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OUR HORIZONS

EDM HK

4TH Annual Meeting

ENDOCRINOLOGY, DIABETES & METABOLISM HONG KONG

HYBRID MEETING

30 - 31 OCT 2021

PROGRAM BOOK

HKU Med

瑪麗醫院 QUEEN MARY HOSPITAL

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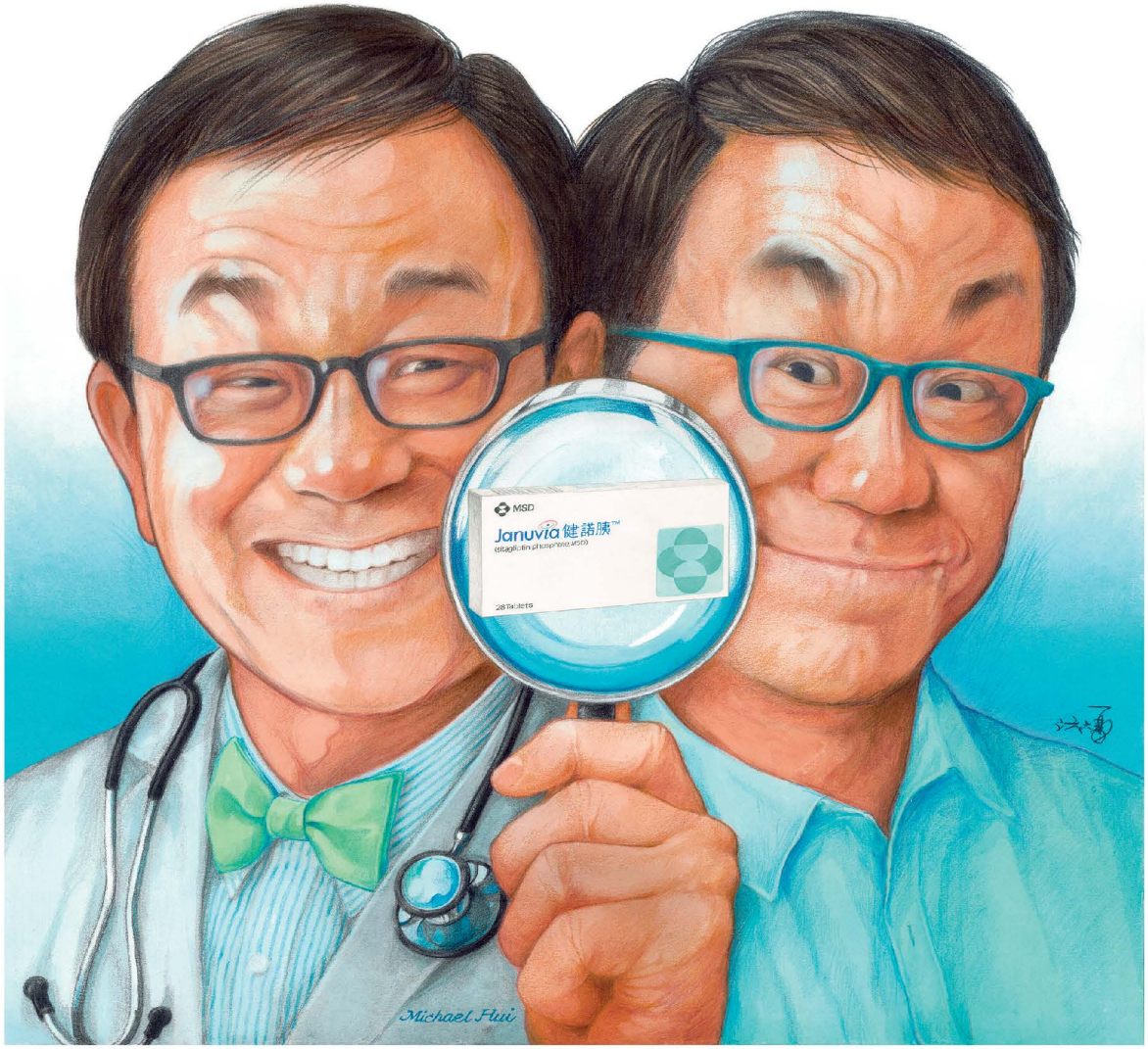

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• JANUVIA健諾胰乃處方藥物，詳情請向醫生或藥劑師查詢。
*根據已公布的研究結果，及其研究設計，跟同類型產品與安慰劑的比較[2,4]
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研究顯示不會增加心血管問題風險(HR=0.98，p<0.001)，並較少需要開始某種傳統針劑的長期治療(HR=0.70，p<0.001)[3]

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資料來源：1. IQVIA HKPA, MAT 4Q2019 (ATIN) 2. McGuire D.K. et al, JAMA Cardi, 2016 May;1(2):126-35 3. Green J.B, Bethel M.A., Armstrong P.W. et al., N Engl J Med., 2015;373(3):232-242 4. Rosenstock, J., et al. JAMA. 2019;321(1):69—79

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HK-DIA-00076 04/20

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WELCOME MESSAGE

On behalf of the organizing committee, we warmly welcome you to the Endocrinology, Diabetes & Metabolism Hong Kong (EDM HK) 4th Annual Meeting. Since our inauguration meeting in 2018, EDM HK has become an iconic yearly event for all healthcare professionals who manage patients with various endocrine and metabolic disorders.

We are glad to present EDM HK this year as a hybrid conference, which enables effective knowledge exchange wherever you might be, and allows face-to-face discussions while abiding to the current social distancing regulations. Following the success of previous meetings, our scientific program this year remains highly diversified and pertinent to this field. We would like to express our heartfelt gratitude to all eminent speakers and honourable chairpersons, our generous sponsors and conference secretariat, various supporting staff, as well as all delegates for your unfailing support to the conference.

We sincerely hope that you will enjoy our exciting program, and let's expand our horizons despite all adversities!

Dr. Alan CH Lee

Co-chairman
EDM HK 2021

Dr. Paul CH Lee

Co-chairman
EDM HK 2021

ORGANIZING COMMITTEE

Co-chairmen

Dr. Alan CH Lee

Dr. Paul CH Lee

Members

Prof. Karen SL Lam

Prof. Kathryn CB Tan

Dr. WS Chow

Dr. YC Woo

Dr. TP Ip

Dr. David TW Lui

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Dr. Johnny YC Chang

Dr. Chariene SL Woo

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Ms. Karen KC Wong

Ms. Amy SW Yee

Ms. SK Leung

Ms. Connie HN Loong

Ms. Michelle HY Lee

ACCREDITATIONS

CME				
Organization	Max. for whole function	30 October	31 October	Group-Category
Hong Kong College of Community Medicine	Pending	Pending	Pending	PP – PP
Hong Kong College of Family Physicians	8	3	5	OEA – 5.02
Hong Kong College of Obstetricians & Gynaecologists	Pending	Pending	Pending	PP – PN
The College of Ophthalmologists of Hong Kong	14.5	6.5	8	CME – PP
Hong Kong College of Orthopaedic Surgeons	8	3	5	PP – B
Hong Kong College of Paediatricans	9	3	6	A – PP
The Hong Kong College of Pathologists	11	4	7	CME – PP
Hong Kong College of Physicians	11	4	7	PP – PP
Hong Kong College of Radiologists	14	5	9	B – PP
The College of Surgeons of Hong Kong	11	5	6	CME – PP
The Medical Council of Hong Kong	10	5	5	CME – PASSIVE CME

CNE		
Organization	30 October	31 October
Hospital Authority Hong Kong West Cluster	4	7.5

PUBLIC LECTURES (BROADCAST ON YOUTUBE)

30 October 2021 (Saturday)

HK Time	Session
10:00 – 10:30	**Public Lecture 1: Foot care in diabetes patients (糖尿病人足部護理小百科)** Chairperson: Ms. Annie Leung Ms. Cynthia Leung (Hong Kong)
10:30 – 11:00	**Public Lecture 2: Relationship between menstrual disturbance and endocrine disorders (經期與內分泌失調)** Chairperson: Dr. Chris Dao Dr. Eunice Leung (Hong Kong)
11:00 – 11:30	**Public Lecture 3: Diet tips for people with diabetes, hypertension, hyperlipidemia and obesity (三高肥胖食乜好？)** Chairperson: Ms. Sarita Chan Ms. Flavia U (Hong Kong)

SCIENTIFIC PROGRAM

30 October 2021 (Saturday)

HK Time	Session
	Room S421
	Sponsored Lecture (1) Chairperson: Professor Karen Lam
13:10 – 13:45	**New era of SGLT2i: looking beyond the A1c in Cardio-Renal disease management** Professor Paola Fioretto (Italy)
13:45 – 13:50	**Q&A**
13:50 – 14:00	**Opening Ceremony**
	Sponsored Lecture (2) Chairperson: Professor Kathryn Tan
14:00 – 14:35	**Management of Hyperlipidemia: where are we now?** Dr. CH Choi (Hong Kong)
14:35 – 14:40	**Q&A**

HK Time	Session	
	Room S421	
	Symposium (1): **Diabetic Complications** Chairpersons: Dr. KF Lee & Dr. Vicki Tam	
14:40 – 15:05	**Diabetic eye disease** Professor WC Lam (Hong Kong)	
15:05 – 15:30	**Diabetic neuropathy** Dr. Jacky Lee (Hong Kong)	
15:30 – 15:40	**Q&A**	
15:40 – 16:00	**Break**	
	Sponsored Lecture (3) Chairperson: Dr. MW Tsang	
16:05 – 16:40	**Overcoming the therapeutic inertia in T2DM: protecting patients from outcomes with SGLT2 inhibitors** Professor Melanie Davies (UK)	
16:40 – 16:45	**Q&A**	
	Room S421	**Room S426 - 427**
	Symposium (2A): **Advances in Obesity Care** Chairpersons: Dr. CH Choi & Dr. WS Chow	**Symposium (2B):** **Disorders of Sex Development** Chairpersons: Dr. PT Cheung & Dr. Elaine Kwan
16:45 – 17:10	**Update on pharmacotherapy of obesity** Dr. Michele Yuen (Hong Kong)	**Practical approach to undervirilized male in the genetic era** Dr. Samantha Lee (Hong Kong)
17:10 – 17:35	**Metabolic surgery: choosing the right procedure for obese patients** Dr. Fion Chan (Hong Kong)	**Surgery in children affected by 45,X/46,XY mosaicism** Dr. YH Tam (Hong Kong)
17:35 – 17:50	**Case sharing**	**Case sharing**
17:50 – 18:00	**Q&A**	**Q&A**

SCIENTIFIC PROGRAM

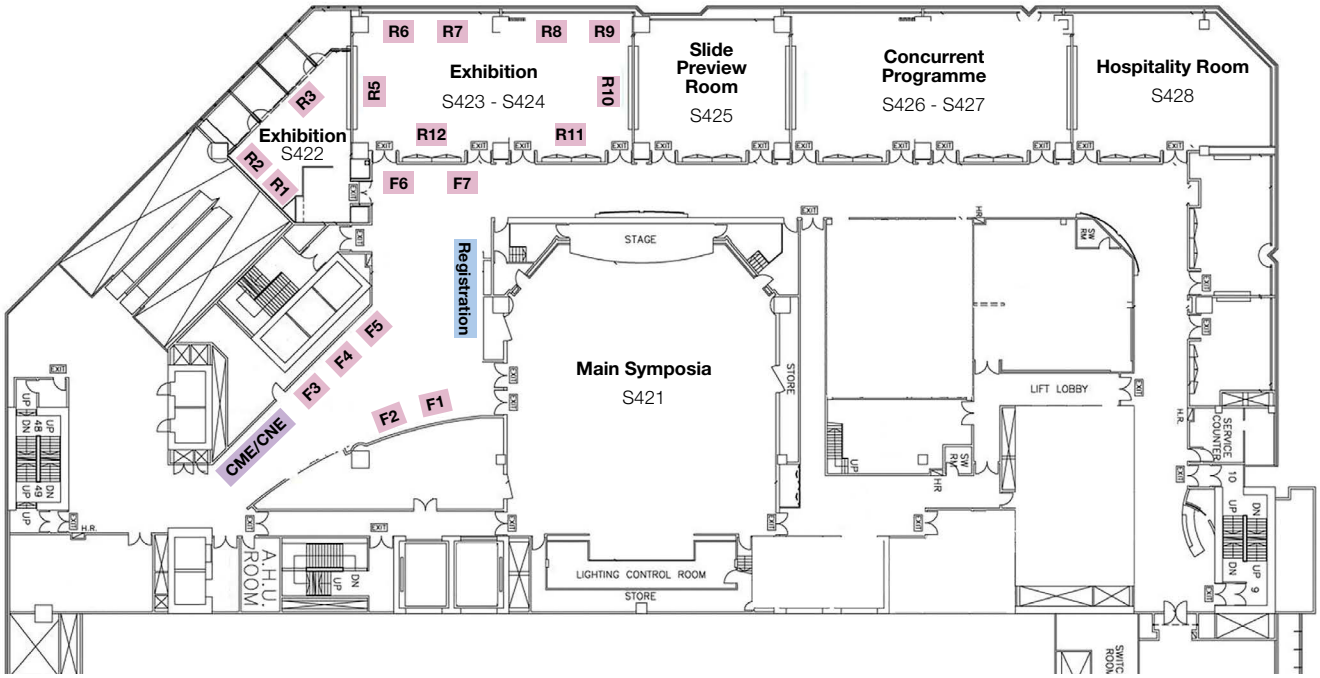
31 October 2021 (Sunday)

HK Time	Session	
	Room S421	Room S426 - 427
	Symposium (3A): Reproductive Endocrinology Chairpersons: Dr. Doris Chan & Dr. Raymond Li	**Symposium (3B): Running Out of Essential Elements** Chairpersons: Dr. Tellus Ng & Dr. YW Ng
09:00 – 09:25	**Fertility preservation and management of infertility in cancer survivors** Dr. Jacqueline Chung (Hong Kong)	**Update on hypothyroidism** Dr. Annette Tso (Hong Kong)
09:25 – 09:50	**Hyperprolactinaemia & prolactinoma** Dr. KK Lee (Hong Kong)	**Spontaneous hypoglycemia: evaluation and management** Dr. Risa Ozaki (Hong Kong)
09:50 – 10:00	**Q&A**	**Q&A**
	Room S421	
	Sponsored Lecture (4) Chairperson: Dr. SC Tiu	
10:05 – 10:40	**Once weekly GLP-1 receptor agonist in T2DM management: From clinical trials to real world evidence** Professor Samantha Hocking (Australia)	
10:40 – 10:45	**Q&A**	
10:45 – 10:55	**Break**	
	Plenary Lecture (1) Chairperson: Dr. Alan Lee	
10:55 – 11:30	**Update on primary aldosteronism** Professor Michael Stowasser (Australia)	
11:30 – 11:35	**Q&A**	
	Sponsored Lecture (5) Chairperson: Professor Ronald Ma	
11:45 – 12:20	**The role of second generation insulin analogues in daily clinical practice** Dr. Alice Cheng (Canada)	
12:20 – 12:25	**Q&A**	
12:25 – 13:25	**Lunch break**	
	Sponsored Lecture (6) Chairperson: Dr. John Ma	
13:30 – 14:05	**The use of GLP1-RA in cardiovascular disease prevention in T2DM** Professor Sten Madsbad (Denmark)	
14:05 – 14:10	**Q&A**	

HK Time	Session	
	Room S421	
	Sponsored Lecture (7) Chairperson: Dr. Vincent Yeung	
14:10 – 14:45	**Management of LDL-C with PCSK9i: Optimizing the outcomes in patients with chronic diseases** Professor Gerald Watts (Australia)	
14:45 – 14:50	**Q&A**	
	Room S421	**Room S426 - 427**
	Symposium (4A): **Cross-specialty Management of Endocrine Disorders** Chairpersons: Professor Alice Kong & Dr. Emmy Lau	**Symposium (4B):** **Endocrine Radiology and Neoplasia** Chairpersons: Dr. Ingrid Mak & Dr. KP Wong
14:50 – 15:15	**Transgender medicine: what endocrinologists need to know** Dr. Tiffany Yau (Hong Kong)	**Application of nuclear medicine in endocrinology** Dr. William Cheung (Hong Kong)
15:15 – 15:40	**Endocrine toxicity of cancer immunotherapy** Dr. David Lui (Hong Kong)	**Multiple endocrine neoplasia** Dr. Paul Lee (Hong Kong)
15:40 – 15:50	**Q&A**	**Q&A**
15:50 – 16:00	**Break**	
	Room S421	
	Plenary Lecture (2) Chairperson: Dr. YC Woo	
16:00 – 16:35	**Cushing's syndrome: diagnostic pitfalls and therapeutic advances** Professor Ashley Grossman (UK)	
16:35 – 16:40	**Q&A**	
	Symposium (5): **Nephrologists and Endocrinologists Are Good Friends!** Chairperson: Dr. Paul Lee & Dr. Maggie Mok	
16:40 – 17:05	**Endocrine conundrum: a nephrologist's perspective** Dr. Gary Chan (Hong Kong)	
17:05 – 17:30	**Crash course on hypophosphataemic disorders** Dr. Alan Lee (Hong Kong)	
17:30 – 17:40	**Q&A**	
17:40 – 17:50	**Closing Remarks**	

FLOOR PLAN

S400, Phase 1 (Old Wing), Hong Kong Convention and Exhibition Centre



Exhibition Booth Registration Counter CME/CNE

LIST OF EXHIBITORS

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

LIST OF OVERSEAS SPEAKERS

Dr. Alice Cheng
Associate Professor
Department of Medicine
University of Toronto, Canada

Professor Samantha Hocking
Associate Professor
Faculty of Medicine and Health
The University of Sydney, Australia

Professor Melanie Davies
Professor of Diabetes Medicine
Diabetes Research Centre
University of Leicester, UK

Professor Sten Madsbad
Professor
Faculty of Health Science
University of Copenhagen, Denmark

Professor Paola Fioretto
Professor in Medicine
Department of Medicine
University of Padova, Italy

Professor Michael Stowasser
Professor
Endocrine Hypertension Research Centre
University of Queensland Diamantina Institute, Greenslopes and Princess Alexandra Hospitals, Australia

Professor Ashley Grossman
Emeritus Professor of Endocrinology
University of Oxford, UK

Professor Gerald Watts
Professor
Departments of Cardiology and Internal Medicine
Royal Perth Hospital, Perth, Australia

LIST OF LOCAL FACULTY

Dr. Doris Chan
Associate Consultant
Department of Medicine & Geriatrics
Pok Oi Hospital

Dr. Fion Chan
Consultant
Department of Surgery
Queen Mary Hospital

Dr. Gary Chan
Associate Consultant
Department of Medicine
Queen Mary Hospital

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Consultant
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Chair Professor
Department of Medicine
The University of Hong Kong

Prof. WC Lam
Clinical Professor
Department of Ophthalmology
The University of Hong Kong

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Consultant
Department of Medicine
Pamela Youde Nethersole Eastern Hospital

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Honorary Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

ABSTRACTS

SPONSORED LECTURE (1)

New era of SGLT2i: looking beyond the A1c in Cardio-Renal disease management

Professor Paola Fioretto

Professor in Medicine
Department of Medicine
University of Padova, Italy

Type 2 diabetes mellitus (T2DM) is a well-established risk factor for chronic kidney disease (CKD). Despite significant advances in diagnosis and treatment over the past two decades, CKD patients still have a poor long–term prognosis including poor mortality and morbidity.

The incidence and prevalence of T2DM, HF and CKD are increasing globally. This epidemic of T2DM, HF and CKD creates an urgent need for effective therapies that can address the expected increased burden of cardiorenal diseases in general and among patients with T2DM. The management of T2DM should be guided by guidelines as well as clinical trial evidence which has shown to be beneficial. This lecture will discuss the role of SGLT2i in cardiorenal disease management.

ABSTRACTS

SPONSORED LECTURE (2)

Management of Hyperlipidemia: where are we now?

Dr. CH Choi

Consultant
Department of Medicine
Queen Elizabeth Hospital

Management of hyperlipidemia:

AFCAPS/TexCAPS; MEGA; WOSCOPS; STELLAR; CARDS; DISCOVERY; J-PREDICT; ALLHAT-LLT; ASCOT-LLA; ACCORD Lipid; AIM-HIGH; HPS2-THRIVE; JELIS; JUPITER; HOPE-3; EMPATHY; REDUCE-IT; STRENGTH; SEAS; ENHANCE; IMPROVE-IT; REVEAL; ACCELERATE; DAL-OUTCOME; ILLUMINATE; PROSPER; STAREE; SPARCL; GOULD; RUTHERFORD; TESLA; TAUSSIG; GAUSS-3; OSLER; EBBINGHAUS; SPIRE; ODYSSEY; FOURIER; CLEAR HARMONY; ORION; COMPASS; ELIPSE; AKCEA-APO(a)-Lrx….

In the jungle of lipid publications, where are we now?

ABSTRACTS

SYMPOSIUM (1): DIABETIC COMPLICATIONS

Diabetic eye disease

Professor WC Lam

Clinical Professor
Department of Ophthalmology
The University of Hong Kong

Diabetic eye disease is a group of eye conditions associated with patient with diabetes. These conditions include diabetic retinopathy, diabetic macular edema, cataracts, and glaucoma. The recent development of the anti-VEGF has revolutionized the management of diabetic retinopathy including the diabetic macular edema. Laser photocoagulation was once the mainstay of the treatment for diabetic retinopathy is replaced by the anti-VEGF.

This presentation will review the latest evidence-based treatment approach for the diabetic retinopathy, and diabetic macular edema.

ABSTRACTS

SYMPOSIUM (1): DIABETIC COMPLICATIONS

Diabetic neuropathy

Dr. Jacky Lee

Associate Consultant
Department of Medicine
Tung Wah Hospital

Diabetes mellitus is increasingly common worldwide, leading to a corresponding surge in the prevalence of diabetic complications including diabetic neuropathy. Diabetic neuropathy most commonly presents as a symmetric distal sensory-predominant polyneuropathy, but can also manifest as focal or multifocal neuropathy, radiculoplexopathy or autonomic neuropathy. Diabetic polyneuropathy is often debilitating and can result in sensory impairment, foot ulceration, amputation, falls and intractable neuropathic pain. Traditional approach with glycemic control has been shown to halt the progression of diabetic neuropathy in patients with type 1 diabetes mellitus, but the effects are only modest in those with type 2 diabetes mellitus. More recently, studies have shown that apart from hyperglycaemia and duration of diabetes, other cardiovascular risk factors also play an important role in the pathogenesis of diabetic neuropathy. However, to date only small-scale intervention studies targeting these risk factors are available and the results suggest that when the diabetic neuropathy becomes detectable by conventional bedside tools, it might be too advanced for any intervention to stop or reverse the disease process. Also, no specific disease-modifying treatment has been proven beneficial at the moment. Guidelines have suggested different classes of drugs as management of painful diabetic neuropathy. Standardized screening and early multifactorial interventions remain crucial for treating diabetic neuropathy and preventing its complications.

ABSTRACTS

SPONSORED LECTURE (3)

Overcoming the therapeutic inertia in T2DM: protecting patients from outcomes with SGLT2 inhibitors

Professor Melanie Davies

Professor of Diabetes Medicine
Diabetes Research Centre
University of Leicester, UK

Sodium-glucose cotransporter 2 (SGLT2) inhibitors, such as empagliflozin, canagliflozin and dapagliflozin, are oral anti-hyperglycemic agents that have shown cardiorenal benefits in patients with type 2 diabetes mellitus (T2DM). However, as SGLT2i has been perceived as a treatment mainly for glycemic control, its initiation for organ protection remains low.

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.

Latest evidence has extended the use of SGLT2 inhibitors for the treatment of heart failure irrespective of the presence of diabetes. In EMPEROR-Reduced, empagliflozin reduced significantly the combined relative risk of cardiovascular death and hospitalization for heart failure by 25%, and significantly reduced the relative risk of first and recurrent hospitalization for heart failure by 30% in adults with HFrEF. The rate of decline in eGFR, a measure of kidney function decline, was slower with empagliflozin than with placebo, and the relative risk of a composite kidney endpoint, including end stage kidney disease and a profound loss of kidney function, was reduced by 50%. The safety profile was similar to the well-established safety profile of empagliflozin. In short, as shown in EMPEROR-Reduced, empagliflozin improved heart failure outcomes and slow down kidney function decline in HFrEF patients with and without diabetes, on top of standard of care. Recent international guideline recommended SGLT2 inhibitor as one of the new foundational therapies for HFrEF management.

As reported in ESC2021, EMPEROR-Preserved study, being the first and only positive HFpEF trial to meet its primary endpoint, demonstrated significant risk reduction in hospitalization for heart failure or CV death. The benefit on empagliflozin on the primary endpoint was consistent across all pre-specified subgroups, including LVEF, sex and diabetes status. Empagliflozin opens the door for improving future HF management regardless of LVEF.

In conclusion, SGLT2i, as proven in multiple studies showing organ protective effects, should be considered beyond glycemic control.

ABSTRACTS

SYMPOSIUM (2A): ADVANCES IN OBESITY CARE

Update on pharmacotherapy of obesity

Dr. Michele Yuen

Honorary Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

Obesity has been defined as a chronic, relapsing and progressive disease with far-reaching health impairment including type 2 diabetes, hypertension, hyperlipidemia, ischemic heart disease, fatty liver and even some cancers. Treating obesity effectively reduces the burden of disease and improves productivity and quality of life. Diet and exercise, along with sustainable behavioral changes, are central to obesity treatment. For non-surgical candidates, pharmacotherapy is often necessary to "kick-start" and maintain the weight loss process. The pharmacotherapeutic options for obesity has seen some changes in 2020 and 2021. Among these changes, locaserin has been withdrawn due to possible increased cancer risk and a new GLP1 agonist, semaglutide, has received approval for use in weight management. This talk will give an overview of current pharmacotherapy of obesity and discuss strategies to choose between different pharmacotherapeutic agents.

ABSTRACTS

SYMPOSIUM (2A): ADVANCES IN OBESITY CARE

Metabolic surgery: choosing the right procedure for obese patients

Dr. Fion Chan

Consultant
Department of Surgery
Queen Mary Hospital

Throughout the last decade, the obesity epidemic continued to get worse. Obesity is associated with significant medical problems such as metabolic syndrome, non-alcoholic fatty liver diseases, obstructive sleep apnoea, cardiovascular diseases, and various cancers, etc. Bariatric surgery has been shown to be a reliable therapy for Asian obesity patients (BMI >30) with metabolic syndromes and it can improve or cure Type II diabetes mellitus and lower cardiovascular risks in a good proportion of patients. Bariatric surgery is therefore well-known as "metabolic surgery". It is, however, associated with a very low risk of mortality and risk of early and late complications including gastrointestinal reflux and nutritional deficiency. With more individuals seeking surgery as a treatment of metabolic syndrome, the choice of procedure has to be considered carefully based on the followings: 1. Weight-loss goals - amount of weight loss expected, and improvement or remission of obesity associated metabolic diseases; 2. Risk tolerance of patients with regard to the medical comorbidities and personal preference; 3. Demand for reversibility of procedures; 4. Willingness for life-long micronutrient supplementation.

Metabolic surgery is safe and effective in achieving sustained weight loss and improvement in obesity related comorbidities. There is no "right" operation for a particular patient. The choice of the surgical procedure should be the balance between benefits and risks.

ABSTRACTS

SYMPOSIUM (2B): DISORDERS OF SEX DEVELOPMENT

Practical approach to undervirilized male in the genetic era

Dr. Samantha Lee

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

Disorder of sex of differentiation (DSD) was reported to affect 5 in 1000, among these 75% were 46XY DSD, and this was a highly heterogenous group with low phenotype-genotype correlations (Audi L et al. Eur J Endocrinol. 2018;179(4):R197-R206). During the diagnostic and management journey of 46XY DSD, not only parents feel perplexed at the first instance of being introduced the term "46XY DSD" or "undervirilized male", physicians and surgeons alike are often stressed by the imminent need to assign for the sex of rearing, facing the challenges in reconstruction of genitalia, predicting the future gender orientation, fertility potential and to look for associated problems in other body system(s) eg. in NR51A, SOX9, HHAT, EMX2, WT1, SLOS etc. Over 60 genes have been reported to be involved in 46XY DSD in accordance to data in DSDNet. With the advances in next generation sequencing, we can gear our investigation and management in a more targeted manner. The aim of this session is to highlight the practical clinical pathway in management of 46XY DSD, and sharing of a real patient scenario to illustrate the importance of an accurate genetic diagnosis.

ABSTRACTS

SYMPOSIUM (2B): DISORDERS OF SEX DEVELOPMENT

Surgery in children affected by 45,X/46,XY mosaicism

Dr. YH Tam

Consultant
Department of Surgery
Prince of Wales Hospital

Disorders of sex development (DSD) are a heterogeneous group of conditions featuring incongruence between phenotypic, gonadal and genetic sex. While it is typical for a potential diagnosis of DSD to be raised when a newborn presents with ambiguous genitalia, children affected by DSD do not always have abnormal external genitalia. This phenomenon is best illustrated by 45,X/46,XY mosaicism, which has an estimated incidence between 1 in 6,000 to 1 in 15,000 live births. In 45,X/46,XY mosaicism, affected individuals present with wide variations in phenotypes ranging from normal female to normal male phenotypes, with ambiguous genitalia in the middle of the spectrum.

Pediatric studies of 45,X/46,XY patients reported in the literature generally focus on phenotypic features, endocrine function, growth and increased gonadal tumor risks. Data on surgical management of 45,X/46,XY children are sparse, and there is no surgical strategy which is universally agreed upon. It is beyond doubt that surgery has a role in the management of children with 45,X/46,XY karyotype given the increased risk of gonadal germ cell neoplasm which dictates a timely and appropriate surgical intervention. Performing genitalia surgery, however, has raised growing ethical concerns when a procedure of an irreversible nature is performed in a child who is too young to bodily autonomy and to participate in the decision-making process.

In the lecture, Dr. Tam would share his surgical experience and approach in 45,X/46,XY children based on their phenotypes, and would share the contemporary evidence in the latest literature including recent studies conducted in Hong Kong Chinese children.

ABSTRACTS

SYMPOSIUM (3A): REPRODUCTIVE ENDOCRINOLOGY

Fertility preservation and management of infertility in cancer survivors

Dr. Jacqueline Chung

Associate Professor
Department of Obstetrics and Gynaecology
The Chinese University of Hong Kong

With the advancement in diagnosis and treatment of cancer, the overall survival rate in young cancer patients has increased. However, anti-cancer treatment including chemotherapy and radiotherapy are often highly detrimental to the female endocrine and reproductive function.

The fecundity of these young cancer survivors becomes the key quality of life issue after their recovery. Despite the existence of multiple international guidelines for clinical practitioners on the issue of fertility preservation, many physicians still initiate anti-cancer treatment without detailed consultation on post-treatment fertility.

Fertility preservation refers to the means to preserve the women's hormonal function as well as fertility from the damage of anti-cancer treatment. A variety of fertility preservation strategies are available and the option of fertility preservation should be individualized for each patient. This presentation aims to discuss the various options of fertility preservation available, especially those involving assisted reproductive technology with embryo and oocyte freezing and an individualized approach will be shared.

ABSTRACTS

SYMPOSIUM (3A): REPRODUCTIVE ENDOCRINOLOGY

Hyperprolactinaemia & prolactinoma

Dr. KK Lee

Honorary Clinical Associate Professor
Department of Medicine
The University of Hong Kong

Hyperprolactinaemia is a common clinical problem faced by an endocrinologist. It is responsible for one third of all cases of female subfertility and prolactinoma accounts for 40% of all functional pituitary tumours. During this symposium, clinical cases will be presented to demonstrate how to approach prolactinoma during pregnancy, prolactinoma in male and the monitoring of side-effects during treatment.

ABSTRACTS

SYMPOSIUM (3B): RUNNING OUT OF ESSENTIAL ELEMENTS

Update on hypothyroidism

Dr. Annette Tso

Honorary Clinical Associate Professor
Department of Medicine
The University of Hong Kong

Thyroid hormones, namely thyroxine (T4) and triiodothyronine (T3), play important roles in the regulation of cell differentiation, neural maturation, growth, protein synthesis, basal metabolic rate, heat production and the utilization of nutrients for energy production. About 5%-10% of the general population is estimated to have some degree of hypothyroidism which, if improperly treated, may result in cardiovascular and metabolic sequelae as well as impact the quality of life.

In this talk, we shall cover the management of hypothyroidism in different contexts, including in pregnancy, in subclinical hypothyroidism and after treatment of thyroid cancer. We shall also discuss controversies surrounding thyroid deiodinase polymorphisms and combination therapy with liothyronine.

ABSTRACTS

SYMPOSIUM (3B): RUNNING OUT OF ESSENTIAL ELEMENTS

Spontaneous hypoglycemia: evaluation and management

Dr. Risa Ozaki

Consultant
Department of Medicine and Therapeutics
Prince of Wales Hospital

Hypoglycaemia has been documented in the literature since the early 19th century. However, it was not until the advent of insulin treatment in the 1920's did it come to light that it is the excess insulin that leads to symptoms we now know to be hypoglycaemia.

Spontaneous hypoglycaemic disorders are rare conditions encountered by clinicians and endocrinologists in the clinical setting. However, before embarking on extensive evaluation and investigations for the underlying aetiology for hypoglycaemia, it is important that the Whipple's triad is firmly documented confirming the presence of hypoglycaemia, so as to avoid unnecessary and costly investigations.

In this session, the approach to the evaluation and management of spontaneous hypoglycaemic disorders will be discussed. Two case studies will be portrayed to illustrate the presentation, investigation and management of hypoglycaemia cases resulting from different pathophysiology.

ABSTRACTS

SPONSORED LECTURE (4)

Once weekly GLP-1 receptor agonist in T2DM management: From clinical trials to real world evidence

Professor Samantha Hocking

Associate Professor
Faculty of Medicine and Health
The University of Sydney, Australia

Once weekly glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are well suited for the management of Type 2 diabetes (T2D). Signalling via its receptors located in various organs, including brain, GI tract and pancreas, GLP-1 provides a number of beneficial effects in the setting of T2D. In the pancreas, GLP-1 acts in a glucose-dependent manner to promote insulin secretion and inhibit glucagon secretion. Apart from this well-known pancreatic effect, GLP-1 enhances satiety and reduces hunger by acting on both central and peripheral receptors in the brain and GI tract. GLP-1 RAs generally improve a number of CV risk markers, including glucose, weight, blood pressure and lipid levels. Moreover, a number of large CV outcome trials (CVOTs) have provided robust evidence concerning CV outcomes. Owing to their glucose-lowering and weight-loss properties and low intrinsic risk of hypoglycaemia, as well as cardiovascular (CV) benefits, GLP-1 RAs are now recommended as second line treatment of T2D after metformin, particularly for individuals with established atherosclerotic cardiovascular disease or with indicators of high risk for cardiovascular disease or in whom weight management is a concern. This presentation will provide an overview of clinical trial evidence and real world data regarding the use of once weekly GLP-1 RAs for type 2 diabetes.

ABSTRACTS

PLENARY LECTURE (1)

Update on primary aldosteronism

Professor Michael Stowasser

Professor
Endocrine Hypertension Research Centre
University of Queensland Diamantina Institute, Greenslopes and Princess Alexandra Hospitals
Australia

During recent decades it has become apparent that primary aldosteronism (PA) is highly prevalent within the hypertensive population and that aldosterone excess has adverse cardiovascular (CV) effects that are in part independent of its effects on blood pressure, resulting in higher rates of CV events in PA compared to essential hypertensives. This cohort therefore represents a potentially enormous contributor to the global burden of CV disease. Not only do patients with PA show impressive BP responses to specific surgical (unilateral laparoscopic adrenalectomy) or medical [usually mineralocorticoid receptor antagonist (MRA)] treatment, but the excess in CV morbidity also resolves, making it essential to identify these patients so that they may benefit from optimal, targeted management. The resulting resurgence of research and clinical interest in PA has led to (1) major improvements in diagnostic workup, including the development of semi-automated mass spectrometric methods of measuring aldosterone and angiotensin II, a highly accurate and streamlined method of confirming the diagnosis (seated saline suppression testing), point-of care cortisol testing to enhance success of adrenal venous cannulation during adrenal venous sampling (AVS, used to differentiate unilateral, surgically correctable from bilateral forms) and metomidate radioisotope scanning (a promising alternative for subtype differentiation); (2) development of new MRAs and their successful wide application among a diverse array of CV conditions and of highly specific CYP11B2 inhibitors; and (3) rapidly expanding knowledge regarding the genetics of PA (familial and sporadic) and its pathogenesis and histopathology as defined by blood and adrenal DNA sequencing and immunohistochemistry using new, highly specific monoclonal antibodies to aldosterone synthase (CYP11B2). Clinicians, however, remain reluctant to look for PA. Given its low cost, reliability, high rate of positivity and substantial clinical significance of a positive test, screening by aldosterone/renin ratio testing should be offered to most hypertensives, and all at initial diagnosis of hypertension.

ABSTRACTS

SPONSORED LECTURE (5)

The role of second generation insulin analogues in daily clinical practice

Dr. Alice Cheng

Associate Professor
Department of Medicine
University of Toronto, Canada

Since the discovery of insulin over 100 years ago, the formulations have continued to evolve to improve clinical utility. Ideal insulin therapy requires a fine balance between achieving glycemic target and minimizing the risk of hypoglycemia. Second generation basal insulin analogues (insulin glargine 300 U/mL and insulin degludec) have been developed to try to better achieve those goals. When compared to first generation basal insulin analogues, both of these insulins have been shown to cause less hypoglycemia whilst providing effective glycemic control.

The first head-to-head study of the second generation basal insulin analogues was the BRIGHT study. Insulin-naïve patients with type 2 diabetes were randomized to receive either glargine 300 U/mL (IGlar U300) or insulin degludec (IDeg) using identical treat-to-fasting-glucose-target tiration algorithms. The primary outcome of noninferiority for HbA1c reduction from baseline was met over the 24-week study period. Overall hypoglycemia was also similar between the 2 groups for the entire study duration. However, there was less anytime hypoglcyemia with IGlar U300 compared to IDeg, during the 12-week titration period when the insulin dose changes and A1c drop were the most rapid. A prespecificed subroup analysis showed that the group with baseline eGFR <60 mL/min/1.73 m2 , achieved lower HbA1c with IGlar U300 with no difference in hypoglycemia. In addition, a post hoc analysis showed that among those age 70 years or older, IGlar U300 allowed for lower HbA1c with no increase in hypoglycemia compared to IDeg. These findings generate the hypothesis that IGlar U300 may be particularly beneficial in these vulnerable populations.

ABSTRACTS

SPONSORED LECTURE (6)

The use of GLP1-RA in cardiovascular disease prevention in T2DM

Professor Sten Madsbad

Professor
Faculty of Health Science
University of Copenhagen, Denmark

Semaglutide is a GLP-1 receptor agonist for once-weekly subcutaneous administration. Gradual dose escalation is recommended with an initial dose of 0.25 mg, which is increased to 0.5 mg after 1 months and in subjects without side effects to 1 mg after one further months of treatment. In semaglutide the similarity to human GLP-1 is well-preserved sharing a 94% homology.

Semaglutide is the most potent GLP-1 receptor agonist. In the phase 3 development program including more than 9000 participants the mean reduction in HbA1c for 1 mg of semaglutide was 1.6% (18 pmol/mol) and the mean body weight reduction was 5.7 kg. In head to head comparisons with DPP-4 inhibitors, other GLP-1 receptor agonists, SGLT-2 inhibitors and basal insulin the results have shown superiority for semaglutide both in relation to reduction in HbA1c and body weight. The risk of hypoglycemia with semaglutide is minimal when not combined with sulfonylurea or insulin.

Safety of semaglutide was studied in comparison with placebo in the cardiovascular endpoint study SUSTAIN 6, where 3297 subjects with type 2 diabetes were followed for 2 years. At baseline 83% had established cardiovascular disease, chronic kidney disease or both. The primary outcome: cardiovascular death, nonfatal myocardial infarction or nonfatal stroke was reduced by 26%, $p<0.001$, nonfatal myocardial infarction by 26%, $p=0.12$ and nonfatal stroke by 39%, $p=0.04$. Revascularization surgery rates were also greatly reduced by semaglutide compared with placebo. Rates of death, including cardiovascular death, were similar in the two groups. Semaglutide 1 mg reduces systolic blood pressure with 2.6 mmHg and increased heart rate with 2.5 beats per minute. In total 45 patients would need to be treated for 2 years to prevent one primary endpoint. The exact mechanism(s) semaglutide reduces CVD risk remain to be established but appears to be mainly linked to anti-arteriosclerotic effects by multiple pathways including reduced inflammatory processes within the atherosclerotic plaque.

Rates of new or worsening of nephropathy were lover, but rates of retinopathy complications significantly higher with semaglutide, and seems to be associated to a fast and great reduction in HbA1c the first 16 weeks of treatment in patients already having retinopathy and treated with insulin.

The most common side effects are gastrointestinal upset, typically nausea, vomiting, diarrhoea or obstipation, which can be seen in about 10-20% of treated, but in most wanes over time and can be minimized using slow up-titration.

In the latest ADA/EASD consensus report semaglutide, liraglutide, and dulaglutide is recommended to reduce the risk of cardiovascular disease in patients with establish cardiovascular disease or in high risk patients (artery stenosis > 50%, left ventricular hypertrophy, eGFR < 60 ml/min) independent of actual HbA1c. GLP-1 RAs do not appear to have consistent effect on hospitalization because of heart failure. Despite the beneficial effects on HbA1c, body weight and cardiovascular events GLP-1 RAs are underused in eligible patients.

ABSTRACTS

SPONSORED LECTURE (7)

Management of LDL-C with PCSK9i: Optimizing the outcomes in patients with chronic diseases

Professor Gerald Watts

Professor
Departments of Cardiology and Internal Medicine
Royal Perth Hospital, Perth, Australia

The role of proprotein convertase subtilisin/kexin type 9 (PCSK9) in the regulation of LDL metabolism was first suggested in families with phenotypic familial hypercholesterolaemia (FH) due to gain-of-function mutations in the PCSK9 gene. Experimental studies then established PCSK9 as a new target for lowering low-density lipoprotein cholesterol (LDL-C) and treating atherosclerotic cardiovascular disease (ASCVD). This led to vigorous pharmaceutical industry programs to develop new drugs for inhibiting PCSK9 in humans.

PCSK9 may be targeted by inhibiting its binding to the LDL receptor (e.g. with monoclonal antibodies (mAbs)), by inhibiting intrahepatic synthesis of PCSK9 at the RNA level (e.g. with nucleic acids) and by inhibiting autocatalytic process of PCSK9 (e.g. with small molecules). The mAb program is by far the most advanced, with two drugs (evolocumab, alirocumab) now integral to the care of patients with FH and patients with established ASCVD, and their use has been recommended for corresponding indications by international clinical practice guidelines. Clear efficacy has been demonstrated in both heterozygous and homozygous FH patients, although PCSK9 inhibitors are ineffective in the extremely rare patients with no LDL receptors due to null/null gene variants.

In essence, clinical outcome trials with PCSK9 mAbs have shown that they effectively lower LDL-cholesterol (by approximately 60%), significantly lower the incidence of further major atherosclerotic cardiovascular events (by approximately 15% against background treatment with statins) and are well tolerated and safe; benefits are on average proportional to the absolute reduction in LDL-C and to the duration of treatment, with greatest effects reported in those with additional risk due to recent MI, polyvascular disease and prior ASCVD events. The value of lowering LDL-C with PCSK9 mAbs has been clearly demonstrated in patients with diabetes, chronic kidney disease and peripheral arterial disease. Clinical benefit with no safety concerns has been reported at very low levels of LDL-C, a recent analysis from the FOURIER trial supporting lowering LDL-C in patients with ASCVD and other risk factors to less than 1 mmol/L (40 mg/dl).

The favourable impact of evolocumab on ASCVD events and progression of structural coronary artery disease is underpinned by an intravascular ultrasound study and, most recently, on unstable plaques in an ACS setting by a trial using optical coherence tomography. The outcome of the two major clinical trials have informed recent lipid guidelines from the ACC/AHA and ESC/EAS. The real challenge at present is how to implement these recommendations.

Despite the impressive success of the use of PCSK9 Mabs, a substantial proportion of patients remain at significantly increased residual risk of ASCVD. From a lipid perspective, future studies will address this continuing gap in care with therapies that target residual elevation in plasma levels of triglyceride-rich lipoproteins and lipoprotein(a).

ABSTRACTS

SYMPOSIUM (4A): CROSS-SPECIALTY MANAGEMENT OF ENDOCRINE DISORDERS

Transgender medicine: what endocrinologists need to know

Dr. Tiffany Yau

Associate Consultant
Department of Medicine and Therapeutics
Prince of Wales Hospital

Transgender people have a gender identity that differs from the sex they were assigned at birth, and this incongruence can result in lifelong gender dysphoria with significant distress or problems in functioning. Many transgender people would seek medical assistance on hormonal therapy, with or without surgery, to better align their physical features to their reaffirmed gender, and to alleviate the dysphoria associated with living in the incongruent gender.

It has been a century since the initiation of hormonal therapy and operations for gender dysphoria globally. Over the past 50 years where transgender treatment has been increasingly accessible worldwide, there remains concerns on the potential adverse effect of hormonal therapy on cardiovascular risk, bone health, and cancer risk etc.

Care for the transgender people requires a multidisciplinary team. In recognition of such, a centralized service for the management of gender identity has been established in the Prince of Wales Hospital since 2016. As of 2021, around 400 people are receiving hormonal therapy in this service. In parallel with the growing awareness of the LGBT community, healthcare professionals should also be more familiar with transgender care and current treatment options.

ABSTRACTS

SYMPOSIUM (4A): CROSS-SPECIALTY MANAGEMENT OF ENDOCRINE DISORDERS

Endocrine toxicity of cancer immunotherapy

Dr. David Lui

Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

Immune checkpoint inhibition is one of the five pillars of cancer therapeutics. Since the first FDA approval of immune checkpoint inhibitor (ICI) ipilimumab for melanoma in 2011, the indications for ICIs continue to expand to include various cancer types. Currently approved ICIs in clinical use include anti-CTLA4, anti-PD1 and anti-PD-L1. It has been estimated that close to half of the metastatic cancer patients may be eligible to receive ICIs. While ICIs possess anti-cancer effects, they are associated with a unique spectrum of side effects named immune-related adverse events (IRAEs).

In contrast to traditional cytotoxic chemotherapy or molecular targeted therapies which rarely cause endocrine dysfunction, ICI-treated patients are particularly susceptible to endocrine IRAEs. Timely treatment is essential not only to improve patients' quality of life, but also to prevent life-threatening complications. Hence, it is crucial for managing clinicians to be familiar with the evaluation and management of endocrine toxicity of cancer immunotherapy.

The incidence, pathophysiological mechanisms, clinical course, risk factors, evaluation and management of various endocrine IRAEs involving the pituitary, thyroid and endocrine pancreas will be discussed in this symposium. Furthermore, case examples will be discussed to illustrate the management algorithms. Relevant local data will be shared to conclude this symposium.

ABSTRACTS

SYMPOSIUM (4B): ENDOCRINE RADIOLOGY AND NEOPLASIA

Application of nuclear medicine in endocrinology

Dr. William Cheung

Honorary Consultant in Nuclear Medicine
Department of Nuclear Medicine and Positron Emission Tomography
Hong Kong Sanatorium and Hospital

Nuclear medicine techniques provide functional as well as morphological information on multiple endocrine organs and were first applied in thyroid disorders. Several radiopharmaceuticals are used for the evaluation of other endocrine conditions such as parathyroid and adrenal diseases and some neuro-endocrine tumors. Novel applications using PET technology are currently expanding the field of nuclear medicine in clinical endocrinology.

The diversity of molecular targets uniquely expressed by endocrine tumors provides opportunities for enhanced characterization of these tumors in terms of hormone synthesis, transporter and receptor expression, mirroring histologic classification on a whole-body, in vivo scale.

Radiopharmaceuticals in endocrine applications serve not only as diagnostic agents but also a platform to treat patients with matched radionuclide therapy agents, using the "theranostic approach". Radioiodine administration is still the standard of practice for the ablation of thyroid remnants after surgery for thyroid cancer, and for patient follow-up and re-staging.

In conclusion, the future of nuclear endocrinology is bright. Advances in nuclear endocrinology will be used together with state-of-art proteomics, functional genomics, and systems biology approaches to identify new targets that will improve diagnosis, offer new treatments, and expand understanding of these diseases.

ABSTRACTS

SYMPOSIUM (4B): ENDOCRINE RADIOLOGY AND NEOPLASIA

Multiple endocrine neoplasia

Dr. Paul Lee

Clinical Assistant Professor
Department of Medicine
The University of Hong Kong

Multiple endocrine neoplasia (MEN) is a group of genetic diseases characterized by the development of tumours in two or more endocrine organs in a single patient. Patients with MEN1 are predisposed to hyperplasia and/or tumours of parathyroid, enteropancreatic, and/or anterior pituitary origin, secondary to the presence of germline mutation in the tumour suppressor gene MEN1 which encodes menin. In MEN2 and MEN3, also known as MEN2A and MEN2B, respectively, patients typically develop medullary thyroid carcinomas and phaeochromocytoma secondary to the presence of germline mutation of the RET proto-ongocene. In contrast to MEN1, strong genotype-phenotype correlations are present in MEN2 and MEN3. Lastly, MEN4 is characterized by the presence of MEN1 phenotype but without an MEN1 mutation. Germline mutations in the cyclin-dependent kinase inhibitor 1b (CDKN1B) gene has been reported in patients with MEN4. This short talk will provide an update on the clinical presentation, management and screening across these MEN disorders.

ABSTRACTS

PLENARY LECTURE (2)

Cushing's syndrome: diagnostic pitfalls and therapeutic advances

Professor Ashley Grossman

Emeritus Professor of Endocrinology
University of Oxford, UK

Cushing's syndrome, once one excludes exogenous causes, is a rare syndrome, or at least it has been classically. A number of diagnostic tests have been proposed and refined over the years, and problems with the differential diagnosis, especially with regards to pituitary-dependent Cushing's syndrome, Cushing's disease, as opposed to the ectopic ACTH syndrome, have been largely resolved. This has been particularly in the context of bilateral inferior petrosal sinus sampling (BIPSS) and new high-resolution cross-sectional (CT, MRI) and functional imaging such as 68Ga-dotatate PET/CT/MRI scanning. However, the accurate diagnosis of the increasing number of patients with very mild Cushing's syndrome, and the awareness of autonomous cortisol-secretion from adrenal "incidentalomas", has led to very problematic decision taking. What do we do with the patient with some clinical and biochemical features of Cushing's syndrome, but no certain diagnosis: Cushing's or pseudo-Cushing's? In essence, the current diagnostic tests provide a probabilistic diagnostic estimate on a background of the pre-test probability, with a clinical decision required as to the most appropriate outcome.

New advances in medical therapies based on the pituitary (pasireotide), adrenal (osilodrostat, levoketoconazole) or glucocorticoid receptor (relacorilant) are expensive and require careful manipulation. In addition, cross-reaction of cortisol metabolites in patients on metyrapone, osilodrostat, and parenteral etomidate, renders careful consideration of assay results mandatory. Surgery (transsphenoidal, unilateral or bilateral adrenalectomy) still remains the cornerstone of treatment. However, recent data on the use of metformin to antagonise many of the adverse effects of exogenous steroids may also be considered in the cases of uncertain mild Cushing's syndrome.

ABSTRACTS

SYMPOSIUM (5): NEPHROLOGISTS AND ENDOCRINOLOGISTS ARE GOOD FRIENDS!

Endocrine conundrum: a nephrologist's perspective

Dr. Gary Chan

Associate Consultant
Department of Medicine
Queen Mary Hospital

Hyponatraemia is the most common electrolyte disorder observed in clinical practice. In severe cases, patients can develop fatal complications from cerebral edema but also permanent neurological disability from overzealous treatment. Management of this disorder rests upon physiological principles and the understanding that a vast majority of cases result from a perturbation of water balance. In this talk, the diagnostic approach as well as the therapeutic aims and options for hyponatraemia will be discussed.

ABSTRACTS

SYMPOSIUM (5): NEPHROLOGISTS AND ENDOCRINOLOGISTS ARE GOOD FRIENDS!

Crash course on hypophosphataemic disorders

Dr. Alan Lee

Associate Consultant
Department of Medicine
Queen Mary Hospital

Phosphate is an essential element of several physiologic pathways, such as skeletal development, bone mineralization, membrane composition, nucleotide structure, maintenance of plasma pH, and cellular signalling. Hypophosphataemia is a common electrolyte disturbance encountered in clinical practice. Acute and chronic hypophosphataemia differ in clinical manifestations and aetiologies. While the causes of acute hypophosphataemia are usually apparent without much investigation, chronic hypophosphataemia requires comprehensive biochemical evaluation. Hypophosphataemia results from 3 major mechanisms, namely intracellular shift, gastrointestinal loss and renal loss. Of note, renal phosphate wasting can be further classified as fibroblast growth factor 23 (FGF-23) mediated and non-FGF-23 mediated. This presentation will provide a concise review on phosphate metabolism and a practical approach to evaluation of hypophosphatemia. The management of important hypophosphatemic disoders, including recent advances in treating FGF-23 mediated pathology, will also be discussed.

SUPPORTING ORGANIZATIONS

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

香港糖尿病聯會
Diabetes Hongkong

Hong Kong Obesity Society
香港肥胖學會

HKSEMR
Hong Kong Society of Endocrinology, Metabolism and Reproduction

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

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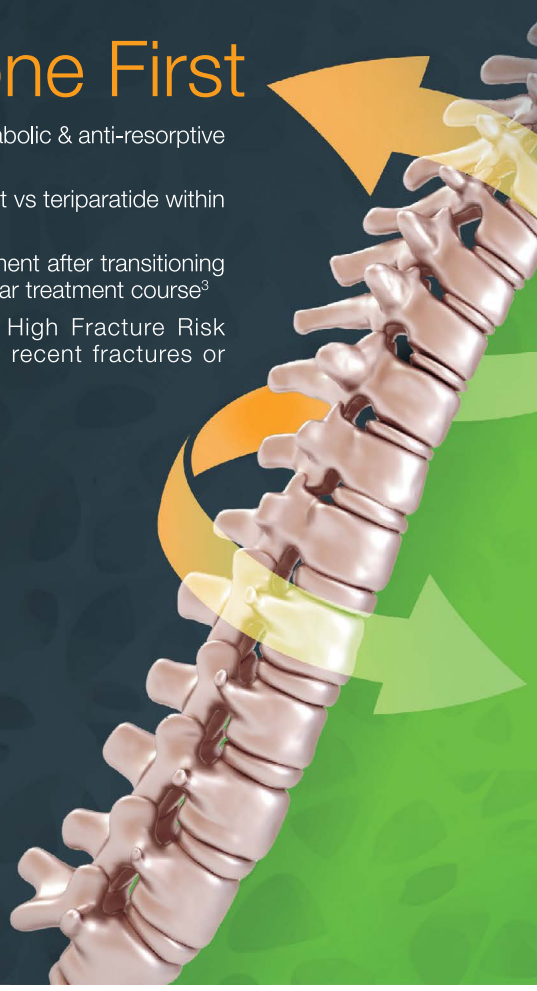

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- Recommended for High to Very High Fracture Risk patients, e.g. those with fracture history or T-score ≤-2.5[4]

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References:
1. Evenity Hong Kong Prescribing Information, Mar 2020, **2.** Langdahl BL, et al, Lancet 2017;390:1585–94, **3.** Saag KG, et al, N Engl J Med 2017;377:1417–27, **4.** Camacho P, et al, Endocr Pract 2020;26:1–46, **5.** Bone HG, et al, Lancet Diabetes Endocrinol 2017;5:513–23, **6.** Kendler DL, et al, J Bone Miner Res 2010;25:72–81, **7.** Brown JP, et al, J Bone Miner Res 2009;24:153–61.

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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 (13.6%) 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
Please read the full prescribing information prior to administration and full prescribing information is available upon request.
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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 overdose with Prolia.
Abbreviated Prescribing Information Version: HKPROPI02
Please read the full prescribing information prior to administration and full prescribing information is available on request.
Prolia® is a registered trademark owned or licensed by Amgen Inc., its subsidiaries, or affiliates.
For medical inquiries or to report adverse events/product complaint, please contact 800 961 142 or email medinfo.JAPAC@amgen.com
For Healthcare Professional Only.

HK-05305-EVE-2020-Nov Date of Approval: April 2021 4iC_1608A

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Contour®
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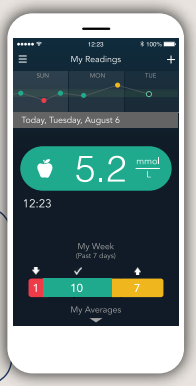
The CONTOUR®PLUS ONE meter and app system combines highly accurate[1] blood glucose testing wth an easy-to-use app

The **smartLIGHT** feature gives patients instant feedback[2] and makes it easier to interpret readings.

HIGHLY ACCURATE

The CONTOUR®PLUS ONE system has been shown to deliver remarkable accuracy within ±8.5% of lab values.[1*]



Second-Chance® sampling

Second-Chance® sampling gives you 60 seconds to reapply blood to the same strip[2] which may help prevent wasted strips.

The **FREE CONTOUR®DIABETES app** is easy to use and intuitive for patients on compatible[†] devices.

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Contour plus ONE

Contour plus

Recommend the CONTOUR® system today

Patients should consult with their health care provider prior to making changes to diet, exercise or treatment regimen and prior to making changes to their meter target ranges.

* Results from an ad hoc analysis of the CONTOUR®PLUS ONE system demonstrated that 95% of the total results were within ±8.5 mg/dL (±0.47 mmol/L) or ±8.5% of the YSI reference glucose values (YSI Life Sciences, Inc., Yellow Springs, OH) for subject-obtained capillary fingertip results from subjects with diabetes, for samples with glucose concentrations <100 mg/dL(<5.55 mmol/L) and ≥100 mg/dL(≥ 5.55 mmol/L), respectively.[1]

† Compatible devices can be found at http://compatibility.contourone.com

References: 1. Bailey TS et al. *J Diabetes Sci Technol* 2017:11(4):736-743. **2.** CONTOUR®PLUS ONE BGMS user guide. Revised January 2016.

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Date of preparation: August 2020. Code: G.DC.04.2020. PP-CPLUS-1-GBL-0013

Ascensia Diabetes Care Hong Kong Limited
Contact No.: 8100 6386 Website: http://diabetes.ascensia.hk

ASCENSIA Diabetes Care

ONE.20200801b

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-JAR-14-V2)

Presentation: Empagliflozin. Film-coated tablets 10 mg; 25 mg. **Indications:** 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. **Dosage and administration:** 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 ≥45mL/min/1.73m² or with hepatic impairment, or for elderly patients. **Contraindication:** Hypersensitivity to empagliflozin or any of the excipients. Patients on dialysis, eGFR <30mL/min/1.73m² or CrCl <30mL/min, or eGFR persistently <45mL/min/1.73m² or CrCl persistently <45mL/min. Rare hereditary conditions that may be incompatible with an excipient. **Special warnings and precautions:** Should not be used in patients with type 1 diabetes. Discontinue immediately when DKA is suspected or diagnosed. Treatment should be interrupted in patients who are hospitalised for major surgical procedures or acute serious medical illnesses. Monitoring of ketones is recommended in these patients. Measurement of blood ketone levels is preferred to urine, and empagliflozin may be restarted when the ketone values are normal and the patient's condition has stabilised. Assess renal function prior to initiation of empagliflozin and periodically thereafter. Discontinue when the eGFR is persistently <45mL/min/1.73m² or CrCl <45mL/min. 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. Patients treated with empagliflozin presenting with pain or tenderness, erythema, or swelling in the genital or perineal area, along with fever or malaise, should be assessed for necrotizing fasciitis. 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, urosepsis, pyelonephritis, necrotising fasciitis of the perineum (Fournier's gangrene), allergic skin reaction, angioedema, phimosis. **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]

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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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REALISE THE POTENTIAL

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80%

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<7%

VS OTHER DIABETES TREATMENT[1,2,7,8,9#]

OZEMPIC®

The only once-weekly treatment unifying superior efficacy and CV benefits[1-5]

SUPERIOR GLYCAEMIC CONTROL[1,2*]

Up to 1.8% HbA_{1c} reduction[2]

SUPERIOR AND SUSTAINED WEIGHT LOSS[1-3*]

Up to 6.5kg weight reduction [2]

PROVEN CV BENEFITS[1,3 †]

26% CV risk reduction[1,3§]

§ When added to SOC, which included oral antidiabetic treatment, insulin, antihypertensives, diuretics and lipid-lowering therapies.[3]

Other diabetes treatments refer to sitagliptin, dulaglutide, exenatide ER, liraglutide, canagliflozin and glargine U100. Target refers to American Diabetes Association target of HbA_{1c} <7%.

† In SUSTAIN 6, Ozempic® reduced CV risk (CV death, nonfatal myocardial infarction [MI] or nonfatal stroke) versus placebo in patients with type 2 diabetes at high CV risk treated with standard of care.[1]

* Results apply to Ozempic® across SUSTAIN trials, which included placebo, DPP-4I, SGLT-2I, GLP-1 RA and basal insulin.[1,2]

For adults with type 2 diabetes with established ASCVD or indicators of high ASCVD risk

2019 ADA/EASD consensus report recommends a GLP-1 RA therapy with proven CV benefit[6]

CV=cardiovascular; CVD=cardiovascular disease; ADA=American Diabetes Association; EASD=European Association for the Study of Diabetes;
GLP-1 RA=glucagon-like peptide-1 receptor agonist.

Abbreviated prescribing information Ozempic® (semaglutide). Ozempic 0.25 mg solution for injection in pre-filled pen; Ozempic 0.5 mg solution for injection in pre-filled pen; Ozempic 1 mg solution for injection in pre-filled pen. **Consult Summary of Product Characteristics before prescribing. Presentation:** Ozempic 0.25 mg & 0.5 mg solution for injection: Each pre-filled pen contains 2 mg semaglutide in 1.5 ml solution. Ozempic 1 mg solution for injection: One pre-filled pen contains 4 mg semaglutide in 3.0 ml solution. **Uses:** Ozempic® is indicated for the treatment of adults with insufficiently controlled type 2 diabetes mellitus as an adjunct to diet and exercise as Monotherapy: when metformin is considered inappropriate due to intolerance or contraindications. Combination therapy: in addition to other medicinal products for the treatment of diabetes. For study results with respect to combinations, effects on glycaemic control and cardiovascular events, and the populations studied, see the full Summary of Product Characteristics. **Dosage and administration:** The starting dose is 0.25 mg Ozempic® once weekly. After 4 weeks the dose should be increased to 0.5 mg once weekly. After at least 4 weeks with a dose of 0.5 mg once weekly, the dose can be increased to 1 mg once weekly to further improve glycaemic control. Ozempic® is to be administered once weekly at any time of the day, with or without meals. Ozempic® is to be injected subcutaneously in the abdomen, in the thigh or in the upper arm. Ozempic® should not be administered intravenously or intramuscularly. When Ozempic® is added to existing metformin and/or thiazolidinedione therapy, the current dose of metformin and/or thiazolidinedione can be continued unchanged. When Ozempic® is added to existing therapy of sulfonylurea or insulin, a reduction in the dose of sulfonylurea or insulin should be considered to reduce the risk of hypoglycaemia. **Elderly:** No dose adjustment is required based on age. Therapeutic experience in patients aged ≥75 years of age is limited. **Renal impairment:** No dose adjustment is required for patients with mild, moderate or severe renal impairment. Experience in patients with severe renal impairment is limited, Not recommended for use in patients with end-stage renal disease. **Hepatic impairment:** No dose adjustment is required for patients with hepatic impairment. Experience in patients with severe hepatic impairment is limited, Caution should be exercised when treating these patients with Ozempic®. **Paediatric population:** The safety and efficacy of Ozempic® in children and adolescents below 18 years have not yet been established. No data are available. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Special warnings and precautions for use:** Ozempic® should not be used in patients with type 1 diabetes mellitus or for the treatment of diabetic ketoacidosis. Ozempic® is not a substitute for insulin. There is no experience in patients with congestive heart failure NYHA class IV and Ozempic® is therefore not recommended in these patients. The possibility of gastrointestinal adverse reactions should be considered when treating patients with impaired renal function as nausea, vomiting, and diarrhoea may cause dehydration, which could cause a deterioration of renal function. Patients should be informed of the characteristic symptoms of acute pancreatitis. If pancreatitis is suspected, Ozempic® should be discontinued; if confirmed, Ozempic® should not be restarted. Caution should be exercised in patients with a history of pancreatitis. Patients treated with Ozempic® in combination with a sulfonylurea or insulin may have an increased risk of hypoglycaemia. Consider reducing the dose of sulfonylurea or insulin when initiating treatment with Ozempic®. In patients with diabetic retinopathy treated with insulin and Ozempic®, an increased risk of developing diabetic retinopathy complications has been observed. Caution should be exercised when using Ozempic® in patients with diabetic retinopathy treated with insulin. These patients should be monitored closely and treated according to clinical guidelines. Rapid improvement in glucose control has been associated with a temporary worsening of diabetic retinopathy, but other mechanisms cannot be excluded. **Interactions:** Ozempic® delays gastric emptying and has the potential to impact the rate of absorption of concomitantly administered oral medicinal products. Ozempic® should be used with caution in patients receiving oral medicinal products that require rapid gastrointestinal absorption. No dose adjustment of paracetamol, oral contraceptives (ethinylestradiol and levonorgestrel), atorvastatin, warfarin, digoxin or metformin is necessary when administered with Ozempic®. For further details of these interaction studies, please see the Summary of Product Characteristics. **Pregnancy and lactation:** Ozempic® should not be used during pregnancy. If a patient wishes to become pregnant, or pregnancy occurs during treatment, Ozempic® should be discontinued. Ozempic® should not be used during breast-feeding. Effect of Ozempic® on fertility in humans is unknown. **Driving or using machines:** When Ozempic® is used in combination with a sulfonylurea or insulin, patients should be advised to take precautions to avoid hypoglycaemia while driving and using machines. **Undesirable effects:** The most frequently reported adverse reactions with Ozempic® in clinical trials were gastrointestinal disorders, including nausea, diarrhoea and vomiting. Adverse reactions by system organ class and absolute frequencies identified in all phase 3a trials listed here as Very common (≥1/10): Hypoglycaemia when used with insulin or sulfonylurea, nausea, diarrhoea; Common (≥1/100 to <1/10): Hypoglycaemia when used with other OADs, decreased appetite, dizziness, diabetic retinopathy complications, vomiting, abdominal pain, abdominal distension, constipation, dyspepsia, gastritis, gastro-oesophageal reflux disease, eructation, flatulence, cholelithiasis, fatigue, increased lipase, increased amylase, weight decreased; Uncommon (≥1/1,000 to <1/100): Dysgeusia, increased heart rate, injection site reactions; Rare (≥1/10,000 to <1/1,000): Anaphylactic reaction.

References: 1. Ozempic® packing insert. **2.** Pratley RE, Aroda VR, Lingvay I, et al. Semaglutide versus dulaglutide once weekly in patients with type 2 diabetes (SUSTAIN 7): a randomised, open-label, phase 3b trial. Lancet Diabetes Endocrinol. 2018;6(4):275-286. **3.** Marso SP, Bain SC, Consoli A, et al. Semaglutide and cardiovascular outcomes in patients with type 2 diabetes. N Engl J Med. 2016;375:1834-1844. **4.** Bydureon® [summary of product characteristics]. Sodertalje Sweden: AstraZeneca AB. http://www.ema.europa.eu/docs/en_GB/document_library/EPAR_-_Product_Information/ human/002020/WC500108241.pdf. Accessed October 10, 2017. **5.** Trulicity® [summary of product characteristics]. Utrecht, The Netherlands: Eli Lilly Nederland B.V. http://www.ema.europa.eu/docs/en_GB/document_library/EPAR_-_Product_Information/human/002825/WC500179470.pdf. Accessed October 10, 2017. **6.** Buse JB, Wexler DJ, Tsapas A, et al. 2019 update to: management of hyperglycemia in type 2 diabetes, 2018. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care. 2020;43(2):487-493. **7.** American Diabetes Association. Standards of medical care in diabetes—2018. Diabetes Care. 2018;41(suppl 1):S1-S159. **8.** Lingvay I, Catarig AM, Frias JP, et al. Efficacy and Safety of once-weekly Semaglutide versus daily canagliflozin as add-on to metformin in patients with type 2 diabetes (SUSTAIN 8): a double-blind, phase 3b, randomised controlled trial. Lancet Diabetes Endocrinol. 2019;7(11):834-844. **9.** Capehorn MS, Catarig AM, Furberg JK, et al. Efficacy and safety of once-weekly Semaglutide 1.0mg Vs once-daily liraglutide 1.2mg as add-on to 1-3 oral antidiabetic drugs in subjects with type 2 diabetes (SUSTAIN 10). Diabetes Metab. 2020;46(2):100-109.

The materials for Ozempic® contained in this virtual exhibition are approved for use only in Hong Kong. Prescribing information may vary depending on local approval in each country. Therefore, before prescribing any product, always refer to local materials such as the prescribing information and/or the Summary of Product Characteristics (SPC).

novo nordisk®

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

ONCE-WEEKLY OZEMPIC® semaglutide injection

OZE-D-20210301

SAMSCA® helps you manage hyponatremia while you are treating your patient's primary condition.

Start Samsca®

When fluid restriction is not enough for clinically significant hypervolemic and euvolemic hyponatremia[1]

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).

Abbreviated Prescribing Information

Presentation: Tablets 15mg or 30mg of tolvaptan. **Indication:** 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). **Dosage:** To be initiated in hospital due to need for evaluation of therapeutic response. The usual starting dose for SAMSCA is 15mg administered once daily without regard to meals. Increase the dose to 30mg once daily, after at least 24 hours, to a maximum of 60 mg once daily, as needed to achieve the desired level of serum sodium. Limit treatment duration to 30 days. **Contraindications:** Hypersensitivity to any component of Samsca. Urgent need to raise serum sodium acutely. Anuria. Hypovolaemic hyponatremia (worsening). Hypernatremia. Patients who cannot perceive or appropriately respond to thirst. Concomitant use of strong CYP3A inhibitors. Pregnancy. Breastfeeding. **Warnings and precautions:** Tolvaptan should be initiated and re-initiated in patients only in a hospital where serum sodium can be monitored closely. Tolvaptan has not been in a setting of urgent need to raise serum sodium acutely. For such patients, alternate treatment should be considered. Osmotic demyelination syndrome is a risk associated with too rapid correction of hyponatremia (eg., >12mEq/L/24 hours). Osmotic demyelination results in dysarthria, mutism, dysphagia, lethargy, affective changes, spastic quadriparesis, seizures, coma and death. Caution should be exercised to ensure patients have adequate access to water and not become overly dehydrated. Urinary outflow must be secured to avoid risk of developing acute urinary retention. If hepatic injury is suspected, discontinue SAMSCA. Avoid use in patients with underlying liver disease. Concomitant use of SAMSCA with other treatments for hyponatremia or other medicinal products that increase serum sodium concentration may result in a higher risk for developing rapid correction of serum sodium and is therefore not recommended. **Drug interactions:** Caution with: co-administration with CYP3A inhibitors, inducers and substrates. P-gp inhibitors, and digoxin. Concomitant use with hypertonic saline is not recommended. The effect of vasopressin analogues such as desmopressin may be attenuated in patients using such analogues to prevent or control bleeding when co-administered with SAMSCA. **Adverse reactions:** The following adverse reactions were reported (>2%) in clinical trials in hyponatremia: Dry mouth, constipation, thirst, asthenia, pyrexia, hyperglycemia, anorexia, pollakiuria or polyuria. See full package insert for further details and other undesirable effect. **Overdosage:** If overdose occurs, estimation of the severity of poisoning is an important first step. Treatment should involve symptomatic and supportive care, with respiratory, ECG and blood pressure monitoring and water/electrolyte supplements as needed. A profuse and prolonged aquaresis should be anticipated. Please refer to full package insert for further details.

References:
1. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure – Web Addenda.
2. Samsca® package insert.

Further information available upon request.

SMC201701 (revised in June 2019)

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

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

Smartly stored data

Wirelessly sends results to the mySugr app

mySugr

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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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DC-2021-AUG-005

Toujeo®
insulin glargine 300U/mL

After a long journey on OADs, give your patients

The Power to Control Diabetes with Reassuring Confidence

When compared with insulin degludec, Toujeo® achieves:

Glycaemic control

Comparable HbA1c reductions in insulin-naïve patients[1,2]

GREATER efficacy among high-risk populations[3,4]

Elderly

Renal impairments

Less hypoglycaemia during titration[1,2]

Up to **43%** LOWER rates

Comparable rates of hypoglycaemia throughout the study

Glycaemic variability

Better stability

20% LOWER within-day fluctuation[5]

13% GREATER nocturnal TIR[6,*]

This advertisement is only intended for Healthcare Practitioners and should not be re-distributed.

* Time-in-range was defined as the percentage of time with blood glucose level from 70 to 180 mg/dL.

OAD=oral antihyperglycaemic drug. TIR=time-in-range.

References: 1. Rosenstock J, et al. Diabetes Care. 2018;41:2147-54. 2. Cheng A, et al. Diabetes Obes Metab. 2020;2:346-54. 3. Halzuik M, et al. Diabetes Obes Metab. 2020;22:1369–1377. 4. Bolli GB, et al. Diabetes Obes Metab. 2021;1–6. 5. Bailey TS, et al. Diabetes Metab. 2018;44:15-21. 6. Congeti, et al. Poster presented at the 56th Annual Conference of the European Association for the Study of Diabetes 2020: September 21 - 25: Virtual meeting. Poster 670.

Abbreviated prescribing information: Presentation: Insulin glargine 300 IU/ml solution for injection. **Indications:** Treatment of diabetes mellitus in adults, adolescents and children from the age of 6 years. **Dosage:** Once daily (preferably at the same time every day up to 3 hours before or after the usual time of administration), with adjusted individual dosage. Please refer to the full prescribing information for guidelines on switching between other insulin preparations. **Administration:** Subcutaneous injection. Toujeo is NOT INTENDED FOR INTRAVENOUS USE since it could result in severe hypoglycaemia. Toujeo must not be drawn from the cartridge of the SoloStar pre-filled pen into a syringe or severe overdose can result. **Contraindications:** Hypersensitivity to insulin glargine or to any of the excipients. **Precautions:** Toujeo has not been studied in children below 6 years of age. **Elderly:** progressive deterioration of renal function may lead to a steady decrease in insulin requirements. **Renal impairment:** insulin requirements may be diminished due to reduced insulin metabolism. **Hepatic impairment:** insulin requirement may be diminished due to reduced capacity for gluconeogenesis and reduced insulin metabolism. Perform continuous rotation of injection site to reduce risk of lipodystrophy and cutaneous amyloidosis. Blood glucose monitoring is recommended after change in injection site. Hypoglycaemia. Intercurrent illness. Combination of Toujeo with pioglitazone. Medication errors prevention. **Interactions:** Effects enhanced by oral antidiabetics, ACEI, disopyramide, fibrates, fluoxetine, MAOIs, pentoxifylline, propoxyphene, salicylates, sulfonamide antibiotics. Effects reduced by corticosteroids, danazol, diazoxide, diuretics, glucagons, isoniazid, oestrogens and progestogens, phenothiazine derivatives, somatropin, sympathomimetics, or 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:** Animal studies do not indicate direct harmful effects with respect to fertility and reproductive toxicity. The use of Toujeo may be considered during pregnancy if clinical needed. It is unknown whether insulin glargine is excreted in human milk. **Overdose:** Insulin overdose may lead to severe and sometimes long-term and life-threatening hypoglycaemia. 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, lipohypertrophy, injection site reactions. For common, uncommon, rare and very rare 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 30°C. Use within 42 days. Do not freeze. **Preparation:** Toujeo 5 x 1.5ml (450IU) pre-filled pens. **Legal Classification:** Part 1 Poison **Full prescribing information is available upon request.**

API-HK-TOU-20.09

MAT-HK-2100905-1.0-08/2021

SANOFI

Sanofi Hong Kong Limited
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WONG CHUK HANG, HONG KONG
Tel: (852) 2506 8333 Fax: (852) 2506 2537

REDEFINING EXPECTATIONS

For Those At Risk Of Cardiovascular Events

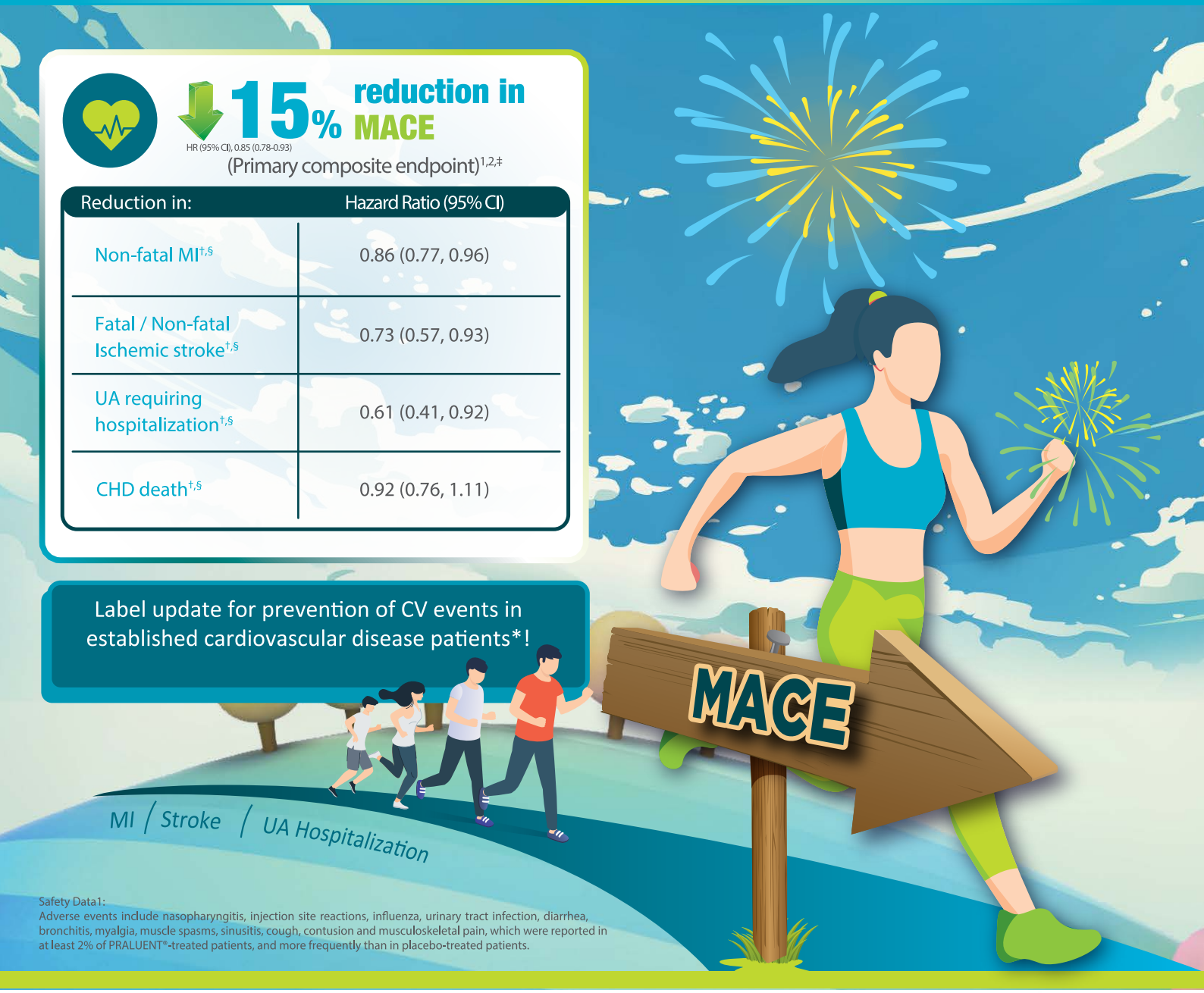
15% reduction in MACE

HR (95% CI), 0.85 (0.78-0.93)

(Primary composite endpoint)[1,2,‡]

Reduction in:	Hazard Ratio (95% CI)
Non-fatal MI[†,§]	0.86 (0.77, 0.96)
Fatal / Non-fatal Ischemic stroke[†,§]	0.73 (0.57, 0.93)
UA requiring hospitalization[†,§]	0.61 (0.41, 0.92)
CHD death[†,§]	0.92 (0.76, 1.11)

Label update for prevention of CV events in established cardiovascular disease patients*!



Safety Data1:
Adverse events include nasopharyngitis, injection site reactions, influenza, urinary tract infection, diarrhea, bronchitis, myalgia, muscle spasms, sinusitis, cough, contusion and musculoskeletal pain, which were reported in at least 2% of PRALUENT®-treated patients, and more frequently than in placebo-treated patients.

* PRALUENT® is indicated to reduce the risk of myocardial infarction, stroke, and unstable angina requiring hospitalization in adults with established cardiovascular disease. PRALUENT® is also indicated as an adjunct to diet, alone or in combination with other lipid-lowering therapies (e.g., statins, ezetimibe), for the treatment of adults with primary hyperlipidemia to reduce low-density lipoprotein cholesterol (LDL-C).

† Statistical testing performed outside hierarchy; therefore not considered statistically significant.

‡ Primary composite endpoint of death from coronary heart disease, nonfatal myocardial infarction, fatal or nonfatal ischemic stroke, or unstable angina requiring hospitalization.

§ Major secondary end points (HR, 95% CI), in order of hierarchical testing, include any coronary heart disease event (0.88, 0.81-0.95), major coronary heart disease event (0.88, 0.80-0.96), any cardiovascular event (0.87, 0.81-0.94), composite of death from any cause, nonfatal myocardial infarction, or nonfatal ischemic stroke (0.86, 0.79-0.93), death from coronary heart disease (0.92, 0.76-1.11, the hierarchical analysis was stopped after the first nonsignificant P value was observed, in accordance with the hierarchical testing plan), death from cardiovascular causes (0.88, 0.74-1.05) and death from any cause (0.85, 0.73-0.98). To adjust for multiplicity, the results of the main secondary end points were to be tested in hierarchical fashion in the sequence listed above if the risk of the composite primary end point was found to be significantly lower in the alirocumab group than in the placebo group.

Study Design[1,2]

ODYSSEY OUTCOMES is a multicenter, randomized, double-blind, placebo-controlled trial involving 18,924 patients who had an acute coronary syndrome 1 to 12 months earlier, had a low-density lipoprotein (LDL) cholesterol level of at least 70 mg per deciliter (1.8 mmol per liter), a non–high-density lipoprotein cholesterol level of at least 100 mg per deciliter (2.6 mmol per liter), or an apolipoprotein B level of at least 80 mg per deciliter, and were receiving statin therapy at a high-intensity dose or at the maximum tolerated dose. Patients were randomly assigned to receive alirocumab subcutaneously at a dose of 75 mg (9462 patients) or matching placebo (9462 patients) every 2 weeks. The dose of alirocumab was adjusted under blinded conditions to target an LDL cholesterol level of 25 to 50 mg per deciliter (0.6 to 1.3 mmol per liter).

MACE=major adverse cardiovascular events. MI=myocardial infarction. UA=unstable angina. PCSK9=Proprotein convertase subtilisin/kexin type 9. CVD=cardiovascular disease. HeFH=Heterozygous Familial Hypercholesterolemia.

Reference:
1. Praluent® Prescribing Information. Mar 2020. 2. Schwartz GG, et al. N Engl J Med. 2018;379:2097-2107.

Presentation: Alirocumab solution for injection. Indications: Prevention of Cardiovascular Events: Reduce risk of myocardial infarction, stroke and unstable angina requiring hospitalization in adults with established cardiovascular disease. Primary Hyperlipidemia (incl. heterozygous familial hypercholesterolemia): As an adjunct to diet, alone or in combination with other lipid-lowering therapies, for the treatment of adults with primary hyperlipidemia to reduce LDL-C. Dosage: 75 mg once every 2 weeks administered subcutaneously. An alternative starting dosage for patients who prefer less frequent dosing is 300 mg once every 4 weeks. If the LDL-C response is inadequate, the dosage may be increased to the maximum dosage of 150 mg administered every 2 weeks. Contraindications: History of serious hypersensitivity reaction to alirocumab. Precautions: Hypersensitivity reactions. Pregnancy and Lactation: There are no available data on use of alirocumab in pregnant women to inform a drug-associated risk. There is no information regarding the presence of alirocumab in human milk, the effects on the breastfed infant, or the effects on milk production. Undesirable effects: Nasopharyngitis, injection site reactions, influenza, urinary tract infection, diarrhea, bronchitis, myalgia, muscle spasms, sinusitis, cough, contusion, musculoskeletal pain, flu-like illness, angioedema. For other undesirable effects, please refer to the full prescribing information. Preparation: 1 x 75mg/ml prefilled pen, 1 x 150mg/ml prefilled pen. Legal Classification: Part 1, First & Third Schedules Poison Full prescribing information is available upon request.
API-HK-ALI-20.07

MAT-HK-2101061-1.0 (09/2021) 4iC_1332

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Praluent® alirocumab

NOTES

FOR TODAY FOR TOMORROW

DECLARE A BETTER TOMORROW

Forxiga® - Your Cardio-renal Guardian

START TODAY BETTER OUTCOME TOMORROW

Largest CVOT of SGLT2i with the **broadest** population from multiple risk factors to established ASCVD[1]

Reduction in cardiorenal events observed in T2DM patients[1]

- ↓17% CV death or hospitalisation for HF*
- ↓24% Cardiorenal composite endpoint†
- ↓47% Renal-specific composite endpoint†

Reassured safety profile of Forxiga®[1]

NEW LABEL AVAILABLE

* hHF alone was a separate, nominally significant exploratory endpoint in the DECLARE trial – the primary endpoint composite of CV death/hHF was driven by hHF.
† Nominally significant, prespecified exploratory outcome.

ASCVD=atherosclerotic cardiovascular disease. CV=cardiovascular. CVOT=cardiovascular outcome trial. hHF=hospitalisation for heart failure. HF=heart failure. SGLT2i=sodium-glucose cotransporter 2 inhibitors. T2DM=type 2 diabetes mellitus.

Reference: 1. Wiviott SD, et al. N Engl J Med 2019;380:347-57.

Abridged 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. **Dosage and Administration:** 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. 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 a GFR < 60 ml/min; with type 1 diabetes; with hereditary problems of galactose intolerance, the total lactase deficiency, or glucose-galactose malabsorption. Discontinue if GFR is persistently below 45 ml/min; 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; 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. 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.0720**

Please contact HKPatientSafety@astrazeneca.com for reporting of Individual Case Safety Report (ICSR) to AstraZeneca Hong Kong Limited.
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HK-4119 14/08/2020 4iC_1441A

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forxiga® dapagliflozin

ACKNOWLEDGEMENTS