P43

**Nutrigenomics: continuing education needs of registered dietitians in Malaysia**

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**Background** – Nutrigenomics or diet-gene interactions have become the focus of much nutritional research in recent years. Little is known about Malaysian dietitians’ involvement and education needs regarding nutrigenomics.

**Objective** – The aim of this survey was to assess the awareness and continuing education needs for registered dietitians regarding nutrigenomics.

**Design** – A validated questionnaire was developed to assess continuing education needs on nutrigenomics among Malaysian dietitians. The questionnaire was distributed to all (n=90) registered dietitians who attended the Malaysian Dietitians’ Association Scientific Conference in year 2007 which resulted with a 46% response rate (n=41).

**Outcomes** – Majority of the respondents (91.5%) graduated from local universities between the years 1988 to 2007. For the same purpose, a high percentage of respondents (91.4%) used family history but only two respondents (5.7%) experienced relying on genetic tests. Although 85.7% respondents are aware on nutrigenomics, only 22.3% understood its definition. More than 80% dietitians are interested to learn and increase their knowledge on human genetics, nutrigenomics and ways to communicate this knowledge to the public. Three highest barriers to the application of nutrigenomics are lack of patient education materials (91.5%), lack of background knowledge among dietitians (85.7%) and lack of continuing education for dietitians (80%). The most preferred learning activity for continuing education was seminars.

**Conclusion** – This survey showed great interests among dietitians to increase and update their knowledge on nutrigenomics. There is a need to include genetics and nutrigenomics knowledge in the continuing education programs for dietitians in Malaysia. This may help dietitians to make informed decision about the possible use of nutrigenomics in their practice.

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P44

**Meta-analysis of the effects of zinc supplementation on high density lipoprotein concentrations in healthy subjects**

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**Background** – High-density lipoprotein (HDL) cholesterol is inversely associated with coronary heart disease (CHD). The effect of zinc on plasma HDL levels requires further elucidation (J Am Coll Nutr 2006; 25: 285-291).

**Objective** – To determine the effects of zinc supplementation on plasma HDL concentrations in apparently healthy subjects using random effects meta-analysis.

**Design** – An electronic database search of Medline, PubMed, Web of Science, and CENTRAL identified 22 English-language, human, controlled trials that investigated the effects of zinc supplementation, alone or in combination, on plasma lipids. Of these, 10 studies specifically examined the effects of zinc on HDL cholesterol levels in healthy subjects. Two reviewers independently assessed risk of study bias against pre-determined criteria, including appropriateness of allocation, randomisation, blinding, and intention-to-treat protocols, and extracted data for meta-analysis. Forest plots showing random effects were generated using endpoint values for supplement minus control group. Standard error of difference was calculated using the independence of supplement and control groups. Standard error of difference was calculated using the independence of supplement and control groups.

**Outcomes** – Participant numbers totalled 13298 with an age range of 18-89 years and a mean baseline HDL concentration of 1.49 mmol/L. Duration of eligible trials ranged from 5 weeks to 7.5 years. Zinc supplementation ranged from 15-160 mg elemental zinc/d. Overall, the random effects analysis showed that zinc supplementation decreased HDL concentration by a mean value of 0.1 mmol/L (P < 0.001). A sensitivity analysis was performed, which excluded one long-term (7.5 year) study with 12741 participants. The outcome confirmed the significant negative effect of zinc supplementation on mean HDL levels (-0.13 mmol/L, P < 0.001).

**Conclusion** – Zinc supplementation decreased HDL cholesterol concentration by an average of 7% in healthy individuals, representing an increased risk of CHD of approximately 15% (J Am Coll Cardiol 2004; 43: 717-724).