Preventing metabolic syndrome: the role of diet and activity during childhood

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Obesity, particularly central obesity, hypertension, glucose intolerance, and dyslipidemias are key components of what has become known as the metabolic syndrome. Whether treated as a syndrome or not, this clustering of risk factors has been shown to be strongly associated with an increased risk of cardiovascular disease and diabetes. Excessively high levels of these physical and metabolic risk factors have been found increasingly among children and adolescents. The effects of dietary habits and physical activity on levels of body fat, blood pressure, lipids, glucose, and a range of inflammatory markers of metabolic risk have been studied in adults but little is known about these effects among children and adolescents. This talk will focus primarily on the effects dietary patterns early in life on factors associated with the development of the metabolic syndrome. In particular, the effects of foods consumption patterns will be explored using data from such studies as the Framingham Children’s Study, the National Health and Nutrition Examination Surveys, and the National Growth and Health Study. The combined effects of diet and activity will also be explored.

Selenium, selenoproteins and prostate cancer risk

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Background - Selenium is an essential micronutrient, at low levels in the New Zealand soil. Although import of Australian wheat has helped to raise the levels of this micronutrient in the New Zealand diet, our own local studies suggest that up to half of the Auckland population could be taking in sub-optimal levels in the diet. We have suggested that this increases the risk of cancer and other chronic diseases, and could well be involved in susceptibility to prostate cancer in New Zealand.

Objectives - We wished to consider whether low serum selenium, and polymorphisms in certain genes that encode selenoproteins, could 1) interact with one another and 2) affect the risk of prostate cancer in New Zealand, and whether these effects might be reduced by selenium supplementation.

Methods - A group of high risk individuals for prostate cancer were identified through patient records at Auckland Hospital, and invited to enter a double blind, placebo-controlled trial, the “negative biopsy trial”, that was coordinated through the University of Arizona in the United States. Blood samples were taken at trial entry, after 1 months randomisation, and again after 6 months of supplementation with 0, 200 or 400mg/day of selenium, in the form of selenised yeast. Selenium levels before and after 6 months supplementation were related to biological indicators (biomarkers) associated with prostate cancer susceptibility. Subjects were also genotyped, and prostate cancer recorded during the course of 5 years of supplementation.

Results - One of the gene variants associated with glutathione peroxidise appears to be associated with prostate cancer risk in this group. It leads to high levels of DNA damage, which can be alleviated through selenium supplementation.

Conclusion - Selenium supplementation may reduce some of the detrimental consequences of a high risk genotype, associated with prostate cancer risk.