•Hyperkalemia:** Treatment should not be initiated if the serum potassium level is >5.4 mmol/l. Medications known to raise potassium levels (e.g. potassium-sparing diuretics, potassium supplements) should be used with caution. If clinically significant hyperkalemia occurs, measures such as adjustment of concomitant medicinal products, temporary down–titration or discontinuation should be considered. Monitoring of serum potassium is recommended especially in patients with risk factors such as renal impairment, diabetes mellitus, hypoaldosteronism, receiving a high potassium diet or mineralocorticoid antagonists. If serum potassium level is >5.4 mmol/l discontinuation should be considered. **•Angioedema:** If angioedema occurs, ENTRESTO should be immediately discontinued and appropriate therapy and monitoring should be provided until complete and sustained resolution of signs and symptoms has occurred. ENTRESTO must not be re-administered. Patients with a prior history of angioedema were not studied. As they may be at higher risk for angioedema, caution is recommended if ENTRESTO is used in these patients. ENTRESTO must not be used in patients with a known history of angioedema related to previous ACE inhibitor or ARB therapy. Black patients may have increased susceptibility to develop angioedema. **•Patients with renal artery stenosis:** Caution is required in patients with renal artery stenosis and monitoring of the renal function is recommended. **•Patients with NYHA functional classification IV:** Caution should be exercised. **•B-type natriuretic peptide (BNP):** BNP is not a suitable biomarker of heart failure in patients treated with ENTRESTO. **•Hepatic impairment:** Caution is recommended when using ENTRESTO in patients with moderate hepatic impairment (Child-Pugh B classification) or with AST/ALT values more than twice the upper limit of the normal range. ENTRESTO is contraindicated in patients with severe hepatic impairment, biliary cirrhosis or cholestasis (Child-Pugh C classification). **Pregnancy:** The use of ENTRESTO is not recommended during the first trimester of pregnancy and is contraindicated during the second and third trimesters of pregnancy. **Breast-feeding:** It is not known whether ENTRESTO is excreted in human milk. Because of the potential risk for adverse drug reactions in breastfed newborns/infants, ENTRESTO is not recommended during breastfeeding. **Adverse drug reactions:** ***Very common (≥1/10):*** Hyperkalemia, hypotension, renal impairment. ***Common (≥1/100 to <1/10):*** Anaemia, hypokalemia, Hypoglycaemia, Dizziness, Cough, Headache, Syncope, Vertigo, Orthostatic hypotension, Diarrhoea, Nausea, Gastritis, Renal failure (renal failure, acute renal failure), Fatigue, Asthenia ***Uncommon (≥1/1,000 to <1/100):*** Hypersensitivity, Dizziness postural, Pruritis, Rash, Angioedema. **Interactions:** ***•Concomitant use contraindicated:*** aliskiren in patients with diabetes mellitus or in patients with renal impairment (eGFR <60 ml/min/1.73 m^2); Use with ACE inhibitors: ENTRESTO must not be started until 36 hours after taking the last dose of ACE inhibitor therapy. ACE inhibitor therapy must not be started until 36 hours after the last dose of ENTRESTO. ***•Concomitant use not recommended:*** ARB containing products ***•Caution when used concomitantly*** with OAT1B1 and OATP1B3 substrates(e.g. statins), PDE5 inhibitors (e.g. sildenafil), lithium, potassium-sparing diuretics (triamterene, amiloride) , mineral corticoid antagonists (e.g. spironolactone, eplerenone), potassium supplements, salt substitutes containing potassium, other agents that may lead to increased serum potassium level (e.g. heparin), non-steroidal anti-inflammatory agents (NSAIDs) including selective cyclooxygenase-2 inhibitors (COX-2 Inhibitors), inhibitors of OATP1B1, OATP1B3, OAT3 (e.g. rifampin, cyclosporine),OAT1 (e.g. tenofovir, cidofovir) or MPR2 (e.g. ritonavir), furosemide, nitrates (e.g. nitroglycerine), metformin. **Packs:** 50mg: 28's; 100mg: 28's and 56's; 200mg: 56's. Not all pack sizes may be marketed. **Legal classification:** P1S1S3. **Ref:** EMA Nov 2015. **FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.**

NOVARTIS

Novartis Pharmaceuticals (HK) Ltd
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Tel: 2882 5222 Fax: 2577 0274

HK2209152844

For adults with T2D uncontrolled on basal insulin + OADs

SoliMix

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:[1‡]

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[1]

SUPERIOR HbA1c REDUCTION

Relative reduction by SOLIQUA™ vs premix

-18.2%

LS mean difference (97.5% CI): -0.2% (-0.4, -0.1)%; p<0.001§

SUPERIOR WEIGHT CHANGE

-1.86 KG

LS mean difference (95% CI: -2.28, -1.43); p<0.001

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)

LESS HYPOGLYCAEMIA

LEVEL-2 HYPOGLYCAEMIA

-55%

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

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

*SoliMix was a multicentre, open-label, parallel group, randomised, controlled trial to compare SOLIQUA™ vs BIAsp 30 in patients with T2D (n = 887) who failed to achieve glycaemic control with BI and OADs. Primary endpoints were non inferiority of SOLIQUA™ vs BIAsp 30 on HbA1c change from baseline to Week 26 and superiority of SOLIQUA™ vs BIAsp 30 on weight change from baseline to Week 26. Key secondary endpoints included proportion of patients reaching HbA1c target <7% without weight gain at Week 26; proportion of patients reaching target without hypoglycaemia (plasma glucose <70 mg/dl) and weight gain at Week 26; and superiority of SOLIQUA™ vs BIAsp 30 in HbA1c reduction.[1]

†BIAsp 30 (30% insulin aspart + 70% insulin aspart protamine).

‡Adults who have been treated with any BI combined with 1 or 2 OADs that could be metformin alone or metformin + an SGLT2 inhibitor.[3]

§Non-inferiority p-value was calculated using a non-inferiority margin of 0.3%.

‖Hypoglycaemia was defined as plasma glucose (PG) <70 mg/dl having occurred at any point within the 26-week treatment period. Post hoc analysis of nocturnal hypoglycaemia, defined as occurring between bedtime and waking, and between 00:00 h – 06:00 h, is not included here. Severe hypoglycaemia, an event requiring external assistance for recovery, was rare with only 1 episode for SOLIQUA™ and 2 in the premix insulin group. Overall safety and tolerability profiles of SOLIQUA™ and premix insulin were consistent with the known safety profile of each product. Gastrointestinal (GI) disorders were more common in the SOLIQUA™ group vs premix insulin: nausea 7.7% vs 0.0%, vomiting 1.1% vs 0.2%, and dyspepsia 0.9% vs 0.2%, respectively.[1,4‡]

¶The higher incidence of AEs observed for SOLIQUA™ vs premix insulin was due to nausea and led to treatment discontinuation in 0.5% of patients. Over the 26-week treatment period, 32.6 % of SOLIQUA™ patients and 27.7% of premix insulin patients reported treatment-emergent adverse events; 2.7% vs 2.9%, respectively, reported serious adverse events. The rate of discontinuation due to any adverse event was 0.9% for each group.

BI, basal insulin; CI, confidence interval; LS, least squares; OADs, oral antidiabetic drugs; SGLT2, sodium-glucose cotransporter-2; T2D, type 2 diabetes.

References: 1. Rosenstock J, et al. Diabetes Care. 2021:dc210393. 2. SOLIQUA™ SmPC as of July 2020. 3. McCrimmon RJ, et al. Diabetes Obes Metab. 2021;23(6):1221-1231. 4. DoF 15017 study results.

Presentation: 100 units of insulin glargine and 33 micrograms lixisenatide in prefilled pen AND 100 units of insulin glargine and 50 micrograms lixisenatide in prefilled pen. Indications: For the treatment of adults with insufficiently controlled type 2 diabetes mellitus to improve glycaemic control as an adjunct to diet and exercise in addition to metformin with or without SGLT-2 inhibitors. Dosage: The dose must be individualised based on clinical response and is titrated based on the patient's need for insulin. The lixisenatide dose is increased or decreased along with insulin glargine dose and also depends on which pen is used. Please refer to the full prescribing information for guidelines. Administration: Subcutaneous injection in the abdomen, deltoid, or thigh. Injection sites should be rotated within the same region from one injection to the next. Soliqua must not be drawn from the cartridge of the pre-filled pen into a syringe. Contraindications: Hypersensitivity to the active substances or to any of the excipients. Patients with type 1 diabetes mellitus. Treatment of diabetic ketoacidosis. 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. Hepatic impairment: Frequent glucose monitoring and dose adjustment may be necessary. Hypoglycaemia may occur if dose is higher than required. Advise patients to take precautions to avoid hypoglycaemia while driving and using machines. Discontinue Soliqua if pancreatitis is suspected. Restart lixisenatide if acute pancreatitis is confirmed. Exercise caution in patients with pancreatitis history. Not recommended in patients with severe gastrointestinal disease. Use with caution in patients receiving oral medicinal products that require rapid gastrointestinal absorption. Potential risk of dehydration. Use may cause formation of antibodies against insulin glargine and/or lixisenatide. Always check pen label before each injection to avoid accidental mix-ups. Soliqua was not studied in combination with DPP-4 inhibitors, sulfonylurea, glinides, and pioglitazone. Interactions: Effects enhanced by anti-hyperglycaemics, ACEI, disopyramide, fibrates, fluoxetine, MAOIs, pentoxifylline, propoxyphene, salicylates, sulphonamide antibiotics. Effects reduced by corticosteroids, danazol, diazoxide, diuretics, glucagon, isoniazid, oestrogens and progestogens, phenothiazine derivatives, somatropin, sympathomimetics, thyroid hormones, atypical antipsychotics and protease inhibitors. Beta-blockers, clonidine, lithium or alcohol may either potentiate or weaken the effects of insulin. Pentamidine may cause hypoglycaemia, followed by hyperglycaemia. The signs of adrenergic counter-regulation may be reduced or absent under the influence of sympatholytic medicinal products such as beta-blockers, clonidine, guanethidine and reserpine. Fertility, pregnancy and lactation: Soliqua should not be used during pregnancy and breast-feeding. It is unknown whether insulin glargine or lixisenatide is excreted in human milk. Overdose: Overdose may lead to hypoglycaemia and gastrointestinal adverse reactions. Mild episodes of hypoglycaemia can usually be treated with oral carbohydrates. More severe episodes with coma, seizure or neurologic impairment may be treated with glucagon (intramuscular or subcutaneous) or concentrated glucose solution (intravenous). Undesirable effects: Hypoglycaemia is very common. For common, uncommon and not known undesirable effects, please refer to the full prescribing information. Storage: Before first use: Store in a refrigerator (2°C - 8°C). Do not freeze. Protect from light. After first use: Store below 25°C. Use within 28 days. Do not refrigerate or freeze. Preparation: Soliqua 3 x 3ml prefilled pen, 5 x 3ml prefilled pen. Legal Classification: Part 1, First & Third Schedules Poison Full prescribing information is available upon request.

MAT-HK-2101068-1.0-09/2021

API-HK-SOL-21.06

sanofi

Sanofi Hong Kong Limited
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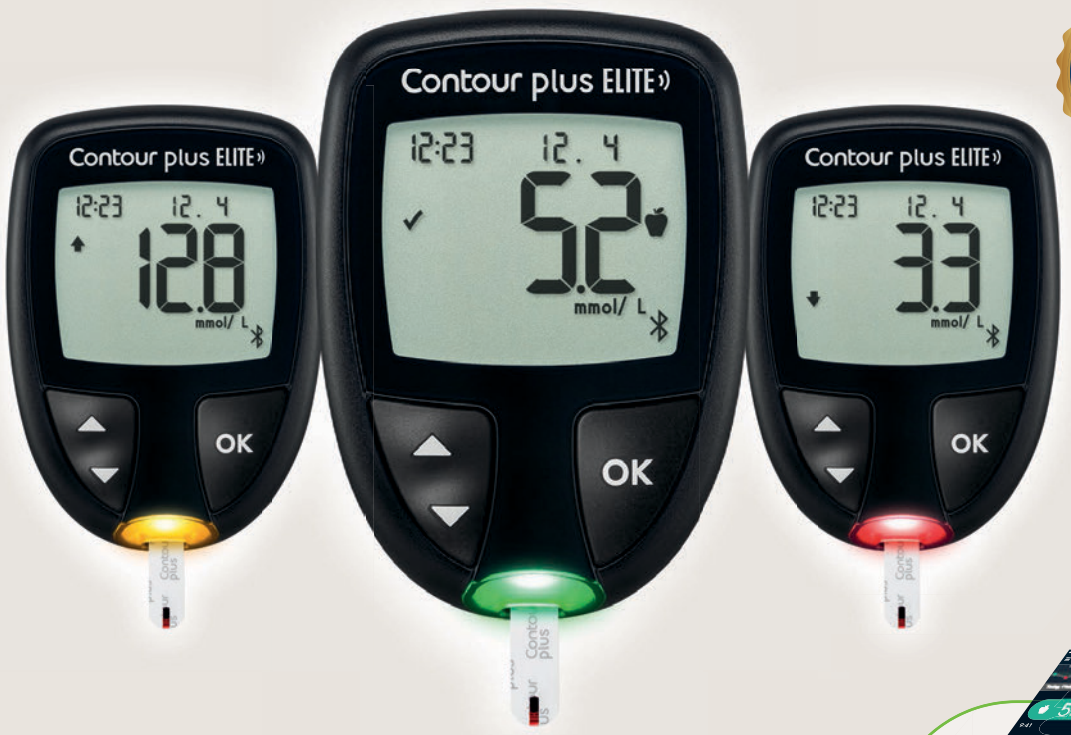
Your smartLIGHT™ for Blood Glucose Management



CONTOUR®PLUS ELITE ISO 15197:2013 Exceeds standard

Free CONTOUR® Diabetes App
(Traditional Chinese available now)

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

Contour plus ELITE Blood Glucose Monitoring System

- **Readings you can trust** to be highly accurate[1] 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®.**

Ascensia Diabetes Care Hong Kong Limited
Hotline : 8100 6386
Website : http://diabetes.ascensia.hk

1. Klaff L et al. Accuracy and User Performance of a New Blood Glucose Motoring System [published online ahead of print, 2020 Nov 26]. J Diabetes Sci Technol. 2020; https://doi.org/10.1177/1932296820974348. 2. CONTOUR®PLUS ELITE User Guide, November 2019, Revision 11.19. 3. Richardson JM et al. Clinical Relevance of Reapplication of Blood Samples During Blood Glucose Testing. Poster presented at the 20th Annual Diabetes Technology Meeting (DTM); November 12-14, 2020.

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1. Brazg, R. L., Klaff, L. J. and Sussman, A. M. New Generation Blood Glucose Monitoring System Exceeds International Accuracy Standards. J Diabetes Sci Technol. 2016, 10(6): 1414-15

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ACKNOWLEDGEMENTS