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New Zealand research in diet-gene interactions - an overview

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Background

Nutrigenomics New Zealand is tasked with developing a competency, which could be utilised by the food industry, to develop gene-specific personalised foods. On strict definitions, this programme is one of nutrigenetics. A considerable amount of published work involves the response of a single gene, usually in the form of a single nucleotide polymorphism (SNP), to a single nutrient. We are utilising Inflammatory Bowel Diseases in particular Crohn's disease (CD), as proof of principle, taking a tiered approach that begins and ends with the human population (1).

Objective

To establish the range of methodologies that would be necessary to develop personalised foods, tailored to genotype, and to apply these methods to developing diets appropriate for individuals with CD in New Zealand.

Design

A case-control study design is being used to identify key genes associated with Crohn's disease in New Zealand. Individuals with the disease are contacted, asked for a blood sample or buccal swab, and invited to fill out a detailed food tolerance questionnaire. Once key genes are established, knowledge of key human disease SNPs is incorporated into the design of paired reporter gene constructs, whereby isogenic cell lines, with and without the variant SNP of interest, are tested for phenotypic effects of nutrients, bioactive compounds and food extracts. Lead compounds are then tested in relevant animal models, before developing into foods. A substantial component of the program relies on high quality data management, bioinformatics and biostatistics. International linkages will be essential for enhanced success of this programme.

Outcomes

This approach has led to the identification of a number of foods associated with beneficial and with detrimental effects in CD. What is apparent is that the same food which benefits one individual is detrimental to another with disease, and no single dietary regime is beneficial. We are beginning to see gene-diet interactions. For example, mushroom intolerance is found in individuals carrying variants in an organic cation transporter gene (OCTN1) (2). A range of different foods associate with the more common variants in the main CD disease gene, NOD2. Food components that lead to a reduction of relevant disease symptoms in our animal models include monounsaturated fatty acids and various polyphenols including curcumin. Since gluten free products benefit a number of individuals with the disease, we have developed methods for incorporating biologically relevant levels of bioactive compounds into gluten free products (3). We are planning to use individuals carrying the relevant genetic variants but without signs of the disease in order to do human intervention studies.

Conclusion

Gene-diet interactions are highly relevant to CD, and will form the next generation of dietary advice to benefit this group of individuals with a debilitating disease. A pathway to developing gene-specific foods has been established (4-6). Strategic international alliances will be essential for the success of this field (7).

Acknowledgements

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References

1. Ferguson LR, Philpott M, Dryland P. Nutrigenomics in the whole-genome scanning era: Crohn's Disease as example. Cell Mol Life Sci.2007 Dec. 64, 3105-18.
2. Petermann I, Triggs CM, Huebner C, Han DY, et al. Mushroom intolerance: a novel diet-gene interaction in Crohn's disease. Br J Nutr. 2009 Aug;102(4):506-8. PubMed PMID: 19660151.
3. Sun-Waterhouse D, Chen J, Chuah C, et al. Kiwifruit-based polyphenols and related antioxidants for functional foods:

kiwifruit extract-enhanced gluten-free bread. Int J Food Sci . 2009 Jun 22:1-14. [Epub ahead of print] PubMed PMID: 19548162.
4. Ferguson LR. Nutrigenomics approaches to functional foods. J Am Diet Assoc.2009 Mar;109(3):452-8. Review. PubMed PMID: 19248861.
5. Ferguson LR. Application of Nutrigenomics in Gastrointestinal Health. To appear in: Debasis Bagchi and Francis C. Lau (Eds). "Genomics, Proteomics and Metabolomics in Nutraceuticals and Functional Foods" Blackwell Publishing, USA, 2009
6. Ferguson LR, Philpott M, Barnett M. Nutrigenomics: Integrating genomic approaches into nutrition research. To appear in: Patrinos G., Ansorge W, Molecular diagnostics, second edition. Elsevier/Academic Press.Oxford, UK, 2009
7. Kaput J, Ordovas JM, Ferguson LR, et al. The case for strategic international alliances to harness nutritional genomics for public and personal health. Br J Nutr. 2005 Nov;94(5):623-32. Review. PubMed PMID: 16277761.

Is the efficacy of dietary fat manipulation influenced by common polymorphisms?

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Common single nucleotide polymorphisms (SNPs) are known to influence the responsiveness to dietary fat composition at a number of levels. SNPs influence fat absorption, tissue partitioning and metabolism, and therefore the relationship between intake and tissue status. For example, data from both *candidate genotyping* and *genome wide association* (GWA) studies indicates that SNPs in the fatty acid desaturase (FADS1 & 2) gene locus influence the elongation of both linoleic acid and α-linolenic acid to the longer chain PUFAs, arachidonic acid and eicosapentaenoic acid. The impact of tissue fatty acid status on physiological risk factors of disease is also influenced by a number of SNPs, with much of the research in this area focussed on the impact of fatty acid composition on blood lipid levels. Furthermore it is becomingly increasingly apparent that SNPs influence appetite, satiety and food preference, and therefore fat intake.

However, a recognised concern in the field of genotype-dietary fat-phenotype associations, and the field of nutrigenetics in general, is a perceived lack of consistency between studies, which is causing some individuals to lose faith in the potential impact of this branch of nutrition. As will be discussed this apparent lack of consistency is likely to be attributable to the influence of factors such as ethnicity and gender on the 'size' of nutrigenetics interactions, a clear understanding of which needs to be gained.

Although not yet ready for widespread use in clinical or public health, in the future a greater use of genetic profiling is likely to enhance current strategies of disease prediction, and allow the provision of more personalised dietary advice to minimise risk in the individual (1).

Reference

1. Rimbach G, Minihane AM. Nutrigenetics and personalised nutrition: how far have we progressed and are we likely to get there? Proc Nutr Soc 2009:1-11.

The interplay between bioactive soy isoflavones the food matrix and the intestine in influencing the nutritional and clinical actions at the clinical, biochemical and genomic level - considerations in the development of functional foods in the 21st Century

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Interest in the potential health benefits of soy foods and more specifically the constituent isoflavones continues even though more recent clinical intervention studies have shown disappointing results, that are inconsistent with the fact that isoflavones possess potent and wide-ranging biological properties, including their ability to bind to estrogen receptors in a manner consistent with selective estrogen receptor modulators, rather than estrogens. This apparent lack of effectiveness when compared with the well-recognized nutritional benefits of soy foods is difficult to reconcile because driving interest in soy was the idea that a high intake of isoflavones from soy foods could explain the relatively low rates of many hormone dependent disease in Asian countries. Many foods that contain soy protein and delivered isoflavones have flooded the market, but most of these soy foods consumed in western populations differ significantly in composition from those consumed in Asia where a soy inclusive diet appears to have beneficial effects. Clinical trials conducted have largely ignored this difference, which could account for the variability in outcome measures. In the future development of any functional or novel food designed to deliver specific phytochemicals or bioactive ingredients, such as soy isoflavones, consideration must be given to the important role that the intestine and bacteria play in altering the metabolism of dietary constituents among individuals, how differences in the chemical composition of isoflavones can influence their bioavailability, whether the interaction with the food matrix can modify in modifying the bioavailability and hence physiological actions at biochemical and genomic levels. In the case of isoflavones, intestinal metabolism is crucial to their bioavailability, biological actions and efficacy. Our recent development of a novel functional food, a pasta, designed to deliver the potential benefits of soy isoflavones, illustrates the importance of taking into account all of the above considerations. Placebo-controlled dietary intervention studies conducted in patients hypercholesterolemia, diabetes and gastroparesis will be presented that show how there is a strong interplay between all of the above factors and why these need

consideration in the future development of nutrition in the 21st century.

Gene expression and physical activity

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Background

Skeletal muscle is the predominant regulator of mobility, whole body energy use and hormone action, including insulin. In response to exercise, muscle has a remarkable capacity for remodelling, regulated by the coordinated induction of many hundreds of genes. The remodelling that occurs is, however, highly dependent upon the frequency, duration and intensity of the imposed exercise stimuli.

Objective

Emerging research is addressing both the upstream signals that coordinate the genes that are expressed, and how nutrient influences these processes.

Outcomes

Metabolic and oxidative stresses during exercise strongly influence signalling events in skeletal muscle. Most notably, oxidative stress activates complex free radical-sensitive signalling cascades. These signalling cascades activate genes, which in turn encode proteins that elicit inflammation and stress responses within muscle. The inflammatory response has been described as a negative consequence of strenuous physical activity, causing muscle damage and soreness. Exercise-induced inflammatory mediators (e.g., cytokines, chemokines, prostaglandins) may, however, play more fundamental roles in attracting immune cells that assist with the remodelling and adaptation of muscle. Indeed, these inflammatory mediators are potent activators of cellular growth, suggesting a more fundamental role in muscle adaptation. It remains popular for athletes to ingest antioxidant minerals and vitamins to counteract the inflammatory response to exercise. These antioxidants suppress the signaling cascades that respond to oxidative stress, and therefore modify the inflammatory responses elicited within skeletal muscle. Recent data demonstrating that antioxidants are detrimental to signalling mechanisms and muscular adaptation will be described.

Conclusion

Exercise is a potent regulator of muscle gene expression. This gene expression response includes the rapid and marked activation of genes that code for inflammatory factors. The ingestion of antioxidants suppresses this response. Recent data highlights that the ingestion of antioxidants reduces the beneficial adaptations that are accrued with exercise training. These data suggest a more cautious approach is required in recommending antioxidant supplements for those individuals undertaking regular exercise.

Concurrent Session 1: Dietary Fats and Oils

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Evaluation of gastrointestinal and gustatory sensitivity to oleic acid in lean and obese men

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Background

Failure to regulate the intake of dietary fat is a major contributing factor in the pathogenesis of obesity. Changes in the body's physiological responses to dietary fats may contribute to excessive fat intake. In humans, fat intake may be regulated by *1)* orosensory stimulation, i.e. taste perception, and *2)* inhibitory postingestional feedback from the gastrointestinal tract.

Objective

To evaluate taste and gastrointestinal sensitivity to oleic acid (C18:1) in lean and obese males.

Design

20 males (10 obese, BMI 33.2±0.8, age 42±4 yrs, 9 lean, BMI 23.7±0.6, age 37±5 yrs) were studied on 3 occasions in single-blind, randomised order. On 2 visits, antropyloroduodenal pressures were measured during a 90-min intraduodenal infusion of saline (control, "C") or C18:1 (0.78 kcal/min). At t=90 min, energy intake was quantified at a buffet lunch. On the third visit, taste sensitivity to C18:1 was established using 3-Ascending Forced Choice procedure, and habitual fat (fat intake questionnaire) and food (by 2-day dietary recall) intake was assessed.

Outcomes

In lean subjects, C18:1 stimulated the number (C: 1.6±0.3, C18:1: 5.8±0.7) and amplitude (C: 19±3, C18:1: 31±5 mmHg) of isolated pyloric pressure waves compared with saline (both $P<0.05$). This stimulation was absent in the obese (number; C: 2.2±0.3, C18:1: 2.9±0.7, amplitude; C: 20.6±3, C18:1: 23.5 mmHg). Similarly, C18:1 suppressed energy (C: 5195±505 kJ, C18:1: 4564±509) and fat (C: 45±5, C18:1: 35±5g) intake in lean subjects compared with saline (both $P<0.05$), but not in the obese (energy; C: 5985±479, C18:1: 5628±483, fat; C: 55±5, C18:1: 51±5). Obese subjects had significantly higher taste thresholds for C18:1 (obese: 8.7±1, lean: 3.8±1 mM), which were related directly to BMI ($r=0.7$, $P<0.05$). Habitual energy (obese: 13476±1984, lean: 8006±503 kJ) and fat (obese: 127±27, lean: 67±6 g) intakes were also greater in the obese (both $P<0.05$).

Conclusion

The data suggest that taste and gastrointestinal perception of fat is compromised in obesity. The observation that habitual fat intake was greater in obese subjects suggests that dietary habits influence sensitivity, and may contribute to excessive fat consumption and the consequent development of obesity.

Dietary monounsaturated fat (from macadamia nuts) added isoenergetically to usual diets can reduce coronary risk in overweight subjects

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Background

Excess adiposity (overweight) is one of a number of factors which increase risk for a range of cardiometabolic diseases. Most risk reduction strategies rely on weight reduction through dietary energy restriction. However, long-term intentional weight reduction in overweight populations has proven to be very difficult to achieve. Therefore, it is important to identify strategies to reduce risk which do not necessarily rely on weight loss.

Objective

To compare the effects of adding monounsaturated fat (MUFA) from macadamia nuts on coronary risk compared to usual diet in overweight adults.

Design

We used a randomised controlled trial design to study the effects of maintaining usual energy intake, but manipulating dietary lipid profile in a group of 64 overweight (BMI>25), otherwise healthy, subjects. For the intervention group, energy intakes of usual (baseline) diets were calculated from multiple 3 day diet diaries, and saturated fat was replaced with MUFA to 50%E by adding macadamia nuts to the diet. Both control and intervention groups received advice on national guidelines for physical activity and adhered to the same protocol for diet diary record keeping and trial consultations. Anthropometric and clinical measures were taken at baseline and at 10 weeks.

Outcomes

No significant changes in any parameters were noted for the control group over the 10 week study period. In the macadamia group, significant ($p<0.05$) reductions in true waist (100.0 to 97.5cm) and total cholesterol (5.38 to 5.10mmol/L), and increases in blood glucose (5.27 to 5.43mmol/L) and percentage change in brachial artery flow-mediated dilation diameters (7.77 to 10.43) were noted. No

significant changes were observed for blood pressure, BMI or inflammatory markers.

Conclusion
In groups where adherence to dietary energy-reduction is poor, isoenergetic interventions which address cardiometabolic risk factors other than body weight may be beneficial.

Blood pressure is associated with markers of endogenous fat metabolism

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Background
The type of fat consumed is one of many dietary determinants of blood pressure (BP). The mechanism of action is unknown but may relate to the role of desaturase activities. Stearoyl-CoA desaturase (SCD) and delta6-desaturase (D6D) catalyse the endogenous synthesis of long chain unsaturated fatty acids into 16:1 n7 and 20:3 n6 fatty acids. In epidemiological studies these fatty acids have been shown to be associated with high CVD risk. Red blood cell (RBC) fatty acid ratios may be used as surrogate measures of desaturase activities, bearing in mind that fatty acid levels are influenced by both dietary fat and endogenous metabolism of fatty acids. Identifying associations between these factors and blood pressure may be informative.

Objective
To assess the association between RBC fatty acids, including SCD ratio (16:1 n7/16:0) and D6D ratio (18:3 n6/18:2 n6), and blood pressure and heart rate.

Design
Baseline data from n=112 overweight adults in a 12 month trial [ACTRN12608000425392] were available for the analysis. Office BP 24-hour ambulatory systolic and diastolic BP, awake and asleep BP, heart rate, and mean arterial pressure (MAP) (n = 75) were measured. Readings were taken every 30 minutes during awake time (06h00-22h00) and every 60 minutes during asleep time (22h00-06h00). RBC fatty acids were measured in a quality assured laboratory (Analytical Reference Laboratories Pathology, Melbourne) and Spearman's correlation was investigated.

Outcomes
The SCD ratio correlated positively and significantly ($P<0.05$) with office diastolic BP ($r=0.266$) and with 24h systolic BP ($r = 0.288$), diastolic BP ($r = 0.237$), MAP ($r=0.300$), day-time systolic BP ($r = 0.266$) and night-time MAP ($r = 0.250$), while no association with D6D was found. The RBC fatty acids, 16:1 n7 and 20:3 n6 (both formed endogenously, by SCD and D6D) were associated with increased BP.

Conclusion
The fatty acids 16:1 n7 and 20:3 n6, as well as the SCD ratio, were significantly and positively associated with BP measurements. This suggests that dietary fat intake, as well as endogenous fat metabolism, may play a role in blood pressure regulation. These findings require further study.

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Saturated fatty acid-induced inflammatory signalling in preadipocytes

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Background
Insulin resistance is the major risk factor in the development of type 2 diabetes. Diets rich in saturated fatty acids (SFA) promote both insulin resistance and inflammation, mediated in part by elevated release of pro-inflammatory factors form adipose tissue. Adipose tissue is comprised of a mixture of mature adipocytes and the stromal vascular (SV) fraction. The SV fraction, containing mainly preadipocytes and macrophages, is suggested to play a significant role in the development of inflammation in adipose tissue. However few studies have addressed whether saturated fatty acids activate an inflammatory response in the preadipocytes cell population. Major pathways of SFA-mediated inflammation include the activation of the toll-like receptors (TLRs) and nuclear factor κB (NF-κB) pathways. Additionally, preadipocytes are able to secrete pro-inflammatory adipokines, including interleukin 6 (IL-6) and monocyte chemoattractant protein 1 (MCP-1).

Objective
To determine whether saturated fatty acids activate inflammatory intracellular signalling and adipokine synthesis in preadipocytes.

Design
Preadipocytes were cultured and treated with or without SFA (0.5, 0.75mM; 0.5, 2, 4 hrs) compared with LPS (10ng/ml). Protein and gene expression of NF-κB/ TLR4 signalling cascades and cytokines were assessed.

Outcomes
Treatment with SFA in preadipocytes, in a dose-dependent mechanism activate TLR4 signalling ($p \leq 0.05$). These intracellular signalling kinases correlated with a marked activation of MCP-1(1.5 ± 0.09-fold, $p \leq 0.05$) and IL-6 mRNA (1.5 ± 0.03-fold, $p \leq 0.05$). These data also demonstrated that adipokines are highly expressed in preadipocytes.

Conclusion
High levels of SFA induce inflammatory signalling in preadipocytes, suggesting that these abundant adipocyte progenitor cells contribute to the net inflammatory response of adipose depots.

The dietary implications of fat taste sensitivity

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Background

Excessive consumption of dietary fat contributes to weight gain and appears to play a role in obesity. Animal studies show that fat taste hypersensitivity is associated with decreased fat intake and preference and may influence body weight. For example, animals that are hypersensitive to fat taste consume less fat and resist weight gain when exposed to a high-fat diet; whereas hyposensitive animals consume excess fat and develop obesity when exposed to the same diet. Whether or not a similar relationship exists in humans remains unclear.

Objective

To investigate the extent of phenotypic variation in oral sensitivity to oleic acid (C18:1) in humans and determine its role in dietary fat and energy consumption, fat perception and anthropometry.

Design

51 subjects (10 males, age 22 ± 0.9yrs, BMI 23.6 ± 1.0; 41 females, age 20 ± 0.5yrs, BMI 21.4 ± 0.5) attended a single laboratory session during which they were screened for their sensitivity to 3.8mM C18:1 and identified as hyper- (3 out of 3 correct identifications of C18:1) or hyposensitive (<2 out of 3 correct identifications of C18:1). Subjects also completed a fat ranking task using custard made with 0, 2, 6 and 10% oil. A 4-day diet recall, food variety questionnaire and a food and diet questionnaire were used to determine habitual fat and energy intake, dietary variety and dietary habits. BMI was calculated from height and weight.

Outcomes

25% (n=13) of subjects were hypersensitive to C18:1 and, 75% (n=38) were hyposensitive. Hypersensitive subjects had lower energy intakes (7187 ± 790 kJ) and BMI values (20.7 ± 0.5 kg/m^2) than hyposensitive subjects (energy; 9171 ± 515 kJ; BMI, 22.2 ± 0.5 kg/m^2) (both P<0.05). Hypersensitive subjects consumed less oil, red meat and dairy products, (all P<0.05) and more hypersensitive subjects perceived fried foods as unhealthy (P<0.05). Hypersensitive subjects also scored significantly higher on the fat ranking task (P<0.05).

Conclusion

The data confirms variation in taste sensitivity to fat (C18:1). Subjects who were hypersensitive to the taste of C18:1 consumed less energy, were more capable at detecting small changes in the fat content of foods, consumed less high fat foods and had lower BMI values. These observations raise the possibility that fat taste sensitivity may be a contributing factor in energy and fat consumption and may influence BMI.

The effects of a fatty meal and cocoa antioxidants, on catalase and superoxide dismutase activity in healthy subjects

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Background

Foods that have high energy content can also be rich in oxidised or oxidisable substrates, and are said to have a high oxidative load. Consumption of these foods may lead to increases in markers of oxidative damage. Superoxide dismutase (SOD) and catalase are important endogenous antioxidants present within the circulation.

Objective

To determine if consumption of a fatty meal increases SOD or CAT activity. Also to observe if consumption of antioxidant rich cocoa at the same time as the fatty meal changes SOD or CAT activity.

Design

A repeated measures crossover design was used. A total of 21 subjects participated with four meals being consumed in a random order. The meals were: water, cocoa drink, water and a fat loaded mashed potato meal, or the cocoa drink and fat loaded mashed potato. Fasting blood samples were collected before the meal and at 1, 2 and 3 hours post-consumption, and plasma was separated. These samples were then analysed for SOD and CAT activity.

Outcomes

There was no significant change in plasma SOD or CAT between baseline and post-meal consumption within meal groups. The cocoa and mashed potato meal caused significant increases in CAT activity at two hours post consumption (p=0.014) and three hours post-consumption (p=0.041), when compared to water consumption.

Conclusion

Consumption of a fatty meal and cocoa at the same time leads to increased CAT activity, but not SOD activity.

The effect of canola oil intake on life span and oxidative stress in SHRSP rats

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Background

Canola oil ingestion as the only dietary fat source (added at 10% w/w to standard rat chow) has been reported to shorten the life span of stroke-prone spontaneous hypertensive rats (SHRSP). The mechanism by which canola oil shortens the life span of SHRSP is still unknown. Evidence suggests that canola oil is detrimental to SHRSP and leads to pathological changes. Oxidative stress leads to vascular damage and plays a critical role in the pathogenesis of cardiovascular diseases such as hypertension. The effect of

canola oil on oxidative stress in the circulation of SHRSP has not been examined previously.

Objective
To investigate the effect of canola oil intake on the life span of SHRSP, and on oxidative stress in the circulation over the course of their life span.

Design
Male SHRSP aged 5 weeks old were fed a defatted diet containing either 10 wt/wt% soybean oil or canola oil along with 1% NaCl in their drinking water, and life span was determined. Over the course of their life span blood pressure and the activities of catalase, glutathione peroxidase (GPx) and superoxide dismutase (SOD) were measured in red blood cells.

Outcomes
Life span was significantly reduced in the canola oil group compared to soybean oil, 12.3 ± 1.1 vs 14 ± 3.4 weeks, respectively ($p < 0.001$). Systolic blood pressure increased over time with a significant difference between the diets at week 11 ($p < 0.05$). Body weight was significantly different between the diets from weeks 6 to 10 ($p < 0.05$). SOD activity was significantly elevated in the canola oil group at week 7 compared to the other weeks. Catalase activity decreased between weeks 5 to 7, and between weeks 9 to 11 in canola oil and soybean oil groups. GPx activity decreased between weeks 5 to 7, with an increased at week 9 in canola and soybean oil groups.

Conclusion
Canola oil significantly reduces life span in SHRSP. Whereas, circulating levels of endogenous antioxidants are modulated over time in SHRSP fed canola oil or soybean oil. These findings question the role of circulating oxidative stress contributing to a shortened life span in SHRSP fed a canola oil rich diet.

Rapid screening of bioassay-guided elucidation of toxic substances in canola oil which shorten life of SHRSP rats

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Background
Previous studies have reported that canola oil shortens the life span of Stroke-Prone Spontaneously Hypertensive Rats (SHRSP) compared with other vegetable oils. Subsequent studies were able to conclude that fatty acid composition and plants sterols (phytosterols) in oil have no effect with longevity. Another approach to explore toxic substances in canola oil used supercritical gas fractionation of canola oil with subsequent testing in SHRSP also did not characterise the compounds responsible for life shortening of the rats. The conventional approach to screen toxic substances in oil using SHRSP rats takes more than six months and involves large number of animals.

Objective
In this work we report an alternative rapid screening method that incorporates sequential fractionation of oil and subsequent treatment of human cell lines that can be used in place of animal studies to elucidate toxic substances in canola oil.

Design
Non-polar and polar fractions of both canola and soybean oil were obtained by column chromatography. Cytotoxicity of the oils and oil fractions was determined by measuring cell viability of NRK52E and HEK 293T kidney cell lines. Cells were treated with each oil, non-polar or polar fraction at the concentration of 10-100 µg/ml.

Outcomes
Treatment of NRK52E and HEK 293T kidney cells with canola oil and a polar fraction that consisted of 3.7% of total canola oil at the concentration of 100 µg/ml showed decreased cell viability compared to that of with soy oil or the polar fraction of soy oil. The non-polar lipid fraction which is 93.6% of total canola oil showed similar cell viability as control.

Conclusion
The cytotoxicity bioassay employed in the current project needs only two week to test a number of fractions compared to 6 months per fraction in animal experiments are used to screen toxic effects. The non-polar fraction which represent the majority of the lipids in canola oil appear to be safe compared with polar fraction, mostly phyto-compounds in canola oil.

The concentration of oleocanthal in olive oil waste

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Background
Oleocanthal is a natural non-steroidal anti-inflammatory phenolic compound present in extra virgin olive oil (EVOO). It has been proposed that the biological activity of oleocanthal is partially responsible for the beneficial health effects of the Mediterranean diet. However, during the processing of olive oil, a large portion of phenolics are lost in the waste portion. Due to oleocanthal's healthful properties, its concentration in waste makes this usually disposed product a potentially valuable commodity to the nutrition/functional food industry.

Objective
In this study we determined the concentration of oleocanthal in olive oil waste and compared this to its concentration in EVOO.

Design
The concentration of oleocanthal in freshly pressed EVOO from Barnea olives and its subsequent waste was analyzed

at three time points (i.e. early, middle and late harvest) during the processing period for this cultivar. Oleocanthal concentrations were quantified using high performance liquid chromatography (HPLC).

Outcomes

At the time points, early and middle harvest, the concentration of oleocanthal in waste was comparable with that in the oil (128.25 ± 14.67 mg/kg and 123.24 ± 6.48 mg/kg, 112.15 ± 1.51 mg/kg and 114.20 ± 19.9 mg/kg respectively). However at late harvest, there was a decline in the concentration of oleocanthal in the waste compared to the oil (62.35 ± 7.97 mg/kg and 152.22 ± 10.54 mg/kg respectively).

Conclusion

Olive oil waste, particularly from early harvest olives is potentially a valuable commodity to the nutrition/functional food industry.

Concurrent Session 2: Salt

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Iodine status during pregnancy and lactation in Palmerston North, New Zealand

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Background

Recent studies have reported that iodine deficiency is re-emerging in New Zealand. Iodine requirements increase during pregnancy and lactation, increasing the risk of deficiency during these periods. Inadequate iodine status during pregnancy can affect fetal mental development and can lead to psychomotor, speech and hearing defects, and also mental retardation. Iodine deficiency during infancy can impair both mental and physical development.

Objective

To explore the current iodine status during pregnancy and lactation in a self-selecting population within the Palmerston North area of New Zealand.

Design

Pregnant and breastfeeding women were recruited from the Palmerston North area via advertisements in the local media and posters at medical and health centres. Twenty-four hour urine samples were obtained from pregnant women (n=24) after 28 weeks gestation and breastfeeding women (n=28) three weeks after delivery. Breast milk samples were also collected (n=27). Iodine concentration was determined in urine and milk samples using inductively-coupled plasma mass spectrometry.

Outcomes

During pregnancy median urinary iodine concentration was 45 µg/L (range 13-121 µg/L) and during lactation median urinary iodine concentration was 36 µg/L (range 9-89 µg/L). None of the participants met the urinary iodine levels recommended by the World Health Organisation for adequacy of 150-249 µg/L during pregnancy and ≥100µg/L during lactation. The median iodine concentration for the breast milk samples was 41 µg/L (range 16-288 µg/L), below the concentration considered to be adequate of 75 µg/L.

Conclusions

This study shows iodine deficiency is a problem within this population of pregnant and breastfeeding women. This has potential adverse consequences for both mothers and their infants. From September 2009 mandatory fortification of bread with iodised salt will come into effect throughout New Zealand. This research provides baseline data which can be used to asses the extent to which mandatory fortification improves iodine status within this vulnerable population.

Achieving salt intake of 6g per day in the current food supply in free-living adults using two dietary education strategies

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Background

The national target for salt intake is specified at 6g of salt/day by many countries including Australia. However there is limited knowledge of the effectiveness of simple dietary education in reducing salt intake.

Objective

To investigate whether the information provided by two different education strategies enables a reduction in salt consumption using foods available in the current food supply.

Design

This was a parallel study conducted over eight weeks. Forty-nine healthy free-living adults were recruited from the community via a newspaper advertisement. Two education strategies were tested. Participants received dietary education at baseline and week four to choose foods identified by either Australia's National Heart Foundation (NHF) Tick symbol or by the Food Standards Australia and New Zealand (FSANZ) low salt guideline of 120 mg sodium/100 g of food. Sodium intake was assessed by 24 h urinary sodium excretion. Experiences regarding adherence to the strategies were recording via a self-administered questionnaire.

Outcomes

Forty-three men and women completed the study. Urinary sodium excretion decreased from 121 ± 50 to 106 ± 47 mmol/24h in the Tick group and from132 ± 44 to 98 ± 50 mmol/24h in the FSANZ group ($p<0.05$, with no between group difference). Barriers to salt reduction included limited variety and food choice, eating-out and increased time associated with identifying products.

Conclusion

Dietary sodium reduction is possible among free-living individuals receiving simple dietary advice.

The relationship between sodium concentration and liking of salty foods

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Background

Population wide sodium (Na) reductions have been recommended due to the established link between high Na intake and hypertension, and hypertension and cardiovascular disease. Despite this Australians are still consuming Na above dietary recommendations, primarily through consumption of processed foods. Na, in the form of sodium chloride (salt), is added to processed foods to increase palatability; however it may be possible that Na concentrations can be reduced without altering the liking of a salty food.

Objective

To determine if a relationship exists between liking, salt taste intensity and Na concentration and to assess if Na concentration can be reduced without affecting liking of a food.

Design

Four variations of hash-browns were produced, identical except for the concentration of sodium (mg/100g): 40 mg – no added Na; 120 mg – Heart Foundation tick criteria; 170 mg- low commercial; 220 mg- high commercial). Subjects (n=56, 48 female) rated their liking and perceived salt taste intensity of the hash-browns. In a separate session, taste sensitivity to Na was determined in accordance with -ISO 3972:1991-Method of investigating sensitivity of taste. 1-way ANOVA and Pearsons product moment correlations were used to analyse results. P values <0.05 were considered statistically significant

Outcomes

There was a significant difference in saltiness and liking between the 220 mg and 40 mg Na hash-brown (P<0.05). There was a significant correlation between liking and salt taste intensity in hash-browns (r=.547 P<0.01). However, while the 220 mg hash-brown was most liked, there was no significant difference in salt taste intensity or liking between the 220, 170 or 120 mg Na hash browns. No significant associations were found between oral sensitivity to Na and liking or perceived intensity of salty foods.

Conclusion

The liking of hash browns can be maintained while significantly reducing the concentration of Na (up to 46%) to a level that meets the Heart Foundation 'tick' criteria of 120 mg Na/100 g.

Sodium and potassium intakes in an Australian population sample: Has there been a change over the past 15 years?

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Background

A high sodium low potassium diet typically consumed in Western countries is associated with adverse health effects including elevated blood pressure and cardiovascular disease. The last time population sodium and potassium intakes were measured in Australia was fifteen years ago and potassium intakes were below recommended and sodium intakes well above. It is unknown if intakes have changed since this time and this information is crucial to understanding Australians' health risk.

Objective

To determine if there has been a change in sodium and potassium intakes from 1992-1994 to 2007-2008 in a Victorian sample of the Australia adult population.

Design

A subsample of participants (n=261) from the Melbourne Collaborative Cohort Study provided 24 h urine collections in 1992-94 and a different subsample of participants, matched to the original sample for sex, date of birth and ethnicity (n=264), provided 24 h urine collections between 2007-08. Urine samples were assayed for sodium, potassium and creatinine.

Outcomes

The mean (SD) age of participants in 1992-94 was 54.3 (7.2) y and in 2007-08 was 68.3 (6.2) y. Mean daily excretion of sodium was not different between 1992-94 and 2007-08 (168 (102) vs 156 (57) mmol, P>0.05). Compared with the 1992-94 cohort, the 2007-08 cohort had a significantly higher mean daily potassium excretion (71 (44) vs 82 (27) mmol, P=0.001) and markedly lower sodium potassium ratio (2.6 (1.1) vs 2.0 (0.8) P<0.001).

Conclusion

Sodium intakes in Australia remain well above the recommended upper level of intake of 100 mmol/day and although potassium intakes appear to have improved over the last 15 years, they are still below the recommended daily adequate intake of 100 mmol for men and 72 mmol for women. These data indicate that public health measures need to be taken in Australia to improve intakes of sodium and potassium in the population.

Ethnic variation in dietary sodium intakes within an Australian population sample

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Background

One of the leading causes of cardiovascular disease is hypertension. Diet has been shown to influence blood pressure, especially salt or sodium. Excess salt consumed throughout life causes blood pressure to rise with age. There is no recent data on the sodium intake of Australians that includes 24hr urinary sodium, the gold standard method for accurately determining sodium intake, and the extent to which ethnicity will affect intake.

Objective

To characterise the variation in dietary sodium intake in an Australian population sample, using 24 hr urine collection and dietary analysis (food frequency questionnaire and 24hr diet recalls).

Design

A cross-sectional analysis of participants who provided 24hr urine samples (2007/08) and complete dietary data that were enrolled in the Melbourne Collaborative Cohort study.

Outcomes

From 790 participants, mean urinary sodium for males was 178.3 (66.7) mmol/day and 133.7(51.0) mmol/day for females. Nearly 90% of men and 70% of women had intakes greater than the recommended 100mmol sodium (6g salt)/day. Sodium excretion was 19% higher for participants who added salt to cooking than those who did not,158.8(64.3) mmol/day versus 129.4(48.8) mmol/day (P <0.001), however no difference was seen with salt usage at the table. Participants with a Greek/Macedonian (30% sample) and an Italian heritage (32% sample) reported using salt in cooking more than those with an Australian/New Zealander background (37% sample) 97% and 98% versus 62% respectively (P < 0.001).

Conclusions

The majority of individuals within this sample exceed the recommended upper levels of 6 grams salt (per day) and ethnic variations in discretionary salt intakes were seen. To achieve population wide reductions in salt intakes, a reduction in the sodium content of processed foods is needed alongside educational campaigns targeting specific ethnic groups in discretionary salt use.

Dietary sodium and sodium potassium ratio predicts blood pressure in an Australian population sample

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Background

Raised blood pressure (BP) is a major risk factor for cardiovascular disease. Excess sodium consumed throughout life contributes to the age-related rise in BP. Reducing dietary sodium or the dietary sodium to potassium ratio lowers BP. The relationship between dietary sodium and potassium intake and BP within an Australian population group has not previously been assessed.

Objective

To assess the relationship between dietary sodium and potassium intake and blood pressure in an Australian population sample, using the gold standard measurement of 24 hr urinary excretion.

Design

A cross-sectional study was conducted using participants enrolled in the Melbourne Collaborative Cohort study. Daily intakes of sodium and potassium were measured from 24 hr urine samples provided by participants (2007/08). BP was assessed under standard conditions in a subgroup of this population.

Outcomes

The mean age of participants (men n=376, women n= 408) was 64.0 (6.3) (SD) years. The mean urinary sodium was 155.1 (63.1) mmol/day (8.9 (3.6) g salt/day), mean urinary potassium was 82.3 ± 27.9 mmol/day and the sodium to potassium (Na:K) ratio was 2.0 ± 0.8 /day. Seventy nine percent of participants consumed more than the recommended intake of sodium (100 mmol/day). In the 584 participants who provided blood pressure measurements, sodium and the Na:K ratio were both predictors of systolic BP (β coefficient (se) β=0.29(0.01) P=0.011, β=2.13(0.86) P=0.014) and diastolic BP (β=0.037(0.01) P<0.001, β=1.57(0.58) P=0.008). After adjustment for age and sex, sodium and the Na:K ratio remained significant predictors for systolic BP (P=0.002, P=0.004), but not for diastolic BP.

Conclusion

In an Australian population sample, most participants were consuming excessive amounts of sodium. Dietary sodium and the Na:K ratio were both significant predictors of BP. These results indicate that a population wide reduction in dietary sodium would be effective in reducing blood pressure in Australia.

When are we consuming all this sodium?

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Background

Reducing dietary sodium (Na) intake is a recommended strategy for reducing chronic disease risk. However sodium intakes in the Australian population remain high. Understanding food related behaviours associated with sodium intake is an important step to help target appropriate dietary advice. Specifically, an estimate of the contribution of different meals and snacks to total dietary sodium is required.

Objective

To determine amount of sodium provided at different meals and snacks, in free living Australian adults.

Design

Two hundred and ninety nine adults (158 women,141 men), who participated in dietary studies, provided 24 hr dietary recalls (analysed using FoodWorks, Version 4) on their usual diet. All foods eaten were classified into 6 meal or snack times: breakfast (BF); morning tea (MT); lunch (L); afternoon tea (AT); dinner (D) and supper (S). Each individual's proportion of Na per meal or snack was calculated and the group average determined.

Outcomes

The mean (SD) age of the participants was 54.6 (9.5) yrs and the mean (SD) BMI was 29.4 (3.9) kg/m^2. Thirty-six percent (n=107) of participants were taking antihypertensive medication. The mean (SD) total daily sodium intake from food was 2725.0 (1176.2) mg.

Sodium contribution per meal or snack						
	BF		MT		L	
	%	mg	%	mg	%	mg
All	16.9	435.9	3.1	88.7	33.9	915.3
F	16.5	345.4	2.9	65.0	33.8	763.9
M	17.2	537.3	3.3	115.3	34.1	1084.9
	AT		D		S	
	%	mg	%	mg	%	mg
All	5.0	134.0	38.4	1084.4	2.8	66.6
F	5.4	123.1	38.2	887.2	3.1	66.4
M	4.6	146.3	38.5	1305.5	2.4	66.8

Conclusion

Although breads and cereals and snack foods have been reported as the main contributors to sodium intake, these data suggest that food choices associated with dinner and lunch contributed the most sodium to the adult diet. Snack foods only contributed 10% of the total daily sodium in this population. Investigating the specific foods consumed at these meals times will enable more targeted public health intervention strategies.

Dietary sources of sodium in the diets of Australian children

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Background

Emerging evidence suggests that dietary sodium contributes to the regulation of blood pressure in children and that blood pressure follows a tracking pattern from childhood into adulthood. Salt added to processed foods is the main source of dietary sodium. It is important to identify the major sources of sodium in children's diets to enable the development of interventions to reduce dietary sodium intake.

Objective

To determine the dietary sodium intake and dietary sources of sodium in a nationally representative sample of Australian children aged 2 – 16 years.

Design

Analysis of the Australian 2007 Children's National Nutrition and Physical Activity Survey. A 24-hour dietary recall was completed for 4826 children from which sodium intake was determined. Children also reported the use of table salt and salt in cooking.

Outcomes

Thirty percent of all children added salt at the table and 39% of children consumed meals where salt had been added during cooking. The greatest contributor to sodium intake across all ages was cereals and cereal based products and dishes (43%), which included bread (13%), pastries (5%) and breakfast cereals and bars (4%). The second major source of sodium intake was meat, poultry and game products and dishes (16%), including processed meats (8%) and sausages (3%). Other moderate sources of sodium include milk products and dishes (11%) and savoury sauces and condiments (7%).

Conclusion

To reduce children's dietary sodium intake those foods that contribute the most sodium such as bread, breakfast cereals, pastries, processed meats, cheese and sauces should be targeted for product reformulation, to decrease the sodium content. Identifying the major sources also means we can target these foods and their lower sodium alternatives in behavioural interventions. Furthermore, continued education aimed at reducing salt addition during cooking and at the table is required.

Monosodium glutamate is not associated with obesity, or a greater prevalence of weight gain over 5 years: findings from the Jiangsu Nutrition Study of Chinese adults

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Background

Animal studies and one large cross-sectional study of 752 healthy Chinese men and women suggest that monosodium glutamate (MSG) may be associated with overweight/obesity, and these findings raise public concern for the use of MSG as a flavour enhancer in many commercial foods.

Objective

The aim of this analysis was to investigate a possible association between MSG intake and obesity, and determine whether a greater MSG intake is associated with a clinically significant weight gain, over five years.

Design

Data from 1282 Chinese men and women who participated in the Jiangsu Nutrition Study (JIN) were analyzed. In this study, MSG intake and body weight were quantitatively assessed in 2002, and followed-up in 2007.

Outcomes

MSG intake was not associated with significant weight gain after adjusting for age, gender, multiple lifestyle factors, and energy intake. When the intake of total glutamic acid intake was added to the model, an inverse association between MSG intake and 5% weight gain was found (P=0.028), but when the model was adjusted for either rice intake or food patterns, this association was abolished.

Conclusions

These findings indicate that when other food items or dietary patterns are accounted for then no association exists between MSG intake and weight gain over a period of five years.

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Maternal high-fat consumption and the risk of disease in the offspring

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Background

Recent evidence suggests that *in utero* nutrition can play a significant role in determining the health of an individual in later life. The nutritional manipulations have included low-protein or caloric restricted diets on one hand, and energy rich or high-fat diets, on the other. A typical North American diet is rich in dietary fats that have been linked to an increased risk of cardiovascular disease (CVD), where both the quantity and the quality of fat play an important role. An increased consumption of saturated fatty acids (SFA) has largely been associated with higher incidence of CVD, whereas a diet rich in polyunsaturated fatty acids (PUFA) can lower the risk of developing CVD.

Objectives

We investigated whether maternal high fat-diets alter the regulation of lipid & lipoprotein metabolism and vascular function of the offspring. We further investigated whether alterations in the regulation of metabolic pathways were due to modifications of gene expression during development. Finally, we investigated whether the programming effects were gender specific and if the effects of pre-weaning diet were influenced by post-weaning diet.

Design

Female C57Bl/6 mice were fed a diet rich in SFA (S), PUFA (P) or regular chow (C) before mating, during pregnancy and lactation. After weaning, their pups received SFA, PUFA or chow diet. Offspring were grouped by sex and according to their maternal diet/postnatal diet combination (S/S, S/C, C/S, P/P, P/C, C/P and C/C). At the end of 11 weeks, plasma and hepatic lipid & lipoprotein profile and other parameters were measured. Aortic vascular function (contractile and relaxation response) and the expression of various genes involved in regulating lipid metabolism were also measured.

Outcomes

Maternal high-fat SFA feeding resulted in higher plasma triglyceride concentrations of male offspring on a chow diet (S/C), whereas the female S/C offspring had higher plasma total- and low-density lipoprotein (LDL)-cholesterol concentrations, as compared to the offspring of mothers fed chow (C/C). Female S/C offspring had reduced mRNA expression of hepatic LDL-receptor than the C/C group. Aortic contractile responses of S/S, S/C and C/S offspring were lower as compared to the C/C offspring. Maternal high-fat PUFA diet resulted in higher plasma HDL-cholesterol, along with aortic contractile dysfunction in the offspring fed chow post-weaning. A continuous exposure to PUFA diet during pre- and post-weaning time period resulted in higher body weight and higher LDL/HDL-cholesterol ratio.

Conclusion

Our novel observation of high maternal SFA intake induced suppression of hepatic LDL-r expression and increased levels of LDL-cholesterol points to mechanistic link between the high fat maternal diet and programming of adult disease in female offspring. Our findings further suggest that a maternal high-fat PUFA diet may not necessarily have beneficial health effects for the offspring.

Activation of metabolic flexibility by exercise prevents prenatally-induced obesity

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Background

Effective regulation of energy metabolism is vital for the maintenance of optimal health, and an inability to make these dynamic adjustments is a recognised cause of obesity and metabolic disorders. Epidemiological and experimental studies have highlighted the role of prenatal factors in the disease process and it is now generally accepted that maternal nutrition during pregnancy significantly influences intrauterine development, shaping postnatal health. Consequences of impaired nutrition during foetal development include intrauterine growth restriction (IUGR) and subsequent obesity development in adult-life.

Objective

We have previously shown that prenatal undernutrition has a lasting effect on behaviour, with IUGR offspring expressing a higher preference for voluntary exercise. This presentation will review our recent research of how moderate daily exercise drives changes in metabolic pathways that promote obesity prevention in IUGR offspring.

Design

Pregnant Wistar rats were either fed chow *ad libitum* or undernourished, generating control or IUGR offspring

respectively. Exercise was available to offspring by running wheel for 1 h every day throughout adult life.

Outcomes

Although red muscle structure indicated higher oxidative capacity in IUGR offspring, obesity prevention was not due to increased fatty acid oxidation. In contrast, increased protein kinase C zeta expression and glycogen content in white muscle of exercised IUGR offspring suggests an enhanced capacity for utilisation of glucose. A higher capacity for anaerobic utilisation of glucose is further supported by stimulation of a lactate shuttle, driven by an increase in monocarboxylate transporters, preventing exercise-induced lactate accumulation.

Conclusion

Activation of metabolic flexibility in IUGR offspring by moderate daily exercise may facilitate muscle contractile performance, and therefore support effective fuel utilization for obesity prevention.

Breast feeding protects against current asthma up to 6 years of age

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Background

The relationship between breast feeding and the development of asthma is controversial.

Objective

To investigate whether breast feeding protects against current asthma in children up to 6 years of age.

Design

1105 infants were enrolled in a prospective birth cohort in two metropolitan centres in New Zealand. Breast feeding was assessed in two ways: duration of “exclusive” breast feeding (age when infant formula, food or other drinks, except water, were introduced) and duration of “any” breast feeding (age when breast feeding was stopped). Current asthma was defined at 2, 3, 4, 5 and 6 years as “ever had a doctor’s diagnosis of asthma” AND [“wheeze in the last 12 months” OR “inhaler use in the last 12 months”]. Logistic regression was used to model associations between breast feeding duration and outcomes after adjusting for relevant confounding variables.

Outcomes

Longer durations of exclusive breast feeding were associated with significant reductions in the risk of current asthma up to 6 years (all, $p<0.026$). Each month of exclusive breast-feeding reduced the risk of current asthma by 17% (0.83, 0.76-0.92) at 2 years; 12% (0.88, 0.80-0.96) at 3 years; 11% (0.89, 0.82-0.97) at 4 years; 12% (0.88, 0.80-0.95) at 5 years; and 9% (0.91, 0.83-0.99) at 6 years. Risk of current asthma was also reduced by longer durations of any breast feeding up to 4 years (all, $p<0.011$). The relationship between exclusive breast feeding and current asthma was modified by atopy at 4, 5 and 6 years ($p=0.018$, $p=0.04$, $p=0.058$ respectively).

Conclusion

Breast feeding, particularly exclusive breast feeding, protects against current asthma up to 6 years of age. Although exclusive breast feeding reduces risk in all children to age 6, the degree of protection beyond 3 years was more pronounced in children with atopy.

Nutrient supplementation in pregnancy: development of evidence-based best-practice guidelines

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Background

Nutrient supplementation in lead up to, and during, pregnancy is common in both Western and developing countries.

Objectives

(i) To derive from published literature data on the safety and efficacy of nutrient supplementation (single and combination supplements, containing macro- and/or micro-nutrients) in lead up to, and during, pregnancy. (ii) To construct a set of evidence-based national nutritional guidelines on an appropriate use of supplements for pregnancy in Australia.

Design

The Cochrane Library and PubMed were searched for previously published meta-analyses and recent systematic reviews (from 1999 onwards) using the key search terms ‘pregnancy’, ‘supplement’, and ‘nutrient’. Hand searching of reference lists was also undertaken. The review of reviews was then translated into evidence-based best-practice guidelines, using the Appraisal of Guidelines for Research and Evaluation Instrument[(1)].

Outcomes

To date, 16 meta-analyses for a range of macro- and micro-nutrients have been identified, and the data extracted. Of these, folate remains the only ubiquitously recommended nutrient for supplementation before and during pregnancy, because of the major relative risk (RR) reduction in neural tube defects (0.28; 95% confidence intervals (CI) 0.13, 0.58). This is despite possible risks associated with an increased incidence of multiple births

(RR 1.4; 95% CI 0.93, 2.11). Prophylactic calcium supplementation may offer benefits in high-risk cases of preeclampsia (RR 0.48; 95% CI 0.33, 0.69), particularly where dietary intake is poor. Iron supplements improve haematological markers, but users also frequently experience adverse gastrointestinal disturbances (RR 1.90; 95% CI 1.09, 3.33).

Conclusions

With the exception of folate, nutrient supplementation before and/or during pregnancy, is not generally recommended, unless dietary intake is inadequate. This is because of significant deficits in the available evidence for their safety and efficacy.

References

1. The Agree Research Trust, http://www.agreetrust.org/instrument.htm, accessed 18 June 2008.

Investigation of casein and whey proteins during a single breast expression session and over a 24-h breastfeeding period

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Background

Breastmilk proteins have both nutritional and physiological significance for infants. Previous long-term studies of casein and whey proteins in breastmilk have not considered short-term (during the day) variations. Understanding these variations has implication for appropriate sample collection for future nutritional studies.

Objective

To investigate the protein concentration in casein and whey fraction of fore and hind milk and between the left and right breasts during a breast expression and over a 24-h period during breastfeeding.

Design

Fore and hind milk samples of 17 healthy term mothers were collected during simultaneous breast expression session (15 min) of the left and right breasts. A sub group of 5 mothers provided milk samples from each breastfeed over a 24-h period, and milk samples were selected from four time points (morning: 0401-1000; day: 1001-1600; evening: 1601-2200; and night: 2201- 0400). Casein and whey were separated from the defatted (skim) milk. After addition of calcium chloride, the pH of skim milk was adjusted to 4.3 followed by ultracentrifugation. The protein concentration of skim, casein and whey fractions was determined by the Bradford protein assay.

Outcomes

The concentration of protein (mean $\pm$ SD g/L) was not significantly different between the fore and hind milk in skim (13.47 $\pm$ 2.39), whey (7.10 $\pm$ 2.01) and casein (2.78 $\pm$ 0.73). In addition, no significant difference between left and right breasts was detected for the protein concentration of skim and whey. However, we found a significant difference ($P = 0.0026$) in the concentration of casein between left and right breasts of seven mothers. Over a 24-h period, within mothers, no apparent differences were found in protein concentrations of casein and whey at the four time points but there was large variation between the mothers with a CV of 18.9% and 32.2% for whey and casein, respectively.

Conclusion

For sampling purposes a single breastmilk sample (fore or hind from each breast) can be used to estimate a protein concentration of skim milk, whey and casein fractions. In addition, preliminary findings of variation between mothers and also between breasts need further investigation.

Maternal macronutrient and micronutrient profiles in pregnancy and postpartum

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Background

Maternal nutrition during pregnancy is known to influence developmental outcomes for the offspring. During lactation, maternal nutrition continues to be important to both mother and child. There are very few studies which report dietary data collected during pregnancy and after birth in Australian women.

Objective

To assess nutritional adequacy of maternal dietary intakes from a Hunter cohort during pregnancy and after birth, in comparison to the Nutrient Reference Values.

Design

Prospective longitudinal study of women during pregnancy and after birth. Maternal dietary intake was assessed using the Dietary Questionnaire for Epidemiological Studies (DQES), a food frequency questionnaire validated in young Australian women. Questionnaires were completed at approximately 18 (*n* 159, response rate (*RR)* 88%) and 36 (*n* 105, *RR* 67%) weeks gestation, and at 13 (*n* 185, *RR* 56%) and 26 (*n* 92, *RR* 61%) weeks postpartum. DQES were analysed using NUTTAB95.

Outcomes

The median energy intake reported by the full cohort ranged from 8160 kJ/day at 18 weeks and 7570 kJ/day at 36 weeks gestation to 7760 kJ/day and 7500 kJ/day at 13

and 26 weeks postpartum respectively (excludes alcohol). Fibre and iron intake during pregnancy and after birth remained constant, with maternal intakes estimated at 20 g/day and 13 mg/day respectively. Folate intake declined progressively between 18 weeks gestation (294µg) and 26 weeks postpartum (255µg), while calcium intake peaked at 36 weeks gestation (1025 mg/day) and progressively declined until 26 weeks postpartum. Mean intakes of iron, folate and calcium during pregnancy and postpartum were less than 50% of the Recommended Dietary Intakes.

Conclusion

Preliminary findings indicate that some key nutrient targets are not being met by this sample of Australian women during pregnancy and after birth. Further work to validate reported dietary intakes for this population group will strengthen its application to providing nutritional guidance in pregnancy and postpartum.

Is the iodine status of pregnant women better in India or New Zealand?

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Background

Iodine deficiency in pregnancy may adversely affect the health of mothers and the normal growth and development of their babies, which has led to a growing realisation worldwide that women have adequate iodine status during pregnancy.

Objective

To compare and contrast the iodine status of pregnant women living in opposing environments: rural India and urban New Zealand.

Design

Two separate observational studies were conducted; one of pregnant women at any stage of pregnancy living in urban centres in New Zealand (NZ) between October and November 2005 and the other of pregnant women in their second and third trimester living in rural centres in central India between March and November 2008. Iodine status in both studies was determined by urinary iodine concentration (UIC) from a casual urine sample and serum Thyroid Stimulating Hormone (TSH) from a blood sample.

Outcomes

The recommended cut-off for median urinary iodine concentration (MUIC) in pregnancy is 150 µg/L. The (MUIC) of pregnant NZ women (n=170) was 38µg/L (Inter-Quartile Range IQR) 24 to 56µg/L), with 70% of NZ women having a UIC <50µg/L. The MUIC of pregnant Indian women (n=215) in the second trimester was 107µg/L (IQR 52 to 188µg/L), with 22% of Indian women having a UIC <50µg/L. The MUIC of these same Indian women in the third trimester was 72µg/L (IQR 42 to 150µg/L), with 30% having a UIC <50µg/L. The concentration of TSH fell within the normal reference range for both groups of women.

Conclusions

Despite different dietary patterns, socioeconomic status and geographical location, both the NZ and Indian women in these studies were iodine deficient. Iodine deficiency in pregnancy can increase the risk of stillbirth, abortion, congenital abnormalities, infant mortality, and may impair the neurocognitive development of their babies.

The food choices of Australian women during pregnancy

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Background

Maternal diet and nutritional status prior to conception and during pregnancy can influence foetal, neonatal and longer-term outcomes. Research has highlighted the importance of maternal nutrition to the health of the next generation. Therefore, examination of the adequacy of dietary practices in childbearing aged women is warranted and can be used to guide development and/or revision of dietary guidelines for this group. Knowledge of whether practices are altered prior to conception or during pregnancy, and if any dietary alterations persist postnatally is required. In Australia there is limited information describing the food choices of pregnant women.

Objective

The study aims to describe the food patterns and specific foods and food groups being consumed by young women according to pregnancy status in comparison to the national intake targets for Australian women.

Design

Food frequency data from a nationally representative sample of Australian women (25-30 yr), who completed Survey 3 of the Australian Longitudinal Study on Women's Health were analysed. The 7486 women with biologically plausible energy intakes, defined as >4.5 but <20.0 MJ/d, were included in the analyses. Pregnancy status was defined as pregnant (n 606), trying to conceive (n 454), had a baby in the last 12 months (n 829) and other (n 5597). Food group and nutrient intakes were compared with the Australian Guide to Healthy Eating and the Nutrient Reference Values for Australia and New Zealand, respectively.

Outcomes

A large proportion of pregnant women were not meeting current food group recommendations or dietary guidelines for key nutrients required in pregnancy. Significant differences (P<0.05) were found in the consumption of breads/cereals, fruit, dairy and meat groups between 'pregnant' and 'other' women. Only 8.1% of pregnant women met nutrient targets for folate, iron, calcium, zinc and fibre and they achieved this through

higher serves of fruit, dairy and meat but lower serves of vegetables compared to current recommendations.

Conclusion

Further research linking diet during pregnancy with pregnancy outcomes is required to allow the development of evidence based dietary guidelines for pregnancy. The current eating patterns of pregnant women will play an important role in this process and will ensure the nutritional needs of pregnant women are addressed.