DPP 2014

Diabetes Preventing the Preventables Forum 2014

亞洲糖尿病基金會
Asia Diabetes Foundation

23-25 May 2014

www.adf.org.hk/dpp2014

Courtyard by Marriott Hong Kong Sha Tin

In Partnership With:

International Diabetes Federation
IDF Centre of Education 2011-2015

The Chinese University of Hong Kong
Prince of Wales Hospital
International Diabetes Federation Centre of Education

Contents

Welcome Message

Dear Faculty and Delegates,

Welcome to Hong Kong, a beautiful city with a rich history and culture. Just like most cities in Asia undergoing rapid transition, diabetes has come a long way over the last two decades. From a simple classification of type 1 and type 2 diabetes, there is now an explosion of information on the phenotypes and genotypes of this complex disease. From a handful of medications, we are now overwhelmed with a growing number of compounds for the treatment, of not just diabetes, but many of its associated conditions and comorbidities.

Diabetes is a lifelong disease and the most challenging aspect in managing diabetes is to help patients manage their disease for the rest of their life. To do this effectively, the care team has to systematically collect and manage a large amount of information, collected over time, in order to assess and advise their patients accordingly.

Our health care systems were never designed to manage these chronic problems and information, which are new health care challenges. This DPP Forum is a meeting designed to address these needs. It aims to foster collaborations amongst all relevant stakeholders, who see the need of a new paradigm shift to change the way chronic care should be delivered, in order to bring out the best of our expertise and technologies to make chronic care accessible, sustainable and affordable.

To this end, we are fortunate to have invited a faculty of experts and thought leaders with a diversity of experiences who will share with us their views and insights into this health care challenge.

We shall also share with you the successes and challenges in implementing the Joint Asia Diabetes Evaluation (JADE) Program, which aims to create a structured environment to allow a professional team to personalize care augmented by information technology.

We hope you will enjoy this meeting and that you will continue to be part of this growing network to prevent and control diabetes and chronic disease.

Juliana Chan

Co-director, IDFCE
CEO, ADF

Francis Chow

Co-director, IDFCE
Executive Councillor, ADF

Greg Lyubomirsksy

Executive Councillor, ADF

Organizer

Organizer

亞洲糖尿病基金會
Asia Diabetes Foundation

Asia Diabetes Foundation

Flat 4B, Block B, Staff Quarters, Prince of Wales Hospital,
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www.adf.org.hk

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Organizing Committee

Chairman Prof. Juliana CHAN

Co-Chairman Dr. Francis CHOW
Dr. Greg LYUBOMIRSKY

Members Ms. Sally CHAU
Dr. Alice KONG
Dr. Christine KWAN
Ms. Vanessa LAU
Dr. Andrea LUK
Dr. Risa OZAKI
Mr. Patrick WONG
Dr. Rose YEUNG
Ms. Rebecca YUE

Faculty

Prof. Philip Clarke

Professor of Health Economics, School of Population Health,
The University of Melbourne, Australia

Professor Philip Clarke leads an active health economic research group at the University of Melbourne. He has had previous appointments at the University of Oxford and the University of Sydney. While serving as Research Fellow at the University of Oxford, he was involved in the economic analysis of the United Kingdom Prospective Diabetes Study (UKPDS), a landmark trial of policies to improve the management of people with type 2 diabetes. The culmination of this research was the development of the UKPDS Outcomes Model, a computer simulation model for predicting outcomes for patients with type 2 diabetes. He has since worked on two other large diabetes clinical trials and several other long-term studies involving people with diabetes. He has over 70 peer review publications and has recently contributed to books on cost-effectiveness analysis and cost-benefit analysis published by Oxford University Press.

Prof. Ronald Ma

Professor, Department of Medicine and Therapeutics,
The Chinese University of Hong Kong, Hong Kong

Professor Ronald Ma is currently Professor at the Department of Medicine and Therapeutics, The Chinese University of Hong Kong, and Honorary Consultant at the Prince of Wales Hospital, Hong Kong. His research interests include the epidemiology and genetics of diabetes and its complications, gestational diabetes and polycystic ovary syndrome.

Prof. Hung-Yuan Li

Associate Professor and Attending Physician, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan

Deputy Secretary-General, Diabetes Association of R.O.C.

Professor Hung-Yuan Li is currently Associate Professor and Attending Physician of the Department of Internal Medicine, National Taiwan University Hospital, Taiwan. Since March 2007, Dr. Li has been Deputy Secretary-General in Diabetes Association of R.O.C. He participates in various activities including drafting of the Taiwan Declaration on Diabetes, lighting buildings in blue for world diabetes day, production of a song for diabetes, as well as drafting and editing several clinical guidelines. In research, Dr. Li is interested in epidemiology of diabetes and the application of biomarkers. He received Professor Fang-Wu Chen Outstanding Research Award from Diabetes Association of R.O.C. and Endocrine society of R.O.C. in 2011. Dr. Li has published 55 papers in peer-reviewed journals including Diabetes, Diabetes Care, Obesity, Clinical Endocrinology, Diabetic Medicine, and Pediatric Diabetes.

Prof. Soo Lim

Associate Professor, Internal Medicine, Seoul National University College of Medicine and Seoul National University Bundang Hospital, Korea

Professor Soo Lim graduated from Seoul National University College of Medicine and completed his residency and fellowship at the Seoul National University Hospital. He studied diabetes at MGH, Harvard Medical School, Boston from 2011 to 2012. His major fields are diabetes, dyslipidemia, and cardiovascular complications. He is an active member of the Korean Diabetes Association and the Korean Society of Lipidology and Atherosclerosis. He has published more than 120 papers in peer-reviewed journals. He is currently actively involved in clinical and laboratorial research.

Prof. Alice Kong

Associate Professor, Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong

Professor Alice Kong is currently Associate Professor of the Department of Medicine and Therapeutics at The Chinese University of Hong Kong. Her research focuses on type 2 diabetes and obesity, in particular lifestyle factors and cardiovascular risk factors clustering in adults and in adolescents. Her recent publications describe the epidemiology and associated cardiovascular risk factors in Hong Kong Chinese diabetic patients and in the youth population.

Prof. Björn Eliasson

Professor, Department of Medicine, Sahlgrenska University Hospital, Sweden

Professor Björn Eliasson is Senior Consultant at Sahlgrenska University Hospital and Professor in Internal Medicine at the University of Gothenburg, Goteborg, Sweden. He is Director of the Diabetes Centre at Sahlgrenska University Hospital as well as Regional Obesity Centre in the Region of Western Sweden. He has previously been President of the Swedish Society for Diabetology, and has also been involved in developing the Swedish National Diabetes Register for 15 years.

Prof. Jacques Bédard

Full Professor of Internal Medicine, Faculty of Medicine, Université de Sherbrooke, Canada

Professor Jacques Bédard has been a specialist in internal medicine since 1976. He is Fellow of both the Canadian Royal College of Physicians and American College of Physicians, and serves as an Internal Medicine Consultant at Sherbrooke University Hospital.

Professor Bédard is also Full Professor of Internal Medicine at Université de Sherbrooke and Principal Investigator at London Clinical Research Center. He has participated in more than 50 multi-centre international clinical trials, and has been a guest speaker at more than 1200 CME-related conferences.

Dr. Daphne Gardner

Consultant, Department of Endocrinology, Singapore General Hospital, Singapore

Dr. Gardner graduated from the University of Oxford (UK) in 2001 [BA (Physiological Sciences), BMBCh (Oxon)]. She was a Clinical Lecturer in Plymouth (UK). She attained specialist accreditation in Endocrinology in Singapore in 2011 and spent her fellowship year at the Oxford Centre for Diabetes, Endocrinology and Metabolism, UK. She now serves as Consultant Endocrinologist and lead clinical for sgDAFNE (Dose Adjustment for Normal Eating) in Singapore General Hospital. Her subspecialty interests include self-management in type 1 diabetes, transition services for young adults/adolescents and monogenic diabetes.

Dr. Enyu Imai

Director, Nakayamadera Imai Clinic, Takarazuka, Hyogo, Japan

Dr. Imai is both a nephrologist and an educator, and has published more than 200 papers during his years at the Nagoya University. He is currently Director of Nakayamadera Imai Clinic, and Board of Director of Kidney Disease - Improving Global Outcome (KDIGO). He is also heavily involved in editing various journals include American Journal of Kidney Diseases, Nephrology Dialysis Transplantation, Journal of the American Society of Nephrology, Nature Review Nephrology, and Clinical Nephrology.

Dr. Parinya Chamnan

Doctor of Department of Social Medicine and Deputy Director of Medical Education Center, Sanpasitthiprasong Hospital, Thailand

Dr. Parinya Chamnan received his medical degree from Chulalongkorn University and later completed his Ph.D. in Epidemiology at the University of Cambridge, England in 2010. Dr Parinya is now working as a clinician, research scientist and clinical instructor at the Department of Social Medicine, Sanpasitthiprasong Hospital, an affiliated hospital of Khon Kaen University and Ubon Ratchathani University. His research interest is on the prevention of diabetes and cardiovascular disease, with particular focus on population risk stratification.

Programme 23–25 MAY 2014

FRIDAY 23 MAY

14:00-17:00	JADE principal investigators meeting	
18:00-21:00	Dinner (by invitation)	

SATURDAY 24 MAY

Plenary Symposium (1)

Co-chair: Dr. Francis Chow and Dr. Su-Yen Goh

09:00-09:10	Welcome	Juliana Chan and Francis Chow
09:10-09:55	Health economics – A case for type 2 diabetes	Philip Clarke, Australia
09:55-10:25	Future of genomic medicine and personalized care	Ronald Ma, Hong Kong
10:25-10:40	Coffee break sponsored by Takeda	

Symposium (1)

Co-chair: Prof. Yook-Chin Chia and Dr. Ma Mercedes Reyes-dela Rosa

10:40-11:10	Screening strategy for gestational diabetes	Hung-Yuan Li, Taiwan
11:10-11:40	Association of vitamin D deficiency with cardiovascular and metabolic risk	Soo Lim, Korea
11:40-12:10	Hypoglycemia identifies vulnerable patients with type 2 diabetes	Alice Kong, Hong Kong

Pfizer Lunch Symposium - Recognizing Painful Diabetic Peripheral Neuropathy in Diabetic Patients

Co-chair: Prof. Juliana Chan and Prof. Ronald Ma

13:00-13:30	Neurobiology of pain and mood in diabetic peripheral neuropathy	Wing-King Lee, Hong Kong
13:30-14:00	Recommendations for the management of painful diabetic peripheral neuropathy	Stephen Wong, Hong Kong

Plenary Symposium (2)

Co-chair: Prof. Larry Ho and Dr. Wing-Yee So

14:00-14:45	How to set up a national diabetes registry	Björn Eliasson, Sweden
14:45-15:30	Behaviour change: from conviction to confidence	Jacques Bédard, Canada
15:30-15:45	Coffee break sponsored by Takeda	

Symposium (2)		Co-chair: Dr. Alexander Tan and Dr. Roseanne Yeung
15:45-16:15	MODY and type 1 diabetes – An Asian perspective	Daphne Gardner, Singapore
16:15-16:45	Albuminuria predicts cardiovascular-renal outcome – insights from the ORIENT study	Enyu Imai, Japan
16:45-17:15	Using A1c to predict diabetes and cardiovascular disease	Parinya Chamnan, Thailand
17:15-17:25	Closing remarks	Greg Lyubomirsky and Juliana Chan

By Invitation MSD Satellite Dinner Symposium - Diabetes Integrated Care Model (Venue: YMCA of Hong Kong)		Co-chair: Prof. Clive Cockram and Prof. Alice Kong
18:30-19:15	Using logistics and information technology to integrate care	Juliana Chan, Hong Kong
19:15-19:45	JADE program in Vietnam	Thy-Khue Nguyen, Vietnam

SUNDAY 25 MAY

Post-forum JADE Program Training Workshop	
09:00-10:00	Experiential learning to improve care
10:00-10:30	What can you learn from the JADE report?
10:30-10:45	Coffee break
10:45-11:30	JADE Portal demonstration and hands-on experience
11:30-12:00	Can we use JADE/DIAMOND/LANDSCAPE to change practice and improve clinical outcome?
12:00-12:30	Closing remarks

Plenary Symposium (1)

Health economics – A case for type 2 diabetes

Saturday | 24 May 2014 | 9:10 a.m.

Philip Clarke

Professor of Health Economics, School of Population Health, The University of Melbourne, Australia

Health economic simulation models are increasingly being worldwide for the assessment of cost-effectiveness of treatments and prevention strategies for people with type 2 diabetes. The purpose of this presentation is to review the working of diabetes simulation models primarily focusing on the UKPDS Outcomes Model. Unlike single equation models such as the UKPDS Risk Engine, the Outcomes Model uses multiple equations to represent both macro-vascular (e.g. myocardial infarction, other ischaemic heart disease, congestive heart failure, stroke) and selected micro-vascular (e.g. blindness) complications. While the Model can estimate the risk over time of each complication and death, it can also estimate outcomes in terms of life expectancy and Quality Adjusted Life Years (QALYs), as these are the most commonly used metrics in economic evaluation. Reduced rates of complications may also reduce health care costs, producing savings which may offset some of the costs of improving treatment. To capture these benefits, a simulation model can also be used to estimate the lifetime health care costs that are related to diabetes-related complications.

A key challenge is to develop similar diabetes simulation models for Asian populations, particularly as studies such as JADE and ADVANCE have demonstrated significant differences in the profile of complications among Asian populations. The presentation will review ways to adapt existing models and opportunities to develop models based on emerging studies that collect patient level data in the Asian region. The presentation will conclude with discussion of how they can be used to assist in evaluation and thereby improve the process of decision making. It is important for academics and public-section decision makers to have access to independently developed simulation models in order to rigorously evaluate costly new technologies as well as plan for the future.

Future of genomic medicine and personalized care

Saturday | 24 May 2014 | 9:55 a.m.

Ronald Ma

Professor, Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong

Diabetes is a complex disease with heterogeneity in disease phenotype, variable patient response to treatment and possible risk associated with particular therapeutic strategies. The latest guidelines from professional organizations including the ADA and EASD have highlighted the need for more personalized treatment strategies compared to the "one-size-fits-all" approach of old. Recent advances in genome-wide association studies have led to the identification of more than 70 common genetic variants associated with type 2 diabetes, and a similar number for obesity. Nevertheless, the risk conferred by each individual marker remains limited, and collectively, the variants offer only limited improvement in the prediction of diabetes above that given by clinical risk factors. Increasing number of genetic variants have been identified to be associated with the risk of diabetic complications, or different response to glucose-lowering treatment, thereby opening up the possibility of personalized treatment regimes based on the genetic profile of patients. Recent advances in sequencing technology have provided researchers with unparalleled ability to interrogate the genome, though much of the heritability of diabetes and other NCDs remain unexplained. Emerging insights in epigenetics have revealed previously unsuspected complexities in the regulation of gene expression, providing great opportunities to discover important markers to guide prediction and treatment. The integration of these different layers of genomic information into clinical care should provide truly "personalized" diabetes care, and hopefully, improved outcome.

Symposium (1)

Screening strategy for gestational diabetes

Saturday | 24 May 2014 | 10:40 a.m.

Hung-Yuan Li

Associate Professor and Attending Physician, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan

Deputy Secretary-General, Diabetes Association of R.O.C.

The International Association of the Diabetes and Pregnancy Study Group (IADPSG) criteria for gestational diabetes (GDM) were proposed in 2010, and it is recommended by the American Diabetes Association as an alternative apart from the Carpenter and Coustan criteria this year. Based on the findings from a retrospective study, we found that the IADPSG criteria are associated with a significant increase in GDM prevalence (13.32% vs. 2.56%). However, the diagnosis can be made two weeks earlier, and there are significant reductions in maternal weight gain and birth weight. Besides, adopting the IADPSG criteria is associated with reduced risk of primary cesarean section, macrosomia, jaundice, and admission to neonatal ICU. In Taiwan, adopting the IADPSG criteria is associated with slightly increased costs, but costs less to identify women with GDM. Taken together, our findings suggest that adopting the IADPSG criteria improves perinatal outcomes and is cost-efficient, despite the increased prevalence of GDM. Therefore, adopting the IADPSG criteria may be reasonable in Taiwan. Moreover, we have also explored the optimal cutoffs to diagnose GDM by using 75g oral glucose tolerance test and proposing an algorithm to reduce the need of OGTT. These findings will be discussed in this symposium.

Association of vitamin D deficiency with cardiovascular and metabolic risk

Saturday | 24 May 2014 | 11:10 a.m.

Soo Lim

Associate Professor, Internal Medicine, Seoul National University College of Medicine and Seoul National University Bundang Hospital, Korea

Vitamin D is a multifunctional hormone that can affect many essential biological functions ranging from immune regulation to mineral ion metabolism. Although the major function of vitamin D is to maintain calcium and phosphate homeostasis and to promote bone mineralization, many nontraditional roles for vitamin D have been identified recently. A close association between altered vitamin D concentration and vascular calcification has been reported recently. We investigated vitamin D status and its association with subclinical atherosclerosis in a population-based cohort study, the Korean Longitudinal Study on Health and Aging (KLoSHA). Participants were 439 men and 561 women aged 65 year or older who were recruited by random stratified sampling for KLoSHA. Anthropometric and biochemical parameters, the concentration of 25-hydroxyvitamin D (25-OHD), and intact PTH were measured. We evaluated the coronary artery calcium score and stenosis using multidetector-row cardiac computed tomography, the intima-media thickness using carotid sonography, pulse wave velocity, and the ankle-brachial index. Among the participants, 49.8, 44.2, and 6.0% had 25-OHD deficiency (< 15 ng/ml), insufficiency (15–29.9 ng/ml), and adequacy (>= 30 ng/ml), respectively. The frequency of coronary artery stenosis (>= 50%) differed between 25-OHD categories: 18.5, 12.9, and 1.9% in the 25-OHD-deficient, -insufficient, and -adequate groups, respectively ($P < 0.05$). After adjusting for cardiometabolic risks and intact PTH concentration, multivariate regression analysis showed that participants with a low 25-OHD concentration had a higher risk of significant coronary artery stenosis; the odds ratios were 2.08 for 25-OHD concentration of 15–29.9 ng/ml vs. at least 30 ng/ml and 3.12 for 25-OHD concentration below 15 ng/ml vs. at least 30 ng/ml (both $P < 0.05$). Conclusions: The association between 25-OHD inadequacy and subclinical atherosclerosis underscores the clinical implications of vitamin D status. An intervention strategy to increase vitamin D level through vitamin D-fortified diet and adequate sun exposure may mitigate the consequences of vitamin D deficiency.

In a different context, recent studies suggest an association between 25-hydroxyvitamin D (25-OHD) and type 2 diabetes (T2D) risk. However, prospective studies investigating the relationship between vitamin D inadequacy and incidence of T2D incorporating obesity and dynamic measures of insulin resistance (IR) and pancreatic β-cell function are limited. So, we tested the hypothesis that baseline 25-OHD is associated with incidence

of T2D in high-risk subjects for up to 5 years of follow-up, independently of obesity, and baseline IR and β-cell function. We recruited 1080 nondiabetic Korean subjects (mean age = 49.5 ± 11.4 years), based on the presence of one or more risk factors for T2D including obesity, hypertension, dyslipidemia and/or family history of T2D. We measured anthropometric and biochemical parameters, the homeostasis model assessment 2 of IR (HOMA2-IR), and insulinogenic index (IGI) calculated by ΔInsulin30/ΔGlucose30 from a 75-g oral glucose tolerance test. Among participants, 10.5% had serum 25-OHD deficiency (<10 ng/ml), 51.6% had insufficiency (10.0-19.9 ng/ml), and 38.0% had sufficiency (≥20 ng/ml), with 32.3±15.6 months incidence of T2D declining accordingly: 15.9%, 10.2%, and 5.4% in corresponding groups (P<0.001). After adjusting for age, sex, blood pressure, lifestyles, family history, season, parathyroid hormone and hsCRP, participants with 25-OHD deficiency had increased risk of T2D independently of BMI, HOMA2-IR, and IGI; the hazard ratios were 2.06 for 25-OHD 10-19.9 ng/ml vs. ≥20 ng/ml (95% CI:1.22, 3.49) and 3.23 for 25-OHD <10 ng/ml vs. ≥20 ng/ml (95% CI:1.66-6.30).

In conclusion, these study findings suggest an association between vitamin D activity and cardiovascular and metabolic risk.

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Hypoglycemia identifies vulnerable patients with type 2 diabetes

Saturday | 24 May 2014 | 11:40 a.m.

Alice Kong

Associate Professor, Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong

Hypoglycemia is an important but often neglected aspect in diabetes management. In both the ADVANCE(1) and ACCORD(2) study, intensive blood glucose lowering was associated with increased risk of severe hypoglycemia. Although in both studies, researchers had reported high rates of multiple adverse events including hospitalizations and all-cause death in the 12 months after the incident event of hypoglycemia, detailed analysis did not reveal increased risk of cardiovascular disease (CVD)(1). Furthermore, while intensive treatment was associated with increased risk of hypoglycemia, in both ADVANCE(1) and ACCORD(2) study, intensively-treated patients tended to have a lower risk of death than those treated conventionally(2). These findings suggested that intensive monitoring in the former group might have prompted corrective actions to reduce adverse clinical outcomes. On the other hand, hypoglycemia is known to increase risk of CVD and mortality in diabetic patients(3) due to reasons including but not limited to arrhythmia(3), abnormal hemostasis and neurohormonal dysregulation. Other host factors such as age and comorbidities, notably, chronic kidney disease (CKD) might also influence clinical outcomes(4). Against this background, professional bodies and experts recommended comprehensive assessments and individualizing treatment goals based on patients' risk profiles, comorbidities, coping skills, cognitive states, and social support in order to maximize benefits and minimize harm(5, 6).

A consecutive cohort of 8,767 type 2 diabetic patients from the Hong Kong Diabetes Registry (mean age 57.4 years), with and without severe hypoglycemia in the 12 months prior to enrolment, were recruited between 1995 and 2007 with follow-up until 2009(7). We explored if type 2 diabetic patients with severe hypoglycemia exhibited particular phenotypes which might predict future events including premature death and all-site cancer. Severe hypoglycemia was defined as hospitalizations due to hypoglycemia using the International Classification of Diseases (ICD-9) codes. At enrolment, patients with and

without severe hypoglycemia had similar cancer rates. During follow-up, patients with severe hypoglycemia had higher incidence of all-site cancer (13.4% vs. 6.4%, p<0.0001) and mortality (32.8% vs. 11.2%, p<0.0001) than those without severe hypoglycemia. After adjusting for confounders, old age, low body mass index (BMI), high glycated hemoglobin (HbA_{1c}), low triglyceride, low LDL-C (low density lipoprotein cholesterol), albuminuria and chronic kidney disease (CKD) were independent predictors for severe hypoglycemia. In conclusion, severe hypoglycemia in type 2 diabetic patients is associated with advanced age, renal dysfunction, poor glycemic control and cancer-subphenotypes (low BMI, low LDL-C and low triglyceride).

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Plenary Symposium (2)

How to set up a national diabetes registry

Saturday | 24 May 2014 | 2:00 p.m.

Björn Eliasson

Professor, Department of Medicine, Sahlgrenska University Hospital, Sweden

Swedish law states that care must be quality assured. This and the St. Vincent declaration in 1989 led to the start of the Swedish National Diabetes Register, NDR. In parallel, evidence-based guidelines for diabetes care were developed in 1996, and updated in 1999, 2010 and are currently being revised again.

NDR was launched in 1996 after negotiations between organisations representing diabetologists, endocrinologists, specialists in internal and general medicine, diabetes educators and the patients. The main aim originally was to monitor clinical results on a local level, to compare with national means.

Patient data are reported at least annually, via online registration or transferral of patient or clinic data. Clinical characteristics are reported, as well as risk factor control, complications and treatments. The data can be used with the patient at the consultation, to optimize treatment and discuss follow-up and plans.

The aggregated results of the clinic are used to monitor the results continuously, and to compare with previous results and national means. Are there areas of the diabetes care that must be improved? Are the treatments in accordance with guidelines? Are the processes working: are feet and eye examinations carried out at regular intervals; are the medications appropriate?

The results are available online in real-time. Standard statistics are readily available, and it is easy to tailor statistics to study subgroups. A written comprehensive and widely distributed report is published in the spring every year. It covers the national and regional results but also results on hospital level. Due to the small number of patients in general practices and possible case mix, results in primary care has only been presented on a regional level.

In some regions, the care-giver, which exclusively fund diabetes care, to a minor degree uses the results of the clinics as a basis for funding. The national authorities also use the data to monitor adherence to treatment guidelines, and can promote efforts to improve problem areas. Such a program is currently being launched. Furthermore, there is also a continuous surveillance of severe complications on national and regional levels, by link with national registers with information on diagnoses associated with hospitalizations, as well as causes of death.

Today NDR is an indispensable part of diabetes care in Sweden.

Behaviour change: from conviction to confidence

Saturday | 24 May 2014 | 2:45 p.m.

Jacques Bédard

Full Professor of Internal Medicine, Faculty of Medicine,
Université de Sherbrooke, Canada

Behaviour change: from conviction to confidence

Behavioural changes (physical activity, diet, compliance, smoking cessation) are fundamental for prevention **and** active treatment of diabetic patients.

The classical (professional-oriented) approach identifies the behaviours that we think the patient should change, information about the **Why** (thinking, falsely, that knowledge equals motivation) and instructions on **How** to change (a traditional directive approach).

Patients, however, don't change!

In response to this problem, we present a practical **universal** (patient-centered) intervention tool (identical for **all** professionals and **all** behaviours) that leads to behavioural change.

This targeted intervention uses recognition of the apparent Stage of Change (Prochaska's model), confirmed by the Conviction level, to develop one of three specific intervention scenarios with the proper closing technique for each scenario.

It uses a variation of the "Motivational Interviewing" communication technique. Through the skilful use of **open questions**, it provokes, reinforces and accelerates progress along the path to change rather than directing it.

Used by different members of the **same therapeutic team** (physicians, nurses, pharmacists, nutritionists, kinesiologists), it creates a **synergy** that increases the acceleration of patient progress as they move from one professional to the next.

Across the spectrum of medical interventions, we have spent more than 30 years focusing on the **WHY** of changing patient behaviours – the time has now come to promote the **HOW**!

Symposium (2)

MODY and type 1 diabetes – An Asian perspective

Saturday | 24 May 2014 | 3:45 p.m.

Daphne Gardner

Consultant, Department of Endocrinology, Singapore General Hospital, Singapore

The Western Pacific region currently has the highest prevalence of diabetes in the world. What is worrying is a projected increase in diabetes prevalence worldwide to 592 million by 2035. Furthermore, the estimated fourfold increase in diabetes prevalence in the youth spells a grave outlook for the future burden of diabetes.

Young onset diabetes (<45 years) presents a wide spectrum of aetiologies: type 1 and 2 diabetes (T1D & T2D) and less well-defined subtypes e.g. LADA and monogenic DM. Each subtype portends a unique clinical course, optimum treatment modality and risk of transmission. In Asians, delineating these subtypes is challenging; the lower prevalence of β cell autoantibodies (Abs) (40%) compared to Caucasians (>85%), lower age of onset of T2D and lower body mass index (BMI) at onset of T2D increases this difficulty.

Clinical assessment should therefore be individualised to ascertain those who are insulinopenic, are at higher risk of developing ketoacidosis and need insulin replacement. Conversely, continual re-assessment is necessary to determine which individuals do not have T1D, and who may do better without insulin. Asian T1D patients often do not receive education in carbohydrate counting and self-management. Both these aspects of T1D management are covered in the structured education programme Dose Adjustment for Normal Eating (DAFNE); this course has been demonstrated in a randomised controlled trial to improve glycaemic control significantly without an increase in severe hypoglycaemia, as well as having positive effects on quality of life, and psychological well-being.

A smaller proportion of patients with young adult onset diabetes harbour rare variants that lead to monogenic forms of diabetes like Maturity onset Diabetes of the Young (MODY). Its prevalence has been estimated to be 1-2% of the diabetes population, although this is largely underestimated, particularly in the Asian population where little is known of its prevalence. Applying pharmacogenomics in MODY demonstrates the classic paradigm of personalised medicine that targets disease aetiology. Individuals with Glucokinase (GCK) MODY demonstrate lifelong mild fasting hyperglycaemia, yet maintain β-cell insulin production ability and do not require treatment; those with HNF1A and HNF4A may often respond well to sulphonylurea therapy, allowing insulin therapy to be stopped.

A big challenge in managing Asian patients with diabetes therefore includes developing diagnostic algorithms to better classify diabetes subtypes amongst a heterogeneous pool of patients, in order to better prognosticate and treat.

Albuminuria predicts cardiovascular-renal outcome – insights from the ORIENT study

Saturday | 24 May 2014 | 4:15 p.m.

Enyu Imai

Director, Nakayamadera Imai Clinic, Takarazuka, Hyogo, Japan

Diabetic nephropathy is the leading cause of end stage renal disease (ESRD) worldwide and 40-50% of ESRD is caused by diabetes. Albuminuria is a sensitive biomarker for prediction of cardiovascular outcome as well as renal one. ACE inhibitor and angiotensin II receptor blockers (ARB) attenuate proteinuria, slow rate of decline of renal function and reduce the incidence of ESRD in patients with diabetic nephropathy. In these type 2 diabetic patients with overt nephropathy, greater percentage reduction of proteinuria at 6-months also predicted better renal outcome. However, the effect size of percentage reduction of proteinuria and impact of remission of proteinuria had not been well established in diabetic nephropathy. In a post hoc analysis of the ORIENT, a double-blind randomized trial of 566 type 2 diabetic patients with nephropathy, we examined the risk association of composite renal outcome (end stage renal disease, doubling of serum creatinine and death) with baseline, change and residual urinary protein/creatinine ratio (UPCR). We estimated the respective hazard ratios (HR) with 95% confidence interval (CI) of with composite renal outcome with baseline UPCR (low:<1.0 g/gCr; moderate:≥1.0 g/gCr,<3.0 g/gCr; and high:≥3.0 g/gCr) as well as percentage reduction of UPCR (Δ) (worsening:<0%; moderate:≥0%, <30%; and high:≥30%) and residual UPCR at 24-week (remission:<1.0 g/gCr; moderate:≥1.0 g/gCr, <3.0 g/gCr; and heavy:≥3.0 g/gCr). Compared to the low group with baseline UPCR<1.0g/gCr, the respective HR with 95% CI in the moderate and high UPCR groups were 3.02 (1.76-5.19) and 9.24 (5.43-15.73). Compared to patients with worsening UPCR (<0%) at 24-weeks, the HR was 0.54 (0.39-0.74) in those with ≥0%, <30% ΔUPCR, and 0.43 (0.31-0.61) in those with ≥30% ΔUPCR. Compared to the remission at 24-week, the HR was 2.12 (1.28-3.49) in moderate residual proteinuria and 4.59 (2.74-7.69) in heavy residual proteinuria. Compared to patients with residual UPCR≥1.0 g/gCr and ΔUPCR<30%, the HR in those with ΔUPCR≥30% and residual UPCR<1.0 g/gCr was 0.38 (0.22-0.64). In conclusion, patients with type 2 diabetes and overt nephropathy, over 30% reduction of UPCR compared to baseline and/or residual UPCR<1.0 g/gCr at 24-week predicted renoprotection. These values may be used as targets to guide anti-proteinuric and renoprotective therapy in diabetic nephropathy.

Using A1c to predict diabetes and cardiovascular disease

Saturday | 24 May 2014 | 4:45 p.m.

Parinya Chamnan

Doctor of Department of Social Medicine and Deputy Director of Medical Education Center, Sanpasitthiprasong Hospital, Thailand

My talk will cover issues related to the use of HbA1c to predict diabetes and its main complication, cardiovascular disease. HbA1c had been used for monitoring of diabetes control for many years before it was recommended to be included as diagnostic criteria for diabetes in 2011. I will discuss the association between HbA1c and diabetes observed in different populations and whether HbA1c predicts diabetes better than other measures of blood glucose. When categorizing non-diabetic individuals by their HbA1c, we found that a small proportion had a baseline HbA1c in the range 6.0-6.4% but one-third of incident cases arose in this group. The cumulative incidence of diabetes in this group over 3 years was 15 times higher than in those with a baseline HbA1c of <5.0%. However, we found that the majority of incident cases of diabetes arose from those with HbA1c in a normal range, underlining the importance of the use of population-based in addition to high risk approaches to prevention of diabetes. Furthermore, the ability of HbA1c and other measures of blood glucose for predicting cardiovascular disease will be discussed. A conventional approach to risk prediction is to use information on risk factors including blood glucose at a single time point for prediction of cardiovascular disease, a little is known about the predictive value of using blood glucose from different time points or changes in blood glucose over time in a non-diabetic population. Although many studies show that HbA1c was strongly associated with the risk of cardiovascular disease, the addition of information on change in HbA1c over 3 years did not improve the prediction of cardiovascular disease over and above information on HbA1c and other major cardiovascular risk factors from a single time point.

Notes

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See you next year!

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Acknowledgments

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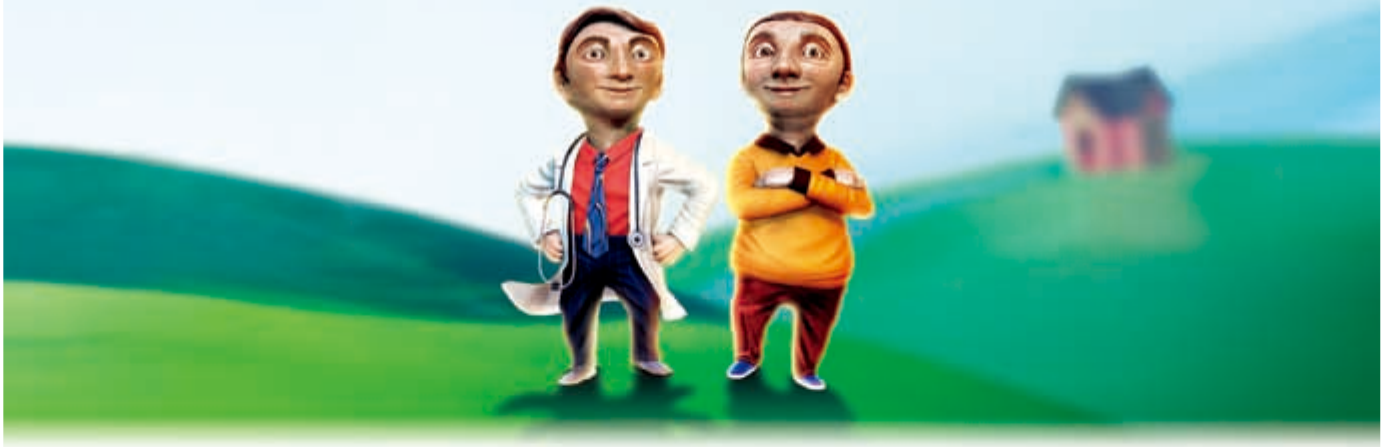

* In patients with baseline HbA1c ≥ 10% coadministered with saxagliptin and metformin IR

References:
1. Kombiglyze™ XR prescribing information. 2. Jadzinsky M, Pfützner A, Paz-Pacheco E, et al. Saxagliptin given in combination with metformin as initial therapy improves glycaemic control in patients with type 2 diabetes compared with either monotherapy: a randomized controlled trial. Diabetes Obes Metab 2009;11:611-622.

KOMBIGLYZE XR (saxagliptin and metformin HCl extended-release) tablets
INDICATIONS AND USAGE: KOMBIGLYZE XR is a dipeptidyl peptidase-4 inhibitor and a biguanide combination product indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus when treatment with both saxagliptin and metformin is appropriate; **DOSAGE FORMS AND ADMINISTRATION**: Tablets: 2.5 mg saxagliptin/1000 mg metformin HCl extended-release, 5 mg saxagliptin/500 mg metformin HCl extended-release, 5 mg saxagliptin/1000 mg metformin HCl extended-release. Administer once daily with evening meal; swallow whole; starting dose based on patient's current regimen then adjust the dose based on effectiveness and tolerability; limit saxagliptin dose to 2.5 mg daily for patients taking strong cytochrome P450 3A4/5 inhibitors. **CONTRAINDICATIONS:** Renal impairment; metabolic acidosis, including diabetic ketoacidosis; hypersensitivity to the active substance or to any of the excipients. **WARNINGS AND PRECAUTIONS:** ◊ Lactic acidosis: warn patients against excessive alcohol intake; ◊ not recommended in hepatic impairment; ◊ temporarily discontinue in patients undergoing radiologic studies with intravascular administration of iodinated contrast materials; ◊ if pancreatitis is suspected, promptly discontinue; ◊ metformin might lower vitamin B12 levels in patients with vitamin B12 deficiency; ◊ when used with an insulin secretagogue or insulin, a lower dose of insulin secretagogue or insulin may be required to minimize risk of hypoglycemia. **ADVERSE REACTIONS:** ◊ Adverse reactions reported in ≥ 5% of patients treated with metformin extended-release and more commonly than in patients treated with placebo are: diarrhea and nausea/vomiting; ◊ Adverse reactions reported in ≥ 5% of patients treated with saxagliptin and more commonly than in patients treated with placebo are: upper respiratory tract infection, urinary tract infection, and headache; ◊ Adverse reactions reported in ≥ 5% of treatment-naïve patients treated with coadministered saxagliptin and metform and more commonly than in patients treated with placebo are: headache and nasopharyngitis; ◊ hypoglycaemia when in combination with SU and insulin; ◊ hypersensitivity-related events were reported more commonly in patients treated with saxagliptin than in patients with placebo. Version Nov 2013.

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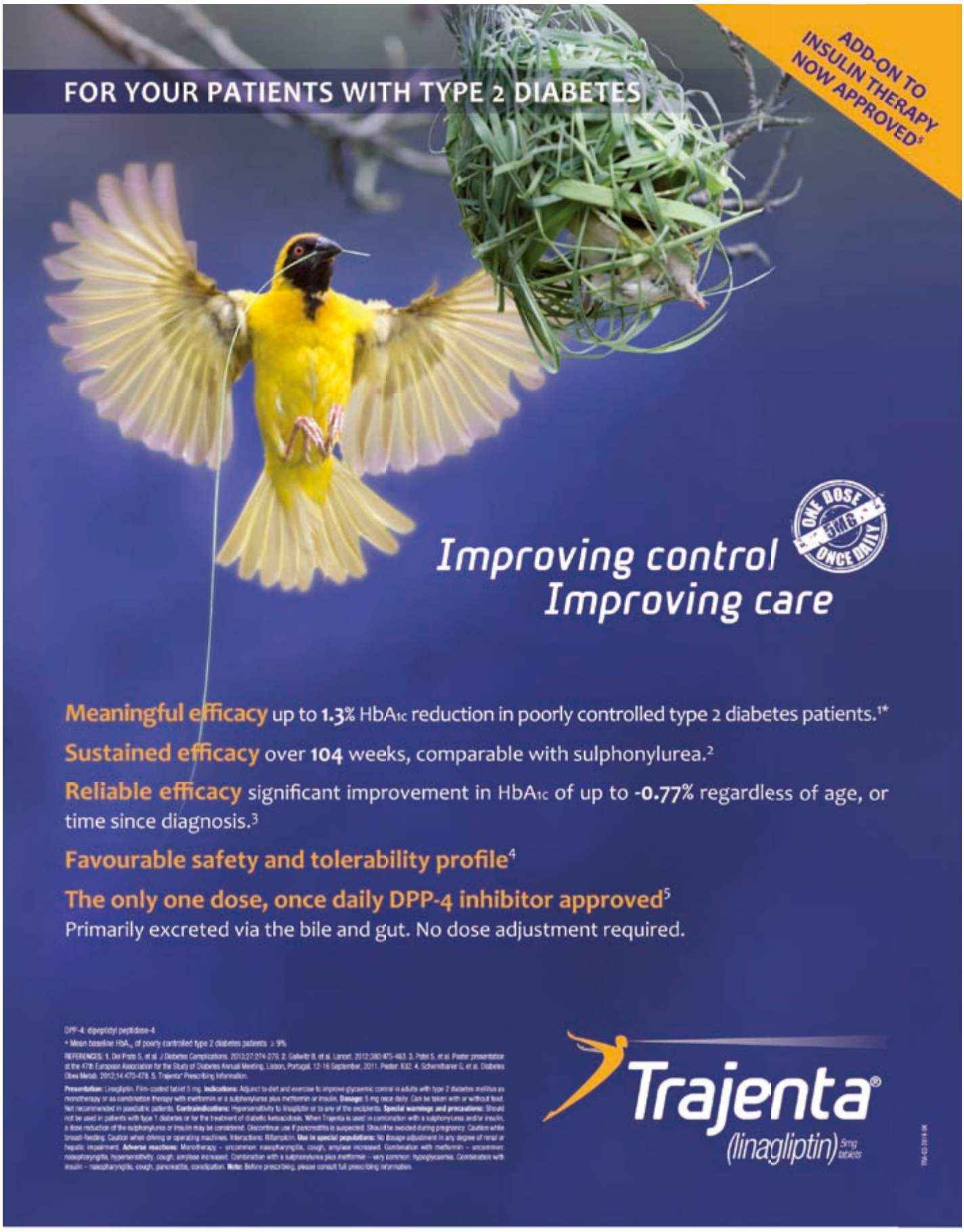

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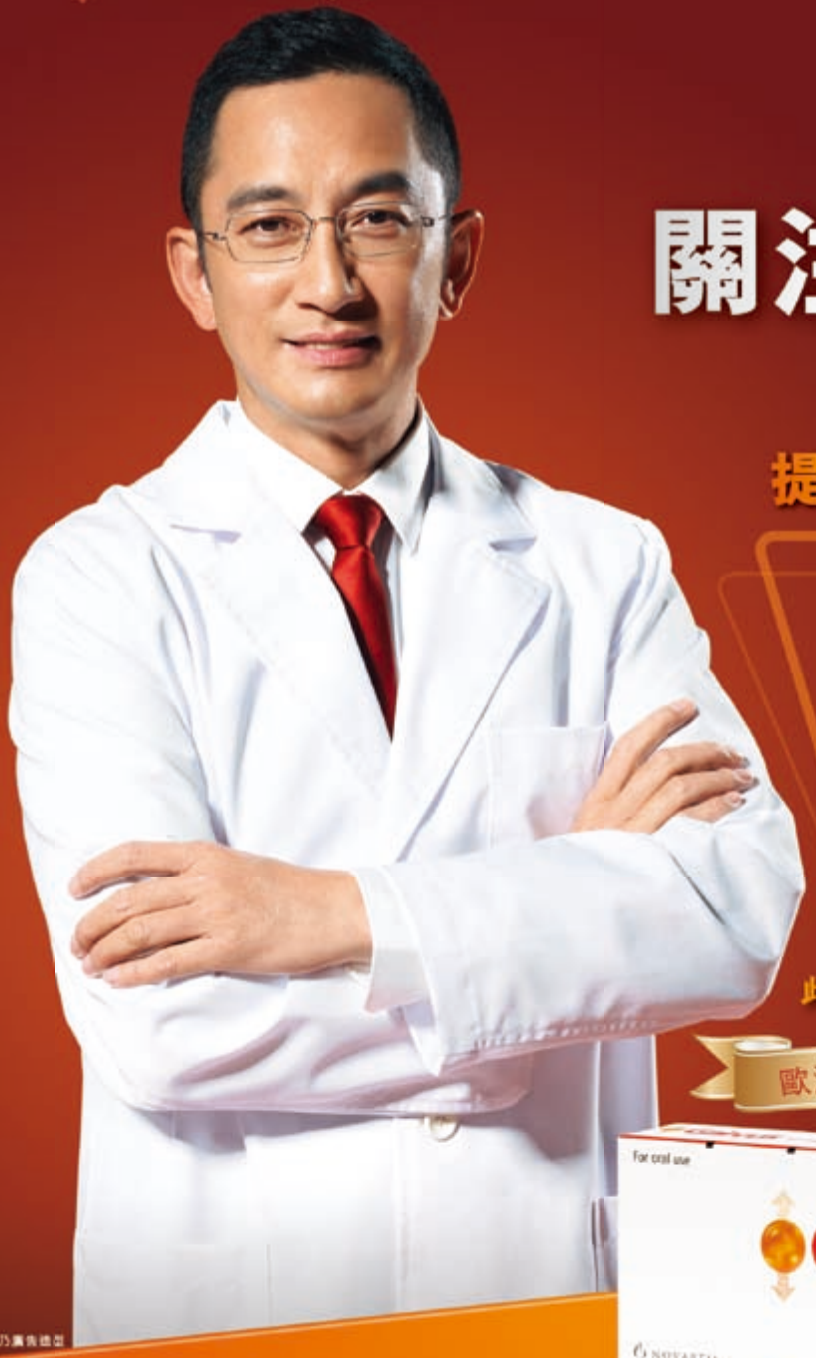

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+ Ahrén B. Dipeptidyl Peptidase-4 Inhibitors: Clinical data and clinical implications. Diabetes Care 2007 Jun; 30(6): 1344-1350

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Abbreviated Prescribing Information

INDICATIONS : 1)Treatment of ischemic symptoms, including ulceration, pain, and coldness of the extremities, in chronic arterial occlusion. 2) Prevention of recurrence of cerebral infarction (excluding cardiogenic cerebral embolism)

CONTRAINDICATIONS

1. Patients with hemorrhage (e.g. hemophilia, increased capillary fragility, intracranial hemorrhage, hemorrhage in the digestive tract, hemorrhage in the urinary tract, hemoptysis, and hemorrhage in the vitreous body) (Bleeding tendency may be increased.) 2. Patients with congestive heart failure (Condition may be worsened.) 3. Patients with a history of hypersensitivity to any ingredient of the drug. 4. Women who are pregnant or may possibly become pregnant

DOSAGE AND ADMINISTRATION

The usual adult dose of Pletaal tablets is 100 mg of cilostazol, twice daily, by the oral route. The dosage may be adjusted according to the age of the patient and the severity of symptoms.

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*For initial and adjunct use in patients with type 2 diabetes,**

JANUVIA® for substantial efficacy in a broad range of patients[1]

In clinical studies:

- **Substantial HbA1c reduction** through a physiologic mechanism of action[2]
- Generally **weight neutral** therapy with a **low risk of hypoglycemia**[2,a]
- **Once-daily** oral treatment[2]

Before prescribing, please consult the prescribing information.

* JANUVIA is indicated for initial use as monotherapy or in combination with metformin, sulfonylurea, PPARγ agonist, insulin (with or without metformin), as an adjunct to diet and exercise.

† As typical with other antihyperglycemic agents (eg, metformin, thiazolidinediones) used in combination with a sulfonylurea, adding JANUVIA increased the incidence of sulfonylurea- or insulin-induced hypoglycemia compared to a placebo. A lower dose of sulfonylurea or insulin may be considered to reduce the risk of sulfonylurea- or insulin-induced hypoglycemia.

JANUVIA® is contraindicated in patients who are hypersensitive to any components of this product and in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis. A dosage adjustment is recommended in patients with moderate or severe renal insufficiency or with end-stage renal disease requiring hemodialysis or peritoneal dialysis. Discontinue if pancreatitis is suspected and not recommended for use in children below 18 years old, during pregnancy or breast-feeding. The adverse experiences reported regardless of causality assessment in >1% of patients and more commonly than placebo or the active comparator included hypoglycemia, diarrhea, dyspepsia, flatulence and headache.

For appropriate patients with type 2 diabetes,

JANUMET® provides powerful HbA1c reductions to help patients who need more than metformin alone[1,4#]

In clinical studies:

- **Powerful HbA1c, PPG, and FPG reductions** to help patients get to goal (HbA1c goal <7%)[4]
- **Comprehensive mechanism of action targets 3 key defects** of type 2 diabetes[5]

Before prescribing, please consult the prescribing information.

* HbA1c goal<7% † For JANUVIA and JANUMET ‡‡ For JANUVIA only

JANUMET is indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus when treatment with both sitagliptin and metformin is appropriate. JANUMET should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis, as it would not be effective in these settings.

JANUMET® is contraindicated in patients with: Renal disease or renal dysfunction, eg, serum creatinine levels ≥1.5 mg/dL (males), 1.4 mg/dL (females); Known hypersensitivity to any other component of JANUMET; acute or chronic metabolic acidosis, including diabetic ketoacidosis with or without coma. In clinical studies as monotherapy and in combination with other agents, the adverse experiences reported regardless of causality assessment in >5% of patients and more commonly than placebo or the active comparator: hypoglycemia, nasopharyngitis, upper respiratory tract infection, headache, and peripheral edema. There have been postmarketing reports of serious hypersensitivity reactions: anaphylaxis, angioedema, and exfoliative skin conditions including Stevens-Johnson syndrome. When JANUMET® is used in combination with a sulfonylurea, a lower dose of the sulfonylurea may be considered to reduce the risk of hypoglycemia. For additional adverse experience information, see the product circular.

Illustration is an artistic rendition. Not necessarily representative of clinical effects.

References: 1. Data on File. MSD Hong Kong. 2. Hong Kong Product Circular (JANUVIA: MSD). 3. IMS Health, NPA™ Monthly TRx's, October 2006 - January 2013. 4. Goldstein BJ, Feinglos MN, Lunceford JK, et al, for the Sitagliptin 036 Study Group. Effect of initial combination of sitagliptin, a dipeptidyl peptidase-4 inhibitor, and metformin on glycemic control in patients with type 2 diabetes. Diabetes Care. 2007;30:1979-1987. 5. Hong Kong Product Circular (JANUMET: MSD).

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