The International Expert Movement (IEM) for the Health Significance of Fat Quality of the Diet is pleased to announce the scientific symposium.

“ROLE OF DIETARY FATS IN PREVENTION AND TREATMENT OF THE METABOLIC SYNDROME”
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PROGRAMME:

Chair: Mrs Connie Diekman, M.Ed., RD, CSSD, LD, FADA, Director of University Nutrition at Washington University in St. Louis, Missouri, USA

Metabolic syndrome is a term applied to a group of risk factors that increase risk of heart disease, diabetes, stroke and other diseases. Five risk factors makeup the syndrome but having even just one risk factor increases the risk of heart disease. Those who have metabolic syndrome are at five times the risk of developing diabetes as is someone who does not have the syndrome.

The first line of treatment for metabolic syndrome is a focus on weight loss. While the amount of fat consumed impacts total calories and therefore weight, the type of fat consumed influences disease risk.

During this symposium we will hear the latest research on fat, fat quality, the impact of fat choices during early life and how the total diet composition impacts metabolic syndrome.

The focus of this symposium supports the mission of the International Experts Movement on the Health Significance of Fat Quality in the Diet or the IEM, which is an initiative from the IUNS. IEM started in February of 2009 with a goal of disseminating sound scientific information about food and nutrition, especially fat quality in the diet to health professionals and the general public. The first meeting was attended by 40 leading experts in the field of fats and health. The IEM has held four global symposiums before this along with a variety of country and region events. The Activities of the IEM are held under the auspices of the International Union of Nutritional Sciences (IUNS) and funded by an unrestricted educational grant from Unilever NV. Find out more on www.theiem.org.

DIETARY FAT AND OBESITY

Professor Susan Jebb, Professor of Department of Primary Care Health Sciences, University of Oxford, UK (susan.jebb@phc.ox.ac.uk)

For many years now the role of dietary fat as a specific risk factor for obesity has been contentious. This presentation will review evidence relating to the macronutrient content of the diet from experimental research, observational studies, controlled interventions and more pragmatic weight loss trials.

Experimental studies in which the macronutrient content of the diet is covertly manipulated strongly suggest that fat is only weakly sating, leading to passive overconsumption of energy from meals high in fat. Moreover a meta-analysis of data from cohort studies and randomised trials suggests that lower fat diets are associated with small but significantly lower body weight. This implies that reducing the fat content of the diet may be an effective strategy to prevent weight gain. However, data from pragmatic weight loss trials often shows that the macronutrient content of the diet is a poor predictor of success. This implies that the effects of the fat content of the diet are modulated by broader dietary patterns or that behavioural factors may be more important determinants than diet composition.

Academic debates, often using different types of evidence, have contributed to consumer confusion regarding the role of dietary fat and weight control and it is vital that we resolve the apparent conflicts and reach a clear message for consumers. Ultimately, any dietary strategy to prevent or treat obesity must address total energy intake rather than fat content per se and must also acknowledge the importance of diet quality, including the fatty acid composition of the diet to maximise the wider health benefits.

FAT QUALITY IN THE CONTROL OF OBESITY AND INSULIN RESISTANCE

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The role of dietary fat quality in the development of obesity and insulin resistance is an area where the knowledge is still rather limited, although several controlled studies have studied the effects of dietary fats and insulin sensitivity. Fat oxidation rate is partly depending on fatty-acid chain length and saturation, providing one potential mechanism by which the source of dietary fat may influence body fat storage.

In the present overview, data in humans will mainly be presented although some animal findings will be discussed. For example, animal models suggest differences in body fat accumulation after feeding different types of fats, i.e. saturated fats more fattening than polyunsaturated fatty acids (PUFA). In vitro studies suggest that fatty acids also can have hormone-like effects and regulate gene expression in preadipocytes and adipocytes, and thereby affecting adipocyte proliferation and differentiation. Long-chain fatty acids may also regulate transcription factors (e.g. PPARs) and other adipose-specific genes involved in adipocyte development. These effects have the potential to affect fat cell number and maturity. Whether this translates to in vivo situations in humans is unknown, but there are some recent indications from observational studies and controlled dietary interventions suggesting distinct effects of fatty acids on abdominal fat accumulation, where PUFA may be less adipogenic as compared with saturated fats. The latter data are in line with some results suggesting slightly improved insulin sensitivity after PUFA (mainly n-6) when compared with saturated fat. The data on monounsaturated fat (MUFA) versus saturated fat are inconsistent with regard to influence on insulin sensitivity, but some data indicate small beneficial effects of replacing saturated fat with MUFA.

In summary, the data are limited but there are suggestive data indicating that dietary fat quality might play some role in the development of adipose tissue accumulation and distribution. Also, the role of dietary fat quality in modulating ectopic fat is of clinical interest since e.g. liver fat is closely linked to type 2 diabetes. It will be important to gain more knowledge in this field since it might help us in the future to compose optimal diets that minimize abdominal and ectopic fat accumulation and thus may help prevent obesity and its related metabolic disorders (including insulin resistance) in the long-term.

EARLY LIPID NUTRITION AND LATER METABOLIC SYNDROME RISK

Professor Berthold Koletzko, Dr. von Hauner Children’s Hospital, University of Munich Medical Center, München, Germany, for the EarlyNutrition Research Project

Nutrition during pregnancy, lactation and early childhood markedly modulates the long-term risk of obesity and associated metabolic disorders, including insulin resis-tance and diabetes. Pre- and postnatal overfeeding with either carbohydrate or fat increases a child’s risk for obesity and associated disorders. It was hypothesized that perinatal dietary fat quality also matters, i.e. a high intake of n-6 polyunsaturated fatty acids (PUFA) and a high n-6/n-3 PUFA ratio might contribute to the intergenerational cycle of obesity. Plausible biological mechanisms may exist, e.g. the arachido-nic acid (n-6) metabolite prostacyclin may promote differentiation of preadipocytes to adipocytes, with antagonistic effects of n-3 PUFA; n-3 PUFA may decrease adipose tissue cellularity and reduce lipid synthesis, reducing both adipocyte...
hyperplasia and hypertrophy; and different PUFA may modulate PPARg and SREBP1c thereby affecting adipocyte differentiation. Animal studies show inconsistent effects of PUFA supply on body fat mass in the offspring but differ in nature and dose of interventions, and in many other aspects. Likewise, human observational and intervention studies do not show consistent effects. The human intervention studies are highly heterogeneous, i.e. regarding intervention composition, dosage, timing and duration, as well as methodology, sample size, compliance, and outcome assessments. Overall, the available animal and human data do not provide conclusive evidence for univocal effects of a high pre- and postnatal n-6 PUFA intake, or a high n-6/n-3 PUFA ratio, on offspring obesity. The available data do not justify changing current recommendations on dietary fat quality in pregnancy, lactation and infancy.

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HIGH OR LOW-FAT DIET (AND MACRONUTRIENT DISTRIBUTION) TO COUNTERACT THE METABOLIC SYNDROME?

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The metabolic syndrome is a clustering of risk factors that increase the risk of developing cardiovascular disease (CVD). While there is agreement that lifestyle changes to promote weight reduction are the primary focus for patients with metabolic syndrome, the optimal macronutrient content of the diet remains uncertain. Current evidence recognizes that, in the short term, any calorie reduced dietary pattern will achieve modest weight loss. However, a substantial body of scientific evidence has shown that by simply varying the macronutrient distribution and composition of dietary factors, longer term weight maintenance and beneficial changes in metabolic syndrome risk factors may be achieved. Current dietary guidance no longer advises against fat, but rather that saturated fatty acids (SFA) and trans fatty acids be replaced with unsaturated fatty acids, both monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids. This recommendation is a paradigm shift from a low fat/reduced fat message, which has been a long-standing dietary recommendation for the reduction of chronic diseases. Similarly, there is substantial evidence to suggest cardiometabolic benefit by replacing refined and/or high glycemic carbohydrates, typical of a Western diet, with unsaturated fats and lean proteins in the diet. The new focus on a moderate-fat/moderate carbohydrate diet that emphasizes unsaturated fatty acids and low glycemic carbohydrates is based on an impressive evidence base has demonstrated the remarkable health effects of MUFA’s, PUFA’s and dietary fiber.

Panel discussion and concluding remarks