

Nutrition Lecture

**Anniversary Symposium** 

## **Nutrition and Ageing**

Wednesday 19 November 2014

The Riverside Theatre, Ulster University









### **Nutrition and Ageing**

4.00	Professor Richard Barnett, Vice-Chancellor,
	Ulster University
	Welcome

- 4.10 Professor Sean Strain, Director of NICHE and Professor of Human Nutrition,Ulster UniversityChairperson's Introduction
- 4.15 Professor Kevin Cashman, Professor of Food and Nutritional Sciences,
   University College Cork
   Bone Health
- 5.00 Professor Joshua Miller, Professor of Nutritional Sciences,
   Rutgers, State University of New Jersey
   Cognitive Health
- 5.45 **Buffet Supper**
- 6.30 Professor Helene McNulty, Professor of Human Nutrition and Dietetics, Ulster University

  Cardiovascular Health
- 7.15 Professor Carol Curran, Dean of Life and Health Sciences, Ulster University Concluding Remarks



# Nutrition and ageing from a perspective of bone health

### **Professor Kevin Cashman**

Professor of Food and Nutritional Sciences, University College Cork

The skeleton is a dynamically metabolic tissue throughout life, but like many other tissues, can be impacted adversely by ageing. While the first three decades of life allow for bone accrual and reaching peak bone mass, the following decades represent a time when bone is relatively stable and then bone loss occurs at later adulthood. This bone loss is a naturally-occurring process but the rate of which can be increased by a number of factors. Significant loss of bone mass (bone mineral density) is the characteristic of osteopenia and the more debilitating disease, osteoporosis. Fragility fractures are the hallmark of osteoporosis and depending on the site of occurrence (with the hip being the most serious) can contribute to significant morbidity and even mortality in older adults. These fractures place an enormous medical and personal burden on aging individuals and a major economic toll on the World's nations.

In the not too distant past, osteoporosis was an under-recognized disease and considered to be an inevitable consequence of ageing. Today osteoporosis is an established and well-defined disease and is known to affect more than 75 million people in the United States, Europe and Japan. Prevention of osteoporosis is high on the agenda of health authorities of most Westernized countries and is considered a priority in terms of maintenance of health, quality of life, and independence in the elderly population. Although nutrition is only one of many factors that influence bone mass and fragility fractures, there is an urgent need to develop and implement nutritional approaches and policies for the prevention and treatment of osteoporosis that could, with time, offer a foundation for population-based preventive strategies. However, to develop efficient and precocious strategies in the prevention of osteoporosis, it is important to determine which modifiable factors, especially nutritional factors, are able to improve bone health throughout life. There are potentially numerous nutrients and dietary components that can influence bone health, and these range from the macronutrients to micronutrients as well as specialized bioactive food ingredients. The evidence-base to support the role of these nutrients and food components in bone health ranges from very firm to scant, depending on the nutrient/component. This presentation will set the scene in terms of the public health importance of prevention of osteoporosis and will

overview the potential impact of a number of select nutrients on its prevention. Recent key reports and reviews have placed particular focus on calcium and vitamin D and these will be highlighted but also some consideration will be given to protein, vitamin A and some B vitamins.

Where possible the presentation will place particular emphasis on the evidence base for the benefits of selected nutrients to bone health.



# Homocysteine and cognitive function in older adults: Do B vitamin supplements prevent cognitive decline?

### **Professor Joshua Miller**

Professor of Nutritional Sciences, Rutgers, State University of New Jersey

By the year 2050, the projected number of people worldwide who will be 85 years or older is about 220 million. It is estimated that almost 50% of individuals ≥85 years of age have dementia primarily due to Alzheimer's disease and cerebrovascular disease. Therefore, in just 35 years, >100 million people in this age group will be suffering from dementia and will require significant medical, monetary, and human resources for their care. This is a sobering prospect of epidemic proportions.

As there is currently no cure for Alzheimer's disease and dementia, strategies for prevention or slowing of progression are highly desired. Diet and nutrition represent promising avenues in this regard. Of particular interest is the sulfur amino acid, homocysteine. Elevated concentration of homocysteine in the blood (hyperhomocysteinemia) is a confirmed independent risk marker for cerebrovascular disease, dementia, and Alzheimer's disease. Because of their involvement as substrates and cofactors in homocysteine metabolism, supplements of the B vitamins, folate, B12, B6, and riboflavin, are effective in lowering blood homocysteine concentrations. Thus, B vitamin supplements have the potential to reduce the risk of dementia associated with hyperhomocysteinemia. Indeed, recent findings indicate that B vitamin supplements slow the rate of brain atrophy and prevent cognitive decline in individuals with mild cognitive impairment and hyperhomocysteinemia (Smith et al, PLoS One, 2010; de Jager et al, Int J Geriatr Psychiatry, 2012; Douaud et al, PNAS, 2013). However, these finding are tempered by a recent meta-analysis that concluded B vitamin supplements have no significant effect on cognitive function in older adults (Clarke et al, Am J Clin Nutr, 2014). This meta-analysis has been highlighted in a press release from Oxford University that concluded, "Taking B vitamins will not prevent Alzheimer's Disease." Thus, it would seem that we have the final proclamation on the issue and the book is closed. But is it?

In this presentation, the evidence linking homocysteine to cognitive decline, Alzheimer's disease, and dementia will be reviewed and the conclusion that B vitamin supplements will not prevent Alzheimer's disease will be critically examined. The case will be made that this conclusion is premature and that there are indeed older adults who could benefit cognitively from homocysteine lowering with B vitamin supplements.



# Nutrition and ageing - cardiovascular health

### **Professor Helene McNulty**

Professor of Human Nutrition and Dietetics, Ulster University

The most common cause of death in people over 65 years is cardiovascular disease (CVD), with heart disease and stroke being the two major forms. This lecture will consider the potential for preventing CVD through nutritional means, with a particular focus on the latest evidence for a protective effect of folate (folic acid) and the related B vitamins. This is an area of active research at Ulster and elsewhere in the world. Much of the research being described tonight was made possible through the Joint Irish Nutrigenomics Organisation (JINGO)\* initiative.

The latest scientific evidence to support protective roles for folate and the related B-vitamins in cardiovascular health will be presented. The international evidence at this time is stronger for stroke than for heart disease, with meta-analyses of randomised trials showing that folic acid reduces the risk of stroke, particularly in people with no history of a previous stroke (i.e. primary prevention). Genetic studies also provide important clues to explain why low B-vitamin status might lead to the development of CVD and how improving B-vitamin status could lead to better cardiovascular health as people age.

Recent work led by the team at Ulster shows that hypertension (high blood pressure), a major risk factor for CVD, may be key to explaining the link between B-vitamins and cardiovascular health. Hypertension (defined as a systolic/diastolic blood pressure of 140/90 mmHg or greater) is estimated to carry a 3-fold increased risk of developing CVD, and stroke in particular. Among the many health and lifestyle factors involved, there is much recent interest in the role of genetic factors that could predispose to the development hypertension. Analysis of two JINGO cohorts: the TUDA Ageing study and the NANS cohort, between them providing comprehensive data of relevance to blood pressure in over 6000 Irish adults from aged 18 to 90 years, has offered us a unique opportunity to examine the role of a novel genetic risk factor in hypertension. We show that 12% Irish adults are genetically at increased risk of developing high blood pressure because of a variant in the MTHFR gene required for folate re-cycling in the body. Importantly, an optimal status of the folate-related B vitamin, riboflavin (vitamin B2), can protect against the development of high blood pressure in these adults who are at-risk of developing high blood pressure and will not be aware of it. Therefore increasing riboflavin intake

Population-based and individual strategies for improving nutritional status can play a major role in preventing hypertension and other major risk factors for stroke and heart disease. Ideally these changes should begin in earlier adulthood in order to prevent the first stroke or heart attack.

(through food sources or supplements) in this genetically at-risk group may offer a

personalised non-drug approach to preventing (or treating) hypertension.

<sup>\*</sup>JINGO is the umbrella body that incorporates four universities (including Ulster) working together to create a National Nutrition Phenotype Database. JINGO combines dietary, physical activity, body measurements, lifestyle and clinical data with nutrigenomics technology data. The JINGO program has resulted in the creation of a series of nutritional phenotype databases including participants spanning the island of Ireland from 18 to 90 years, of which the Trinity, Ulster, Department of Agriculture (TUDA) Ageing study focusses on nutrition in older age. JINGO was funded through government initiatives in the North and South of Ireland (2008-2013).



## Notes







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