EFFECTS OF PROTEIN ON GASTROINTESTINAL FUNCTION AND APPETITE REGULATION

A thesis submitted by
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Abstract

The prevalence of obesity and associated diseases, including type-2 diabetes mellitus, continues to increase at an alarming rate. The available therapies have largely ignored the key role of the gastrointestinal tract in determining appetite and blood glucose regulation in responses to ingested nutrients. A detailed understanding of these gastrointestinal mechanisms is critical in aiding development of new and effective interventions for obesity.

The research presented in this thesis focuses on the complex gastrointestinal mechanisms involved in the regulation of glycaemia, appetite and energy intake in response to protein in lean and obese individuals. In particular, this research explores the gastrointestinal motor and hormonal responses to nutrients involved in energy intake regulation and blood glucose control in both healthy lean and obese individuals. Using the novel, non-invasive technique of 3-dimensional ultrasound, the study described in chapter 5 reports that, in lean individuals, the rate of gastric emptying of drinks containing 30g and 70g of protein was comparable (kcal/min; 30g: 2.6±0.2, 70g: 2.9±0.3), and within the ranges previously observed for fat and carbohydrate (1-4 kcal/min). This was reflected by similar releases of cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), glucose-dependent inhibitory polypeptide (GIP), insulin and glucagon, for ~45 min following the drinks. Beyond 45 min, the 70g load resulted in more sustained hormone release, reflecting greater total calories and thus prolonged delivery of nutrient to the small intestine. Energy intake was comparable between the two loads, suggesting that a threshold amount of protein may exist, beyond which no additional appetite-suppressive benefit occurs.

In the studies described in chapters 6-8, intraduodenal infusions, combined with high-resolution manometry, were used to evaluate the effects of nutrients in the small intestine on
antropyloroduodenal motility and gastrointestinal hormone release. Nutrients were infused directly into the duodenum at standardised rates, reflecting the normal range of gastric emptying; intraduodenal infusion bypasses orosensory and gastric influences, isolating the effects of nutrient to the small intestine.

The first of these studies reported that intraduodenal protein has load-dependent effects on antropyloroduodenal motility, ghrelin, CCK, GLP-1, peptide tyrosine tyrosine (PYY), insulin and glucagon, glycaemia, and energy intake at a subsequent meal in lean individuals. The second study reported that load-dependent effects of protein on antropyloroduodenal motility and CCK, GLP-1, GIP, insulin and glucagon release are also apparent in obese individuals, suggesting that small intestinal sensitivity to protein remains intact in obesity. The final study demonstrated, in lean individuals, that intraduodenal lipid modulates gastrointestinal motor responses and CCK and GLP-1 concentrations more potently than an equicaloric protein load. In contrast, protein had more pronounced effects on insulin and glucagon release. Despite these differences, protein and lipid suppressed energy intake comparably, suggesting that different mechanisms may underlie the suppression of energy intake by these nutrients.

These data provide novel insights into the roles that gastrointestinal motor and hormone responses to dietary protein play in the regulation of blood glucose, appetite and energy intake in lean and obese individuals. These observations provide potential mechanistic explanations for the effects of high-protein diets on glycaemic control, and appetite. Importantly, they provide a basis for future development of nutrition-based interventions for the treatment of obesity.
Declaration of Originality

I, Amy Hutchison, certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name for any other degree or diploma in any university or other tertiary institution without the prior approval of the University of Adelaide and where applicable, any partner institution responsible for the joint award of this degree.

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Amy Hutchison

November 2015
Publications Arising From This Thesis

The data presented in this thesis have formed the basis of the publications listed below:


Other Publications


Dedication

Ko te manu e kai ana i te miro, nōna te ngahere.
Ko te manu e kai ana i te mātauranga, nōna te ao.

The bird that partakes of the miro berry reigns in the forest.
The bird that partakes of the power of knowledge has access to the world.

This thesis is dedicated to
my husband, Rob,
for always inspiring and challenging me,
and my whānau,
for your unwavering love and support.
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The studies reported in this thesis were performed at the Discipline of Medicine, Royal Adelaide Hospital. While conducting this research, I was financially supported by a Faculty of Health Sciences PhD Scholarship.

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Thank to you all of my friends and family for your love and encouragement, especially those that have helped make our home Adelaide. Finally, the biggest thank you of all must go to Rob, my husband, for your unconditional love, support and encouragement over the years.
### List of abbreviations

<table>
<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>APD</td>
<td>Antropyloroduodenal motility</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
</tr>
<tr>
<td>ARC</td>
<td>Arcuate nucleus</td>
</tr>
<tr>
<td>AUC</td>
<td>Area under the curve</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>CCK</td>
<td>Cholecystokinin</td>
</tr>
<tr>
<td>CHO</td>
<td>Carbohydrate</td>
</tr>
<tr>
<td>CV</td>
<td>Coefficients of Variance</td>
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<tr>
<td>DPP-IV</td>
<td>Dipeptidyl peptidase-IV</td>
</tr>
<tr>
<td>EDTA</td>
<td>ethylenediaminetetraacetic acid</td>
</tr>
<tr>
<td>ELISA</td>
<td>Enzyme-linked immunosorbance assay</td>
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<tr>
<td>FFM</td>
<td>Fat free mass</td>
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<td>GI</td>
<td>Gastrointestinal</td>
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<td>GIP</td>
<td>Glucose-dependent insulinotropic peptide</td>
</tr>
<tr>
<td>GLP-1</td>
<td>Glucagon-like peptide-1</td>
</tr>
<tr>
<td>HbA1c</td>
<td>Glycated haemoglobin</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>Homeostatic model assessment of insulin resistance</td>
</tr>
<tr>
<td>ID</td>
<td>Intraduodenal</td>
</tr>
<tr>
<td>IPPW</td>
<td>Isolated pyloric pressure wave</td>
</tr>
<tr>
<td>LCD</td>
<td>Low calorie diet</td>
</tr>
<tr>
<td>MI</td>
<td>Motility index</td>
</tr>
<tr>
<td>MMC</td>
<td>Migrating motor complex</td>
</tr>
<tr>
<td>mV</td>
<td>Millivolt</td>
</tr>
<tr>
<td>NIDDM</td>
<td>Non-insulin dependent Diabetes Mellitus</td>
</tr>
<tr>
<td>NS</td>
<td>Not significant</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
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<td>--------------------------------------</td>
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<tr>
<td>PWs</td>
<td>Pressure waves</td>
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<tr>
<td>PYY</td>
<td>Peptide tyrosine tyrosine</td>
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<tr>
<td>RIA</td>
<td>Radioimmunoassay</td>
</tr>
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<td>Total Free Amino Acids</td>
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<td>TFEQ</td>
<td>Three Factor Eating Questionnaire</td>
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<td>TMPD</td>
<td>Transmucosal potential difference</td>
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