

## Investigation of mechanisms of protein induced satiety: meal structure and protein content effects on gastric emptying and gut hormone release

M. Groenen<sup>1</sup>, C.L. Fyfe<sup>1</sup>, G. Holtrop<sup>2</sup>, G.W. Horgan<sup>2</sup>, C.L. Meek<sup>3</sup>, F. Gribble<sup>3</sup>, P. Morgan<sup>1</sup> and A.M. Johnstone<sup>1</sup>

<sup>1</sup>The Rowett Institute, University of Aberdeen, Foresterhill, Aberdeen, UK, AB25 2ZD, <sup>2</sup>Biomathematics and Statistics Scotland, Foresterhill, Aberdeen, UK, AB25 2ZD and <sup>3</sup>University of Cambridge, Cambridge, CB2 1TN, UK.

High protein diets have beneficial effects on satiety, particularly during energy deficit<sup>(1)</sup>, with growing evidence emphasising the role of the gut in the mechanisms of protein-induced satiety<sup>(2)</sup>. Nutritional factors such as protein type and amount and non-nutritional factors (meal structure and viscosity)<sup>(3,4)</sup> influence gastric emptying (GE) and thus the release of gut hormones related to acute satiety, such as Glucagon-Like Peptide (GLP)-1 and Glucose-dependent insulinotropic polypeptide (GIP). These hormones have been found to inhibit GE and have a negative effect on food intake via the central nervous system<sup>(5,6)</sup>. We examined the effect of meal composition (protein content; 15% or 30% of calories) and meal structure (solid or liquid) of an isocaloric meal on GE, release of the hormones GLP-1 and GIP, subjectively rated appetite, and interactions between these mechanisms of satiety. Ethics approval was obtained from the North of Scotland Research Ethics Committee and informed consent from participants was acquired.

The study was implemented as a 2 × 2 randomized, crossover design in healthy males (age 26–72). Each participant received, on separate days, 5 test meals relating to: control (egg yolk on toast) or either a high (30%) or low (15%) protein milk smoothie (liquid) or jelly (solid). GE data was acquired through isotope ratio mass spectrometry analysis of breath samples collected using the <sup>13</sup>C Octanoic Acid stable isotopic technique<sup>(7)</sup>. The data was fitted to the mkβ model presented in Ghooos *et al*<sup>(8)</sup> to acquire parameters for statistical analysis. Blood plasma was analysed; –5, 10, 20, 30, 45, 60, 90 and 120 minutes after eating, for GLP-1 and GIP using the MESCO scale technique in a clinical laboratory with concurrent self-reported hunger/satiety related ratings using a 100 mm visual analog scale. The different satiety ratings were combined to form one appetite score<sup>(9)</sup>. Data was analysed with ANOVA allowing for random variation between volunteers. Contrasts were used to reflect the experimental design.

A high protein liquid breakfast was the most effective in decreasing GE. Increased protein content decreased the duration of fast GE ( $t_{asc}$ ) ( $p = 0.03$ ). Similarly, liquid food, in addition to decreasing  $t_{asc}$  ( $p = 0.032$ ), also increases the initial delay (latency) in <sup>13</sup>CO<sub>2</sub> excretion ( $p < 0.001$ ). Both these parameters indicate slower passage of food. Analysis of the AUC for GLP-1 ( $p = 0.006$ ) confirmed a significant increase after ingestion of a high protein meal whereas GIP decreased ( $p = 0.02$ ). GLP-1 also displayed a negative interaction with appetite score ( $r = -0.30$ ,  $p = 0.043$ ). However, meal structure, not protein content, tended to decrease self-reported appetite ( $p = 0.073$ ) and high protein solid meals might contribute to a greater reduction in appetite than high protein liquid meals, suggesting meal structure is more important in self-reported satiety.

Protein content influenced mechanisms of protein induced satiety. Increased protein content decreased gastric emptying rate and increased plasma GLP-1. GLP-1 also interacted with appetite score. However, meal structure was of greater influence on self-reported satiety.

This was funded by The Scottish Government and the European Union Seventh Framework Programme (FP7/2007–2013) under grant agreement n° 266408 for the “Full4Health” grant.

1. Gilbert JA *et al.* (2011) *Nutr, Met and Cardiovascular Dis* **21**, B16–B31.
2. Janssen S & Depoortere I (2013) *Trends in Endocrin & Met* **24**(2), 92–100.
3. Brennan IM *et al.* (2012) *Am J of Physiology-Gastrointestinal and Liver Phys* **303**(1), G129–G140.
4. Camps G *et al.* (2016) *AJCN* **104**(1), 73–80.
5. Seino Y, Fukushima M & Yabe D (2010) *J of diabetes investigation* **1**(1–2), 8–23.
6. Valassi E, Scacchi M & Cavagnini F (2008) *Nutr, Met and Cardiovascular Dis* **18**(2), 158–168.
7. Lacroix *et al.* (1973) *Science* **181**(4098), 445–446.
8. Ghooos *et al.* (1993) *Gastroenterology* **104**, 1640–1640. 17.
9. Anderson *et al.* (2002) *AJCN* **76**(5), 1023–1030.