

Concurrent Session 4: Gastrointestinal Tract

An investigation of unsubstantiated 'low GI' claims for Australian foods

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Background: The glycemic index (GI) concept is widely used as a mark of product differentiation by food manufacturers and consumers. In Australia, several cases of deceptive practice have been investigated by the Australian Competition and Consumer Commission (ACCC) and by Australia's consumer advocate organisation, *Choice*. Under the wider perspective of the protection of public health, a false GI value claim of a product could affect consumers that rely on that statement as part of their therapeutic management of a specific disorder. Food Standards Australia New Zealand (FSANZ) developed a new draft Standard for Nutrition, Health and Related Claims (P293), in which GI claims are treated as a hybrid nutrition content/health claim, and must include a scientifically substantiated numerical GI value that is determined using the Australian Standard methodology.

Objective: To determine the glycemic index (GI) of 10 products in the Australian market with unsubstantiated GI claims to verify the validity of such claims.

Design: Ten healthy subjects consumed 50 g available carbohydrate portions of the reference food (glucose sugar) and the 10 products. Fingerprick blood samples were collected at -5, 0 (fasting), 15, 30, 45, 60, 90 and 120 min and analysed for plasma glucose concentration. The incremental area under the plasma glucose response curve (iAUC) was used to calculate the GI value of each test food, using glucose as the reference food.

Outcomes: GI values for the products varied from 31 ± 4 to 85 ± 12 . Only two out of 10 products were identified as low GI ($GI \leq 55$). Eight of the 10 products were therefore found to make a false claim.

Conclusion: Many products on the Australian market make misleading and false claims around their GI. Greater surveillance and monitoring of GI claims with appropriate regulatory action is necessary for the safeguard of public health.

Microbiota and protein profiles of interleukin-10 gene-deficient mice are altered when fed diets enriched in n-3 and n-6 polyunsaturated fatty acids

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Background: Inflammatory bowel diseases (IBD) are characterised by chronic intestinal inflammation due to an abnormal immune response towards intestinal microbiota. The effect of dietary fats in relation to microbial communities, especially in relation to IBD, is not well defined.

Objective: The aim was to identify differences in the large bowel microbiota in response to diets containing polyunsaturated fatty acids (PUFA) and to correlate those with histology and protein data of interleukin-10 gene-deficient (*Il10*^{-/-}) vs. C57BL/6 control mice fed PUFA diets.

Design: Denaturing gradient gel electrophoresis (DGGE), qRT-PCR and cluster analyses of caecal bacterial DNA as well as histopathological, 2D-DIGE LC/MS-MS and pathway analyses of the colon were performed.

Outcomes: The DGGE profiles of *Il10*^{-/-} and C57 mice fed AA or EPA diet showed differences ($P < 0.05$) in the presence of DNA fragments identified as *Bacteroides vulgatus* and *E. coli* ssp. AA and EPA showed only mild reduction in colon inflammation. Bacteria-influenced proteins associated with actin cytoskeleton and tight junction signalling were more up-regulated with AA which might affect tight junction integrity and migration of bacteria.

Conclusion: These data clearly underline the necessity for a "multi-omics" approach to define the complex bacteria-host interaction networks and to identify mechanistic effects of dietary fats in colon inflammation.