Posters

**High glycemic index carbohydrate mediates an acute proinflammatory process as measured by NF-κB activation**

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**Background** - Some high-carbohydrate diets may increase the risk of cardiovascular disease (CVD) by promoting hyperglycemia, oxidative stress, endothelial dysfunction and low-grade inflammation.¹ In this context, the classification of carbohydrates according to their postprandial effects (ie the glycemic index, GI) may be relevant to prevention and management of CVD.

**Objectives** - The present study was designed to detect differences in postprandial NF-κB activation (an acute inflammatory marker) in mononuclear cells and nitrotyrosine levels (a marker of oxidative stress) after high vs low GI meals in 10 lean, young, healthy European Caucasian subjects (5 male, 5 female) matched for age, BMI, waist circumference, diet and physical activity.

**Design** - A 50 g portion of a high GI (white bread) was compared with an isoenergetic, macronutrient-matched portion of a low GI food (pasta) consumed in random order after an overnight fast. Glycaemia, insulinemia, NF-κB and nitrotyrosine levels were determined at 0, 60, 120 and 180 min and quantitated using the area under the curve (AUC).

**Outcome** - Glycemia and insulinemia were within the normal range but 3- and nearly 4-fold higher respectively after the bread meal compared with the pasta meal. As hypothesised, the NF-κB response was 3-fold greater after the bread meal (mean ± SEM: 69 ± 16 optical density (OD) · h) compared with the pasta meal (23 ± 4.7 OD · h). Nitrotyrosine levels increased after the bread meal (0.67 ± 0.49 nmole/L) and decreased after the pasta (-0.81 ± 0.30 nmole/L) but the difference did not reach statistical significance.

**Conclusion** - The present study shows that high GI carbohydrate, but not low GI carbohydrate, mediates an acute proinflammatory process as measured by NF-κB activity. Blunting postprandial glycemia via low GI carbohydrate may lower CVD risk.

**References**

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**Displacement of adhered enteropathogens from human mucus by selected lactobacilli**

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**Background** - A probiotic has been defined as a viable microbial food supplement which beneficially influences the health of the host (1). Adhesion to and colonization of the mucosal surfaces are possible protective mechanisms against pathogens through competition for binding sites and nutrients or immune modulation.¹

**Objectives** - The aim of this study was to assess the ability of selected *Lactobacillus* strains to displace pathogens from human intestinal mucus, providing a basis for the selection of new probiotics with the ability to competitively exclude intestinal pathogens.

**Design** - The *Lactobacillus* strains included in this study have been pre-selected on the basis of their resistance to acid and bile and their ability to induce the production of pro- and anti-inflammatory cytokines.² An intestinal mucus model³ was used to assess the displacement of pathogens by the selected *Lactobacillus* strains.

**Outcomes** - The levels of pathogen displacement varied between 15 and 68% depending on both the pathogen and the lactobacilli used, indicating the need of a case-by-case characterization of each probiotic strains.

**Conclusion** - Selection of probiotics that inhibit or displace a specific pathogen can be based on further assessment, product development and human clinical interventions on prevention or treatment of infection caused by that pathogen.

**References**