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### An *in-vitro* digestion model to investigate the release of organic acids from sweet potato

H Sabboh, S Le Rouzic, MJ Gidley

University of Queensland, Centre for Nutrition and Food Sciences, Australia.

**Background** – Sweet potato (*Ipomoea batatas*) is an important micronutrient resource in many tropical areas, and it is becoming more and more relevant because of its increasing importance in human nutrition. As far as we know, no studies have been conducted on the organic acid digestibility in sweet potato. Yet, it has been shown that organic acids from plant food, especially citric and malic acid have been shown to exert alkalinizing effects after absorption and metabolism, with a potential to prevent low-grade metabolic acidosis characteristic to western diet consumption.

**Objective** – The study of the release of organic acids from raw and cooked sweet potato, related to the texture and their absorption in the small intestine.

**Design** – Raw and 3 min. or 6 min-boiled sweet potato are cut into slices of 4g. An *in-vitro* mimic of human digestion is used, comprising oral (human  $\alpha$ -amylase 100 units/L, pH 6.9), gastric (porcine pepsin 800-2,500 units/mg, pH 2), and pancreatic (pancreatin activity 4 X USP specification and bile extract, pH 6) digestion models followed by an *in-vitro* model of small intestine absorption using Caco-2 cell monolayers. Moreover, texture analysing has been conducted on the different sweet potato samples to relate the organic acid digestion release to the cell wall structure.

**Outcomes** – The preliminary kinetic results of *in-vitro* digestion indicate that raw sweet potato samples have the lower percentage of release of oxalic and tartaric acids compared to the 3 and 6 min-boiled samples. Moreover, the release of these two organic acids during the digestion increases in function of the boiling time. As expected the texture analysing shows that the stiffness, i.e. the resistance to deformation (N/mm<sup>2</sup>/s), is the highest with the raw sample and the lowest with the 6 min-boiled sample, which would explain the *in-vitro* release patterns.

**Conclusion** – This information will be useful in defining the opportunity for sweet potato to contribute to micronutrient nutrition. Indeed, studies on organic acid digestibility can show how sweet potato organic acids are digested and made available for absorption for maximum nutritional impacts of sweet potato.

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## P36

### A high fat challenge activates the innate immune response in asthma

LG Wood<sup>1,2</sup>, ML Garg<sup>3</sup>, A Wood<sup>1</sup>, J Smart<sup>1</sup>, PG Gibson<sup>1</sup>

<sup>1</sup>School of Medicine and Public Health, University of Newcastle, NSW, 2308

<sup>2</sup>Respiratory and Sleep Medicine, Hunter Medical Research Institute, John Hunter Hospital, NSW, 2305

<sup>3</sup>Nutraceuticals Research Group, Biomedical Sciences, University of Newcastle, NSW, 2308

**Background** – Dietary fat has been shown to activate the innate immune response. As the innate immune response has been shown to cause asthma in some individuals, we hypothesised that a high dietary fat intake may exacerbate inflammation and contribute to worsening asthma.

**Objective** – To examine the effect of dietary fat on inflammation in asthma.

**Design** – Non-obese (BMI <30) subjects with asthma were randomized to receive a high fat/ high energy (AHIFHE) (n=8) or low fat/ low energy (ALoFLE) (n=10) food challenge. Non-obese healthy controls (n=10) also underwent a high fat/ high energy (CHIFHE) food challenge. Subjects on the AHIFHE and CHIFHE challenge consumed 200% daily energy requirement in 24 hours, including 50% energy from fat. Subjects on the ALoFLE challenge consumed 75% daily energy requirement in 24 hours, including 20% energy from fat. Clinical assessment and blood samples were collected at 0, 2, 3, 4 and 24 hours. Inflammatory markers, including plasma TNF $\alpha$ , CRP and IL-6, were analysed by high sensitivity ELISAs.

**Outcomes** – At 4 hours after the commencement of the food challenges, subjects on the AHIFHE challenge, had a significantly higher increase in plasma CRP concentrations, compared to subjects on both the ALoFLE and CHIFHE challenge (p= 0.021). In subjects on the AHIFHE challenge, there was also a significantly higher increase in plasma TNF $\alpha$  concentrations at 3 hours, compared to subjects on ALoFLE challenge (p = 0.034).

**Conclusion** – A high fat/ high energy intake causes an increase in systemic inflammation in subjects with asthma. This effect was not observed in healthy controls, suggesting that subjects with asthma are more susceptible to fat-induced inflammation.

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