Addition of different fats to a carbohydrate food: Impact on gastric emptying, glycaemic and satiety responses and comparison with in vitro digestion

Miriam E. Clegg a,⁎, Megan Pratt a, Oonagh Markey b, Amir Shafat b,c, C. Jeya K. Henry a,d

a Functional Food Centre, School of Life Sciences, Oxford Brookes University, Gipsy Lane, Oxford, OX3 0BP, United Kingdom
b Faculty of Education and Health Sciences, University of Limerick, Ireland
c Physiology Department, National University of Ireland, University Road, Galway, Ireland
d Clinical Nutritional Sciences, Brenner Centre for Molecular Medicine, Singapore 117609, Singapore

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A B S T R A C T

In vitro, the addition of lipids to a carbohydrate food has been found to increase the digestibility of starch. In contrast, in vivo studies have shown that the addition of fat to a food can reduce the glycaemic response (GR).
The aim of this study was to assess if delayed gastric emptying (GE) causes reduced GR with the addition of lipids to a carbohydrate food and if a relationship between GR and in vitro digestion of starch exists for high fat foods. Ten healthy volunteers were tested on five occasions after consuming pancakes containing 50 g of available carbohydrate and 202 kcal of sunflower oil, olive oil, butter, medium chain triglyceride (MCT) oil or a control containing no oil. GR was measured using fingerpick blood samples, satiety using visual analogue scales and GE using the 13C octanoic acid breath test. There was a significant difference in GR between the different pancake breakfasts (p = 0.05). The highest GR was observed following the control pancakes and the lowest following the olive oil pancakes. There were significant differences in GE half time, lag phase and ascension time (p < 0.05) between the different pancakes with the control pancakes having the shortest GE time and the MCT pancakes the longest. There was a significant difference in satiety parameters fullness (p = 0.003) and prospective consumption (p = 0.050), with satiety being lowest following the control pancakes. There was a significant inverse correlation between the GR and all satiety parameters. A significant inverse correlation (p = 0.009) was also observed between the digestibility of starch and GE. The paper indicates that the digestibility of starch in vitro does not predict the GR for high fat containing foods.

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1. Introduction

Glycaemic response (GR) is based both upon the digestibility of the carbohydrate and also on the delivery of the carbohydrate for digestion. The effect of fats on GR has been extensively researched with a variety of different results. Many studies have highlighted that the addition of fat to a carbohydrate food does reduce the GR to that food (Henry, Lightowler, Newens, & Pata, 2008; Thomsen, Storm, Holst, & Hermansen, 2003; Thomsen et al., 1999). The majority of studies have found no difference between different lipids (Brynes, Frost, Edwards, Ghatel, & Bloom, 1998; Burdge, Powell, & Calder, 2006; Henry et al., 2008; MacIntosh, Holt, & Brand-Miller, 2003; Mekki et al., 2002; Pedersen, Marckmann, & Sandstrom, 1999; Thomsen et al., 1999; Thomsen et al., 2003), whilst others have shown differences between saturated, polyunsaturated and monounsaturated fats (Gatti et al., 1992; Joannic et al., 1997; Rasmussen, Lauszus, Christiansen, Thomsen, & Hermansen, 1996). Addition of monounsaturated fatty acids appears to both increase and reduce GR compared with saturated fatty acids (Gatti et al., 1992; Rasmussen et al., 1996). Addition of polyunsaturated fatty acids reduces GR more than saturated fatty acids (Gatti et al., 1992) and polyunsaturated fatty acids have a lower GR than monounsaturated fatty acids (Joannic et al., 1997). To-date however, no research has assessed the effect of medium chain triglycerides (MCT) on GR. MCT are triglycerides whose fatty acids have a chain length varying between 6 and 10 carbon atoms in length (Babayan, 1987; Bach & Babayan, 1982). MCT are more water soluble than long chain triglycerides and can hence be absorbed when there are decreased intraluminal concentrations of pancreatic enzymes and bile salts (Fernandes, van de Kamer, & Weijers, 1962), making them easier to digest. MCT are also characterized by a more rapid gastric emptying (GE), both characteristics are likely to impact on the GR to a food.

Several papers examining the effect of different fats on GR have highlighted the role that delivery, in essence GE, may play in the differences in GR between fats. However, to-date no studies have tried to characterize the effect of different saturations of fats on GE and the subsequent relationship with GR and satiety (Joannic et al., 1997; Rasmussen et al., 1996). MCT have been shown to have an accelerated GE (Beckers, Jeukendrup, Brouns, Wagenmakers, & Saris,
when compared to long chain triglycerides primarily due to gut hormones that are released in response to long chain triglycerides but not medium or short chain (de Jong, Hopman, Jansen, & Lammers, 1985). In exercise studies, MCT taken in parallel with glucose resulted in a greater glucose response than glucose alone prior to the beginning of exercise, and was believed by the authors to be due to accelerated GE (Beckers et al., 1992). This accelerated GE with MCT may result in a larger glycaemic response.

Once the delivery of nutrient into the duodenum has been achieved, the next important parameter influencing glycaemic response is the digestibility of the carbohydrate. Our diets contain foods that consist of matrices of merged starch and lipids; for example plant based cereals and legumes. Lipids and emulsifiers form strong complexes with starch that alter its physical and chemical properties (Kaur & Singh, 2000). The result being lipid-starch complexes in which the hydrophobic part of the lipid resides inside the helical structure of the starch molecule disallowing α-amylase access and therefore restricting the biodegradation of starch (Gidley, 2001; Liu, 2005). Considerable research has amassed on understanding how lipid-starch complexes influence the physical and chemical properties of a food, and how the individual nutrients alter physiological functions. However, limited research exists on how lipid–starch complexes may impact physiological parameters such as GE and GR. Since different saturations of fat form lipid-starch complexes to varying extents, the rate of digestibility of starch may differ depending on the type of lipid used (Kaur & Singh, 2000; Raphaeides, Arsenoudi, Exarhopoulos, & Xu, 2010). Previously, in in vitro we have found that addition of lipids to a carbohydrate food does not reduce its digestibility, in fact the opposite occurred (M. E. Clegg et al., 2011). Results from this study indicated that the addition of fats to a starch rich food increases the digestibility of the starch even though the presence of the lipid-starch complex was confirmed through the analysis of viscosity possibly due to bile rapidly breaking down the lipid-starch complex. The digestibility was even more rapid in the lipid-starch emulsion that contained medium chain triglycerides (MCT). However, in in vivo the effect of the addition of fats to GR has resulted in a lower glycaemic response for most oils (Henry et al., 2008), though MCT has not previously been included in any analysis.

Given that the digestibility of lipid-starch complexes in vitro is increased compared to starch alone, it is interesting that the GR in vivo is reduced with the addition of lipid. This apparent contradiction indicates that the effect of fats on GR may be predominantly because of the transit and absorption of the fat as opposed to the digestibility of the starch. The objective of this study was to examine the role that GE plays in the GR to lipid-starch complexes. This was achieved through measurement of GR, satiety and GE following meals containing sunflower oil, olive oil, butter, MCT oil and a control containing no oil.

2. Methods

Ten healthy volunteers (eight female, two males; 26.4 ± 3.9 years; 1.70 ± 0.09 m; 61.8 ± 9.6 kg) were recruited for the study by means of advertisements, flyers and personal communications. Before inclusion in the study, potential volunteers were briefed regarding all aspects of the experiment and were given the opportunity to ask questions. This was followed by a health assessment, which included anthropometric measurements and a health questionnaire (giving details of food allergies/intolerances, metabolic diseases, special dietary needs and smoking habits). Those who fulfilled all the acceptable criteria (BMI: 18.5–24.99 kg/m²; blood pressure: between 110–120 and 75–85 mmHg; age: 18–35 years; fasting blood glucose: 4–6 mmol/l; not on prescription medication; non-smoking; no genetic or metabolic diseases) were included in the study. Physical activity was quantified using Baecke's questionnaire (Baecke, Burema, & Frijters, 1982) and only those not partaking in competitive sports and endurance events were included. On the day prior to a test, volunteers were asked to restrict their intake of alcohol and caffeine-containing drinks and to restrict their participation in intense physical activity (e.g. long periods at the gym, intensive swimming, running, aerobics etc). Volunteers were requested to come to the laboratory between 7 and 8:30 am after a 12-hour overnight fast.

All volunteers gave written informed consent prior to starting and were compensated for their time at completion. The study was conducted according to the guidelines laid down in the Declaration of Helsinki, and was initiated following approval by the Oxford Brookes University research ethics committee.

2.1. Experimental design

Volunteers participated in a randomized, balanced, single-blind study. Volunteers were tested five times in total with a minimum of two days between each test. Prior to the first test, volunteers recorded their diet the day before using a weighed food diary. This diet was then repeated for the day prior to subsequent tests. Volunteers fasted for 12 hours overnight before attending the laboratory in the morning. On arrival in the laboratory the volunteer's height was measured using a stadiometer (Seca Limited, Birmingham, West Midlands, Middlesex, UK) and body weight using the Tanita BC-418 MA (Tanita UK Limited, Yiewsley, UK) before baseline measurements were taken.

2.2. Test foods

Following baseline measurements, volunteers ate the test food. The test food consisted of three pancakes and 200 ml of water. The test foods contained 50 g of available carbohydrate and 202 kcal of either — sunflower oil (22.4 g), olive oil (22.4 g), butter (26.9 g), MCT oil (24.3 g) or a control containing no oil. The pancakes were made up of 58.9 g white flour (Tesco plain flour, Tesco PLC, Cheshunt, Hertfordshire), 54.4 g egg (Tesco free range medium eggs, Tesco PLC, Cheshunt, Hertfordshire), and 90.6 g milk (Tesco semi skimmed milk, Tesco PLC, Cheshunt, Hertfordshire). The pancakes were based on the exact medium fat meal used in our previous in vitro research (M. E. Clegg et al., 2011) (Table 1). The pancakes also contained 100 mg 13C-octanoic acid (Europ-top, France) for measurement of GE. The volunteers consumed the test food and the water at a comfortable pace, within a 15 min period.

2.3. Test procedures

2.3.1. Gastric emptying

The addition of 13C-octanoic acid results in 13CO2 appearance in the breath which was used to calculate emptying from the stomach (Chos et al., 1993). Breath samples were taken prior to meal consumption and every 15 minutes for four hours after the test meal. Breath samples were collected by blowing gently into a 10 ml Exetainer® (Labco, Buckinghamshire, UK) with a drinking straw and replacing the cap just prior to the end of exhalation. Breath samples were analysed using isotope ratio mass spectrometry (DeltaV, Thermo-Fisher Scientific, Hemel Hempstead, UK) and results were expressed relative to Vienna PeeDee Belemnite (V-PDB), an international standard

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for known $^{13}$C composition. The $^{13}$CO$_2$ values were expressed as the excess amount in the breath above baseline and converted into moles. Data were then displayed as percentage of $^{13}$CO$_2$ dose recovered per hour and cumulative percentage $^{13}$CO$_2$ recovered over time. CO$_2$ production was assumed to be 300 mmol/m$^2$ body surface area per hour (Shreewe, Cerasi, & Luft, 1970). Body surface area was calculated using a validated weight-height formula (Haycock, Schwartz, & Wisotsky, 1978). This was then fitted to a GE model developed by Ghoos et al. (1993). For each GE test, the coefficient of determination ($r^2$) between the modeled data from Ghoos et al. (1993) and raw data was calculated and $r^2 >0.95$. From this model several parameters were measured. Lag phase (Tlag) and half time (Thalf) were calculated using the formulae derived by Ghoos et al. (1993). Tlag is the time taken to maximal rate of $^{13}$CO$_2$ excretion (Jackson, Bluck, & Coward, 2004) and is equivalent to the time of the inflection point (Schommartz, Ziegler, & Schadewaldt, 1998). Thalf is the time it takes 50% of the $^{13}$CO$_2$ dose to be excreted (Jackson et al., 2004). Latency phase (Tlat) is the point of intersection of the tangent at the inflection point of the $^{13}$CO$_2$ excretion curve representing an initial delay in the excretion curve (Schommartz et al., 1998). Ascension time (Tasc) is the time course between the Tlat and Thalf, representing a period of high $^{13}$CO$_2$-excretion rates (Schommartz et al., 1998). Illustration of these time points can be seen in Clegg and Shafat (2010).

2.3.2. Glycaemic response

The protocol used to measure the blood glucose response was adopted from that described by Brouns et al. (2005) and is in line with procedures recommended by the FAO/WHO (1998). Blood samples for glucose analysis were obtained at 15, 30, 45, 60, 90 and 120 min subsequent to the start of the meal. Blood was obtained by finger prick using the Unistik 3 single-use lancing device (Owen Mumford, Woodstock, UK). Prior to a finger-prick, volunteers were encouraged to warm their hand to increase blood flow. In order to minimize plasma dilution, fingertips were not squeezed to extract blood but were instead gently massaged starting from the base of the hand moving toward the tips. The first two drops of expressed blood were discarded and the next drop was used for testing. Blood glucose was measured using the HemoCue® 201+ Glucose analyzer (HemoCue Ltd, Dronfield, UK). The HemoCue® is a reliable method of blood glucose analysis (Stork, Kemperman, Erkelens, & Veneman, 2005). To ensure accuracy of data, the HemoCue® instruments used in the study were calibrated against blood glucose analyzer (YSI 2300 stat, YSI Inc., Yellow Springs, Ohio, USA). Fasting blood samples were taken at —5 and 0 min, and the test food was consumed immediately afterwards. Further blood samples were then taken at the times mentioned in the protocol above.

The GR data was converted to ‘the change in GR’ values. The ‘change in GR’ was calculated by computing the difference between the blood glucose concentration at a time point and mean baseline blood glucose concentration. Since it represented the relative increment in the GR at any time point compared to the baseline value, it was this ‘change in GR’ that was used for all further analyses, including incremental area under the curve (IAUC) calculations, blood glucose response curve construction and statistics. The total blood glucose response was expressed as the IAUC ignoring the area beneath the baseline, and was calculated geometrically by adding together the individual areas of the triangles and trapezoids for the entire 120 min testing period (trapezoidal rule) (Brouns et al., 2005; Wolever, 2006).

2.3.3. Satiety

One hundred millimeter continuous line visual analogue scales (VAS) were utilized to measure subjective feelings of hunger, fullness, desire-to-eat and prospective food consumption. The volunteers provided VAS data at baseline (0 min) and at 30, 60, 90, 120, 150, 180, 210 and 240 min after the commencement of eating the test food and after lunch. The specific questions asked were, ‘How hungry do you feel?’; ‘How full do you feel?’; ‘How strong is your desire to eat?’ and, ‘How much food do you think you can eat?’.

2.4. Statistical analysis

Studies on in vivo assessment of GR have been based on 10 volunteers, as reviewed by Brouns et al. (2005) and the FAO/WHO (1998), to take account of inter-individual variations. A sample size of 10 was therefore used in the current study.

Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS) version 17 (SPSS Inc., Chicago, Illinois), and data and figures processed in a Microsoft Excel spreadsheet (2006, Reading, UK). Data are presented with descriptive statistics (mean, standard deviation, standard error, coefficient of variation) where appropriate. Differences in total GR (calculated as IAUC) and GE between test meals were analysed using repeated measures analysis of variance (RM-ANOVA). Comparisons between the trials were done by examining the contrasts within the ANOVA. A Kolmogorov-Smirnov test prior to analysis indicated that all the data sets were normally distributed. VAS data were transformed by natural log and was analysed using a two-way RM-ANOVA with two within subject factors—time and meal. Pearson’s correlation was completed to ascertain correlations between GE, satiety and GR and between in vivo digestion from Clegg et al. (2011) and in vivo GR. The level of significance was set to $p \leq 0.05$.

3. Results and discussion

The current study showed that changes occurred in GR, satiety and GE with the addition of fats to a starchy food. These changes were greater for some fats types than for others and was dependent on the physiochemical properties of the fat.

3.1. Glycaemic response

There was a significant difference in GR between the different pancake breakfasts ($p=0.05$; Fig. 1). The GR was reduced with the addition of certain fats in the current study. The control with no oil had the highest GR and the monounsaturated oil, olive oil had the lowest (Fig. 2). The overall significance between the pancake breakfasts was primarily due to differences between control and MCT and control and olive oil ($p=0.05$).

It has previously been found that GR is reduced with the addition of lipids (Henry et al., 2008; Thomsen et al., 1999; Thomsen et al., 2003). However our results contradict the finding that polysaturated fatty acids tend to lower the GR more than monounsaturated (Joannic et al., 1997). In the current study, butter, the saturated fatty acid had quite a similar GR to the control which is in keeping with the finding...
3.2. Gastric emptying

that, when compared to unsaturated fatty acids, the addition of saturated fats to a carbohydrate food is not as beneficial to GR (Gatti et al., 1992; Rasmussen et al., 1996). However the majority of studies have failed to find a distinction between the effects of different oils on GR (Brynes et al., 1998; Burdge et al., 2006; Henry et al., 2008; MacIntosh et al., 2003; Mekki et al., 2002; Pedersen et al., 1999; Thomsen et al., 1999, 2003).

Research suggests that the change in GR with the addition of different fats may be mediated by several mechanisms. One of these mechanisms includes changes in the insulin response depending on the degree of fat saturation; however research on this topic is somewhat contradictory. Similar to the current finding, Gatti et al. (1992) found no effect on the postprandial blood glucose response after adding 35 g saturated fat to a white bread meal, whereas ingestion of 35 g olive oil or corn oil reduced the GR by 70% compared with white bread, without altering the insulin responses. In contrast, Rasmussen et al. (1996) found that the addition of 100 g butter to potato suppressed the blood glucose response area in volunteers with non-insulin-dependent diabetes mellitus whereas neither 40 nor 80 g olive oil had any influence. Furthermore, intake of butter but not olive oil stimulated insulin release. The contrast of these two studies indicates that, when compared to unsaturated fatty acids, the addition of saturated fats, primarily polyunsaturated fats, increase satiety and the satiety hormone cholecystokinin (CCK) more than saturated fats (Maljaars, Romney, Haddeman, Peters, & Masclee, 2009). CCK is also known to be a strong inhibitor of GE (French, Murray, Rumsey, Sepple, & Read, 1993; Fried et al., 1991) and hence the more rapid GE of the butter pancake breakfast may have been due to a lower CCK response. The GE of the meals showed that the MCT had a much slower GE compared to the other oils except the sunflower oil. This is in direct contrast to previous literature that has shown that GE is accelerated with MCT (Beckers et al., 1992; Kossena et al., 2007). The release of gut hormones that delay GE are increased with triglycerides of chain lengths of 12 or greater (de Jong et al., 1985; McAulughlin et al., 1999). The current MCT oil contained primarily caprylic (C8) and capric (C10) acids. However, the majority of studies that have assessed transit of MCT versus long chain triglycerides have looked at transit within the small intestine as opposed to GE (de Jong et al., 1985; Ledeboer, Masclee, Jansen, & Lamers, 1995) and very few studies have taken into account that when the energy content of each fat is controlled, that MCT has a higher osmolarity. Verkijk, Vecht, Gielkens, Lamers, and Masclee (1997) found that interdigestive motility was the same between MCT and LCT regardless of whether osmolarity or calories were assessed. Lasdas, Isaacs, Murphy, and Sladen (1984) indicated that the addition of MCT to a meal may not result in delayed GE in the same way as LCT, due to the lack of production of gut hormones. However, the higher osmolarity of MCT may also delay GE which could cancel out the effect of the gut hormones on GE.

The finding that MCT delayed GE may be supported by what we found previously in vitro (M. E. Clegg et al., 2011; Maljaars et al., 2009). In vitro, it was found that in the gastric section of the digestion process, the MCT had a rapid release of the starch compared to the other oils. This may have been due to the water soluble nature of the MCTs allowing the lipid-starch complex to dissociate in the stomach and for the MCT to be stabilized within the watery contents of the stomach. However for the other complexes, this did not occur until the bile was added in the in vitro intestinal phase when the other oils rapidly caught up with the MCT pancake in terms of starch digestion. If the starch and lipid were released in the stomach this may have slowed the rate of emptying due to the increased osmolarity of the chyme (MCT having the higher osmolarity already) or by stabilizing the MCT within the watery environment of the stomach, that can cause delayed GE (Marciani et al., 2009).

3.3. Satiety

There was a significant difference in VAS parameters fullness (p = 0.003) and prospective consumption (p = 0.050) but not hunger (p = 0.054) and desire to eat (p = 0.102). For fullness the primary differences existed between the control pancakes and the MCT, olive oil and sunflower pancakes (p < 0.05; Fig. 3). For prospective consumption the differences were between the control pancakes and the MCT, olive oil and sunflower pancakes (p < 0.05). The addition of fat to the pancakes caused them to decrease hunger in most instances. This is not surprising given that 202 kcal of additional energy was incorporated into the meal. The addition of butter, the saturated fat, did not have this effect indicating that that saturated fat does not have the satiating properties of other unsaturated fats. This may be due to a diminished secretion of gut hormones that increase satiety.

Table 2

| Gastric emptying time (half time (Thalf), lag phase (Tlag), latency time (Tlat) and ascension time (Tasc)) for pancakes containing medium chain triglycerides (MCT), sunflower oil, olive oil, butter and control containing no added fat (n=10). | p<0.05 for ANOVA. |
|-----------------|-----------------|-----------------|
| Thalf (min)*    | MCT 182.5 ± 54.1 | Olive Oil 134.3 ± 34.3 | Sunflower 158.2 ± 41.7 | Butter 114.1 ± 28.9 | Control 95.3 ± 12.8 |
| Tlag (min)*     | 68.6 ± 11.9     | 54.8 ± 10.2     | 60.1 ± 11.3     | 61.1 ± 11.3     | 64.3 ± 8.9     |
| Tlat (min)      | 59.3 ± 14.2     | 50.6 ± 5.6     | 52.0 ± 11.3     | 46.7 ± 10.2     | 48.8 ± 11.8     |
| Tasc (min)*     | 211.0 ± 61.4    | 165.8 ± 36.0   | 191.2 ± 36.7   | 147.2 ± 29.0    | 124.8 ± 17.5   |

Fig. 2. Glycaemic response curve for pancakes containing medium chain triglycerides (MCT), sunflower oil, olive oil, butter and control containing no added fat (n=10).
and decrease GE following consumption of saturated fat (Maljaars et al., 2009). The current study has highlighted that saturated fat does not decrease GE times, reduce GR or increase satiety. This builds further support for the argument to reduce dietary saturated fat.

3.4. Correlations

There was a clear correlation between all of the area under the curve (AUC) for the satiety parameters and GR (p=0.004) with the higher the GR the lower the satiety (Fig. 4). The relationship between satiety and GR is clearly established (Warren, Henry, & Simonite, 2003) and the current study even further confirms this with the lower GI the more satiating the food.

There were no correlations between any GE parameters and GR. There was a correlation between GE Tlag and hunger and desire to eat (p = 0.048 and p = 0.042) but not to any other GE parameters. As outlined in the introduction, several papers (Joanic et al., 1997; Rasmussen et al., 1996) have highlighted a need to understand if GE plays a role in the GR to fat containing foods. The current study is the first one to do this. The relationship between GE and GR has previously been confirmed in the extant literature (Lodefalk, Aman, & Bang, 2008) however it appears that this relationship is negated in the current study where fats were added to the meal. And although trends existed between the two, in that the control had the highest GR and fastest GE and the MCT had amongst the lowest GR and slowest GE, there was no significant relationship between the cumulated GR and GE data. This may be due to the differing physical properties and physiological responses caused by the variety of fats, these responses may not just influence delivery of nutrients but also hepatic glucose release, insulin secretion and lipid-starch complex formation which were unfortunately not measured in the current study. However it must also be noted that the GR measurement time, although it followed the FAO/WHO (1998) guidelines, may not have been sufficiently long enough to account for all of GR and providing a meal that had a faster emptying meal or a longer GR test may have obtained a better correlation between the GE and GR results. These GE times may indicate that the use of a standard GR test be used with caution when testing HF foods.

3.5. Comparison with in vitro digestion

Araya, Contreras, Alvina, Vera, and Pak (2002) and Monro and Mishra (2010) found a significant positive correlation between food samples digested in vitro and GR. As the current lipid-starch emulsion was both digested in vitro and in vivo we decided to repeat this technique combining the data from Clegg et al. (2011) with the current GR data using the methods described in Araya et al. (2002) and Monro and Mishra (2010). Here we found that there was a significant inverse correlation between the starch digestion in vitro and GR so that the greater the starch digestion in vitro the lower the GR using the methods described in Araya et al. (2002); p = 0.015, R = −0.346; Fig. 5A) and Monro and Mishra (2010); p = 0.004, R = −0.402; Fig. 5B). Although previous studies have shown a direct relationship between in vivo and in vitro data for GR (Araya et al., 2002), the current study tells us that in vitro digestion models for GR should be used with some caution especially in mixed foods containing high amounts of fat. These two methods use different techniques for comparison of in vitro data to in vivo data, one compares the area under the under the digestion curve (Monro & Mishra, 2010), the other compares the rapidly digesting

![Fig. 3. Visual analogue scale response to the question “How full do you feel?” for pancakes containing medium chain triglycerides (MCT), sunflower oil, olive oil, butter and control containing no added fat (n=10).](image1)

![Fig. 4. Relationship between glycaemic response and area under the visual analogue scale curve for the parameter “hunger” (n = 50; p = 0.004).](image2)

![Fig. 5. A: Correlation between carbohydrate digestion rate, expressed as A/B ratio where A = rapid digestion carbohydrates (percentage carbohydrate hydrolyzed at 20 min) and B = lente digestion carbohydrate (percentage carbohydrate hydrolyzed at 120 min), and glycaemic response. B: Correlation between carbohydrate digestion rate expressed as total incremental area under the glucose curve (iAUC), and glycaemic response.](image3)
starch to the slow digesting starch (Araya et al., 2002). For both methods the inverse relationship between in vitro and in vivo data holds true. However, the results from the previous study (Clegg et al., 2011) used a set amount of bile and pancreatin as outlined in their standard protocols. This may have stifled the results as the amount that would be released in vivo would be representative of the amount of fat released in the food rather than a fixed amount. In the current comparison all of the pancakes had the same macronutrient content with the exception of the control pancakes. It could be interpreted that the control data still the findings in Fig. 5 so that the inverse relationship between the starch released in vitro and the GR in vivo does not exist. Yet, when the data is reanalysed without the control data a trend towards this significant relationship still exists (Araya et al., 2002; p = 0.083; R — 0.285 and Mono & Mishra, 2010; p = 0.056, R = — 0.305).

4. Conclusion

The current study indicates that the interpretation of GR is difficult as it cannot be easily predicted nor can the physiological factors that influence be simply defined unless the full extent of transit and glucose uptake and disposal are taken into consideration (Mono, Mishra, & Venn, 2011). Our findings suggest that that GE is not a good predictor of GR and that in vitro assays cannot be used to predict GR in vivo for high fat foods. The current study highlights that the impact of a food during digestion can dramatically influence the physiological outcome of the nutrients in it. It must, however, be remembered that the current study examined a wide range of different fats, more than has been used in previous studies which are each known to evoke different responses and interact in different ways with their surrounding environments.

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