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Relative glycaemic impact of customarily consumed portions of eighty-three foods measured by digesting *in vitro* and adjusting for food mass and apparent glucose disposal

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Practical values to guide food choices for control of postprandial glycaemia need to refer to entire foods in amounts customarily consumed. We tested an *in vitro* method for determining the relative glycaemic impact (RGI) of customarily consumed portions of foods. Sugars released during *in vitro* pancreatic digestion of eighty-three foods were measured as glucose equivalents (GE) per gram of food, adjusted by the glycaemic indexes of the sugars to obtain glycaemic GE (GGE) per gram and multiplied by food portion weight to obtain the GGE contribution of the food portion, its RGI. The results were compared with clinical GGE values from subjects who consumed the same food amounts. *In vitro* and *in vivo* GGE values were significantly correlated, but the slope of the regression equation was significantly less than one, meaning *in vitro* GGE values overestimated *in vivo* GGE values. Bland–Altman method comparison showed the *in vitro*–*in vivo* disparity to increase as mean GGE increased, suggesting the need to allow for different rates of homeostatic blood glucose disposal (GD) due to different GGE doses in the customarily consumed food portions. After GD correction, Bland–Altman method comparison showed that the bias in predicting *in vivo* GGE values from *in vitro* GGE values was almost completely removed ($y = 0{\cdot}071x - 0{\cdot}89$; R^2 0·01). We conclude that *in vitro* food values for use in managing the glycaemic impact of customarily consumed food quantities require correction for blood GD that is dependent on the GGE content of the food portions involved.

Carbohydrate: Glycaemic impact: Glucose disposal: *In vitro* digestion: Foods

With growing evidence that postprandial blood glucose responses have a direct role to play in the disease complications associated with glucose intolerance[1], there is an increasing demand for food values that will facilitate food choices for blood glucose control, by being expressed as grams per serving or grams per reference amount customarily consumed[2,3]. Available carbohydrate values are not an accurate guide to the potential of a food to raise blood glucose levels because they are determined on samples that have been finely ground to ensure complete release of carbohydrate. In contrast, the glycaemic impact of a food, which in the present context means the blood glucose-raising potential of the glycaemic carbohydrate released in digestion, depends on the degree to which food structure survives mastication and on other factors that affect the rate and extent of carbohydrate digestion. To overcome the limitations of available carbohydrate in glycaemia management, the glycaemic index (GI) was introduced as an adjunct to available carbohydrate values[4]. Although usually referred to as the 'glycaemic index of a food', GI is derived from the measured glycaemic effect of a food, calculated to an available carbohydrate basis, and is expressed as a percentage of the effect of glucose equal in weight to the carbohydrate. So, it becomes an imputed carbohydrate-based and not a food-based index.

GI was designed specifically to compare foods of equal carbohydrate content after they had been classified into food exchange categories of similar composition for intensive diabetes management[4]. It is more difficult and less appropriate to use GI for accurate glycaemic control under the everyday conditions of food purchasing, cooking and eating, in which most foods, even within marketed food categories, do not have the same carbohydrate content, and are not presented or consumed in carbohydrate-based portion sizes. And because GI is a fixed carbohydrate-based index, it does change with the amount of food consumed, so it cannot indicate the effects of different food intakes on glycaemic impact[5].

The glycaemic load, calculated as the product of GI and the amount of carbohydrate consumed in a food[6], is an intake-responsive measure of the relative glycaemic impact (RGI) of an entire food. Glycaemic load has been expressed

Abbreviations: GD, glucose disposal; GE, glucose equivalents; GGE, glycaemic GE; GI, glycaemic index; iAUC, incremental area under the blood glucose response curve; RGI, relative glycaemic impact.

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as glucose equivalents (GE)[7], but in terms of glycaemic effect, it is not generally the true GE of a food[8] because it has been calculated from GI, and so it is based on glycaemic responsiveness per gram of glucose at the 50 g glucose reference intake, not at a glucose intake equivalent in effect to the 50 g carbohydrate food portion[9]. And because the glucose dose–glycaemic response curve is non-linear, the response per gram of glucose at an intake of 50 g glucose may be much less than that at an intake of 50 g carbohydrate in food, especially with low GI foods. The effects of non-linearity on the determination of the GE of a food in terms of glycaemic potency and the need for values that reflect the effects of customarily consumed food portions have been addressed by measuring RGI of a standard glucose dose–glycaemic response curve, using relevant portion sizes and expressing the results as grams of glycaemic GE (GGE)[10].

However, all the clinical procedures for measuring the glycaemic potency of foods have drawbacks in common: they are very costly to perform, they face the logistical and ethical demands typical of clinical trials and, importantly, they suffer from intra- and inter-subject variation, which means that large subject numbers are required to adequately power tests of differences between foods[11,12].

One possible way around the difficulties of clinical trials is to measure glycaemic impact *in vitro* using a method that mimics the digestive actions of the gut. In this paper, we have applied a method for determining the GGE content of foods, and tested its performance against the results of clinical trials. The elements of the *in vitro* digestion are similar to those in other methods that simulate gut processes[13], but to obtain values of true glucose equivalence that are relevant, the present trial differed from others in a number of respects: the clinical arm used portions similar to those customarily consumed, the *in vivo* GGE values were directly determined off a glucose standard curve, and the *in vitro* GGE values included the contribution of non-glucose glycaemic sugars. Most importantly, initial results in the study indicated that an adjustment was required for the effects of dose dependence of rates of glucose disposal (GD) that would occur in response to the different GGE intakes in the portions of food consumed. The effects of making such an allowance for homeostasis then became an important aspect of the analysis, and they are presented here.

Method

Food sampling

Foods were purchased from single outlets, but the same sample was used in the *in vitro* and *in vivo* analyses.

***In vitro* digestion**

Reagents

All the reagents that were used, HCl, Na_2HCO_3, ethanol, sodium acetate 3-hydrate, maleic acid, D(+)-glucose, NaOH and sodium azide, were of high purity. Enzymes that were used were pepsin EC 3.4.23.1 from porcine stomach mucosa (Sigma, P 7000; 800–2500 U/ml; St Louis, MO, USA), pancreatin (Sigma, P7545; 8 × USP specifications), amyloglucosidase EC 3.2.1.3. from *Aspergillus niger* (Megazyme, E-AMGDF; 3260 U/ml) and invertase (BDH concentrate; 39 020 3D).

The 3,5-dinitrosalicylic acid reagent consisted of 10 g 3,5-dinitrosalicylic acid dissolved in a solution of 16 g NaOH plus 300 g Na–K tartarate in 1 litre, and it was allowed to stand for 2 d before use.

Sample treatment

All soft or crisp foods such as bakery products (Group A), breakfast cereals (Group B), and crackers, cakes and bars (Group C) were rubbed through a 4 mm square wire mesh, which provided a crumbled sample. Sundry cereals (Group D), snack foods (Group E), fruits (Group G), vegetables (Group H), meat (Group I), combination foods – savoury (Group J), and combination foods – sweet and meals (Group K) were processed through a Kenwood chef model A720 electric mincer with a 9 mm aperture plate, which subjected the samples to a shearing plus cutting action and produced a mechanically 'chewed' sample. Groups A–C were crumbled to avoid compaction, which occurred in some soft bakery products. Dairy-based foods (Group F) were analysed as is.

In vitro *digestion*

Food samples were digested in 70 ml open specimen pots inserted to their full depth in an aluminium heating block, placed on a 15 place magnetic stirrer, and covered with an insulating sheet. The digestion consisted of a simulated gastric digestion followed by small intestinal digestion, with timed sampling during the small intestinal phase. Briefly, 30 ml of water and 0·8 ml of 1 M-HCl were added to the sample to attain a pH of 2·5 (±0·2), with pH adjustment if necessary, 1 ml of 10 % pepsin that was dissolved in 0·05 M-HCl was added, and the mixture was stirred slowly (130 rpm) for 30 min at 37°C to accomplish gastric digestion. The small intestinal phase was initiated by adding 2 ml of 1 M-$NaHCO_3$ and 5 ml of 0·1 M-sodium maleate buffer, pH 6/0·02 % sodium azide/1 mM-$CaCl_2$, followed by the addition of 0·1 ml amyloglucosidase and 5 ml of 2·5 % pancreatin in 0·1 M-maleate buffer, pH 6 in quick succession to start amylolysis, and the pots were quickly made to the 55 ml mark with distilled water. Digesta aliquots of 1·0 ml were removed before adding the amyloglucosidase–pancreatin ($T = 0$) and at 20, 60 and 120 min from the start of amylolysis, and were each added to 4 ml absolute ethanol in a tube and mixed.

Measuring sugars released during digestion

After at least 30 min, the tubes containing the timed samples in ethanol were centrifuged for 10 min at 2000 rpm (Centrifuge Omnifuge 2.0 RS Heraeus Sepatech, Osterode, Germany) to clarify. A 0·05 ml aliquot of ethanolic supernatant, or glucose standard (1 mg/ml), was added to 0·25 ml of acetate buffer, pH 5·2, containing 1 % invertase + 1 % amyloglucosidase, and was incubated at 37°C for 10 min to complete depolymerisation to monosaccharides, which were measured as reducing sugars by a scaled-down dinitrosalicylic acid colorimetric method[14]. Fructose was measured by the thiobarbituric acid method[15], or values were taken from the New Zealand Food Composition Database[16]. All the samples were measured in duplicate. Rapidly available carbohydrate

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was calculated as reducing sugar in the 20 min sampling of pancreatic digest.

Glycaemic sugars may also be measured as glucose, measured by a glucose oxidase assay, plus fructose.

Clinical glycaemic glucose equivalents determination

The study was conducted according to the guidelines laid down by the Declaration of Helsinki, and all procedures involving human subjects were approved by the Canterbury Ethics Committee. Written informed consent was obtained from all the subjects.

The *in vivo* trials were carried out over 4 years. Participants were all males aged between 20 and 64 years, and none had diabetes according to the WHO classification (fasting glucose $> 6{\cdot}2$ mmol/l). None suffered from any diagnosed gastrointestinal or hepatic conditions during or immediately before the measurement period.

Glucose references and test foods

Glucose was supplied as glucose beverages bottled as 75 or 50 g anhydrous glucose in 300 ml. The beverage containing 50 g glucose was diluted with soda water (to maintain electrolyte balance) in the ratio of 3:1 and 1:1 to make solutions containing 12·5 and 25 g glucose, respectively, so the final range of doses was 0 (soda water) 12·5, 25, 50 and 75 g glucose in 300 ml.

Procedure

The effect of each food on blood glucose concentrations was measured once in each individual. The foods tested are given in Table 1, along with the number of participants tested for each food. The number of individuals who were tested for each food varied from seven to twenty. On average, the glucose standard curves were measured at approximately three monthly intervals for each individual. However, for the first twenty-seven of the foods, marked with an * in Table 1, a single 25 or 50 g glucose reference was used, whichever was thought to give a response in the vicinity of that expected of the test food. Subsequently, glucose references of 0 (soda water), 12·5, 25, 50 and 75 g glucose were used to allow construction of glucose dose–glycaemic response standard curves. The order in which the glucose references and foods were measured was randomised and balanced so that the glucose references were spread evenly over the course of the testing, and the sequence for each individual was varied.

All the tests were carried out at the Lipid and Diabetes Research Group following a standard protocol. Participants were asked to eat a meal containing carbohydrates, to refrain from drinking alcohol the night before each test and from physical exercise on the morning of the test and to report to the clinic after having fasted. Capillary blood samples were taken using a lancet. A drop of blood was collected into a HemoCue® cuvette, and blood glucose concentration was measured using a Hemocue® Glucose 201 Analyser (Helsingborg, Sweden)[17]. The average of two fasting blood glucose concentrations, determined 5 min apart, was used as a baseline measure. Test foods and glucose references were consumed within 15 min, and capillary blood samples were taken at 15, 30, 45, 60, 90 and 120 min after the person began consuming the test food or glucose reference. If the blood glucose concentration had not returned to within 0·2 mmol/l of the baseline concentration at 2 h, further blood samples were taken at 150 and 180 min after the start time. Participants were asked to remain seated for the duration of the tests with the exception of visits to the toilet.

Analysis of results. Two different *in vivo* methods were used. Initially, the method used one glucose reference of either 25 or 50 g amount, and this method was used for twenty-seven foods (marked with an * in Table 1). As a result of progress in our research, a more accurate method was then developed for measuring GGE from a standard curve[10], and the remaining sixty-three foods were tested by this method.

Calculation of incremental area under the blood glucose response curve

For all the foods, the incremental area under the blood glucose response curve (iAUC) to a maximum of 180 min was calculated geometrically using the method described by FAO/WHO for each of the test foods for each participant[18]. Areas where the curve dropped below baseline were excluded.

Calculation of slopes and intercepts for glucose curves

The iAUC for each of the five glucose reference intakes at each time point were calculated as described earlier. The iAUC and the glucose intakes (0, 12·5, 25, 50 and 75 g) were then log transformed, and the slope and intercept were determined.

Calculation of the in vivo *glycaemic glucose equivalents values of foods*

The curves describing the relationship between reference glucose intakes and iAUC were used to estimate the glycaemic response for all foods measured with glucose curves. This was done by using the derived slope and intercepts from the individual's glucose response curve to estimate the GGE from the individual iAUC for each food. The GGE for each food at the specified portion size was then taken as the average GGE over the individuals for whom it was measured.

For the foods where a single 25 or 50 g glucose reference rather than a glucose standard curve was used, the GGE of the test food was calculated as GGE/portion size by dividing the $iUAC_{test\ food}$ by the $iAUC_{average\ glucose}$, and by multiplying the result by the amount of glucose in the reference drink. Each glucose reference was measured at least three times. The average GGE/serve for each food by each method was taken as the average of all the individuals.

GGE values calculated from a single 25 or 50 g glucose reference value (marked with an * in Table 1) were adjusted to the value that would have been obtained directly from a glucose reference curve, by multiplying the ratio of the single reference value and the corresponding value on a glucose dose–glycaemic response curve, using the equation:

$$y = -0{\cdot}007275x^2 + 1{\cdot}339x + 1{\cdot}778;\quad R^2\ 0{\cdot}998. \qquad (1)$$

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Table 1. Glycaemic impact as glycaemic glucose equivalents (GGE) per serving of foods determined *in vivo* and *in vitro*
(Mean values with their standard errors)

		GGE						
		In vivo				*In vitro*		
Food	Serving size (g)	*n*	Mean (g)	SE	CV (%)	Mean (g)	SE	CV (%)
A Breads								
1. Bread, pita white, Gianni's*	90	7	14·6	3·3	60	32·7	1·8	7·7
2. Burgen bread mixed grain*	90	8	13·0	0·8	18	20·8	0·7	4·8
3. North's Swiss rye bread*	100	11	13·4	2·3	56	18·7	0·3	2·0
4. Pam's Swiss grain bread*	100	11	17·0	2·3	44	22·3	0·1	0·9
5. Vogel's Kibbled rye*	100	11	17·4	4·0	77	25·5	0·9	5·0
6. Crop and food bread1*	100	11	15·6	2·5	52	19·8	0·6	4·6
7. Crop and food bread 2*	100	11	12·7	1·3	34	16·9	0·3	2·9
8. Crop and food bread 3*	100	11	16·0	1·7	36	18·1	0·6	4·6
9. Crop and food bread 4*	100	11	14·3	2·8	65	17·8	1·0	8·2
10. Crop and food bread 5*	100	11	18·3	3·7	67	18·2	0·9	7·1
11. Crop and food bread 6*	100	11	11·3	2·2	64	12·8	0·3	3·9
12. Crop and food bread 7*	100	11	11·4	2·0	58	15·5	0·6	5·8
13. Low GI concept bread†	100	12	18·9	3·3	60	16·8	0·7	6·1
14. Tip Top family fresh white	100	20	28·3	2·6	41	34·6	1·3	5·2
15. Morph bread*	100	10	34·8	4·8	44	37·0	0·7	2·8
16. Epic bread*	100	11	34·5	4·1	39	32·3	1·1	4·8
17. Bakker Gold bread*	100	11	31·5	4·1	43	37·4	0·9	3·6
B Breakfast cereals								
18. Kellogg's cocoa pops*	40	8	18·9	2·5	38	16·5	0·3	2·2
19. Fruity bix, wild berry*	50	8	16·2	2·3	41	25·3	0·7	3·9
20. Kellogg's All bran†	45	10	13·9	1·9	43	20·0	0·6	4·3
21. Skippy Cornflakes†	31	10	27·2	3·1	36	23·9	0·2	1·5
22. Hubbard's fruitful lite muesli†	30	10	15·7	1·0	20	15·0	0·3	2·7
C Crackers/cakes/bars								
23. Bluebird crazy caramel yum bar*	50	8	13·5	2·3	49	16·2	1·5	13·4
24. Ryvita crackers, original*	25	8	7·6	0·8	30	4·7	0·2	5·9
25. Mother earth oat bar	100	20	23·0	3·1	60	36·7	1·2	4·7
26. Russian slice	100	14	29·4	6·7	86	38·4	1·6	6·0
27. Fruit and nut loaf	100	14	28·3	2·4	32	38·8	2·3	8·3
28. Apricot slice	100	14	23·2	2·2	35	38·5	1·5	5·4
29. Pam's apricot fruit bars	100	14	30·6	3·5	43	40·7	2·8	9·8
30. Uncle Toby's Fruit twist	100	14	42·8	3·7	33	40·5	1·7	5·9
31. Ernest Adams luncheon cake†	40	10	12·3	0·9	22	14·1	0·4	4·3
32. Arnott's Salada crackers†	35	10	14·3	1·2	28	18·7	0·2	1·3
33. Arnott's Vita wheat crackers†	35	10	14·1	1·8	40	20·4	0·2	1·7
34. Griffins Fruitli Fingers (apricot)	100	14	21·4	2·8	48	41·3	1·1	3·7
35. Ernest Adams Anzac biscuits	100	14	21·7	2·1	36	34·0	2·8	11·6
36. Griffins Fruit Digestive biscuit	100	14	26·4	3·9	56	37·1	0·9	3·4
D Sundry cereals								
37. Parboiled rice	100	11	15·5	3·0	64	7·5	0·7	12·3
38. Spaghetti pasta	100	12	16·0	4·8	103	17·0	0·4	3·7
39. Moroccan Couscous	100	11	25·3	2·8	37	30·2	0·2	1·1
40. Pasta spirals	100	12	10·6	3·1	102	17·1	0·6	5·3
41. Lasagna sheets	100	13	16·7	2·5	55	22·5	0·9	5·9
E Snack foods								
42. Tasty whole cashew nuts	100	12	4·3	1·4	113	10·5	0·0	0·3
43. Eta salted peanuts cooked in canola	100	13	2·6	1·2	172	11·2	0·3	3·7
44. Pam's corn chips	100	13	26·2	4·1	56	46·5	0·6	1·8
45. Cadbury's dairy milk chocolate	100	12	14·5	2·6	63	32·6	0·6	2·8
F Dairy-based foods								
46. Custard-Swiss maid vanilla	100	13	9·2	1·5	58	16·5	0·5	4·3
47. Ice cream – Cadbury's vanilla	100	13	5·2	1·3	93	24·8	0·6	3·2
48. Ice cream – Pam's Swiss caramel	100	13	12·1	2·6	76	26·4	0·4	2·3
49. Trim milk- Meadowfresh	250	13	5·2	1·1	76	13·5	0·0	0·4
50. Yogurt Deluxe lemon	100	13	6·8	1·2	65	18·6	0·7	5·4
51. Sanitarium Up and Go	250	12	14·8	3·1	72	18·5	0·8	5·8
G Fruits								
52. SPC pear halves in juice*	192	8	4·9	1·2	70	6·3	0·1	3·3
53. Dole ripe banana	100	13	8·5	2·6	112	7·0	0·1	1·4
54. Sunreal dried apricots	100	13	9·4	2·2	86	25·8	0·7	3·6
55. Californian orange	100	13	4·5	1·5	121	3·4	0·0	1·5
56. Braeburn apple	100	13	3·8	1·0	95	3·0	0·1	3·8

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Table 1. *Continued*

Food	Serving size (g)	GGE						
		In vivo				*In vitro*		
		n	Mean (g)	SE	CV (%)	Mean (g)	SE	CV (%)
57. Pam's pitted dates	100	11	30·1	3·6	40	45·4	0·3	0·8
58. Sun-Maid raisins	100	12	27·6	6·7	84	38·7	0·3	1·0
H Vegetables								
59. Cinderella instant mashed potato	100	20	15·3	1·5	44	18·0	0·6	4·7
60. Edgell's chickpeas	100	20	4·9	1·0	91	3·2	0·1	5·3
61. Masterfoods Borlotti beans	100	13	5·9	1·4	84	5·1	1·0	28·1
62. Craig's red kidney beans	100	12	7·7	2·6	118	7·7	0·5	8·3
63. Craig's lentils in brine	100	13	4·5	1·5	118	4·4	0·4	11·6
64. Potato cooked and cooled overnight	100	13	10·0	1·7	61	14·2	0·0	0·4
65. Broccoli florets	100	13	4·3	2·1	176	1·9	0·0	2·4
66. Carrots (peeled)	100	13	5·6	2·7	172	3·9	0·2	7·0
67. Yams with ends off	100	13	7·5	1·7	84	6·7	0·1	1·5
68. McCain's Super juicy sweet corn	100	13	7·5	2·1	102	9·1	0·0	0·5
69. Parsnip	100	14	7·2	1·3	66	13·7	0·2	1·8
70. Potato cooked	100	14	15·2	2·3	56	18·7	0·2	1·7
71. Orange kumara	100	14	11·6	1·5	48	5·8	0·1	2·2
72. Pumpkin	100	14	4·6	0·8	68	4·6	0·0	0·5
73. Wattie's garden peas	100	14	6·6	1·0	57	3·2	0·1	5·3
I Meat								
74. Steak (beef cooked medium)	100	12	2·5	1·0	138	2·3	0·3	17·0
J Combination foods – savoury								
75. Wattie's tomato soup (packet mix)	250	13	12·1	2·6	76	15·5	0·4	4·0
76. Campbell's leek and potato soup	250	12	15·9	2·9	63	18·4	0·9	7·3
77. Irvines Thai Chicken snack meal	270	12	26·3	6·9	90	39·2	2·4	8·5
78. Irvines Beef casserole snack meal	270	12	13·0	1·3	34	24·1	0·4	2·1
79. Irvines Cottage pie snack meal	270	13	9·4	2·6	101	32·3	1·0	4·3
80. Irvines Macaroni cheese snack meal	270	13	6·3	1·6	93	39·1	0·7	2·6
81. Irvines Fish pie snack meal	270	12	11·9	2·0	57	25·5	3·2	17·6
K Combination foods – sweet								
82. Crofters strawberry cheesecake	100	13	13·2	2·0	55	38·7	0·5	1·8
83. Sara Lee Apricot Danish pie	100	13	23·1	2·6	41	38·7	0·5	1·8

GI, glycaemic index.

* Fifty or twenty-five grams of glucose references used in initial measurement of *in vivo* GGE values, but values subsequently adjusted to their equivalent values on the glucose dose–glycaemic response standard curve using equation 1.

† Fifty grams of glucose references used in initial measurement of *in vivo* GGE values, but values were subsequently adjusted to their equivalent values on the glucose dose–glycaemic response standard curve using equation 1. All other GGE *in vivo* values were obtained directly from the standard curve.

This equation was based on the results of six published studies of the relationship between glucose dose and glycaemic response normalised to a response of 50 GGE at an intake of 50 g glucose[8].

Calculation of in vitro *glycaemic glucose equivalents values*

GGE values were based on rapidly available carbohydrate (20 min digestion) measured as total GE. The GE values were converted to *in vitro* GGE values by reducing the GE values by the proportion of fructose in the rapidly available carbohydrate multiplied by 0·78. Multiplying by 0·78 allows for the fact that fructose has 0·22 of the glycaemic potency of glucose (GI fructose = 22)[19], that is, it contributes 0·22 GGE/g. Thus, where the proportion of fructose is P_f and the total amount of GE measured is GE, the *in vitro* GGE value is obtained as

$$\mathrm{GGE} = \mathrm{GE} - (0{\cdot}78 P_{\mathrm{f}}\mathrm{GE})\,\mathrm{g}. \qquad (2)$$

If free glucose and fructose are measured enzymatically rather than as total GE and fructose, as in the present study, the *in vitro* GGE value is calculated as glucose + 0·22 fructose.

No allowance was made for the presence of lactose (GI = 80) in the foods given in Table 1.

Allowance for apparent glucose disposal

The theoretical balance between GGE released *in vitro* from a portion of food after a given duration of digestion and GD that would have occurred *in vivo* in the same time in response to the GGE loading by the food portion was determined using the relationship: GD rate = $-0{\cdot}000104\mathrm{GGE}^2 + 0{\cdot}0169\mathrm{GGE}$ g/min[20] determined from previous clinical trials[21]. As the *in vitro* GGE value was determined after 20 min digestion, theoretical GD at 20 min was

$$\mathrm{GD} = 20(-0{\cdot}000104\mathrm{GGE}^2 + 0{\cdot}0169\mathrm{GGE})\,\mathrm{g}. \qquad (3)$$

So, the net GGE contribution, being the difference between GGE release from the food and GGE disposal (Table 2), was

$$\mathrm{Net\,GGE} = \mathrm{GGE} - 20(-0{\cdot}000104\mathrm{GGE}^2 + 0{\cdot}0169\mathrm{GGE})\,\mathrm{g}. \qquad (4)$$

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Table 2. Steps in transforming *in vitro* glucose equivalents (GE) values to net glycaemic GE (GGE) values: GGE is calculated from GE by allowing for the relative glycaemic potency of fructose, and then net GGE is calculated from GGE by subtracting apparent glucose disposal at 20 min*

	GE (g)	GGE (g)†	Glucose disposal (GD, g)	Net GGE (GGE − GD, g)‡	Net GGE −GGE *in vivo* (Table 1, g)
Breads					
1. Bread, pita white, Gianni's	32·7	32·7	8·9	23·8	9·24
2. Burgen bread mixed grain	20·8	20·8	6·1	14·7	1·65
3. North's Swiss rye bread	18·7	18·7	5·6	13·1	−0·31
4. Pam's Swiss grain bread	22·3	22·3	6·5	15·8	−1·23
5. Vogel's Kibbled rye	25·5	25·5	7·3	18·2	0·79
6. Crop and food bread 1	19·8	19·8	5·9	13·9	−1·71
7. Crop and food bread 2	16·9	16·9	5·1	11·8	−0·9
8. Crop and food bread 3	18·1	18·1	5·4	12·6	−3·36
9. Crop and food bread 4	17·8	17·8	5·4	12·4	−1·88
10. Crop and food bread 5	18·2	18·2	5·5	12·7	−5·58
11. Crop and food bread 6	12·8	12·8	4·0	8·8	−2·48
12. Crop and food bread 7	15·5	15·5	4·8	10·8	−0·65
13. Allied Mills low GI concept bread	16·8	16·8	5·1	11·7	−7·24
14. Tip Top family fresh white bread	34·6	34·6	9·2	25·4	−2·93
15. Morph bread	37·0	37·0	9·7	27·3	−7·5
16. Epic bread	32·3	32·3	8·8	23·5	−10·98
17. Bakker Gold bread	37·4	37·4	9·8	27·6	−3·86
Breakfast cereals					
18. Kellogg's cocoa pops	22·6	16·5	5·0	11·5	−7·41
19. Fruity bix, wild berry flavour	27·8	25·3	7·2	18·1	1·87
20. Kellogg's All bran	22·5	20·0	6·0	14·1	0·19
21. Skippy Cornflakes	24·3	23·9	6·9	17·0	−10·25
22. Hubbard's fruitful lite muesli	17·2	15·0	4·6	10·4	−5·35
Crackers/cakes/bars					
23. Bluebird crazy caramel yum bar	16·2	16·2	4·9	11·2	−2·28
24. Ryvita crackers, original	6·1	4·7	1·5	3·2	−4·44
25. ME sultana oat honey bars	53·6	36·7	9·6	27·0	4·04
26. Russian slice	60·9	38·4	9·9	28·5	−0·94
27. Fruit and Nut loaf	56·3	38·8	10·0	28·8	0·5
28. Apricot slice	56·1	38·5	10·0	28·5	5·3
29. Pam's apricot fruit bars	56·0	40·7	10·3	30·4	−0·25
30. Uncle Toby's Fruit twist	58·4	40·5	10·3	30·2	−12·58
31. Ernest Adams Fruit luncheon cake	21·5	14·1	4·4	9·7	−2·56
32. Arnott's Salda crackers	18·7	18·7	5·6	13·1	−1·19
33. Arnott's Vita wheat crackers	20·7	20·4	6·1	14·4	0·28
34. Griffins Fruitli Fingers (apricot)	56·1	41·3	10·4	30·9	9·45
35. Ernest Adams Anzac biscuits	47·7	34·0	9·1	24·9	3·22
36. Griffins Fruit Digestive biscuit	48·8	37·1	9·7	27·4	0·98
Sundry cereals					
37. Parboiled rice	7·5	7·5	2·4	5·1	−10·45
38. Spaghetti pasta	17·0	17·0	5·1	11·8	−4·19
39. Moroccan Couscous	30·2	30·2	8·3	21·9	−3·4
40. Pasta spirals	17·1	17·1	5·2	11·9	1·31
41. Lasagna sheets	22·5	22·5	6·6	15·9	−0·78
Snack foods					
42. Tasty whole cashew nuts	12·8	10·5	3·3	7·1	2·84
43. Eta salted peanuts cooked in canola	13·0	11·2	3·5	7·7	5·05
44. Pam's corn chips	49·8	46·5	11·3	35·2	9·04
45. Cadbury's dairy milk chocolate	56·4	32·6	8·8	23·7	9·23
Dairy-based foods					
46. Custard-Swiss maid vanilla	23·0	16·5	5·0	11·5	2·27
47. Ice cream – Cadbury's vanilla	31·3	24·8	7·1	17·7	12·47
48. Ice cream – Pam's Swiss caramel	32·9	26·4	7·5	18·9	6·8
49. Trim milk- Meadowfresh	13·5	13·5	4·2	9·3	4·1
50. Yogurt Deluxe lemon	22·9	18·6	5·6	13·1	6·25
51. Sanitarium Up and Go	26·2	18·5	5·6	13·0	−1·82
Fruits					
52. SPC pear halves in juice	6·3	6·3	2·0	4·2	−0·67
53. Dole ripe banana	11·4	7·0	2·3	4·8	−3·75
54. Sunreal dried apricots	36·0	25·8	7·4	18·5	9·05
55. Californian orange	4·9	3·4	1·1	2·3	−2·22
56. Braeburn apple	7·5	3·0	1·0	2·0	−1·8
57. Pam's pitted dates	72·7	45·4	11·1	34·3	4·2
58. Sun-Maid raisins	66·9	38·7	10·0	28·7	1·11

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Table 2. *Continued*

	GE (g)	GGE (g)†	Glucose disposal (GD, g)	Net GGE (GGE − GD, g)‡	Net GGE −GGE *in vivo* (Table 1, g)
Vegetables					
59. Cinderella instant mashed potato	18·1	18·0	5·4	12·6	−2·7
60. Edgell's chickpeas	3·2	3·2	1·1	2·1	−2·76
61. Masterfoods Borlotti beans	5·1	5·1	1·7	3·4	−2·47
62. Craig's red kidney beans	7·8	7·7	2·5	5·2	−2·48
63. Craig's lentils in brine	4·5	4·4	1·5	3·0	−1·54
64. Potato cooked and cooled o/n	14·2	14·2	4·4	9·8	−0·17
65. Broccoli florets	2·5	1·9	0·6	1·3	−3·05
66. Carrots (peeled)	5·2	3·9	1·3	2·6	−3·01
67. Yams with ends off	6·7	6·7	2·2	4·6	−2·95
68. McCain's Super juicy sweet corn	9·3	9·1	2·9	6·2	−1·3
69. Parsnip	14·9	13·7	4·2	9·4	2·23
70. Potato cooked	18·7	18·7	5·6	13·1	−2·11
71. Orange kumara	6·4	5·8	1·9	3·9	−7·67
72. Pumpkin	4·6	4·6	1·5	3·1	−1·51
73. Wattie's garden peas	3·2	3·2	1·1	2·1	−4·46
Meat					
74. Steak (beef cooked medium)	2·3	2·3	0·8	1·5	−0·97
Combination foods – savoury					
75. Wattie's tomato soup (packet mix)	15·5	15·5	4·8	10·8	−1·34
76. Campbell's leek and potato soup	18·4	18·4	5·5	12·8	−3·07
77. Irvines Thai Chicken snack meal	39·2	39·2	10·1	29·1	2·78
78. Irvines beef casserole snack meal	24·1	24·1	6·9	17·1	4·11
79. Irvines cottage pie snack meal	32·3	32·3	8·8	23·6	14·16
80. Irvines macaroni cheese snack meal	39·1	39·1	10·1	29·0	22·7
81. Irvines fish pie snack meal	25·5	25·5	7·3	18·2	6·29
Combination foods – sweet					
82. Crofters strawberry cheesecake	38·7	38·7	10·0	28·7	15·5
83. Sara Lee Apricot Danish pie	38·7	38·7	10·0	28·7	5·59

GI, glycaemic index.
* Precision of the parent means is shown in Table 1.
† GE to GGE adjustment by taking account of the glycaemic potency of fructose (equation 2).
‡ Adjustment for glucose disposal: net GGE = GGE − 20(−0.000104 GGE^2 + 0.0169 GGE) g.

Analysis of glycaemic glucose equivalents in vitro*–glycaemic glucose equivalents* in vivo *relationship*

All GGE calculations, means, standard deviations and coefficients of variation were calculated in a Microsoft Excel spreadsheet. Prediction of *in vivo* GGE by *in vitro* GGE with and without adjusting for GD was tested by regression analysis weighted by the precision of the *in vivo* measures using the Genstat statistical package[22]. A method comparison analysis was conducted, also in Excel, using the Bland & Altman[23] procedure, which involved plotting the means of the *in vitro* and *in vivo* determinations against the difference between the two. The Bland–Altman analyses were applied to the individual food groupings and then in turn to the relationship of GGE *in vitro* with GE, GGE and net GGE (GGE after adjusting for theoretical GD). Because of the heterogeneity of foods within most groups and the small number of foods in some groups, all foods were taken together to show the effects of converting GE to GGE and GGE to net GGE.

Results

The GGE values determined from clinical blood glucose response measurements (*in vivo*) and by *in vitro* digestive

Table 3. Regression equations predicting glycaemic glucose equivalents (GGE) *in vivo* (*y*; Table 1) from net GGE *in vitro* (*x*; Table 2) for food groupings and all the foods taken together

Food groupings	Number of foods	Equation	R^2	*P*
A. Breads	17	$y = 1.069x + 1.14$	0·70	<0·001
B. Breakfast cereals	5	$y = 0.568x + 10.3$	*	0·557
C. Crackers, cakes, bars	14	$y = 0.834x + 3.69$	0·72	0·001
D. Sundry cereals	5	$y = 0.614x + 8.65$	0·507	0·098
E. Snack foods	4	$y = 0.795x - 2.75$	0·983	<0·001
F. Dairy-based foods	6	$y = 0.216x + 5.88$	*	0·632
G. Fruits	7	$y = 0.79x + 1.99$	0·908	<0·001
H. Vegetables	15	$y = 0.783x + 3.59$	0·711	<0·001
I. Combination foods	7	$y = 0.113x + 11.3$	*	0·490
All foods	83	$y = 0.741x + 3.77$	0·622	<0·001

* Not statistically significant.

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analysis of GGE as GE corrected for the low GI of fructose present (equation 1) are shown in Table 1, with their standard errors and CV. The corresponding GE values on which they were based are given in Table 2. The precision of measurement was much greater for the *in vitro* measurements than for the *in vivo* measurements within all groups; standard errors for *in vivo* measures ranged from 0·6 of the *in vitro* SEto 72 times (median 4·8 times as large). The CV, being based on the variability of individual observations rather than on the mean, differ more markedly; however, the size

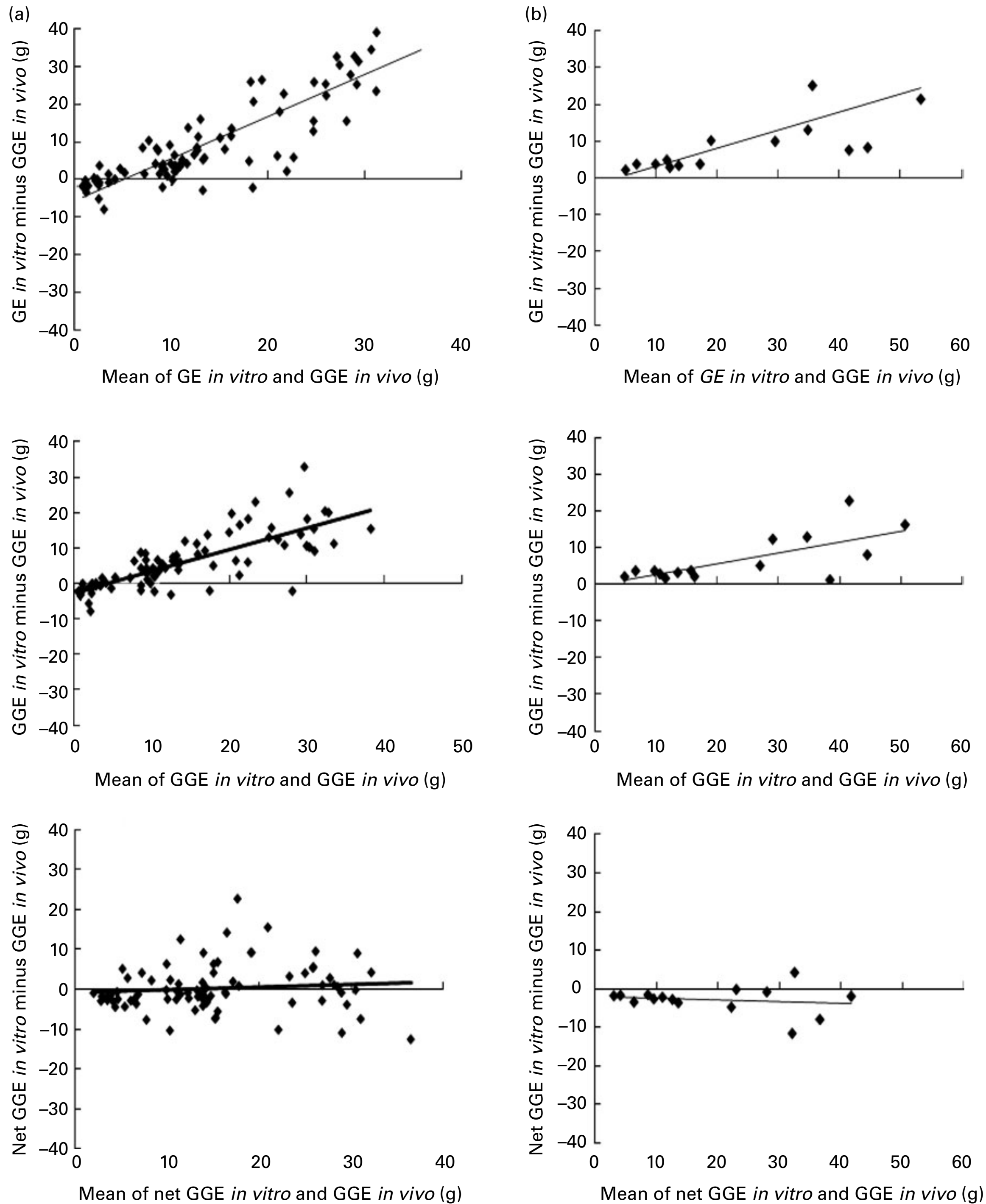

Fig. 1. Bland–Altman method comparisons (mean of *in vitro* net glycaemic glucose equivalents (GGE) and *in vivo* GGE *v.* difference between *in vitro* net GGE and *in vivo* GGE) showing improving correspondence between *in vitro* and *in vivo* measurements by allowing, firstly, for the relative glycaemic potency of fructose (glucose equivalents (GE) to GGE conversion), and secondly, for glucose disposal (GGE conversion to net GGE, by subtracting glucose disposal at 20 min.). (a) Data from GGE *in vivo* (Table 1) and net GGE *in vitro* (Table 2). (b) Data based on net GGE values and glucose disposal baselines in Monro *et al.*[20]. (a) GE: $y = 1·129x - 5·82$; R^2 0·74; GGE: $y = 0·608x - 2·62$; R^2 0·59; net GGE: $y = 0·0705x - 0·8931$; R^2 0·0105. (b) GE: $y = 0·491x - 1·89$; R^2 0·56; GGE: $y = 0·29x - 0·32$; R^2 0·50; net GGE: $y = 0·048x - 1·862$; R^2 0·030.

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Table 4. Equations from Bland–Altman method comparison applied to foods within the food groups; the mean (x) of net GGE *in vitro* (Table 2) and GGE *in vivo* (Table 1) is plotted against the difference (Table 2) between net GGE *in vitro* and GGE *in vivo*.

Food groups	Bland–Altman equation	R^2
A. Breads	$y = -0.026x + 2.43$	0·16
B. Breakfast cereals	$y = -0.634x + 6.18$	0·19
C. Crackers, cakes, bars	$y = 0.024x - 0.548$	0·002
D. Sundry cereals	$y = 0.0173x - 6.11$	0·04
E. Snack foods	$y = 0.221x + 3.18$	0·74
F. Dairy-based foods	$y = 0.105x + 6.23$	0·28
G. Fruits	$y = 0.192x + 1.66$	0·019
H. Vegetables	$y = 0.08x - 2.93$	0·019
I. Combination foods	$y = 0.255x + 2.23$	0·02
All foods	$y = 0.071x - 0.89$	0·01

of the CV (about 5 % for the *in vitro* procedure and about 70 % for the *in vivo* procedure) is not unusual for these types of measurement.

For each of the food groups shown and for all the foods taken together, the equations for predicting the *in vivo* GGE values from the *in vitro* measurements of net GGE and their R^2 values are given in Tables 3 and 4. Five groups (snack foods, fruits, crackers, vegetables and breads) had significant positive relationships between GGE *in vivo* measures and net GGE *in vitro* measures. For most, the slope of the equation was significantly less than one, indicating that the *in vitro* results for the foods in the group covered a wider range than the *in vivo* ones; the bread group was the one exception, with a slope close to one. Given that the five groups showing the positive *in vitro*–*in vivo* relationship account for fifty-seven of the eighty-three samples, it is perhaps not surprising that there is a significant (although not as strong) correlation between *in vivo* and *in vitro* measures for all the foods taken together.

Bland and Altman plot[24] method comparison of GGE *in vivo* values and GGE *in vitro* values before correction for GD had indicated that the *in vitro* measures were generally higher than the corresponding *in vivo* values, and this discrepancy increased with the GGE quantity in the food (Fig. 1). In light of the increasing disparity between *in vitro* and *in vivo* GGE values with increasing GGE intake, we undertook the research, based on clinical measurements[22], which identified equations relating apparent GD, which represents the overall homeostatic adjustment to blood glucose loading, to GGE intake[21]. Applying the GD equation to the unadjusted GGE values allowed a GD value (GD, Table 2) to be calculated for the GGE dose contributed by each food (GGE, Table 2). Subtracting GD from GGE provided GD-adjusted values termed 'net GGE' (Table 2). As the values given in Table 2 are derived from the values given in Table 1, the measures of precision have not been repeated in Table 2.

Comparison of the *in vivo* GGE values with the *in vitro* values for GE, GGE and net GGE showed an improved correspondence between the *in vivo* and *in vitro* methods as one converted successively from GE to GGE to net GGE (Fig. 1 and Table 5). Subtracting apparent GD from GGE to obtain net GGE provided, on average, an almost perfect overall correspondence between the *in vivo* and *in vitro* mean GGE determinations, although the individual data points remained scattered. The disparity, GGE *in vitro* minus GGE *in vivo,* was >5.0 g for forty-six foods ($44 > +5$ g, $2 < -5$ g), while for

Table 5. Changes in relationship between *in vivo* and *in vitro* values for glucose equivalents (GE), glycaemic glucose equivalents (GGE) and net GGE for all that foods based on values given in Table 2

	Correlation equation	R^2	Bland–Altman plot	R^2
GE	$0.39x + 5.37$	0·57	$1.129x - 5.82$	0·74
GGE	$0.49x - 1.89$	0·56	$0.608x - 2.62$	0·59
Net GGE	$0.74x + 4.55$	0·62	$0.071x - 0.893$	0·01

net GGE *in vitro* (GD allowed for) minus GGE *in vivo*, the number of foods showing a >5 g disparity was reduced to twenty-five in total ($10 < -5$ g, $15 > 5$ g). The *in vitro* GGE minus *in vivo* GGE disparity was >10 g for twenty-three foods (no differences <10 g), whereas the net GGE *in vitro* minus GGE *in vivo* disparity was >10 g for only eight foods ($4 > +4$ g, $4 < -4$ g; Table 2).

Discussion

The present study investigated the *in vitro* measurement of the RGI of customarily consumed food portions. It confirmed with a large sample of eighty-three foods that use of a correction for GD, derived in a previous detailed study of responses to fifteen food intakes[20], improves the validity with which *in vitro* determinations of the RGI of customarily consumed amounts of foods may predict relative glycaemic effects *in vivo*. Although the procedure was similar in principle to many other *in vitro* methods, e.g. Brighenti *et al.*[24], Englyst *et al.*[25] and Goni *et al.*[26] there are two particular aspects of the present study that deserve comment.

Firstly, the term 'RGI' was chosen to represent a rapidly acting food property, not a food effect. It is the rapidly imposed dietary loading of glycaemic carbohydrate by a food during digestion, measured relatively as GE by using a glucose reference and adjusted by the relative glycaemic potency (GI) of the constituent monosaccharides, that converts the GE to GGE. RGI therefore states the relative potential of a food quantity to increase blood glucose concentrations expressed as the amount of glucose that would theoretically have the same effect. The term impact is appropriate because the GGE determination was based on rapidly available carbohydrate measured in a time-limited digestion, and not on the exhaustive digestion used in usual available carbohydrate determination for food labelling. The terminology for both RGI and GGE is, therefore, scientific and accurate.

Secondly, the analysis was conducted in terms of usual food intakes, similar to or the same as the 'reference amounts customarily consumed per eating occasion' (US Food and Drug Administration, 2002), now preferred in USA nutrition labelling and dietetics[27]. The data produced were, therefore, relevant to usual food consumption patterns. By comparing foods in portions of realistic size rather than in equicarbohydrate portions, as used in GI determination, the effects of homeostasis in the clinical results that were obtained and their consequent effect on the relationship between *in vitro* and *in vivo* GGE determinations were deduced. The work reported here has demonstrated the need to build an allowance for the emergent effects of homeostasis into the *in vitro* analysis.

There was a large amount of scatter in the data of the present study. The results of the Bland–Altman analyses (Fig. 1)

showed that the approach taken to correcting for homeostasis was valuable, even if not totally sufficient for all the foods. However, two more recent studies that have examined the effect of allowing for apparent GD support the present findings with improved *in vitro–in vivo* correlations. The study in which the equations for the GD baselines were derived[20], based on a sample of fifteen food intakes, produced an *in vivo* and *in vitro* relationship of

$$\text{GGE}\,in\,vivo = 0{\cdot}99\,\text{net}\,\text{GGE}\,in\,vitro + 0{\cdot}75, \quad R^2\,0{\cdot}90.$$

$$(\text{Bland–Altman equation}: \; y = -0{\cdot}065x + 0{\cdot}68),$$

where y is the *in vitro* net GGE–*in vivo* GGE and x is the mean of net GGE *in vitro* and GGE *in vivo,* and the subsequent study of twenty-four British foods (results yet to be published) yielded relationships of

$$\text{GGE}\,in\,vivo = 0{\cdot}96\,\text{net}\,\text{GGE}\,in\,vitro - 0{\cdot}31, \quad R^2\,0{\cdot}90,$$

$$(\text{Bland–Altman equation}\; y = -0{\cdot}009x + 0{\cdot}98).$$

In the present study, the GGE values determined *in vitro* were able to give a statistically significant prediction of GGE values from human blood glucose responses for all the foods taken together and for five out of eleven of the individual food groupings (A, C, E, G and H; Table 3). Those food groupings that did not give a significant correlation generally contained few foods, and may yield significant predictive equations after adding further values. Although the present work has shown the benefit of allowing for homeostasis when establishing an *in vitro–in vivo* relationship, a very close correspondence between the *in vitro* and *in vivo* GGE values is always difficult to achieve because of the enormous variability in clinical blood glucose response measurements[11,12,28].

A number of factors that could act *in vivo* and not *in vitro*[29,30] may have contributed to the modest correlations between *in vitro* and *in vivo* results, and they may have differed between and within the food groups due to the heterogeneity of food types in each group. Further research may yield additional factors, such as a quantity-dependent factor for delay in gastric emptying, which may further improve *in vitro* prediction of relative *in vivo* responses for certain food groups. For instance, delayed gastric emptying may have been a factor in foods containing a relatively high proportion of fat and of relatively large portion size. Combination foods – savoury (Food grouping J), which showed the greatest dependence of *in vitro* overprediction of *in vivo* GGE with increasing mean GGE dose (Bland–Altman slope 0·255, Table 4), consisted of 250–270 g portions and contained relatively high fat contents.

Given the large clinical variability in blood glucose responses seen here, it perhaps makes good sense, for the purposes of comparing foods, to measure RGI independently of the unstable physiological factors that affect measurements of glycaemic responses such as those used in standard GI determination. It is well established that relative rates of digestion are major determinants of glycaemic effects that can be measured with good precision *in vitro*[25], the intrinsic glycaemic potency of major food sugars (their glycaemic indexes) has been replicated in numerous studies[19], and the glucose dose–glycaemic response relationship is very consistent when expressed on a glucose equivalence basis[8]. Now, dose-dependent rates of GD, as GGE/min, have been determined[20], and in so far as they are reflected in the glucose dose–glycaemic response relationship, they are also likely to be consistent intrinsic human responses to blood glucose loading. Therefore, most of the elements necessary to obtain a valid indication of the RGI of amounts of foods customarily consumed, free of physiological fluctuations, are available. Thus, *in vitro* analysis may soon be able to provide predictions of RGI accurately enough to be used routinely in place of *in vivo* analysis. The *in vitro* analysis is, after all, a modified available carbohydrate determination in which the standard factors are included to introduce nutritional relevance.

There is a tendency to reject the use of *in vitro* measures of glycaemic impact when they cannot accurately predict clinical responses, and an assumption is usually made that the *in vitro* measures are unsuitable in guiding food choices for glycaemic control[30]. However, one may conversely argue that glycaemic response is too inconsistent and too state-dependent, for its measurement under one set of clinical conditions (those of GI determination), with portion sizes that are not customarily consumed, to be any more valid than *in vitro* values as food guides in a community setting. Whether imprecise RGI values from such clinical measurements of glycaemic response are any more effective in dietary management of glycaemia than precise values from *in vitro* digestive analyses which have included factors for physiological relevance is yet to be established.

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References

1. Guigliano D, Ceriello A & Esposito K (2008) Glucose metabolism and hyperglycaemia. *Am J Clin Nutr* **87**, Suppl. 1, 217S–222S.
2. Monro JA & Mishra S (2010) Database values for food-based dietary control of glycaemia. *J Food Comp Anal* (In the Press).
3. Miller-Jones J (2007) Glycaemic response definitions. *Cereal Foods World* **52**, 54–55.
4. Jenkins D, Wolever T, Taylor R, *et al.* (1981) Glycaemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* **34**, 362–366.
5. Monro J (2003) Redefining the glycaemic index for dietary management of postprandial glycaemia. *J Nutr* **133**, 4256–4258.
6. Salmeron J, Manson JE, Stampfer MJ, *et al.* (1997) Dietary fiber, glycaemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* **277**, 472–477.
7. Livesey G, Taylor R, Hulshof T, *et al.* (2008) Glycaemic response and health – a systematic review and meta-analysis:

British Journal of Nutrition

https://doi.org/10.1017/S0007114510000589 Published online by Cambridge University Press

relations between dietary glycaemic properties and health outcomes. *Am J Clin Nutr* **87**, Suppl. 1, 258S–268S.
8. Monro JA & Shaw M (2008) Glycaemic impact, glycaemic glucose equivalents, glycaemic index, and glycaemic load: definitions, distinctions and implications. *Am J Clin Nutr* **87**, 237S–243S.
9. Standards Australia (2007) *Glycaemic Index of Foods (AS4694-2007)*. Sydney: Standards Australia.
10. Wallace A, Monro J, Brown R, *et al.* (2008) A glucose reference curve is the optimum method to determine the Glycaemic Glucose Equivalent values of foods in humans. *Nutr Res* **28**, 753–759.
11. Vega-Lopez S, Griffith J, Ausman L, *et al.* (2007) Interindividual variability and intraindividual reproducibility of glycaemic index values for commercial white bread. *Diabetes Care* **30**, 1412–1417.
12. Wolever T, Vorster H, Björk I, *et al.* (2003) Determination of the glycaemic index of foods: interlaboratory study. *Eur J Clin Nutr* **57**, 465–482.
13. Woolnough J, Monro J, Brennan C, *et al.* (2008) Simulating human carbohydrate digestion *in vitro*: a review of methods and the need for standardization. *Int J Food Sci Technol* **43**, 2245–2256.
14. Englyst HN & Hudson GJ (1987) Colorimetric method for routine analysis of dietary fibre as non-starch polysaccharides. A comparison with gas–liquid chromatography. *Food Chem* **24**, 63–76.
15. Blakeney A & Mutton L (1980) A simple colourimetric method for determination of sugars in fruit and vegetables. *J Sci Food Agric* **31**, 889–897.
16. FOODfiles (2001) *Datafiles of the New Zealand Food Composition Database*. Palmerston North: New Zealand Institute of Crop & Food Research.
17. Stork ADM, Kemperman H, Erkelens DW, *et al.* (2005) Comparison of the accuracy of the hemocue glucose analyzer with the Yellow Springs Instrument glucose oxidase analyzer, particularly in hypoglycaemia. *Eur J Endocrinol* **153**, 275–281.
18. Food and Agriculture Organisation of the United Nations (1998) *Food and Nutrition Paper 66. Carbohydrates in human nutrition. Report of a FAO/WHO Expert Consultation on Carbohydrates*. Rome: FAO.
19. Foster-Powell K, Holt SHA & Brand-Miller JC (2002) International table of glycaemic index and glycaemic load values: 2002. *Am J Clin Nutr* **76**, 5–56.
20. Monro J, Mishra S & Venn B (2009) Baselines representing blood glucose clearance improve *in vitro* prediction of the glycaemic impact of customarily consumed food quantities. *Br J Nutr* **103**, 295–305.
21. Venn B, Wallace A, Monro J, *et al.* (2006) Glycaemic load estimated from glycaemic index does not differ greatly from glycaemic load measured using a standard curve in healthy volunteers. *J Nutr* **136**, 1377–1381.
22. Genstat (2007) 9th ed. Hemel Hempstead, UK: VSN International Ltd.
23. Bland J & Altman D (1999) Measuring agreement in method comparison studies. *Stat Methods Med Res* **8**, 135–160.
24. Brighenti F, Pellegrini N, Casiraghi M, *et al.* (1995) *In vitro* studies to predict physiological effects of dietary fibre. *Eur J Clin Nutr* **49**, S81–S88.
25. Englyst K, Englyst H, Hudson G, *et al.* (1999) Rapidly available glucose in foods: an *in vitro* measurement that reflects the glycaemic response. *Am J Clin Nutr* **69**, 448–454.
26. Goni I, Garcia-Alonso A & Saura-Calixto F (1997) A starch hydrolysis procedure to estimate glycaemic index. *Nutr Res* **17**, 427–437.
27. Food and Drug Administration (US) (2005) Reference amounts customarily consumed per eating occasion. 101.12. Code of Federal Regulations: Title 21 – Food and Drugs (4-1-05 ed.) pp 52–61.
28. Venn B & Green T (2007) Glycaemic index and glycaemic load: measurement issues and their effect on diet–disease relationships. *Eur J Clin Nutr* **61**, S122–S131.
29. Louis-Sylvestre J (1999) Glucose utilization dynamics and food intake. *Br J Nutr* **82**, 427–429.
30. Brand-Miller J & Holt S (2004) Testing the glycaemic index of foods: *in vivo* not *in vivo*. *Eur J Clin Nutr* **58**, 700–701.

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