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Gastrointestinal transit, post prandial lipaemia and satiety following three days high-fat diet in men

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Running head: High fat diet accelerates gastric emptying

ABSTRACT

Background: High fat diets of two weeks have been shown to accelerate gastric emptying (GE). To date no studies have shown any alteration in GE following shorter HF diets. **Objective:** To assess if a high-fat, high-energy (HF) diet of three days can adapt gastrointestinal (GI) transit, blood lipids and satiety. **Methods:** Eleven male volunteers participated in a study consisting of three, three-day interventions each separated by a test day. During the first intervention volunteers recorded their diet. In the second and third interventions volunteers repeated their food diary plus either a low-fat (LF) yogurt or HF yogurt supplement in randomized order. Test days involved measurement of GE using the ^{13}C OBT, mouth-to-caecum transit time (MCTT) using the inulin H_2 breath test and satiety using visual analogue scales. Blood samples for measurement of lipaemia were taken using venous cannula. **Results:** MCTT was different between the three test days ($p=0.038$) with the shortest MCTT following the HF intervention. GE was shortest following the HF intervention. There were no differences in satiety between the interventions. The HF intervention reduced triglycerides, total cholesterol and LDL cholesterol and increased HDL cholesterol. **Conclusion:** This study shows that changes in GI transit due to a HF diet can occur in a time period as short as three days.

KEYWORDS: gastric emptying, lipaemia, mouth to caecum transit time, satiety

INTRODUCTION

The obesity epidemic escalated from about 1980 and has been rising relentlessly ever since (James, 2008). Overconsumption of high-energy-dense food is among the contributors to positive energy balance (Rolls, 2000). Our current environment is characterized by an unlimited supply of convenient, relatively inexpensive, highly palatable, energy dense foods (Hill & Peters, 1998).

It is well documented that eating a high-fat (HF) meal in comparison to a low-fat (LF) meal reduces gastric emptying (GE) rates (Cecil *et al.*, 1999) to the extent that it can delay GE of the following meal (Clegg & Shafat, 2010a). HF diet intervention studies have shown the opposite effects in that GE, especially of HF food, is accelerated. However, the time frame over which this occurs has never been closely examined. Cunningham *et al.* (1991) showed that fourteen days on a HF diet significantly decreased GE T_{half} from 147 (88-206) (mean (range)) to 98 (80-116) minutes and mouth-to-caecum transit time (MCTT) from 360 (130-350) to 240 (200-520) min. Similar findings by Castiglione *et al.* (2002) showed a HF diet for 14 days increased GE rate of HF food (0.36 ± 0.05 pre diet vs. 0.47 ± 0.03 %/min post diet (mean \pm SE)) but not LF food. Boyd *et al.* (2003) examined the effect of a duodenal lipid infusion on antropyloroduodenal pressures, following 14 days on either a HF or a LF diet. Results showed the amplitude and tone of the isolated pyloric pressure waves was less following the HF diet compared to the LF diet. This decrease in pyloric contractile force is likely to contribute to acceleration in GE.

The presence of food in the stomach causes both satiation and satiety (Geliebter, 1988) and delaying the emptying of food leads to a prolonged satiety period. There is a school of thought that this increased GE caused by a HF diet can reduce the ability of the stomach to retain chyme

causing reduced satiety, increased food intake (French *et al.*, 1995; Boyd *et al.*, 2003) and subsequent obesity. This may explain the accelerated GE that has been observed in obese individuals (Cardoso-Junior *et al.*, 2007; Clegg & Shafat, 2009). Following the two weeks HF diet, satiety recorded through food diaries and visual analogue scales (VAS) showed tendencies towards decreased fullness, increased hunger and increased food intake after two weeks on a HF diet (French *et al.*, 1995). Following a two week HF or LF diet, hunger was greater during an oral fat tolerance test following the HF diet (Boyd *et al.*, 2003) and two weeks on a high fat diet is also associated with increased maximum tolerated stomach volume (Park *et al.*, 2007). Together, these observations suggest that two weeks HF diet can reduce the satiating effect of fat. It is unclear whether these effects occur after a shorter adaptation period.

Accelerated GE has significant consequences for the development of diabetes and obesity, independent from dysregulation of appetite (Clegg & Shafat, 2009). Accelerated GE may also result in larger and earlier peaks in both plasma triglycerides and glucose concentrations (Liddle *et al.*, 1988; Darwiche *et al.*, 2001). These changes can lead to the development of insulin insensitivity (Temelkova-Kurktschiev *et al.*, 2000; Ridker, 2008) and atherosclerosis.

No studies to date have examined the effect of a HF diet on GE over a time period of only 3 days. The aim of the current study was to examine the effects of short term HF dietary intervention on GE, MCTT, satiety and post-prandial lipid absorption.

METHODOLOGY

VOLUNTEER CHARACTERISTICS

Following ethical approval from University Research Ethics Committee eleven healthy male volunteers (24.7 ± 3.1 yrs; 182 ± 8 cm; 81.7 ± 9.3 kg) were recruited to take part in this study. All procedures were in accordance with the ethical standards of the institution on human experimentation and written informed consent was obtained. Before participation all volunteers completed a health history questionnaire to ensure that they had no medical ailments that would compromise their participation. None had any history of gastrointestinal (GI) disorder or suffered from GI upset before or during the study.

EXPERIMENTAL DESIGN

Volunteers participated in a randomized, single blind, crossover design; attending the laboratory for three test sessions with three days between each test session. During the three days, prior to the first trial (control) volunteers followed their usual diet and completed a weighed food diary. Prior to the remaining two trials volunteers repeated their food diary with the addition of either a LF or HF yogurt given in randomized order.

DIETARY SUPPLEMENTATION

The volunteer's diet was supplemented with either a yogurt only (low-fat, LF) for three days, or yogurt and oil combination for three days (high-fat and high energy, HF). The LF and HF interventions were completed in randomized order. The LF intervention was completed to control for any possible effects of the yogurt itself. The LF intervention consisted of 260g of yogurt; the HF intervention consisted of 260 g of yogurt and 90g of sunflower oil combined to form a homogenate mixture; per day for a three-day period. The LF yogurt consisted of 858 kJ (205 kcal), 8 g of fat, 15 g of protein and 20g carbohydrate. The HF yogurt consisted of 4250 kJ (1015 kcal), 98 g of fat, 15 g protein and 20 g carbohydrate. Yogurts could be eaten at any time during the day as desired by the volunteer except during the 12 hours prior to testing.

TEST PROCEDURE

On the test days volunteers arrived at the laboratory following a 12 hour overnight fast. Upon arrival at the laboratory, measurements of body mass and stature were taken from each of the volunteers. Baseline breath H_2 and breath $^{13}CO_2$ samples were collected. Volunteers were given a HF breakfast meal and 15 minutes in which to consume it. If the meal was finished before the allocated 15 minutes the clock was reset to zero and all subsequent measurements were taken from this point onwards. All times presented here are relative to the end of meal consumption. At six hours volunteers were free to leave the laboratory and could eat *ad libitum* for the remainder of the day whilst weighing and recording all food consumed.

HIGH FAT BREAKFAST

The breakfast test meal consisted of three pancakes made from 50g egg (Dunnes stores free range large eggs, Ireland), 37g plain white flour (Dunnes stores plain flour, Ireland), 65g whole milk (Dawn Dairies, Limerick, Ireland), 40g of sunflower oil (St Bernard sunflower oil, Dunnes stores, Ireland), 12g of inulin (Raftiline HP, Orafiti, Belgium) and 100mg ¹³C octanoic acid (Euriso-top, France). This was served with 30g of chocolate spread (Panda, Boyne Valley foods, Ireland) and 200ml water (Kerry spring water, Dingle, Ireland). The meal consisted of 2504 kJ (599 kcal), 40.4 g of fat, 14.5 g of protein and 48.2 g of carbohydrate.

MCTT, GASTRIC EMPTYING AND SATIETY

Breath hydrogen (Micromedical H₂ meter, UK) and breath air samples were taken at baseline and every 10 minutes throughout the 6 hours following breakfast. These were used for the analysis of MCTT and GE, respectively. The test meal contained 10g non-digestible substrate, inulin. Inulin is present as a plant storage carbohydrate in a number of vegetables and plants including bananas, onion, garlic, wheat and chicory. High performance inulin HP is manufactured to remove the shorter chain molecules so it has an average degree of polymerization of 25 (Niness, 1999). Upon reaching the caecum, inulin is metabolized by colonic bacteria, hydrogen gas is released which can be detected in end exhalation breath. MCTT was defined as a consecutive increase in breath hydrogen over three consecutive readings of at least a cumulative 10 ppm (Bond *et al.*, 1975; Geboes *et al.*, 2003). Geboes *et al.* (Geboes *et al.*, 2003) found that MCTT correlated best with lactose ¹³C-ureide when high performance raftilin was used and that there was no proportional

bias between the two techniques. Inulin has also been shown to effect GE to much lesser extent than other substrates such as lactulose (Clegg & Shafat, 2010b).

Octanoic acid is firmly retained in a standard solid test meal in the gastric environment. However, in the duodenum octanoic acid is rapidly absorbed from the chyme and carried via the portal venous system to the liver. Here it is rapidly and completely oxidized to labelled $^{13}\text{CO}_2$ which is exhaled into the breath which can be used to give a measure of gastric emptying (Ghoos *et al.*, 1993). Following the addition of 100mg ^{13}C octanoic acid to the test meal $^{13}\text{CO}_2$ breath samples were taken every 15 minutes for six hours. Breath samples for measurement of $^{13}\text{CO}_2$ were analyzed using isotope ratio mass spectrometry (ABCA, Sercon LTD, Chesire, UK) and results were expressed relative to V-PDB, an international standard for known ^{13}C composition. $^{13}\text{CO}_2$ values were expressed as the excess amount in the breath above baseline and converted into moles. Data are displayed as percentage of $^{13}\text{CO}_2$ dose recovered per hour and cumulative percentage $^{13}\text{CO}_2$ recovered over time. Carbon dioxide production was assumed to be 300 mmol/m² body surface area per hour. Body surface area was calculated using a validated weight-height formula (Haycock *et al.*, 1978). This was then fitted to a GE model developed by Ghoos *et al.* (1993). For all the data, r^2 coefficient between the modeled and raw data was calculated and $r^2 > 0.95$. From this model several parameters were measured. Lag phase (T_{lag}) and half time (T_{half}) were calculated using the formulae derived by Ghoos *et al.* (1993). T_{lag} is the time taken to maximal rate of $^{13}\text{CO}_2$ excretion and is equivalent to the time of the inflection point. T_{half} is the time it takes 50% of the ^{13}C dose to be excreted. Latency phase (T_{lat}) and ascension time (T_{asc}) were derived from formulae developed by Schommartz *et al.* (1998). T_{lat} is the point of intersection of the tangent at the inflection point of the $^{13}\text{CO}_2$ excretion curve representing an initial delay in the excretion curve. T_{asc} is the time course between the T_{lat} and T_{half} , representing

a period of high $^{13}\text{CO}_2$ -excretion rates. Further graphical representation of these timepoints can be found in Clegg *et al.* (2010b). Scintigraphic half time (T_{halfS}) and lag phase (T_{lagS}) are scintigraphic equivalent values developed by Ghooos *et al.* (1993) to be comparable to data from scintigraphic GE measurements. The total percentage dose recovered, the peak in percentage dose recovered per hour and the time at which the peak (T_{peak}) occurred were obtained from the raw (unmodelled) data.

Satiety was measured using a 150 mm VAS to detect changes in hunger, thirst, desire to eat, tiredness, fullness and cold every 30 minutes throughout the six hours. Variables thirst, tiredness and cold were used to distract volunteers from analysis of their satiety.

BLOOD SAMPLES

Blood samples were collected at baseline and following breakfast every 10 minutes for the first 30 minutes followed by every 30 minutes until six hours. Serum separating clot activator tubes were allowed to clot at room temperature (for 15 minutes) before centrifugation began at 3500 rpm for 5 mins in 4°C . Serum was removed and transferred to 1.5 ml plastic vials; these were stored at -70°C for subsequent biochemical analysis.

BIOCHEMICAL ANALYSIS

Methods for biochemical analysis of blood glucose, total cholesterol, HDL cholesterol (HDL-C) and triglycerides has been described previously (Clegg *et al.*, 2007). Estimates of LDL-C

concentration were calculated using the Friedewald formula (Friedewald *et al.*, 1972). CV was less than 2.5% for all blood glucose and lipid concentrations.

DIETARY ANALYSIS

Food diaries were analyzed for macronutrient content and energy intake using Compeat Pro Version 5.8 using McCance and Widdowson's 6th Edition food tables (Nutrition Systems, Grantham, UK).

STATISTICAL ANALYSIS

Statistical analysis was performed using the SPSS version 15.0 (Surrey, UK). All data except VAS data was tested for normal distribution. Dependent on the results of these tests, data was examined using either a repeated measures analysis of variance (rmANOVA) or the Friedman test for non parametric data. Trials were compared by examining the contrasts within the ANOVA or by the Wilcoxon sign rank test. VAS data were transformed by natural log and analysed using a 2-way rmANOVA with two within subject factors - time and condition. Comparisons between the trials were done by examining the contrasts within the ANOVA. All data are expressed as mean \pm SD unless otherwise stated and the significance level was set at $p < 0.05$.

RESULTS

DIETARY INTAKE

There were significant differences in dietary intake between the interventions for energy ($F(2,10)=255.792$, $p<0.001$), fat ($F(2,10)=2232.310$, $p<0.001$) and carbohydrate ($F(2,10)=18.612$, $p<0.001$) but not for protein ($F(2,10)=3.989$, $p=0.061$). Intakes of energy, carbohydrate and fat were greatest following the HF intervention. Daily intake of dietary fat was 88g higher on the HF intervention indicating volunteers were compliant with the dietary manipulation (Table 1).

GASTRIC EMPTYING

There were significant differences between the parameters T_{lat} ($\chi^2(2,10)=11.091$, $p=0.004$) and T_{peak} ($F(2,10)=3.523$, $p=0.049$) between the three interventions. The LF intervention did not affect GE. The HF intervention accelerated the initial stages of GE as measured by T_{lat} , and T_{peak} compared to the control and LF intervention (Table 2). There were no differences between the other parameters of GE ($p>0.05$).

MCTT

Differences existed in MCTT between the three interventions ($F(2,10)=4.186$, $p=0.038$). MCTT decreased significantly between the control and HF intervention (control 280 ± 60 min; HF intervention 226 ± 84 min; $p=0.025$), no differences existed in MCTT between the LF intervention

(291 ± 47 min) either the control and the HF intervention (both $p > 0.05$). An example of one volunteer's breath hydrogen concentrations can be seen in Figure 1.

BLOOD LIPIDS

Plasma triglyceride concentrations differed following the interventions ($F(2,10)=5.176$, $p=0.015$) and across time ($F(2,10)=16.745$, $p<0.001$). The differences exist between control and high fat ($p=0.004$) and between LF and HF ($p=0.025$) but not between control and LF ($p>0.05$).

Triglycerides were lower following the HF intervention, both in the fasted state and post-prandially.

For total cholesterol there were differences between interventions ($F(2,11)=19.299$, $p<0.001$) and across time ($F(2,11)=3.206$, $p<0.001$). The differences exist between base and HF ($p<0.001$) and control and HF ($p<0.001$) but not between LF and HF ($p>0.05$). Total cholesterol was lowest following the HF intervention.

For LDL cholesterol there were differences between interventions ($F(2,10)=4.165$, $p=0.031$) and across time ($F(2,10)=16.068$, $p<0.001$). HF LDL was lower than control ($p=0.008$) but no differences existed between control and LF or LF and HF ($p>0.05$).

For HDL cholesterol there were no differences between the interventions ($F(2,10)=2.597$, $p=0.99$) but there was a difference across time ($F(2,10)=5.324$, $p<0.001$). HF HDL was

significantly higher than LF ($p=0.045$) but there were no differences between the other tests (Figure 2).

For glucose there were no differences between the interventions ($p>0.05$), there was a differences across time ($F(2,10)=24.745$, $p<0.001$). Glucose increased over the first 30 minutes postprandially and then declined in all three tests. Plasma glucose concentration remained relatively stable around fasting values from 60 minutes to 6 hours in all tests.

SATIETY

Results from the VAS showed that there was no difference between the three interventions for any of the parameters of hunger, desire to eat and fullness ($p>0.05$). However, there was an effect of time for all three parameters, with volunteers became less satiated over the six hour period for hunger ($F(2,10)=39.263$, $p<0.001$), desire to eat ($F(2,10)=42.919$, $p=0.001$) and fullness ($F(2,10)=29.897$, $p=0.005$). There were no significant differences in energy or macronutrient dietary intakes on the evening of each test session ($p>0.05$) (Table 2).

In summary, three days high-fat diet accelerated the initial phase of gastric emptying, reduced MCTT, decreased fasting and postprandial lipaemia without affecting hunger sensation or food intake.

DISCUSSION

The results of this study showed that GE T_{lat} and the T_{peak} were shorter following the HF intervention. These results extend findings from studies that have examined the effects of a two week HF diet (Cunningham *et al.*, 1991; Castiglione *et al.*, 2002). There are two factors that may explain why current data demonstrate acceleration in GE while others (Cunningham *et al.*, 1991) found little or no differences over a similar time period: 1. The intensity of nutritional change and 2. Fatty acid specific adaptation. The percentage energy provided from fat in the Cunningham *et al.* (1991) study was 55% and energy intake was 19.26MJ. In the current study the energy intake from fat increased by 15% from 31% to 46% and daily energy intake during the HF intervention was 15.46MJ. In the Cunningham *et al.* (1991) study, the entire diet of the volunteers was changed compared to the current study where the volunteer's diet was supplemented with yogurt and oil. Hence, the volunteers in Cunningham *et al.* (1991) were subjected to a wide variety of fatty acids. In the current study the volunteers were subjected to a narrow range of fatty acids from sunflower oil hence primarily linoleic acid (18:2, LA). It is known that the GI tract responds very specifically to different fatty acids (Robertson *et al.*, 2002; Maljaars *et al.*, 2009). Any adaptation is hence going to be specific to the effects of that fatty acid. It is interesting to speculate on the potential mechanisms underlying the adaptation of gastrointestinal transit to chronic fat intake. Fat sensing in the gut has, for many years, been an established observation without a mechanistic explanation. Recently, G Protein coupled Receptor 120 (GPR120) was cloned (Moore *et al.*, 2009), shown to be sensitive to fatty acids (Burns & Moniri, 2010) and to induce CCK secretion *in vivo* (Tanaka *et al.*, 2008). Several steps in this sensory cascade may explain desensitisation to fat including, but not limited to, receptor expression down regulation, expression of different isoforms of the receptor (Burns & Moniri, 2010) and further intracellular

steps in the enteroendocrine cells. Furthermore, GPR 120 is just one member of a large family of luminal receptors sensitive to fat and it may well be the anatomical distribution and relative expression of these receptors that determine gastrointestinal sensitivity to ingested fat. In the current study, the major component of fat in the test meal was also in the form of the same oil – sunflower oil. In this way the volunteer's GI system only had to become accustomed to a narrow range of fatty acids, primarily LA (Cummins *et al.*, 1967). The smaller dietary change in the current study may allow faster adaptation of the GI tract as it only had to adapt to a specific set of fatty acids in this controlled HF diet.

As T_{lat} represents the first portion of the meal to empty from the stomach it corresponds with evidence that suggests the feedback mechanisms from the small intestine have been disrupted or in this case perhaps delayed (French *et al.*, 1995). The ileal brake and intestinal transit time have been proposed as targets for the modulation of appetite, food intake and adiposity (Maljaars *et al.*, 2008). MCTT was accelerated in the HF group compared to the control group ($p=0.025$). As MCTT represents the time that the head of the meal reaches the caecum, this is in keeping with the GE data that found that the first portion of the test meal, the latency phase, emptied faster following the HF intervention. The shortening of MCTT is also in agreement with animal studies demonstrating desensitization of the ileal-brake to chronic infusion of fat (Brown *et al.*, 1994). Other mechanisms such as duodenal brake may also explain these observations (Shahidullah *et al.*, 1975). The number and distribution of fatty acid sensitive receptors along the GI tract (Engelstoft *et al.*, 2008) means that classifications into gross anatomical structures may be less useful than actual nutrient sensitivity. In humans, Cunningham *et al.* (1991) found that MCTT was shorter after 14 days on a HF diet. This current data indicates that transit as far as the large

intestine is faster after only three days on a HF diet. What implication does this have for post prandial lipaemia?

Surprisingly, fasted and postprandial triglycerides and cholesterol (total and LDL) were reduced by three days on a HF diet. The main source of fat added to the volunteer's diet was sunflower oil whose primary fatty acid is LA (Cummins *et al.*, 1967). LA is a polyunsaturated fatty acid (PUFA) that has been shown to lower serum levels of LDL cholesterol. The substitution of PUFA (the vast majority being LA, varying from 0.6% to 28.8% energy) for carbohydrates has more favorable effects on the total:HDL-cholesterol ratio than any class of fatty acids (Mensink *et al.*, 2003; Harris, 2008). Although GE was accelerated following the HF intervention, the lack of change in satiety and lipaemia implies that HF diet is unlikely to impact negatively on an individual's health.

The effects of high-fat diets and the intestinal mechanisms leading to dysregulation of appetite have been recently reviewed (Little *et al.*, 2007). In the current study satiety values and food intake were no different on the test day following each intervention. Castiglione *et al.* (2002) similarly found no difference in satiety following either HF or LF test meals or before and after a two week HF diet. Boyd *et al.* (2003) found that following a two week HF or LF diet, hunger was greater during an oral fat tolerance test following the HF diet, however this did not affect subsequent food intake. Similarly, ileal infusion of fatty acids 18:1 or 18:2, but not 18:0, reduced appetite but not food intake (Maljaars *et al.*, 2009). Park *et al.* (2007) found no changes in GI transit or appetite following a high-fat diet intervention. Using food diaries French *et al.* (1995) found increases in food intake following a two week HF diet. However, in the current study there

were no changes in satiety and food intake and it appears that although three days is sufficient to change GE it is not sufficient an adaptation period to alter satiety.

The current set of data shows that a three-day HF diet is sufficient to accelerate GE and MCTT of a HF breakfast. This, however, did not affect satiety and food intake. This is the first study to show changes in GI transit over only three days and indicates that short term changes in diets can impact GI processing. However, the lipaemia data indicates that in the short term the three-day diet was not detrimental to cardiovascular health and cholesterol and triglyceride levels were improved. A longer adaptation may influence lipaemia and satiety as has been previously demonstrated. This study, as with many studies on this topic, consisted not just of a HF diet but also a high energy diet. There is scope here to further understand if the acceleration in GI transit occurs to such an extent with a solely HF (not high energy) diet in such a short time span. However, this study provides a valuable insight into how GI processing is altered following three days dietary modification.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Table 1: Average daily energy and macronutrient consumption during each of the three intervention periods and energy and macronutrient intake on the day of testing following each of the dietary intervention periods: control, LF yogurt and HF yogurt. Data is mean±S.D for n=11

Daily energy and macronutrient consumption			
	Control	LF	HF
Energy (kcal)	2643±414	2865±469†	3693±417†*
Energy (kJ)	11066±1733	11242±1964†	15462±1746†*
Protein (g)	103.6±19.6	108.3±22.0	108.4±20.1†
Fat (g)	89.5±14.7	97.5±15.6†	187.3±15.4†*
Carbohydrate (g)	353.5±71.2	393.3±80.6†	400.3±66.9†
Energy and macronutrient intake post testing			
Energy (kcal)	1833±684	1818±692	1835±967
Energy (kJ)	7674±2864	7612±2897	7683±4049
Protein (g)	69.0±23.0	67.1±28.4	72.2±33.6
Fat (g)	76.9±36.8	72.5±29.7	68.6±46.0
Carbohydrate (g)	225.7±81.5	235.7±112.5	214.6±99.9

† p = < 0.05 vs. control * p = < 0.05 LF vs. HF

Table 2: Gastric emptying parameters from the ^{13}C octanoic acid breath following each of the three intervention periods: control, LF yogurt and HF yogurt. Data is mean \pm S.D for n=11. † p = < 0.05 vs. control * p = < 0.05 LF vs. HF.

Time (min)	Control	LF	HF
T_{halfS}	176 \pm 95	156 \pm 39	150 \pm 48
T_{lagS}	62 \pm 17	59 \pm 11	54 \pm 14
T_{asc}	213 \pm 95	193 \pm 40	194 \pm 49
T_{lat}	50 \pm 13	48 \pm 12	40 \pm 7*†
T_{half}	263 \pm 106	241 \pm 44	234 \pm 54
T_{lag}	159 \pm 53	148 \pm 26	136 \pm 26
T_{peak}	168 \pm 40	160 \pm 34	132 \pm 28* †
Total dose recovered (%)	38.8 \pm 6.8	38.5 \pm 3.4	40.0 \pm 4.2
Peak % dose	9.19 \pm 2.03	9.18 \pm 0.80	9.60 \pm 1.04

T_{halfS} - Scintigraphic equivalent gastric emptying half time; T_{lagS} - Scintigraphic equivalent lag phase; T_{asc} – gastric emptying ascension time; T_{lat} – gastric emptying latency phase; T_{lag} – gastric emptying lag phase; T_{peak} – time of the peak in the gastric emptying curve from the unmodelled data; total dose recovered (%) – total percentage ^{13}C dose recovered taken from unmodelled data; peak % dose – peak in the percentage ^{13}C dose recovered taken from the unmodelled data.

FIGURE HEADINGS

Figure 1: A typical volunteer's breath H₂ data following each of the three interventions: control, LF yogurt supplement and HF yogurt supplement. Mouth-to-caecum transit time is marked by a black arrow.

Figure 2: Blood lipids – (a) total cholesterol, (b) HDL cholesterol and (c) triglycerides prior to and following a high fat breakfast following either periods of control diet, diet supplemented with yogurt (LF) or diet supplemented with HF yogurt (HF) for n=11. Data is given as mean±S.D. Error bars for the LF values were similar in magnitude to other data on the same graph and are not presented to improve clarity.







