WHAT'S NEW?

A conference for health and education professionals

Thursday 1st March 2012

W5 the Odyssey Belfast

The Dairy Council for Northern Ireland

This conference is dedicated
to the memory of

Julie Wallace
BSc PhD RNutr

7th April 1971 - 7th February 2012

CONFERENCE PROGRAMME

09.30 Registration and coffee

10.00 Professor Sean Strain,
University of Ulster, Coleraine
Chairperson's introduction

10.15 Professor Andy Salter,
University of Nottingham
Dietary fatty acids and cardiovascular disease
Where are we now?

10.50 Dr Michelle McKinley,
Queen's University Belfast
Review of nutrition and reproductive disorders
PMS and PCOS

11.25 COFFEE

11. 45 Sarah Bath,
University of Surrey
Emerging evidence on iodine deficiency in the UK
Public health implications?

12.20 Professor Marie Murphy,
University of Ulster, Jordanstown
'Start Active, Stay Active'
New recommendations for physical activity in the UK

12.55 LUNCH

2.00 Dr Tom Hill,
Newcastle University
An update on nutrition, exercise and bone health outcomes

2.35 Professor Barbara Livingstone,
University of Ulster, Coleraine
Energy intakes and childhood obesity
Is the evidence all that it seems?

3.10 General discussion

3.30 Close

Dietary fatty acids and cardiovascular disease: where are we now?

Professor Andy Salter

Division of Nutritional Sciences, School of Biosciences,
University of Nottingham

A reduction in consumption of total, saturated and trans fatty acid, and their replacement with cis- polyunsaturated fatty acids (PUFA), has been the cornerstone of public health policy for over forty years. These recommendations were made largely on the basis of specific effects of these fatty acids on risk of developing atherosclerotic cardiovascular disease (CVD).

The intervening years have seen a plethora of human epidemiological and intervention trials to further elucidate the specific relationship between dietary fatty acid intake, plasma lipids and lipoproteins and cardiovascular morbidity and mortality. A number of recent meta-analyses and systematic reviews have revisited the role of specific dietary fatty acid classes in CVD risk. In general these continue to support the replacement of saturated fatty acids with cis-PUFA in order to reduce CVD morbidity/mortality. They also highlight the potent adverse effects of trans fatty acids derived from partially hydrogenated vegetable oils.

In recent years there has been much emphasis on the ratio between n-6 and n-3 PUFA, with the suggestion that consumption of n-6 PUFA-rich vegetable oils has adversely increased this ratio in modern diets. However, evidence of specific beneficial effects of n-3 PUFA is largely restricted to very long chain fatty acids of marine origin (EPA and DHA), as opposed to plant –derived alpha -linolenic acid. There is also growing recognition that the ability of individuals to synthesize long chain n-3 PUFA can vary considerably, with factors such as age, sex, background diet and genetic polymorphisms in desaturase/elongase enzymes all contributing to such differences. As such, an individual's 'omega-3 status' (tissue levels of EPA and DHA) may be a more meaningful risk factor that dietary intakes.

It is however, important to recognise that fatty acids are consumed as complex mixtures depending on the specific food types selected by the individual. Animal products undoubtedly contribute a significant proportion of total SFA intake in an omnivorous diet. However, a recent meta-analysis concluded that, while intake of processed meat is associated with an increased incidence of coronary heart disease, this is not the case for 'red' meat.

The impact of milk and/or dairy products on CVD risk has also been the topic of recent review and there is no evidence to suggest that high milk intake is associated with increased risk. In fact, milk consumption may actually be protective. Taken together the evidence described above suggests that the consumption of animal products, per se, is not necessarily associated with increased CVD risk and that we should take a more holistic view of dietary fat intakes.

Review of nutrition and reproductive disorders: PMS and PCOS

Dr Michelle Mckinley

Centre for Public Health,
Queen's University Belfast

Two major conditions affecting women of reproductive age are Premenstrual Syndrome (PMS) and the Polycystic Ovary Syndrome (PCOS).

PMS is defined as a consistent pattern of emotional and physical symptoms occurring only during the luteal phase of the menstrual cycle that are of sufficient severity to interfere with some aspects of life. Whilst up to 85% of women experience premenstrual symptoms, the estimated prevalence of PMS, as defined above, is 2-10%. This talk will review evidence regarding the role of nutrients in the management of PMS, focusing on calcium, magnesium, vitamin B6 and essential fatty acids.

PCOS is the most common endocrine disorder among women, affecting approximately one in ten women in the UK. Key features of PCOS include the presence of polycystic ovaries, menstrual cycle disturbance, hyperandrogenism (excessive production and/or secretion of androgen hormones) and obesity. Metabolic syndrome (a cluster of metabolic risk factors, commonly insulin resistance, high blood pressure and cholesterol abnormalities, associated with increased risk of heart disease and type 2 diabetes) is consistently found in PCOS patients who are obese and is also detected in many normal-weight PCOS patients. The presence of insulin resistance, hyperinsulinaemia and obesity has a negative impact on menstrual cycles and fertility rates. The evidence regarding the role of diet and lifestyle in the restoration of metabolic physiology and reproductive potential will be reviewed.

Emerging evidence on iodine deficiency in the UK: public health implications

Sarah Bath
Department of Nutrition and Metabolism,
University of Surrey

Iodine deficiency is of concern because iodine is required for the production of thyroid hormones which are needed for correct brain development, particularly during pregnancy and early life; indeed iodine deficiency is considered by the WHO to be the greatest preventable cause of brain damage worldwide. Historically, iodine deficiency was widespread in the UK and this deficiency persisted until the 1960s. In contrast to most countries, iodine deficiency in the UK was not eradicated through an iodised salt programme but by the accidental increase in the iodine concentration of milk since the 1930s. Milk and dairy products are now the main source of iodine in UK diets, providing up to 40% of UK iodine intakes.

Although the UK has been considered iodine-sufficient for many years, there is now an increased focus on the risk of iodine deficiency. This is due to a nationwide study in 2011 that revealed mild iodine deficiency in UK schoolgirls and also from our own data that showed pregnant women in the South of the UK to be iodine deficient.

There are clear public-health implications of iodine deficiency in the UK and I will present a review of the subject area, summarising the re-emergence of iodine deficiency in the UK, the main sources of iodine in the diet and the role of iodine in brain development. I will present the results of studies by our group showing that the iodine concentration of organic milk is over 40% lower than conventional milk and that iodised salt is available to fewer than 20% of supermarket shoppers. Most importantly, I will present data concerning the effect of iodine status in UK pregnant women on cognitive performance in the child. We have found that children of iodine deficient mothers are 50% more likely to have an IQ score in the bottom quartile and 88% more likely to have a reading score in the bottom 25%, than the children born to iodine-sufficient mothers.

Start Active, Stay Active: new recommendations for physical activity in the UK

Professor Marie Murphy
Sport & Exercise Science Research Institute,
University of Ulster

In July 2011 new physical activity guidelines for the UK were announced in the government publication “Start Active Stay Active”. This is the first time that all 4 home countries have come together to issue a common set of guidelines, endorsed by the four Chief Medical Officers. This presentation will outline the process through which the guidelines were developed and review some of the more recent evidence upon which they were based.

The current guidelines take a life course approach; specifying the amount and type of physical activity that UK children and adults should undertake and, for the first time, providing guidance on the early years (<5), older adults and people with disabilities. The new guidelines provide a stronger recognition of the role of vigorous intensity activity and offer the flexibility to combine moderate and vigorous intensity in meeting weekly targets. In addition, the 2011 guidelines draw upon recent reviews of sedentary behaviour to make recommendations on limiting the amount of sitting that individuals do as part of their daily life.

Recent data from the Northern Ireland Sport & Physical Activity Survey will be used to describe adult physical activity participation rates and the effect of the new guidelines on physical activity surveillance will be considered.

An update on nutrition, exercise and bone health outcomes

Dr Tom Hill

Human Nutrition Research Centre,
Newcastle University

Osteoporosis is a condition of fragility which is characterized by a low bone mass and microarchitectural deterioration of bone with a consequent increase in bone fragility and susceptibility to fracture. In the UK it is estimated that 3 million people are affected with osteoporosis. Furthermore, 1 in 2 British women and 1 in 5 British men >50 years of age will experience an osteoporotic fracture in their lifetime with the estimated costs in the UK being about £1.7 billion annually.

Bone is a dynamic tissue that responds to the external and internal environments to which it is exposed during an individual's lifetime. While a considerable proportion (up to 80%) of the inter-individual variation in bone mass is genetically determined, lifestyle factors such as diet and exercise are also important with the latter estimated to account for up to 17% of the variance in bone mineral density. Osteogenic effects of exercise on bone include mechanical and hormonal influences and it is well recognized that the 'type' of exercise undertaken influences the bone response, with impact type activities such as walking, jogging and jumping being most favourable. However, questions remain regarding the optimal exercise regime (including type and duration) for improving bone strength.

Several dietary components have been linked to bone health throughout the life course including among others calcium, vitamin D, protein, acid-base homeostasis and bioactive compounds such as conjugated linoleic acid. Vitamin D has come to the fore again recently in light of the release of new vitamin D DRVs by the Institute of Medicine in the US. From the UK perspective, a number of key research questions are currently being addressed which will inform the Scientific Advisory Committee on Nutrition report due in early 2014. These questions will be alluded to in this talk.

Evidence is accumulating that peak bone mass is a major determinant of later osteoporosis risk with one study suggesting it accounts for half the variance in bone mineral density at aged 70. Evidence from intervention studies suggests that weight-bearing physical activities, such as jumping, may contribute to bone mineral mass gain in children. Recent research also demonstrates that combining nutrients such as calcium or protein with physical exercise results in greater bone mineral mass accrual during adolescence than either low nutrient intake and exercise or high nutrient intake alone. Thus optimizing peak bone mass acquisition through dietary and physical exercise measures may represent a valuable primary method for the prevention of osteoporosis in later life.

Notwithstanding the important developments in examining the effect of nutrients alone one bone health, more research needs to focus on using foods containing a mixture of bone active compounds (e.g. dairy foods) in combination with different exercise regimes bone health.

This talk will briefly overview the role of key nutrients in bone health across the lifecycle and will discuss the potential synergistic effect of a combination of nutritional factors and exercise types on bone health outcomes.

Energy intakes and childhood obesity: is the evidence all that it seems?

Professor Barbara Livingstone

Northern Ireland Centre for Food and Health,
University of Ulster

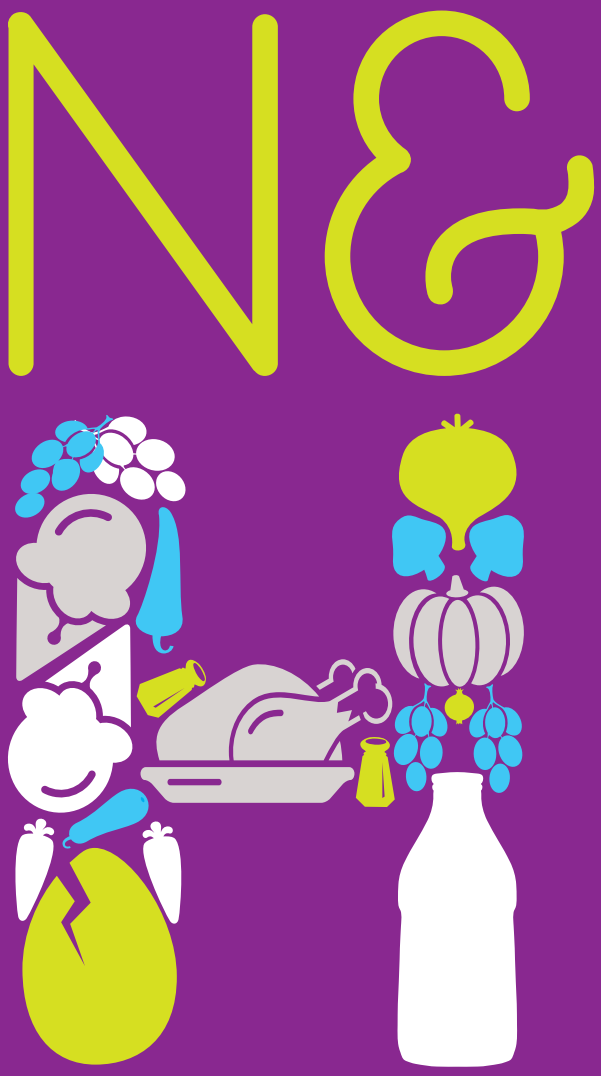
Tackling the burgeoning rates of childhood obesity is now a major public health priority which will require a concerted societal response if the problem is to be attenuated and eventually reversed. Although only the energy intake side of the energy balance equation will be examined in this presentation, it needs to be stressed that the overeating which leads to obesity is a relative phenomenon. The current generation of children may be the most inactive of all time and this factor is at least as important as overeating in accounting for the escalating rates of childhood obesity. In the face of reduced energy needs, children are particularly vulnerable to passive overeating of food in the midst of a food environment which is less than benign. The factors which now conspire to undermine normal appetite regulation include the ready availability and variety of palatable, relatively low cost, energy dense foods served in ever increasing portion sizes.

For better or worse, all of the risk factors for the development of childhood obesity have their initial beginnings in the family of origin. There is now extensive evidence that childhood food preferences and eating patterns may be permanently modelled by parental eating patterns and food beliefs. Insights gained from such work could be used to inform the development of interventions designed to improve child-feeding practices but unfortunately, the current food environment often forces parents into a no-win situation with respect to child feeding practices.

Finding effective strategies to reduce childhood obesity hinges on appropriate and rigorous evaluation of the strength of the evidence with regard to postulated causes of obesity. Although justifiable alarm has been raised about the escalating rates of childhood obesity, this may also have ignited feelings of righteous zeal and fuelled an unhelpful tendency to target culpability at specific food(s). The selective and inaccurate citation of research results is not helpful when there is a need for evidence-based, rather than belief-based public health policy on childhood obesity

There is now consensus that the need to improve children's dietary (and physical) activity) patterns must be tackled more aggressively than hitherto, because failure to do so will have catastrophic economic and health implications in the future. However, clear evidence on successful public health approaches to the prevention of obesity is difficult to identify. Policies targeting single foods (e.g.) sugar sweetened beverages are unproven and unlikely to succeed in isolation. Policy makers should not be rushed into simplistic approaches ahead of credible evidence that they work, that they avoid unwanted side-effects, and that they are cost effective.

notes



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