

An ionic-gelling alginate drink attenuates postprandial glycaemia in males

HARDEN, Charlotte, RICHARDSON, J Craig, DETTMAR, Peter W, CORFE, Bernard M and PAXMAN, Jenny <<http://orcid.org/0000-0003-3596-489X>>

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1 **AN IONIC-GELLING ALGINATE DRINK ATTENUATES**
2 **POSTPRANDIAL GLYCAEMIA IN MALES**

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4 **Charlotte J. Harden ^a, J. Craig Richardson^b, Peter W.**
5 **Dettmar^b, Bernard M. Corfe^{c*}, Jenny R Paxman^a**

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7 ^a Centre for Food Innovation, Sheffield Hallam University,
8 Howard St, Sheffield, S1 1WB, UK.

9 ^b Technostics Limited, The Deep Business Centre, Tower
10 Street, Kingston Upon Hull, HU1 4BG, UK.

11 ^c Department of Oncology, The University of Sheffield,
12 The Medical School, Beech Hill Road, Sheffield, S10 2RX,
13 UK.

14

* Corresponding author: Dr Bernard Corfe, Department of
Oncology, The University of Sheffield, The Medical
School, Beech Hill Road, Sheffield, S10 2RX, UK,

E-mail address: b.m.corfe@sheffield.ac.uk

Telephone: +44 (0)114 271 3004

Fax: +44 (0)114 271 3314

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15 **ABSTRACT**

16 Obese individuals are at increased risk of type 2 diabetes
17 compared to their healthy weight counterparts. Dietary
18 fibre, such as alginate, could attenuate glycaemic
19 disturbances associated with obesity when included in
20 the diet.

21 Forty self-reported, healthy males completed this
22 randomised, single-blinded, controlled, parallel trial to
23 determine the glycaemic response to a controlled test-
24 lunch of mixed composition following an ionic-gelling
25 alginate preload drink compared to an acidic-gelling
26 control.

27 Individual baseline area under the curve was 52% lower
28 ($P=0.010$) and peak glycaemia was 14% lower ($P<$
29 0.0005) after the ionic-gelling alginate drink compared
30 with the control. Body fatness was a predictor of
31 postprandial glycaemia however there was no interaction
32 effect between body fat % and treatment type.

33 We have shown ionic-gelling alginate can attenuate
34 glycaemic response to set lunch of mixed composition.
35 Functional foods that include ionic-gelling alginates may
36 benefit those with elevated postprandial blood glucose.

37 **KEY WORDS**

38 Alginate; glucose; glycemia; gel; body fat

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40 **1.0 INTRODUCTION**

41 As obesity increases, the incidence of associated co-
42 morbidities rises concomitantly, most dramatically in
43 relation to body mass index-related diabetes (McPherson
44 et al., 2007). Abdominal fatness has been linked with
45 elevated fasting blood glucose (Rezende et al., 2006).
46 Pascot et al. (1999) showed visceral adipose tissue
47 accumulation was accompanied by increased plasma
48 glucose in the fasted state and after a 75g oral glucose
49 load in young and middle aged women. In a six year
50 prospective study Kriketos et al. (2003) showed baseline
51 body fatness and increasing fatness over time to be
52 strong predictors of elevated fasting plasma glucose in
53 individuals 'at-risk' of type 2 diabetes
54
55 Epidemiological evidence suggests dietary fibres may
56 have a preventive role in the development of type 2
57 diabetes (Meyer et al., 2000). Several mechanisms by
58 which soluble fibres may modulate glycaemic response
59 have been proposed (Augustin et al., 2000). Soluble fibre
60 ingestion reduces carbohydrate digestion rates, therefore
61 aiding regulation of postprandial glycaemia (Augustin et
62 al., 2000; Kimura et al., 1996; Welch, 1994).

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63 Soluble fibres have been shown to have beneficial effects
64 in controlling glycaemia following carbohydrate ingestion
65 in healthy volunteers (Goñi et al., 2000; Rigaud et al.,
66 1998; Lavin and Read, 1995). Similarly, fibre-rich foods
67 (Flammang et al., 2006) and fibre supplementation
68 (Sierra et al., 2002) have been shown to help attenuate
69 postprandial glycaemic responses in type 2 diabetic
70 adults. Kaline et al. (2007) reviewed the potential
71 mechanisms by which diets rich in dietary fibre can be
72 useful in diabetes prevention.

73 Alginate is an algal polysaccharide found in the cell walls
74 of certain brown seaweed species. This fibre has been
75 used in several relevant human intervention studies. 5.0g
76 of sodium alginate added to a meal significantly
77 attenuated postprandial glycaemic response in type 2
78 diabetics by 31% compared to the control meal
79 (Torsdottir et al., 1991). Wolf and colleagues (2002)
80 demonstrated that 1.5g of sodium alginate, incorporated
81 into a 100g glucose-based preload drink with an acid-
82 soluble calcium source (to produce an acid-induced
83 viscosity complex), elicited a non-significant drop in peak
84 glycaemia and a significant attenuation of incremental
85 change from baseline area under the curve (AUC) in
86 healthy, non-diabetic adults compared to a soluble fibre-
87 based control. Williams et al. (2004) fed a "crispy bar"

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88 containing 5.5g guar gum and 1.6g sodium alginate to
89 healthy adults and measured the resultant glycaemic
90 response compared to an alginate-free bar. Postprandial
91 blood glucose excursions were significantly lower at 15,
92 30, 45, and 120 minutes and the positive incremental
93 AUC was significantly reduced (by 33%) after
94 consumption of the enriched “crispy bar” compared to the
95 alginate-free bar. Paxman et al. (2008a) reported a
96 strong positive correlation between change from
97 individual baseline AUC glycaemia and body fat % when
98 a hypromellose control preload was ingested prior to a
99 test lunch. This positive correlation was not apparent
100 following an ionic gelling sodium alginate preload,
101 providing preliminary evidence to suggest that the
102 enhanced glycaemic response to a meal at higher body
103 fat could be normalised following ingestion of an alginate
104 preload identical to the one used in the present study.

105 Hoad et al. (2004) fed volunteers a strong gelling (high-G)
106 and a weaker gelling (low-G) alginate meal, a guar-based
107 meal or a control (without added fibre) and examined the
108 resultant gastric emptying rates. *In vitro*, both alginate
109 meals formed intragastric gel 'lumps', and in the case of
110 the strong-gelling alginate, this was reportedly associated
111 with a feeling of fullness and a reduction in hunger. Hoad
112 and colleagues (2004) purport that acid-gelling agents

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113 such as alginate may be usefully incorporated into
114 weight-reducing diets/ foods in order to enhance antrum
115 distension and/ or manipulate nutrient uptake from the
116 ileum.

117 Alginate is widely used in the food industry as a thickener,
118 stabiliser and gelling agent (Brownlee et al., 2005). Its
119 constituent sugar residues are D-mannuronic (M) and L-
120 guluronic acid (G). Homopolymeric G blocks (comprising
121 diaxial linkages in the 1C_4 conformation) can react with
122 Ca^{2+} and H^+ ions to yield a strong, cross-linked gel
123 (Brownlee et al., 2005; Seal and Mathers, 2001; Kimura
124 et al, 1996). Consequently, the gel strength of alginate
125 and its consequent biochemical and biophysical
126 properties are determined by its chemical structure.

127 Specific alginates and specific alginate formulations are
128 therefore likely to react differently within the
129 gastrointestinal milieu.

130 The primary objective of the present study was to
131 examine the effect of alginate gelled ionically compared
132 to acidically (control) on glycaemic response to a
133 standard meal of mixed composition. Secondary to this,
134 we investigated how body fatness affects the postprandial
135 glycaemic response when subjects ingest the ionic-
136 gelling formulation compared to the acid-gelling control.

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139 **2.0 MATERIALS AND METHODS**

140 **2.1 Subjects**

141 41 male subjects participated in the study. Only one
142 subject was excluded, due to unusually low fasting
143 glucose levels, leaving complete datasets for 40
144 participants. Subjects aged 18 to 65 years were eligible
145 to take part providing they did not meet any of the criteria
146 for exclusion which were; type 1 or 2 diabetes, history of,
147 or current cardiovascular complaints (or if they had been
148 fitted with a pacemaker or other implantable electronic
149 device) or gastrointestinal complaints (such as irritable
150 bowel syndrome or inflammatory bowel disorder,
151 dumping syndrome or Cushing's syndrome), current fibre
152 supplement use, use of constipation-causing drugs such
153 as codeine or morphine, bowel blockage, bowel muscle
154 weakness or recent food poisoning. In addition, anyone
155 with a known allergy to, or intolerance of, the foods or
156 ingredients used in the experiment was excluded from
157 taking part, as were vegans (due to the nature of the
158 foods used).

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159 Baseline pre-screening took place less than one week
160 prior to the experimental phase, in which subjects
161 completed a general health questionnaire and various
162 anthropometric measures were made. Height and weight
163 were recorded (SECA 709 mechanical column scales
164 with SECA 220 telescopic measuring rod; SECA United
165 Kingdom, Birmingham) and body mass index (BMI) was
166 calculated. Bioelectrical impedance analysis was
167 undertaken following 5 minutes of supine rest on non-
168 conducting foam matting using a BodyStat 1500
169 (BodyStat Ltd., Isle of Man, British Isles). Body fat % was
170 recorded. Subjects completed a 51-item Three Factor
171 Eating Questionnaire (TFEQ; Stunkard and Messick,
172 1985) to determine eating behaviour across three pre-
173 defined factors. Mean values for all three factors;
174 restraint, disinhibition and hunger, were low for the group
175 as a whole (Stunkard and Messick, 1985). Subject
176 characteristics are reported in Table 1. This study was
177 approved by the relevant University Ethics Committee
178 (Ref: FIRC/2006/RE21). All subjects gave informed
179 consent to participate.

51 52 **2.2 Study Design** 53

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181 In this randomised, single-blinded, controlled parallel trial
182 subjects ($n = 40$) were split equally either side of the

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183 median into haptiles by body fatness (lower body fat
184 group: <16.10%, upper body fat group: ≥16.10%).

185 Following a 12 hour overnight fast, all subjects consumed
186 a controlled breakfast at 9am (60g Kellogg's® Hint of
187 Honey Corn Flakes; Kellogg's Company GB Limited,
188 Manchester, 125ml semi-skimmed milk and 200ml 'Drink
189 Fresh' orange juice; DCB Foodservice, Herts). After
190 breakfast subjects were asked to travel to the laboratory
191 using motorised transport to minimise energy expenditure.
192 From breakfast until 11am subjects consumed only
193 bottled spring water (Highland Spring still natural mineral
194 water with a sports cap, 2 x 500ml; Highland Spring Ltd,
195 Perthshire, Scotland) to a maximum volume of 1 litre.
196 Water consumption was *ad libitum* but the bottles were
197 weighed prior to the experiment and at 11am in order to
198 determine the exact amount consumed before the test-
199 lunch. Upon arrival at the facility for the experimental day,
200 subjects were randomly allocated to one of two preload
201 treatments; an ionic-gelling sodium alginate formulation
202 (SA) or an acid-gelling excipient free control (EF).

203 **2.3 Preload Formulations and Glycaemia**

204 The SA formulation contained sodium alginate, calcium
205 carbonate (CaCO₃) and buffering agents. It was
206 specifically formulated to undergo enhanced ionic

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207 intragastric gelation upon ingestion. This is achieved by
208 mixing sodium alginate with an acid soluble calcium salt.
209 Post-ingestion solubilisation of calcium salt in acidic
210 gastric fluid liberates free calcium ions which are then
211 available to cross-link with the sodium alginate. The SA
212 formulation has been described in detail by Paxman et al.
213 (2008b). The EF control is identical in composition to SA
214 with the omission of the CaCO₃. This formulation yields a
215 gel via acid gelation (in the absence of calcium), resulting
216 in weaker intra-molecular hydrogen bonded mass.

217 Prior to preload ingestion, baseline glycaemia (11:45am,
218 0 minutes) was determined using capillary blood taken
219 from the finger. A single use Accu-check® Softclix® Pro
220 lancing device was used to obtain a single droplet sample
221 via OneTouch® Ultra® Test Strips with FastDraw™
222 design. The OneTouch® Ultra® Blood Glucose
223 Monitoring System was used to determine glycaemia
224 (reference range 1.1 to 33.3mmol/l; Lifescan Inc., Bucks).

225 Each preload was served at 12:00pm (15 minutes after
226 baseline glycaemia measurements) in an opaque non-
227 descript plastic cup in standard feeding booths in green
228 light. The coloured light masked a very slight colour
229 difference between preload drinks. The drinks were
230 flavoured with vanilla to yield an orosensory match.
231 Subjects were instructed to drink the entire product. All

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232 preloads were consumed within 5 minutes of their initial
233 hydration with 100ml bottled water.
234 Following ingestion of the product (12:15pm, 30 minutes
235 from baseline), glycaemia was again determined
236 following identical protocol.

237 **2.4 Test-lunch**

238 Volunteers ingested a controlled test-lunch of mixed
239 composition thirty minutes after consuming the preload
240 drink (12:30pm, 45 minutes from baseline) in standard
241 feeding booths in natural light. The test-lunch consisted
242 of 300g pre-cooked then chilled penne pasta (Don Mario
243 100% durum wheat semolina pasta quills', manufactured
244 by Abbey Foods Ltd, PO BOX 178, Liverpool) and 100g
245 Sacla Italia TM vine-ripened tomato and mascarpone stir
246 through sauce (F.lli Sacla S.p.A. Asti Italy; Sacla UK LTD,
247 Basil House, 21 London End, Bucks). This test-lunch
248 was heated to a temperature of at least 72°C in a
249 microwave and was served at a temperature of between
250 60-65°C. Subjects were instructed to consume the entire
251 test-lunch and all subjects adhered to protocol.

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253 The meal provided 57%, 13% and 30% of total energy
254 from carbohydrate, protein and fat respectively, as

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255 analysed by NetWISP (version 3.0 for Windows, Tinuviel
256 Software, Anglesey, UK). The test-lunch protocol used
257 here has been described previously (Paxman et al.,
258 2008a).

259 **2.5 Protocol Postprandially**

260 Further measures of capillary glucose were obtained at
261 90, 120, 150, 180, 210, 240, 270 and 330 minutes from
262 baseline. In total, ten capillary blood samples were taken
263 to determine glycaemia up to 330 minutes from baseline
264 (270 minutes postprandially).

265 **2.6 Statistical analysis**

266 Blood glucose measures were converted to delta area
267 under the curve (AUC) using the trapezoid rule with
268 subtraction of basal values (NCSS; Hintze, 2004, NCSS
269 and PASS Number Cruncher Statistical Systems,
270 Kaysville, Utah). Two-way between groups ANOVAs
271 were performed in order to identify the main effects of
272 treatment and body fat haptile and any interaction effects
273 on glycaemia at each time point, change from individual
274 baseline AUC glycaemia and peak postprandial
275 glycaemia (SPSS; version 15.0 for Windows, SPSS Inc.,
276 Chicago, IL, USA). Graphical presentations were
277 produced using SPSS (version 15.0 for Windows, SPSS
278 Inc., Chicago, IL, USA) and Microsoft Excel 2003

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279 (Microsoft Office, Microsoft Corporation). Significance
280 was set at $p < 0.05$. Data are presented as mean \pm 1 SD.

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282 **3.0 RESULTS**

283 Forty self-reported healthy male subjects (equal numbers
284 in each treatment arm) successfully completed the
285 experiment with no deviation from protocol.

286 **3.1 Ionic gelling sodium alginate attenuates the** 287 **glycaemic response to a meal**

288 Two-way between groups ANOVAs showed a significant
289 effect of treatment type on glycaemia at 90 ($p < .0005$),
290 150 ($p = .003$), 180 ($p = .021$) and 210 ($p = .013$) minutes
291 (see Figure 1). Overall, ingestion of SA compared to EF
292 resulted in a significant reduction in a mean change from
293 individual baseline AUC glycaemia ($\underline{M} = 148.43 \pm 148.65$
294 vs. $\underline{M} = 312.53 \pm 253.60$; $p = .010$) of 52.5% (see Figure 1).
295 Irrespective of treatment type, subjects in the lower
296 haptile for body fat % had a reduced mean change from
297 individual baseline AUC glycaemia (177.68 ± 255.44)
298 compared to those in the upper haptile for body fat %
299 (283.28 ± 171.66 ; $p = .065$; data not shown) however, this
300 was not significant and there was no interaction effect
301 between treatment type and body fat % grouping.

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302 **3.2 Ionic gelling sodium alginate reduces peak**

303 **postprandial glycaemia**

304 Preload type failed to affect the timing of peak glycaemia
305 as shown in Figure 1. However, Figure 2 shows the
306 significant 14% lower mean peak postprandial glycaemia
307 at 90 minutes following SA versus EF (\underline{M} = 6.06 ± .59
308 mmol/L vs. \underline{M} = 6.92 ± .70mmol/L; p < .0005) for the study
309 group as a whole. Subjects in the lower body fat haptile
310 had a lower peak postprandial glycaemia (6.39
311 ± .85mmol/L) than those in the upper body fat haptile,
312 irrespective of treatment type (6.59 ± .70mmol/L; p = .170;
313 data not shown) however this was not significant and
314 there was no interaction effect between treatment type
315 and body fat % grouping.

316 **3.3 Body fat classification determines the**

317 **postprandial glycaemic response to a meal but the**

318 **beneficial effects of alginate remain**

319 Irrespective of treatment type, subjects in the upper body
320 fat haptile had non-significantly elevated peak
321 postprandial glycaemia and non-significantly greater
322 mean change from individual baseline AUC glycaemia
323 compared to those in the lower body fat haptile. In
324 addition, body fat % grouping had a significant effect on
325 delta glycaemia at 120 (p = .005), 150 (p = .012), 180

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326 ($p= .049$) and 210 minutes ($p= .046$) from baseline, with
327 subjects in the upper body fat haptile having higher mean
328 glycaemia than those in the lower body fat haptile at
329 these time points, irrespective of preload treatment
330 (Figure 3).

331 For glycaemia at each time point, change from individual
332 baseline AUC glycaemia and peak postprandial
333 glycaemia however, the two-way between-groups
334 ANOVA showed no interaction effect between treatment
335 type and body fat % grouping in each case. Subjects
336 appeared to respond to the ionic-gelling sodium alginate
337 (SA) treatment in a similar fashion irrespective of body
338 fat %.

339 Examination of the response to treatment type by body
340 fat % grouping showed the lower body fat haptile on SA
341 reduced their change from individual baseline AUC
342 glycaemia by 68.3%, and their peak postprandial
343 glycaemia by 16.2% compared to the lower body fat
344 haptile on EF. A slightly weaker effect was apparent in
345 the upper body fat haptile on SA who reduced their
346 change from individual baseline AUC glycaemia by
347 46.6%, and their peak postprandial glycaemia by 9.7%
348 compared to those in the upper body fat haptile on the EF
349 treatment type (Figure 3). This finding supports our

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350 previous suggestions relating to altered glycaemic
351 response and body fatness (Paxman et al., 2008a).
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353 In summary, glycaemic response to the test-meal was
354 reduced following ingestion of the ionic-gelling sodium
355 alginate drink (SA) compared to the acid-gelling
356 excipient-free formulation (EF) throughout the 330 minute
357 measurement period. Body fatness influenced
358 postprandial glycaemic response but the effect of the
359 ionic-gelling alginate drink was maintained.

360

361 **4.0 DISCUSSION**

362 The literature suggests soluble fibre can alter subjective
363 hunger and fullness ratings (Peters et al., 2011), gastric
364 emptying rate and intestinal nutrient absorption, though
365 the extent and subsequent effect on glycaemia is poorly
366 established (Wolf et al., 2002; Delargy et al., 1997;
367 Fairchild et al., 1996). Contradictory reports are most
368 likely explained by the type, dose, homogeneity and
369 physicochemical properties of fibres used, and differing
370 participant characteristics between studies.

371 The physicochemical properties of alginate have particular
372 potential in terms of attenuating postprandial glycaemic

1 373 response and improving diabetic control (Williams et al.,
2 374 2004; Wolf et al., 2002; Torsdottir et al., 1991). Highly
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4 375 viscous solutions are unpalatable; solutions which form
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6 376 solid gel particles in the gastric lumen may provide a
7
8 377 more feasible alternative for controlling gastric emptying
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10 378 and nutrient uptake. In order to establish an optimum
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12 379 formulation for delivery of a glycaemia-modulating
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14 380 alginate, the physiological response to ionic- and acid-
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16 381 gelling alginates were compared in males of differing
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18 382 body fatness. Physiologic data show greater glucose
19
20 383 intolerance among the obese and numerous prospective
21
22 384 studies support such associations between measures of
23
24 385 obesity and type 2 diabetes risk (Carey et al., 1997).
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26 386 Such differences are postulated to be connected with
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28 387 body fatness.
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31 388 Our data show that the ionic-gelling sodium alginate drink
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33 389 (SA) reduced early-phase and peak postprandial
34
35 390 glycaemia and flattened the postprandial glycaemic curve
36
37 391 in comparison to the acid-gelling control (EF). From
38
39 392 baseline to 30 minutes, the EF preload drink resulted in a
40
41 393 slight elevation of blood glucose, most likely due to the 7g
42
43 394 fructose contained within the formulation. In comparison,
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45 395 the SA preload treatment elicited no change in glycaemia
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47 396 during this period despite containing the same amount of
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49 397 fructose. The difference between these responses can
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398 most probably be attributed to the addition of calcium
399 carbonate in the SA formulation. The acid-soluble
400 calcium salt was expected to facilitate intra-gastric ionic
401 gelation of the drink (Kimura et al., 1996). When alginate
402 formulations are pH dependent there is a known time lag
403 of 25-40 minutes before gelation occurs (Mattes, 2007).
404 The inhibition of a glycaemic response to the SA
405 formulation could have resulted from immediate fructose
406 entrapment, delayed gastric emptying or both. Torsdottir
407 et al. (1991) reported delayed glucose delivery and
408 reduced glycaemic peak in type 2 diabetics by the
409 addition of alginate to meals. They attributed this
410 response solely to delayed gastric emptying, measured
411 by aspirated radioactive stomach contents. There is
412 evidence to suggest alginate ingestion results in 'gel
413 lump' formation, which alters nutrient transport to the
414 small intestine (Hoad et al., 2004). In this study the
415 glycaemic response to a test-lunch of mixed composition
416 following the SA drink was consistently lower throughout
417 the investigation, thus it seems likely that nutrients were
418 captured within the gel matrix to some degree.

419 Hoad et al. (2004) used serial magnetic resonance
420 imaging (MRI) to gather *in vivo* measurements of guar
421 gum and weak and strong gelling alginates that had been
422 incorporated into milk-based drinks. MRI images showed

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423 heterogeneous distribution of alginate formulations in the
424 stomach with the formation of 'lumps', compared to the
425 homogenous distribution of guar gum. Initial 'gel lump'
426 formation was observed 10 minutes postprandially, other
427 'lumps' developed over time, compatible with the pH
428 decrease normally observed following ingestion of a meal.
429 In addition, the strong gelling alginate resulted in
430 significantly increased intragastric gel 'lump' production
431 compared to the weak gelling. Data from 'lump'
432 classification showed liquid filled 'lumps' were formed
433 predominantly with the strong gelling alginate; the
434 researchers hypothesise this gel strength is sufficient to
435 allow layer formation which resist break forces caused by
436 stomach motion.

437 There is a prevailing assumption that BMI measurement
438 is strongly associated with body fatness and consequent
439 morbidity and mortality (Gallagher et al., 2000).
440 Increased postprandial blood glucose is independently
441 related to the risk of cardiovascular disease and all-cause
442 mortality in newly diagnosed type 2 diabetics. Some
443 individuals classified overweight by BMI do not have
444 high % body fat. Conversely, others who have normal or
445 healthy BMIs have a relatively high body fat %.
446 Individuals who are misclassified by BMI are reportedly
447 uncommon relative to the UK population as a whole but

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448 since body fatness is a stronger predictor of increased
449 fasting glucose than BMI (Kriketos et al., 2003) it is more
450 appropriate and meaningful to divide subjects in the
451 present study by body fat %. In support of this, the
452 present study clearly shows subjects in the upper body
453 fat haptile had comparatively elevated early-phase
454 glycaemic excursion to those in the lower body fat haptile.

455

456 **5.0 CONCLUSIONS**

457 We conclude that an ionic-gelling sodium alginate drink
458 can significantly attenuate postprandial glycaemic
459 response in self-reported healthy males in comparison to
460 an acid-gelling control. This effect persisted in subjects in
461 both the lower and upper haptiles of body fatness. The
462 benefits of optimising glycaemic control through the use
463 of ionic-gelling sodium alginate products in patients with
464 morbidity related to body fatness (including type 2
465 diabetic and metabolic syndrome patients) warrant further
466 investigation.

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468 **ROLE OF THE FUNDING SOURCE**

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472

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626 May 2011).

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629 **TABLES**

630 Table 1

631 Subject characteristics

		<i>n</i>	Range	Mean ±	SD
Age (y)		40	18 – 55	30.03 ±	11.21
Sodium Alginate	Lower BF%	9	20 – 31	23.89 ±	4.05
	Upper BF%	11	18 – 51	34.55 ±	10.47
	TOTAL	20	18 – 51	29.75 ±	9.71
Excipient Free	Lower BF%	11	21 – 32	24.00 ±	3.19
	Upper BF%	9	19 – 55	38.00 ±	15.94
	TOTAL	20	19 – 55	30.30 ±	12.78
BMI (kg/m²)		40	18.6 – 39.4	26.02 ±	4.41
Sodium Alginate	Lower BF%	9	21.7 – 24.7	23.34 ±	1.07
	Upper BF%	11	22.7 – 35.2	29.07 ±	3.03
	TOTAL	20	21.7 – 35.2	26.50 ±	3.72
Excipient Free	Lower BF%	11	18.6 – 26.0	22.32 ±	2.33
	Upper BF%	9	23.0 – 35.6	29.47 ±	4.72
	TOTAL	20	18.6 – 39.4	25.54 ±	5.06
Body Fat (BF) %		40	7.1 - 35.6	17.54 ±	7.05
Sodium Alginate	Lower BF%	9	7.1 – 11.9	10.31 ±	1.56
	Upper BF%	11	16.8 – 31.7	22.58 ±	4.94
	TOTAL	20	7.1 – 31.7	17.06 ±	7.29
Excipient Free	Lower BF%	11	9.2 – 15.4	12.76 ±	1.82
	Upper BF%	9	18.1 – 35.6	24.47 ±	5.09
	TOTAL	20	9.2 – 35.6	18.03 ±	6.96

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633

634 **FIGURE CAPTIONS**

635 Figure 1

636 Mean delta AUC glycaemia (± 1 SD)

637 Following ingestion of the SA preload (solid line, filled
638 diamonds), mean delta AUC glycaemia was reduced by
639 52.5% when compared to the EF preload (broken line,
640 open squares). There was a significant effect of preload
641 treatment type on mean delta AUC ($p = .010$). In addition,
642 preload treatment type had a significant effect (*) on
643 mean delta glycaemia at 90 minutes ($p < .0005$), 150
644 minutes ($p = .003$), 180 minutes ($p = .021$) and 210
645 minutes ($p = .013$).

646

647 Figure 2

648 Mean peak postprandial glycaemia (± 1 SD)

649 There was a significant effect of preload treatment type
650 on mean peak postprandial glycaemia (SA solid bars; $\underline{M} =$
651 $6.06 \pm .59$ mmol/L compared to EF shaded bars; $\underline{M} =$
652 $6.92 \pm .70$ mmol/L; * $p < .0005$).

653

654 Figure 3

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3 655 Mean delta AUC glycaemia by body fat haptile
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5 656 When subjects were split by haptiles of body fat % (solid
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7 657 line = upper body fat haptile, broken line = lower body fat
8
9 658 haptile) there was a significant effect of body fat %
10
11 659 classification (§) on mean delta glycaemia at 120 minutes
12
13 660 ($p = .005$) 150 minutes ($p = .012$) 180 minutes ($p = .049$)
14
15 661 and 210 minutes ($p = .046$), irrespective of treatment type
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17 662 (solid diamonds = sodium alginate, open squares =
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19 663 excipient free).
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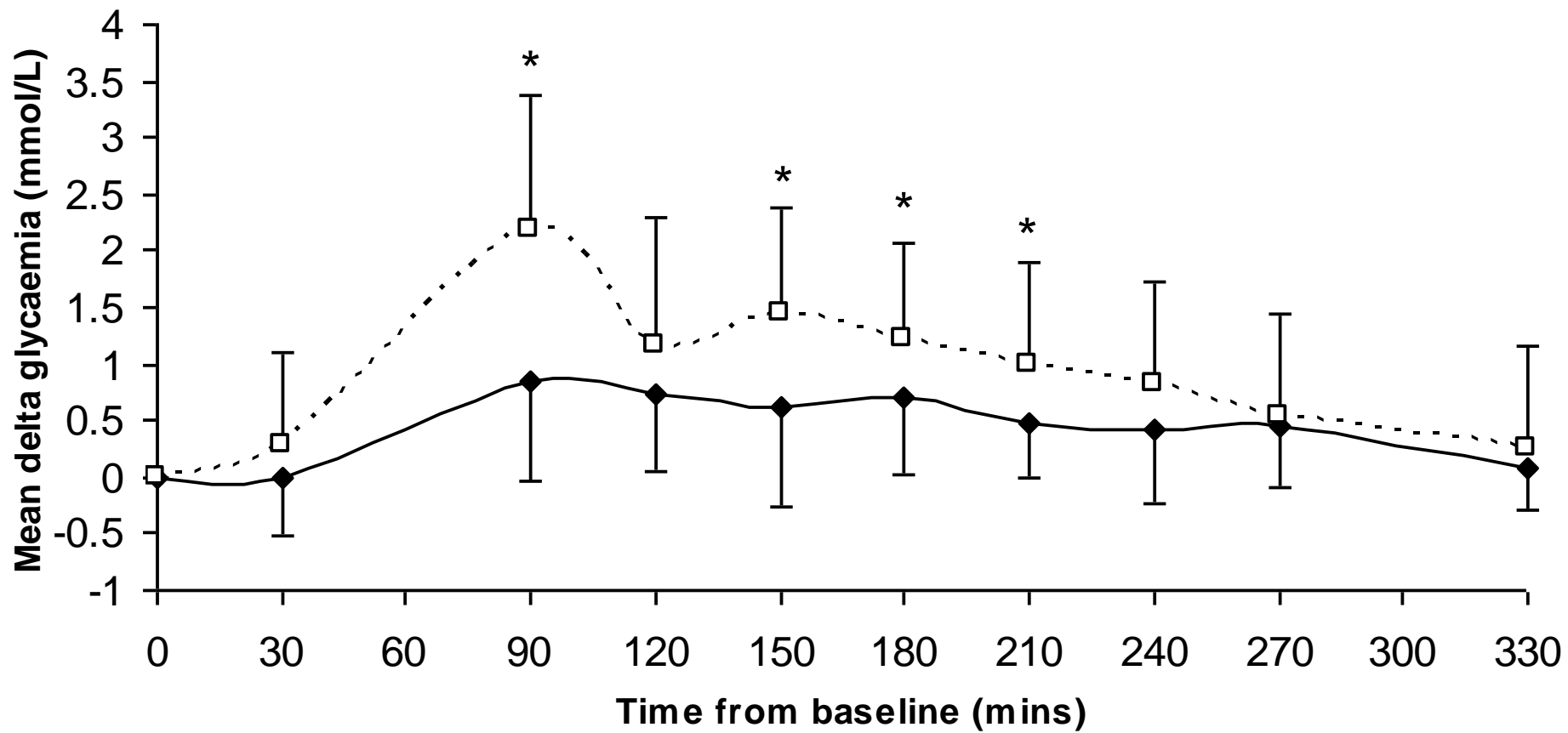
Figure 1

Figure 2

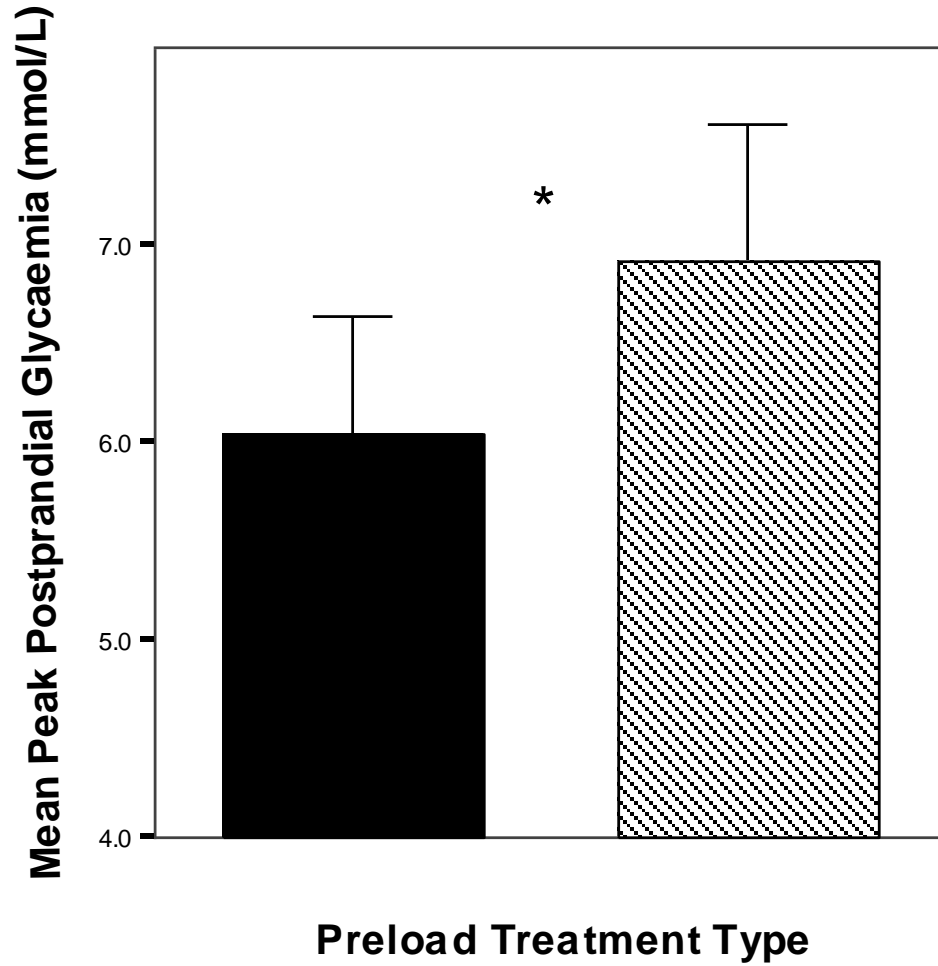


Figure 3

