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Oral versus intubated feeding and the effect on glycaemic and insulinaemic responses, gastric emptying and satiety.

Morey S[a], Shafat A[b] and Clegg M. E[a]

[a] Functional Food Centre, Department of Sport and Health Sciences, Faculty of Health and Life Sciences, Oxford Brookes University, Gipsy Lane, Oxford OX3 0BP, UK

[b] Physiology, School of Medicine, National University of Ireland, Galway, Ireland

Corresponding author: Dr Miriam E Clegg, Functional Food Centre,
Department of Sport and Health Sciences, Faculty of Health and Life Sciences, Oxford Brookes University, Gipsy Lane, Oxford OX3 0BP, UK
Email: mclegg@brookes.ac.uk; Ph: +44 1865 484365

24 **Abstract**

25

26 Cephalic phase responses (CPR) are important in early initiation of digestion and maximal
27 absorption of nutrients prior to ingestion. Bypassing CPR has been shown to have consequences
28 on metabolic responses that may influence satiety. The aim of this study was to investigate if
29 using gastric intubation to bypass oro-pharyngeal and oesophageal exposure would reduce CPR
30 including insulin and blood glucose and whether these impact on gastric emptying and satiety.
31 Ten male subjects were tested on 2 occasions, 3-7 days apart after an overnight fast, in
32 randomized order. Subjects were cannulated and intubated with a gastric tube for both tests. For
33 test one, subjects ate 400ml soup with a spoon and for test two the soup was infused into the
34 stomach at an equivalent rate. Subsequently measurements of glycaemic (GR) and insulinaemic
35 responses (IR) from cannula samples, breath samples for measurement of gastric emptying using
36 the [^{13}C] sodium acetate breath test and visual analogue scales (VAS) for satiety were taken over
37 180 minutes. There were differences in IR over the first 15 minutes (Oral: 169.0 ± 22.1; Gastric
38 124.1 ± 18.8; $t(9)=2.67$; $p= 0.028$) but no difference in GR. There were differences in gastric
39 emptying half time (Oral: 85.0 ± 2.7 ; Gastric 79.4 ± 3.3; $t(9)= 2.40$; $p=0.04$) and ascension
40 time (Oral: 68.2 ± 2.2; Gastric 64.0 ± 2.2 ; $t(9)=2.57$; $p=0.03$) with food taking longer to empty
41 from the stomach on the Oral test day than on the Gastric test day. There was no significant
42 difference in the satiety ratings. This study demonstrated that bypassing oro-pharyngeal and
43 oesophageal exposure decreases the normal physiological CPR with detriment to IR and gastric
44 emptying.

45

46 **Keywords:** glycaemic response, insulin, gastric emptying, cephalic, satiety

47 **Introduction**

48

49 Oral ingestion of food and beverages provides a sensory experience including anticipation, sight,
50 smell, chewing and taste. Bypassing this route removes much of this sensory stimulation and
51 therefore reduces the normal physiological cephalic phase responses (CPR). These responses
52 assist in early initiation of digestion and maximal absorption of nutrients prior to ingestion [1-3].
53 One response of particular interest is the CPR secretion of insulin (CPIR) and its role within
54 glycaemic control [4-8]. Oral stimulation by food initiates fast release of insulin; that peaks
55 between 1-4 minutes returning to baseline within ten minutes, the consequence of which avoids
56 both peak levels of glucose release and subsequent gluconeogenesis and lipolysis [1, 3, 9].

57

58 Work by Teff [10] identified that CPIR caused a 30% reduction in plasma glucose post ingestion
59 of food and Ahren and Holst [6] found that blocking neural pathways for CPR caused a reduction
60 of 73% in CPIR, causing higher plasma glucose levels for longer following food ingestion.
61 Western diets currently contain more energy dense foods with less fibre [11]. These foods may
62 reduce oroosensory stimulation as they need less chewing and have a faster oral transit time [8].
63 This can result in a reduction in both CPRs and satiety [8, 12]. With the loss of oral sensory
64 stimulation and associated CPIR an individual's risk of hyperglycaemia and hyperinsulinaemia is
65 believed to be increased, and if this becomes a consistent metabolic profile there is a greater risk
66 of subsequent metabolic and cardio-vascular disease [12, 13]. To date, many of the studies have
67 focused on looking at the effect on glycaemic and insulinaemic responses directly following
68 sham feeding or oral stimulation feeding [14-16] for up 30 min but have not examined the

69 impact of the CPIR on the entire postprandial period to demonstrate if they would present greater
70 metabolic risk.
71
72 Smeets et al. [8] believes that a loss of adequate oral sensory signalling from dietary choices that
73 are more readily available in western society today are responsible for a reduction in satiety.
74 Earlier work by Cecil et al. [14] identified that it is a combination of early oro-sensory
75 stimulation with gastro-intestinal influences such as motility and distension that provides the
76 greatest sensation of satiety. Furthermore utilising techniques that bypass oral CPR was found to
77 increase rates of gastric emptying and decrease satiety [14]. Increasing rates of gastric emptying
78 is likely to impact on satiety as food will remain in the stomach for a shorter time, decreasing the
79 time that the stomach remains distended which could result in increased food consumption [14,
80 17].
81
82 Although there is much evidence that the loss of CPIR impacts on the metabolic profile of an
83 individual there is some contradiction within the evidence [3-6, 8, 13]. Only recently have
84 studies combining sensory signals and the implications on food consumption been undertaken
85 [16]. Smeets et al. [8] identifies a need for further work to examine the impact of sensory signals
86 on both short term metabolic pathways and satiety. Previous work has primarily focused on the
87 effect of CPR on satiety and gastric emptying [14, 15] or on the role of CPR on immediate
88 insulinaemic responses (IR) and glycaemic responses (GR) [16] but has not looked at the
89 responses of both over the entire postprandial period where it is more likely to have a metabolic
90 implication. This study therefore aimed to examine two components of CPR in combination,
91 which is lacking from the extant literature. The first component that was measured was the effect

92 of oral stimulation on IR and GR. The second component measured was changes in gastric

93 emptying and satiety.

94

95 **Materials and Methods**

96

97 *Subjects*

98 Twelve healthy males not suffering from diabetes or pre-diabetes were recruited for the study by

99 means of advertisements and personal communications. Volunteers were given full details of the

100 study protocol prior to giving their written informed consent. Ethical approval for this study was

101 provided by the Research Officer for Faculty of Health and Life Sciences, Oxford Brookes

102 University, UK, in accordance with the Declaration of Helsinki. All subject's fasting blood

103 glucose was <6.1mmol/l and their BMI was between 18.5 - 30kg/m^2. None of the subjects were

104 taking any medication that would interfere with glucose metabolism or insulin signalling. None

105 of the participants were smokers. Eating behaviour was determined using the Three Factor

106 Eating Questionnaire [18]. Only those who did not consciously restrain their food intake due to

107 psychological reasons, weight concerns and external stimuli were included in the study. All

108 subjects were asked to rate their liking of the soup on a scale of 1-10 with 1 being strongly

109 disliking it. All volunteers reported liking the soup and rated it as 7 or greater and hence were

110 included in the study.

111

112 *Study Design*

113 The study required volunteers to attend the lab on three separate occasions. The first was for

114 initial preliminary assessment and if volunteers were suitable to proceed with the study they were

115 required to attend for two subsequent test days; one consisting of oral ingestion of soup (Oral)
116 and one consisting of gastric infusion of the soup into the stomach via a feeding tube (Gastric).
117 The two tests days were carried out in random order, with a minimum of three, maximum seven
118 days between tests.
119
120 Subjects were requested to record their food intake in a weighed food diary the night before
121 attending for their first test and repeat this prior to their second test. They were requested to fast
122 from 22.00 the night before although were able to drink water. Subjects were also requested to
123 abstain from strenuous exercise and alcohol consumption the day before testing.
124
125 *Preliminary assessment*
126 Volunteers were requested to attend the lab after an overnight fast so a fasting blood glucose
127 measurement could be taken via a fingerprick blood sample (HemoCue 201+ glucose analyzer,
128 Angelholm, Sweden). A health questionnaire pertaining to food allergies and intolerances and
129 any known metabolic conditions or medication was requested. Baseline anthropometric
130 measurements of height, weight, body mass index (BMI) and blood pressure were undertaken
131 from each individual subject. These aimed to screen for any medical conditions or medication
132 that may interfere with glucose metabolism or insulin signalling; in which case the volunteer
133 would be declined from participating further within the study.
134
135 The preliminary test required volunteers to attempt insertion of an oral gastric feeding tube
136 (Vygon, 14 French Levine, length 125, Gastro-duodenal feeding tube, Ecouca, France). This
137 ensured volunteers could satisfactorily undertake the procedure and familiarise themselves with

138 the technique. Self-insertion of the tube makes the process less stressful for the volunteer as it
139 gives them control over the rate of insertion [19]. Tube insertion was required to a length of 50-
140 55 cm and subjects needed to retain the tube placement comfortably. For subjects having
141 difficulty, repeated attempts were not encouraged as this can impact on the gastric response.
142 Accurate placement of the tube was confirmed by inserting 100ml of water and using a 100ml
143 syringe to aspirate the gastric contents. Once 80% of this can be aspirated then the tube was
144 deemed to be in the stomach [20]. The gastric tube was then removed. Following preliminary
145 testing, two volunteers were unable to pass the oral gastric tube (gastric intubation) and
146 withdrew, leaving a subject group of 10 male participants (37.8±3.4 years; 1.77±0.03m;
147 75.4±4.2kg).
148
149 Subjects who were able to proceed with the study were then timed whilst ingesting 400ml of the
150 test soup orally in order to measure the rate of normal feeding. This allowed determination of an
151 individual flow rate in ml/min for gastric tube infusion of the soup using a syringe during the
152 Gastric test session.
153
154 *Experimental Test Protocol.*
155 Subjects attended the lab in the morning between 7-8am after an overnight fasting. On arrival,
156 cannulation of a superficial vein of the upper limb was undertaken to provide collection of blood
157 samples for glucose and insulin measurement throughout the test protocol. Samples were taken at
158 a baseline of – 5 minutes, following the onset of soup ingestion / infusion at 3 minute intervals
159 for first 15 minutes and then subsequently at 15 minute intervals for a total of 180 minutes.

160 Baseline assessment of satiety using 100mm visual analogue scale (VAS) and breath samples for
161 measurement of gastric emptying were also undertaken at -5 minutes.
162
163 Subjects inserted the oral gastric tube for both the Oral and the Gastric test in order to ensure
164 there was no physiological difference between circumstances of each test that may influence
165 findings. The oral gastric tube was removed by the subjects after completion of the first 15
166 minutes of the trial.
167
168 The 400ml of test soup was ingested orally by the subject or infused via the oral gastric tube at
169 the rate of normal eating as determined from the pre-test trial. The test meal and quantity were
170 based on previous similar studies [14, 15]. Timing of each test trial began at the point of
171 initiating food eating / infusion. The test soup (Campbell's Cup Soup, Cream of tomato, Leeds,
172 UK) contained 25g of available carbohydrate and had 100mg of [^{13}C] labelled sodium acetate
173 added. This is a naturally stable carbon isotope which is rapidly absorbed and oxidised within the
174 liver to form labelled CO_2 which subjects then exhale. The soup contained 170 kcal, 2.5g
175 protein, 28.3g total carbohydrate, 4.9 g fat and 0.7g sodium. The soup was prepared by adding
176 the soup powder to 300 ml of boiling water, 100 ml of water at room temperature and serving or
177 intubating immediately.
178
179 *Gastric emptying*
180 Collection of breath via a straw tube into a glass vial (10ml Exetainer, Labco, Bucks, UK) allows
181 analysis at a later stage as an indicator of the rate of gastric emptying. Work by Braden et al. [21]
182 identified that this non-invasive method is reliable, safe and cost-effective for measuring rates of

183 gastric emptying. Subject's breath samples were taken at baseline of -5 minutes, postprandial at
184 3,6,9,12,15 minutes and then every 15 minutes until 180 minutes. Samples were then analysed
185 using isotope ratio mass spectrometry (ABCA, SerCon Limited, Crewe, UK) and results were
186 expressed relative to V-PDB, an international standard for known ^{13}C composition. For breath
187 $^{13}CO_2$ levels using 130 known standard samples, the coefficient of variation across these samples
188 has been shown to be 0.0044% [22]. $^{13}CO_2$ values were expressed as the excess amount in the
189 breath above baseline and converted into moles. Data are then displayed as percentage of $^{13}CO_2$
190 dose recovered per hour and cumulative percentage $^{13}CO_2$ recovered over time. CO_2 production
191 was assumed to be 300 mmol/m^2 body surface area per hour. Body surface area was calculated
192 using a validated weight-height formula [23]. This was then fitted to a gastric emptying model
193 developed by Ghoos et al., [24]. For all the data, r^2 coefficient between the modelled and raw
194 data was calculated and $r^2>0.95$. From this model several parameters were measured. Lag phase
195 and half time were calculated using the formulae derived by Ghoos et al., [24]. Lag phase is the
196 time taken to maximal rate of $^{13}CO_2$ excretion [25] and is equivalent to the time of the inflection
197 point [26]. Half time is the time it takes 50% of the ^{13}C dose to be excreted [25]. Latency phase
198 [26] is the point of intersection of the tangent at the inflection point of the $^{13}CO_2$ excretion curve
199 representing an initial delay in the excretion curve. Ascension time [26] is the time course
200 between the latency phase and the half time representing a period of high $^{13}CO_2$-excretion rates.
201
202 *Blood glucose*
203 Blood samples were used to test for blood glucose and insulin at each time point. Blood glucose
204 was measured using the HemoCue® 201+ Glucose analyzer (HemoCue Ltd, Dronfield, UK). The
205 HemoCue® is a reliable method of blood glucose analysis [27]. The laboratory's CV for 20 or

more duplicate measurements of fasting glucose (i.e. minute-to-minute variation in human subjects) was <5%. The inter-assay CV (i.e. analytical variation) on standard solutions was <3.6%.

Insulin

At each time point, 6 mL was collected into blood collection tubes treated with di Potassium EDTA (BD vacutainer, Oxford, UK) and immediately stored in crushed ice. Samples were then centrifuged 4000rpm, 4 ºC, for 10 minutes. Plasma was removed and stored at -40 ºC until analysis. Insulin concentrations in the plasma samples were determined by electrochemiluminescence immunoassay using an automated analyzer (Cobas® E411; Roche diagnostics, Burgess Hill, UK). The Cobas® system is a reliable method of blood insulin determination [28]. The unit of measurement was µU/ml.

On completion of the 180 minutes testing, the subject's intra–venous cannula was removed.

Visual analogue scales

Throughout the test trials 100mm visual analogue scale (VAS) were utilised by each subject; at baseline, 6, and 15 minutes and then every subsequent 15 minutes for a total of 180 minutes in order to gain some comparison between oral ingestion and gastric infusion on their desire to eat and level of satiety. Each time point required subjects to make a vertical mark across the horizontal VAS line with anchor points of 'not at all to 'extremely' for specific questions to rate their level of hunger, fullness and desire to eat and anchor points of 'nothing at all' to 'large

228 amount' for how much food they thought they could eat. Use of VAS as a reliable measure of
229 subjective appetite and predictability of feeding behaviour is validated by Sorensen et al. [29].
230
231 *Statistical analysis*
232 Results of blood glucose, insulin and VAS data were converted to reflect the change in GR, IR
233 and VAS respectively by subtracting the baseline value from those taken at set time points. It
234 was this change response value that was then used within all subsequent analysis. Incremental
235 area under the curve (IAUC) using the trapezoidal rule [30] was calculated for all GR, IR and
236 the four parameters of the VAS. GR and IR IAUC were calculated for the first 15 min of the test,
237 the first 60 min of the test and the entire 180 in of the test. Statistical analysis was undertaken
238 using Statistical Package for Social Sciences (SPSS, version 19.0, USA). Mean differences
239 between Oral and Gastric IAUC for total GR, IR and the four parameters of the VAS were
240 analyzed using paired sample t-test and the effect size as calculated using Cohen's *d*. Paired
241 sample t-test was also used for comparison of gastric emptying times. Results are expressed as
242 means ± standard error (SE) unless otherwise stated and significance was defined as $p<0.05$.
243
244 **Results**
245
246 *Glycaemic Response*
247 During the 180 minute test the GR peaked at 30 minutes in both Oral (2.35±0.25 mmol/L) and
248 Gastric (2.76±0.35 mmol/L) tests (Figure 1). In both tests, this peak was followed by a rapid
249 decline in glucose concentration with the nadir occurring at 75 minutes in the Oral test (-

250 1.25±0.19 mmol/L) and in the Gastric test (-1.41±0.29 mmol/L). There was no significant
251 difference in the peak GR between the tests (t(9)=1.13; p=0.29; *d*=0.50).
252
253 There were no significant differences in GR IAUC following either 180 min (Oral: 71.3±9.5
254 mmol/L·min; Gastric 79.7±12.7 mmol/L·min; t(9)=0.68; p=0.51; *d*=0.24), 60 min (Oral:
255 64.9±9.5 mmol/L·min; Gastric 74.7±10.4 mmol/L·min; t(9)=0.78; p=0.45; *d*=0.04) or 15 min
256 (Oral: 6.0±1.2 mmol/L·min; Gastric 6.1±1.7 mmol/L·min; t(9)=0.11; p=0.91; *d*=0.31).
257
258 *Insulinaemic response*
259 Over 180 minutes the IR (Figure 2) peaked at 30 minutes in both Oral (51.8±8.1 uU/ml) and
260 Gastric (62.9±8.1 uU/ml) tests with a nadir at 165 minutes for Oral (-0.5±0.5 uU/ml) and at 135
261 minutes for Gastric (-1.2±0.3 uU/ml). There was no significant difference in the peak IR
262 between the tests (t(9)=1.23; p=0.25; *d*=0.38).
263
264 There were no significant differences in IR AUC following 180 min (Oral: 1897.7±309.2
265 uU/ml·min; Gastric 2037.5±205.9 uU/ml·min; t(9)=0.61; p=0.56; *d*=0.19) or 60 min (Oral:
266 1638.7±262.0 uU/ml·min; Gastric 1870.5±196.8 uU/ml·min; t(9)=1.10; p=0.30; *d*=0.36) but
267 there was a difference after 15 min, with the Oral test causing the greater IR (Oral: 169.0±22.1
268 uU/ml·min; Gastric 124.1±18.8 uU/ml·min; t(9)=2.67; p=0.028; *d*=0.63).
269
270 *Gastric emptying*
271 There were significant differences in gastric emptying times between the meals for half time
272 (t(9)= 2.40; p=0.04) and ascension time (t(9)=2.57; p=0.03) but not for latency or lag phase

273 (p>0.05). The food took ~4-5 min longer to empty from the stomach on the Oral test day than on
274 the Gastric test day (Table 1)
275
276 *Satiety*
277 There were no significant differences in satiety ratings following the two different feeding
278 methods for any of the parameters hunger (t(9)=0.38; p=0.71), fullness (t(9)=0.96; p=0.36),
279 desire to eat (t(9)=1.60; p=0.15) and prospective consumption (t(9)=0.68; p=0.52) (Table 2;
280 Figure 3).
281
282 **Discussion**
283
284 This study was the first to examine the entire IR and GR postprandial profile in combination with
285 GR and satiety following Oral and Gastric feeding. The study demonstrated no significant
286 differences between oral or intubated feeding on GR however the Oral method of feeding
287 resulted in a greater IR over the first 15 minutes. There were also no significant differences
288 found between Oral or Gastric feeding on satiety but gastric emptying was significantly
289 accelerated by 4-5min on the Gastric test in comparison to the Oral test.
290
291 In comparing oral ingestion with intubated feeding it was hypothesised within the present study
292 that bypassing oro-pharyngeal and oesophageal exposure would decrease the CPIR [31].
293 Findings from this present study based on the first 15 min of IAUC insulin data appear to
294 replicate the characteristic early CPIR profile reported in previous work from oral ingestion of
295 food [10]. However there were no differences in GR between the oral ingestion compared with

296 intubated feeding. Early CPIR occurs to a peak within the initial 1- 4 minutes after gustatory
297 stimulation returning to baseline within ten minutes [1-3, 9, 32]. Although overall it is a minimal
298 rise in insulin concentration levels compared to those secreted postprandially (5uU/ml [5] for
299 cephalic response compared to a postprandial response that could be ~60 uU/ml (current study)),
300 it is believed to increase digestive secretions, decrease gut motility and decrease food intake [1-
301 3].
302
303 The effects of CPIR on blood glucose appear to differ. Findings in a study by Teff et al. [32]
304 identified that in normal weight healthy males, 4 minutes post ingestion of food there was a
305 significant increase in insulin and also a significant drop in plasma glucose as a result of early
306 CPIR. However as in the current study, this change in GR has not been replicated in all studies
307 perhaps due to difficulty in measuring such small variations in blood samples [5, 8]. Ranawana
308 et al. [33] also identified high variability in glucose absorption between individuals even after
309 eating the same food. This presents some evidence that within-individual variance is also a
310 possibility even in a controlled methodology which may account for the lack of differences seen
311 here. A further consideration is that simultaneous release of glucagon during CPR may prevent a
312 reduction in blood glucose levels caused by CPIR [5] but without measuring glucagon levels this
313 is an unknown factor in the present study.
314
315 Other reasons for disparity within the results of studies on CPR maybe palatability and the
316 duration of oral transit time [8]. To date there is mixed findings as to whether palatability
317 directly impacts vagally activated pathways to increase concentrations of CPIR; which may
318 influence study findings if there is inconsistency within the subject group [9, 29, 34]. However

319 within the present study an initial questionnaire undertaken identified that none of the subjects
320 had a dislike for the tomato soup, utilised. Expert opinion is also divided in relation to the effect
321 the texture or form of food choice may have on oral exposure time. Teff [34] believes there is a
322 lack of CPIR to liquid stimuli; suggesting that chewing is required for adequate vagal stimulation
323 for insulin secretion. However, Cecil et al. [14, 15, 35] validate the use of liquid soup for
324 initiating CPR where they found the influence of sight, smell, and taste played an important part
325 in stimulating both pancreatic and gastric secretions as well as influencing appetite regulatory
326 centres. Cassady et al. [36] and deGraaf [12] also identify that liquid such as soup when eaten
327 with a spoon extends oro-sensory transit time to increase both CPR and satiety. A final
328 explanation for the differences in results seen between studies may come from work by Cecil et
329 al. [15], who found that the macronutrient content of the soup plays an important role as high fat
330 soup suppressed hunger, induced fullness, and slowed gastric emptying more than the high-
331 carbohydrate soup when ingested orally but there was no differences between the soups when
332 they were given intragastically.
333
334 One of the main aims of the current research was to combine satiety and metabolic responses
335 within the one study. Oro-sensory stimulation has been shown to increase the secretion of gastro-
336 intestinal peptides, these peptides slow gastric emptying [5, 34]. The delay in gastric emptying
337 increases satiety by prolonging stomach distention [8, 14, 31]. The present study hypothesised
338 that bypassing oro-pharyngeal and oesophageal exposure would reduce levels of satiety by
339 reducing the insulinaemic response and accelerating gastric emptying. Bypassing oro-pharyngeal
340 and oesophageal exposure did result in a slight acceleration in gastric emptying and increased
341 insulinaemic response however it was not possible to detect any differences from this in satiety.

342 A recent study [16] was able to confirm that gastric infusion of nutrients induced greater appetite
343 ratings than ingestion, alongside increases in satiety hormones however they were unable to
344 detect changes in food intake. Cecil et al. [14, 15, 35] also utilised gastric intubation in a series of
345 studies assessing the impact of bypassing oro-pharyngeal stimulation on satiety and gastric
346 emptying. They identified that loss of oro-sensory stimulation impacted negatively on both
347 satiety and gastric emptying, with subjects feeling fuller earlier with greater suppression of
348 hunger when food was eaten orally. The present study failed to find significant differences
349 between the tests for satiety; this may be due to the gastric emptying changes of only 4-5 min,
350 which although significant may not be sufficient to decrease satiety. It may also be due to the
351 large variability on the VAS data. However it should be noted that VAS assessments are only a
352 measure of perceived hunger not actual food intake and an ad libitum test meal would have
353 provided objective assessment of subsequent food intake [36, 37]. Although other studies have
354 been able to identify changes in VAS with oral stimulation using a similar sample size they have
355 not been able to detect changes in actual food intake [16]. An increased sample size however is
356 hindered by the invasive nature of the intubation test procedure.
357
358 **Conclusion**
359
360 In conclusion this study was able to demonstrate that utilising an oral gastric tube for infusion of
361 food to bypass oro-pharyngeal and oesophageal exposure decreases the normal physiological
362 CPR with detriment to IR and marginal accelerations in gastric emptying but was unable to
363 demonstrate any impact on satiety and GR. Potential future research could use a solid test meal
364 and include an ad libitum test meal.

Conflict of Interest Statement: There are no conflicts of interest associated with this manuscript

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388

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390

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481 **List of figures:**

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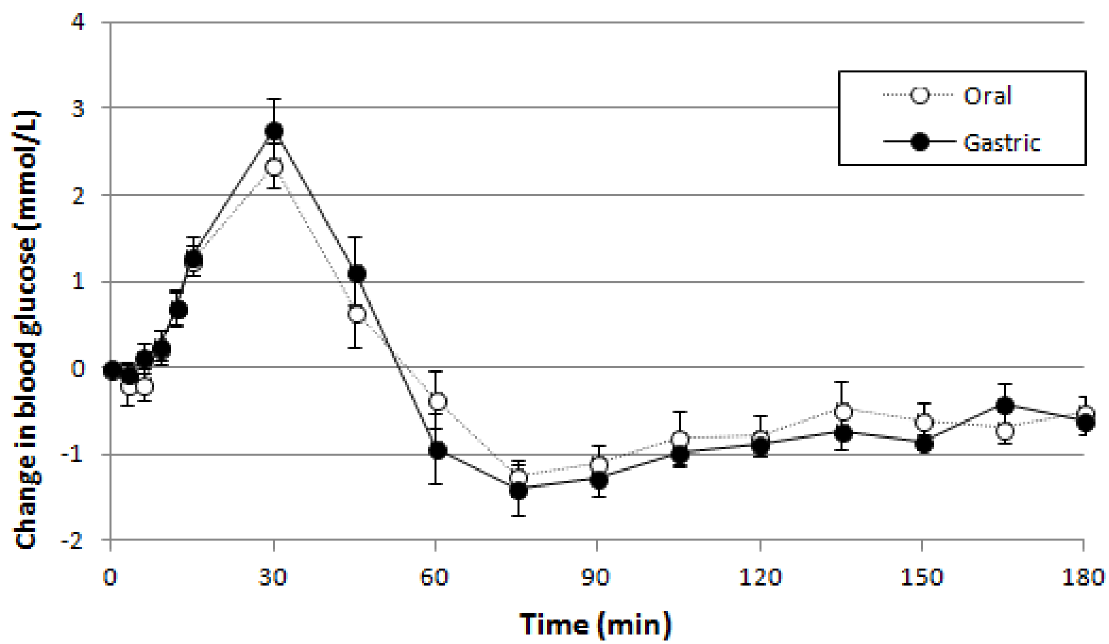
483 Figure 1. Change in blood glucose response (mean ± standard error) following Oral and Gastric

484 tests over 180 minutes (a) and over 15 minutes (b).

485 a)



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487 b)

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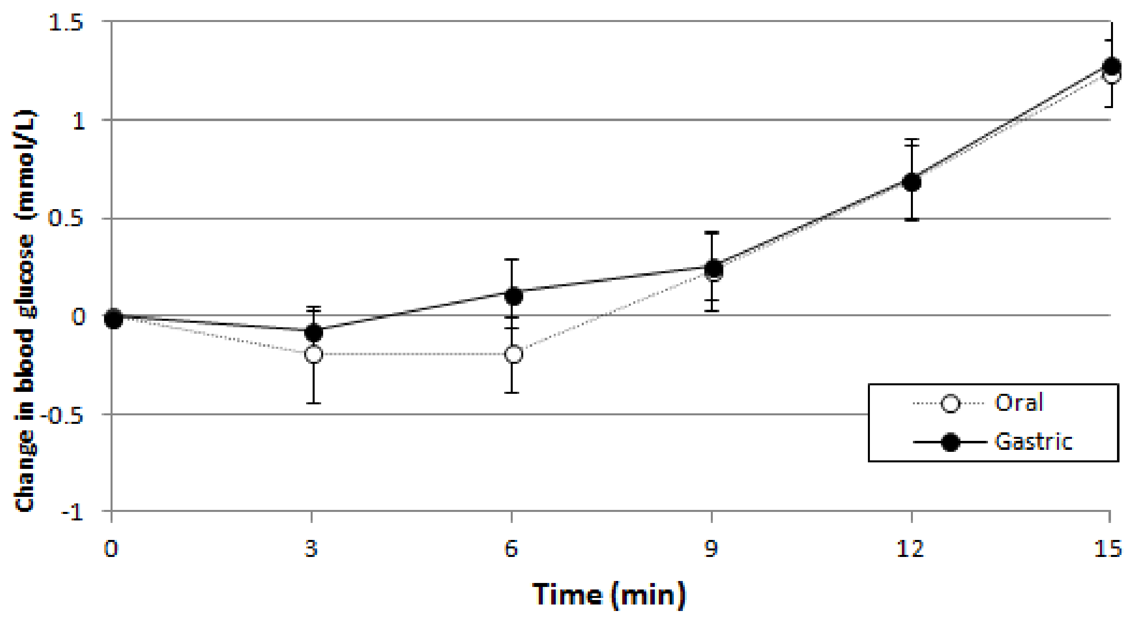

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509 Figure 2. Change in insulinaemic response (mean ± standard error) following Oral and Gastric

510 tests over 180 minutes (a) and over 15 minutes (b).

511 a)

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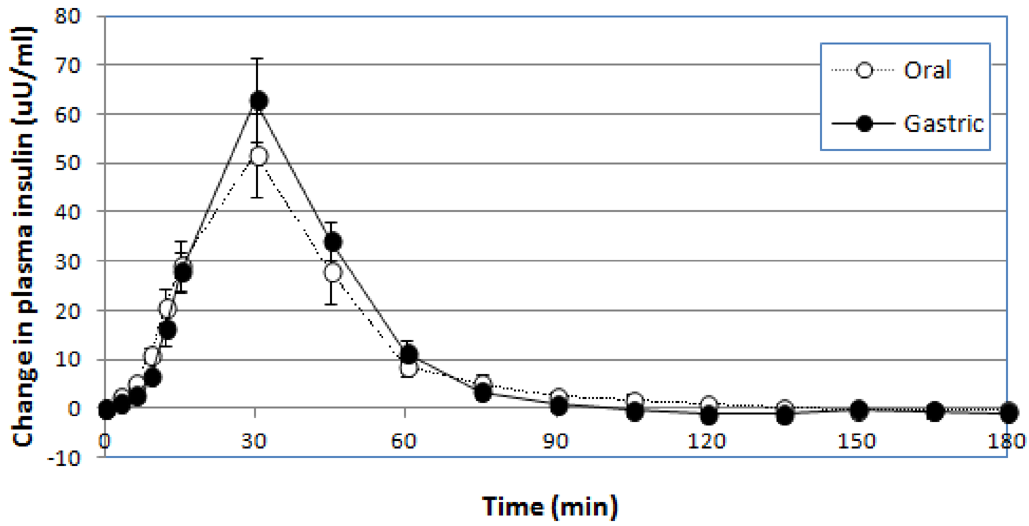

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514 b)

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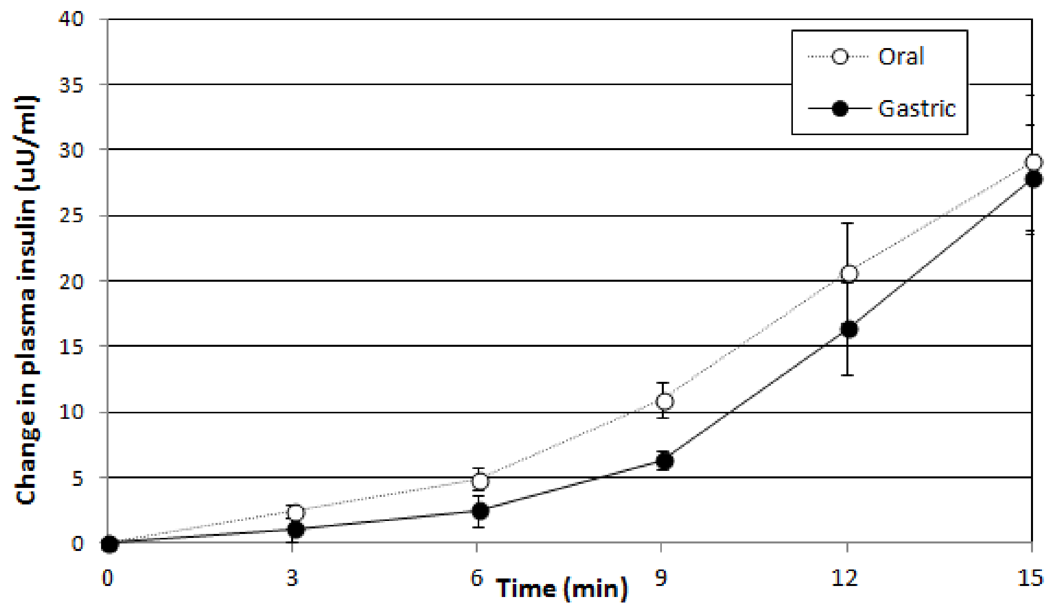

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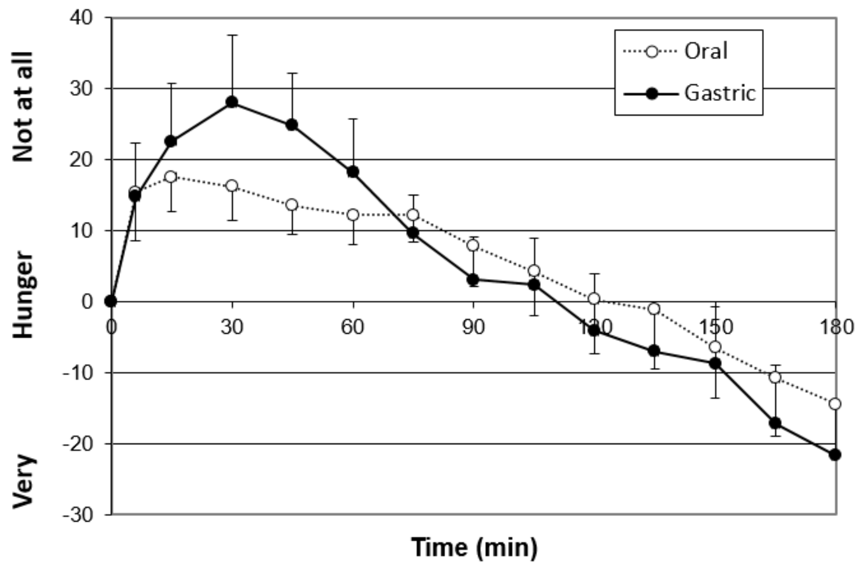
518 Figure 3. Change in satiety ratings from visual analogue scales (mean ± standard error) following

519 Oral and Gastric tests for hunger (a), fullness (b), desire to eat (c) and prospective consumption

520 (d).

521 a)



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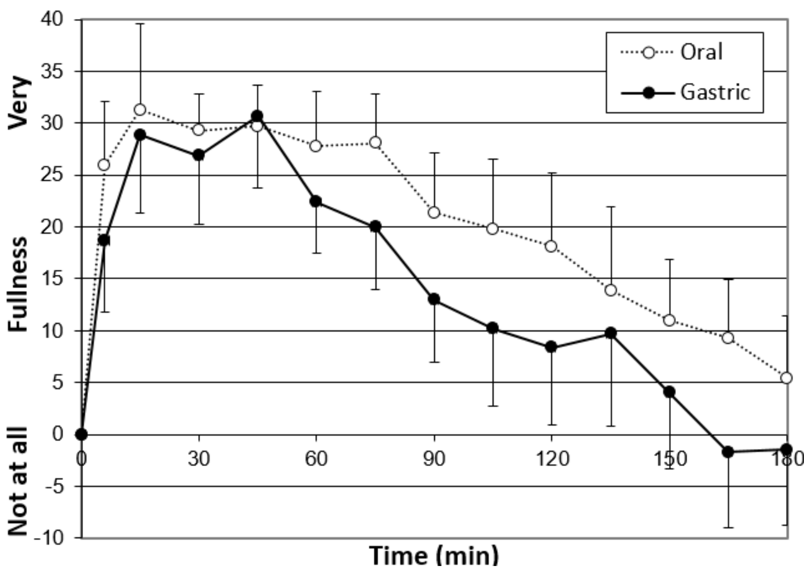
523 b)



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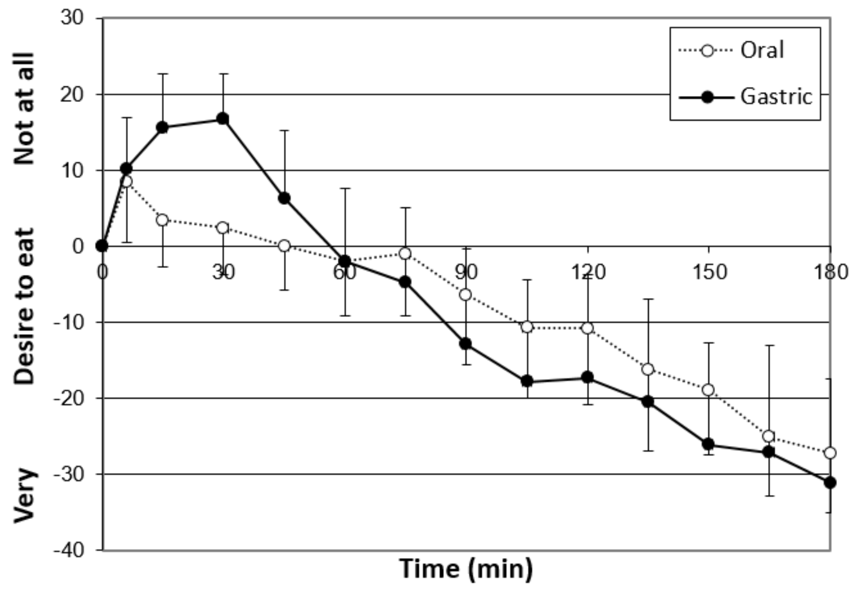
526 c)



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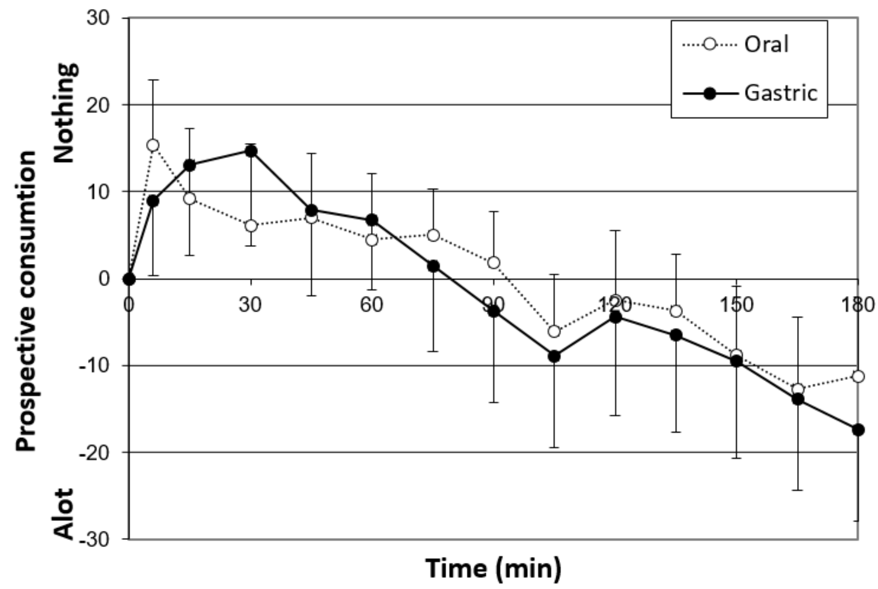
528 d)



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534 **List of Tables**

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536 Table 1: Gastric emptying times (mean ± standard error) following Oral and Gastric infusion of

537 soups. *=p<0.05

Time	Oral	Gastric	*d*
Latency Phase (min)	16.8 ± 0.7	15.4 ± 1.3	0.44
Lag phase (min)	52.4 ± 1.8	48.4 ± 2.8	0.55
Half time (min)	85.0 ± 2.7	79.4 ± 3.3*	0.59
Ascension time (min)	68.2 ± 2.2	64.0 ± 2.2*	0.60

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554 Table 2: Satiety rating, hunger, fullness, desire to eat and prospective consumption (mean ±

555 standard error) following Oral and Gastric infusion test.

	Oral	**Gastric**	***d***
Hunger (mm.min)	1914 ± 489	2298 ± 731	0.20
Fullness (mm.min)	3964 ± 711	3330 ± 808	0.26
Desire to eat (mm.min)	1149 ± 712	2050 ± 802	0.38
Prospective consumption (mm.min)	1680 ± 621	2233 ± 848	0.24

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