

PROGRAM BOOK

2nd INTERNATIONAL SYMPOSIUM ON HUMAN'S HEALTH AND AGING SCIENCES

*"The Role of Hormone and Hormonal Disorder
Management for Healthy Human Kind in Aging Population"*



January 27 - 29th, 2017
Aston Semarang Hotel & Convention Centre
Central Java - INDONESIA

SECRETARIAT

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Are You Addicted to Sugar? **The Role of Sugar Intake in the Obesity Pandemic**

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In 2016, the European Society of Endocrinology (ESE), the American Endocrine Society (ENDO) and the International Society of Endocrinology (ISE) united in a joined effort to combat the obesity pandemic.

Part of that joined program was the kick-off symposium at the 2016 International Congress of Endocrinology in Beijing. Every year at one of the major international congresses, the three societies will again organize such dedicated symposia as we are convinced that obesity can only be successfully concurred by a global approach.

In line with this joined program, my presentation addresses obesity and focusses on the effects of the increase in sugar intake by people around the globe.

The role of fructose and sucrose in obesity and fatty liver disease

In the Western world, consumption of soft drinks has increased the last three decades and is partly responsible for the epidemic-like increase in obesity. Soft drinks, originally sweetened by sucrose, are now sweetened by other caloric sweeteners, such as fructose. In a study by Lindqvist et al, they investigated the short-term effect of sucrose, glucose or fructose solutions on food intake and body weight in rats, and on peripheral and central appetite signals (1). All rats offered the sugar solutions increased their total caloric intake. The increased caloric intake occurred even though the rats offered either of the sugar solutions consumed less chow. Because of the increased caloric intake, the sugar-drinking rats had elevated serum levels of free fatty acids, triglycerides and cholesterol (1).

Stanhope and coworkers also addressed these effects of high sugar intake (2). High-fructose corn syrup (HFCS) has replaced sucrose as the predominant sweetener in beverages. They compared the metabolic/endocrine effects of HFCS with sucrose and, in a subset of subjects, with pure fructose and glucose by studying 34 men and women who consumed 3 isocaloric meals with either sucrose- or HFCS-sweetened beverages (2). Eight of the male subjects were also studied when fructose- or glucose-sweetened beverages were consumed. Unexpectedly, postprandial triglycerides (TG) profiles after HFCS or sucrose were not intermediate but comparably high as after pure fructose (2). Apparently, short-term consumption of sucrose and HFCS results in postprandial TG responses

Management of Thyroid Dysfunction in the Aging Population

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Elderly patients with thyroid dysfunction tend to have other co-existing medical conditions and co-morbidities. Of concern are the pre-existing cardiovascular diseases (CVD) and the associated CVD risk factors such as hypertension, hyperlipidemia and diabetes mellitus, all of which are aggravated by thyrotoxicosis or hypothyroidism. In addition they are also prone to serious complications such as atrial fibrillation, cardiac failure from thyrotoxicosis and pericardial effusion in the case of hypothyroidism, complications which are rarely seen in the young.

Hyperthyroidism

In view of the above complications and the increase risk of relapse with medical therapy, radioactive iodine(RAI) and surgery are preferred over oral anti-thyroid drugs(ATD) as a definitive therapy for thyrotoxicosis in the elderly. Between the two, RAI is favoured simply due to the ease of its administration and the absence of any serious long term surgical sequelae such as hypoparathyroidism and vocal cord palsy. When contemplating RAI therapy for the elderly especially those with cardiomyopathy it is recommended that they are pre-treated with ATD till the thyroid hormone levels are just above normal values. It is important to administer adequate doses of RAI between 10-15 mCi to completely render the patient hypothyroid since this is the only assurance against relapse. The 2016 ATA guidelines suggested a shorter break of only 2-3 days before administering radioiodine so as to reduce the risk of severe thyroiditis and thyroid storm as a result of rebound in thyroid hormone levels upon stopping the ATD. The ATD may be resumed 3-7 days after the RAI administration in those with complications. It is important to continue beta-blockers throughout the whole period of treatment. Hypothyroidism may occur as early as a month following RAI with 40% becoming hypothyroid within 2 months of RAI. If on the other hand hyperthyroidism persists after 6 months, retreatment with RAI is suggested.

For those who opt for surgery, a very important consideration is the availability of a high-volume thyroid surgeon (who performs at least 25 thyroidectomies a year) as this is associated with 3-4 times less risk of complications. Near-total or total thyroidectomy is the procedure of choice and patients should be rendered euthyroid prior to the procedure with ATD pretreatment. An iodine containing

Type I Diabetes in Older Adults: Current Concepts on Pathogenesis and Management

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ABSTRACT

The evolution of natural history, pathogenesis and management of type 1 diabetes (T1D) has been greatly advanced by a significant number of recent studies over the past decade.

It is well established that pathogenesis of T1D begins years prior to clinical diagnosis. This latent period leaves valuable room from trials aiming at prediction and prevention.

The traditional concepts of pathogenesis, based on autoimmunity, utilization of immunosuppressive agents, with records of failures, have been challenged by a significant series on prediction, prevention and reversal trials, that portray T1D as a T-cell-mediated autoimmune disorder, in the context of genetic susceptibility, with "triggering" insults, likely environmental, that mediate β -cell destruction.

T1D in older adults, a growing but underevaluated population, has different characteristic features as compared to T1D in children and adolescents, with differences in presentation, course of disease, and management, which should be carefully individualized.

Reasonable goals for research trials and treatment modalities should take in consideration the variable comorbidities and functional status of patients, aiming to achieve the lowest HbA_{1c} levels that do not cause problematic hypoglycemia.

Recent advances in T1D prediction markers, prevention, reversal, islet transplantation and artificial pancreas will be addressed in this presentation.

Keywords: Type 1 diabetes, autoimmunity, T-cell-mediated disorders, problematic hypoglycemia, islet transplantation.

Evidence of Incretin Physiology in Diabetes: Difference Between Asia and the West?

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Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder with multiple pathophysiologic abnormalities. Insulin resistance in muscle, liver and adipose tissue and B cell dysfunction represent the core defects (also known as "Harmonious Quartet") in T2DM. In addition to "Harmonious Quartet", incretin defect, increased glucagon secretion, increased glucose reabsorption in the kidney, neurotransmitter dysfunction all play important roles in the development of T2DM. Collectively, these eight players comprise the "Ominous Octet" (Figure 1). Research done over the last several decades have demonstrated that incretin hormones play an important role in glucose homeostasis.

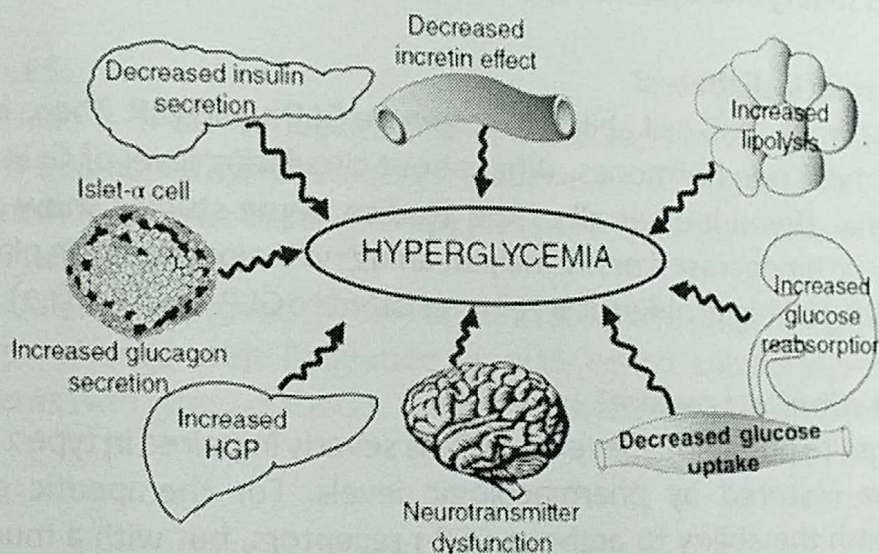


Figure 1: Ominous Octet: Eight distinct pathophysiological defects contribute to the development of T2DM. (Adapted from DeFronzo with permission. Ref. 1).

What is the incretin effect?

The term incretin effect (**IN**testinal **se**CRETion of **IN**sulin) is used to describe the finding that oral glucose induces a more pronounced insulin response in comparison with intravenous glucose administration. Incretin hormones are gut peptides released after nutrient ingestion.

Where do these hormones come from?

GIP is secreted from K cells in the small intestine, located primarily in the