Science for
Better Nutrition

Satellite Symposium

Nutritional Management of Obesity in Diabetes - An Outlook into the Future

Thursday December 5th 2013
17:15 – 19:15 | Room 203 / 204

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Nestlé
Nutrition Institute

Science for
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Program

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Chairperson:

Prof. Mike Lean | Scotland
University of Glasgow

Speakers:

Substantial Weight Loss Reverses the Metabolic Syndrome: Why is it Not Used More as a Therapeutic Strategy?
Prof. Joseph Proietto | Australia

Low and Very Low Calorie Diets as Treatment Options for Managing and Preventing Diabetes
Prof. Mike Lean | Scotland

Ketogenic Diets for Weight Loss - Effect of Ketosis on Appetite Hormones
Dr. Priya Sumithran | Australia

Incorporating Behaviour Modification Strategies in Weight Management Programs
Prof. David Sarwer | USA

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Substantial Weight Loss Reverses the Metabolic Syndrome: Why is it Not Used More as a Therapeutic Strategy?

Prof. Joseph Proietto

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It has been clearly demonstrated that substantial weight loss using adjustable gastric banding reverses the metabolic syndrome in most of those who undergo the procedure (1). A similar degree of weight loss can be achieved using a VLED. Why is this approach not used more often? The reason is that medical weight loss strategies almost always end in failure. This is because body weight is vigorously defended.

Weight is regulated by the hypothalamus. In the Arcuate Nucleus (ARC) there exist two types of neurons, one type express NPY and AgRP while the other type express POMC and CART. NPY neurons cause hunger while the POMC neurons inhibit it. The balance of the activity of these neurons determines if an individual has the desire to eat or not.

A major influence on the relative activity of these ARC neurons is the balance of hunger regulating hormones in the blood. These hormones are produced by the gut (Ghrelin, CCK, PYY, Oxyntomodulin, GLP-1), fat (leptin) and the pancreas (Insulin, Amylin and Pancreatic Polypeptide). Of these, only Ghrelin stimulates hunger, all the others inhibit the desire to eat. Following weight loss, the levels of these hormones change predominantly in a direction to make the individual hungrier (Ghrelin level rises while the levels of the other hormones fall). This results in increased hunger. These changes in hormone levels are long lasting [(2)]. In addition there is a persistent reduction in energy expenditure.

These findings suggest that following weight loss, it is necessary to prescribe appetite suppressing drugs or hormones. At present we have few such agents to choose from. Available agents and those under investigation will be discussed.

References:

1. P E O'Brien et al. Treatment of Mild to Moderate Obesity with Laparoscopic Adjustable Gastric Banding or an Intensive Medical Program. Ann Intern Med. 2006; 144: 625-633
2. Sumithran P, et al. Long-term persistence of hormonal adaptations to weight loss. N Engl J Med, 2011; 365:17 1597-604

Low and Very Low Calorie Diets as Treatment Options for Prevention and Treatment of Type 2 Diabetes

Prof. Mike Lean

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Type 2 diabetes is primarily a disease of nutrient storage - specifically fat storage. It is part of the 'Metabolic Syndrome' spectrum of metabolic decompensation whose clinical features can be traced to the ectopic accumulation of lipid, and consequent functional interference, in organs such as heart, liver and pancreas. While there is undoubtedly an important familial, and presumed genetic/epigenetic, component conferring susceptibility on individuals as they age, far and away the most important element promoting T2DM is an increase in total body fat. Even very modest net positive energy balance generates weight/fat gain and thus T2DM in susceptible individuals. Most people with T2DM also develop other features of Metabolic Syndrome.

Current management guidelines for treatment/intervention in T2DM aims to reduce risk of premature CVD (which affect 50-70% of patients), and to delay or avoid microvascular complications (which will affect 15-25%, depending on age).

In view of health-economic impacts, there is increasing interest in interventions to prevent T2DM in susceptible individuals.

Large studies have demonstrated significant, but small, reductions in microvascular complications from treatments to reduce blood glucose, and valuable improvements in secondary prevention of CHD for patients with T2DM. However, application of current clinical guidelines results in polypharmacy (often 6-8 drugs), and the net effect is estimated to be only a 10-15% reduction in CHD. People with T2DM still die 6-8 years younger than non-diabetic subjects.

At BMI 20-21 kg/m^2, irrespective of genes, T2DM is virtually non-existent, but it increases sharply as BMI rises about 23 kg/m^2. The mean BMI of newly diagnosed patients in UK is now 30kg/m^2. Reducing weight by 4-8kg by the DPP/DPS diet-lifestyle intervention cut new T2DM by 58%: weight loss is the dominant element, as demonstrated by the added effect of orlistat in the XENDOS trial, without the need for LELD/VLED. There is little evidence, but no reason to withold liquid-formula diets for prevention. Blood glucose and BP fall rapidly on LELD/VLED, with negative energy balance, ahead of weight change.

For treatment of T2DM, VLEDs have shown normalisation of blood glucose and insulin with weight loss >15kg. Weight loss >15kg is also the amount effective in reversing T2DM to normal glucose tolerance following bariatric surgery, where the effective intervention is also negative energy balance. The latest SIGN guideline from Scotland has declared weight loss >15kg as a new target for management of severe and complicated obesity, including the obese T2DM patient. This is seldom achievable using conventional diet-lifestyle measures, but was maintained at 12 months by 33% of all patients who entered a programme with 810kcal/day LELD and the Counterweight structured weight maintenance programme.

This programme was greatly more cost-effective than bariatric surgery for generating weight losses >15kg.

References:

Lean MEJ et al Feasibility and indicative results from a 12-month low-energy liquid diet treatment and maintenance programme for severe obesity. Br J Gen Pract 2013; DOI: 10.3399/bjgp13X663073

Ketogenic Diets for Weight Loss – Effect of Ketosis on Appetite Hormones

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During fasting or restriction of dietary carbohydrate intake, fatty acid oxidation in the liver results in the production of ketones. Low-carbohydrate ketogenic diets are a popular means of weight loss, and in the short-term, often result in greater weight loss than low-fat diets [1]. The mechanism of efficacy of ketogenic diets is not clear, but it is commonly thought that ketones suppress appetite.
The homeostatic regulation of body weight involves signals from numerous circulating hormones originating from the gut, pancreas and adipose tissue, which are integrated in the hypothalamus to influence appetite and energy expenditure. These peripheral factors include ghrelin, which stimulates appetite, and the satiation signals cholecystokinin, peptide YY, glucagon-like peptide-1 and leptin. Diet-induced weight loss is accompanied by a number of compensatory changes which encourage weight regain, such as an increase in ghrelin, and reductions in energy expenditure and several hormones involved in satiation. Recent studies have shown that the circulating concentrations of several hormones and nutrients which influence appetite are altered after weight loss induced by a low carbohydrate ketogenic diet, compared with after reintroduction of regular foods [2,3], suggesting a possible mechanism by which ketogenic diets may reduce appetite.

References:

1. Nordmann AJ et al. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. Arch Intern Med 2006;166:285-293.
2. Chearskul S et al. Effect of weight loss and ketosis on postprandial cholecystokinin and free fatty acid concentrations. Am J Clin Nutr 2008;87:1238-1246.
3. Sumithran P et al. Ketosis and appetite-mediating nutrients and hormones after weight loss. Eur J Clin Nutr 2013;67:759-764.

Incorporating Behaviour Modification Strategies in Weight Management Programs

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Lifestyle modification, which targets changes in diet and physical activity, is the cornerstone of weight loss treatment. In efficacy trials conducted at academic medical centers and with interventions often delivered by registered dietitians, persons treated with a 1200-1500 kcal/d diet, recommendations to increase physical activity, and behavior modification strategies (taught either to patients individually or in small groups during weekly sessions), lose 7%-10% of their initial weight in 20 to 26 weeks. This magnitude of weight loss has been shown to be associated with improvements in weight related co morbidities (including features of the metabolic syndrome). These weight losses also are associated with significant changes in psychosocial status, including improvements in quality of life, self-esteem, and body image as well as reductions in depressive symptoms. Recently, a number of investigators have demonstrated that lifestyle modification for weight loss can be delivered effectively outside of highly specialized obesity clinics and by individuals who have been trained in the delivery of these interventions. At the same time, a number of preliminary studies have suggested that these interventions can be delivered electronically--via telephone calls, websites or smart phone applications. The use of this technology can reduce the significant burden of regular, repeated visits to the clinic and, at the same time, promote increased patient-provider contact, which is critical for weight loss and long-term weight maintenance. The presentation will review the research studies in these areas and provide specific recommendations for the use of lifestyle modification strategies in clinical practice for patients with obesity and type 2 diabetes.

Notes: