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Title: Influence of Habitual Physical Activity on Gastric Emptying in Healthy Males and Relationships with Body Composition and Energy Expenditure

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Running Head: Activity, Body Composition, Gastric Emptying

ABSTRACT

Although a number of studies have examined the role of gastric emptying (GE) in obesity, the influences of habitual physical activity level, body composition and energy expenditure (EE) on GE have received very little consideration. In this study, we have compared GE in active and inactive males, and we have characterised relationships with body composition (fat and fat free mass) and EE. Forty-four males (Active: n=22, Inactive: n=22; range BMI 21-36kg/m^2; range percent fat mass 9-42%) were studied, with GE of a standardised (1676 kJ) pancake meal being assessed by ^{13}C-octanoic acid breath test, body composition by air displacement plethysmography, resting metabolic rate (RMR) by indirect calorimetry and activity EE (AEE) by accelerometry. Results showed that GE was faster in active compared to inactive males (mean ±SD half time ($t_{1/2}$): Active: 157±18 and Inactive: 179±21 min, $p<0.001$). When data from both groups were pooled, GE $t_{1/2}$ was associated with percent fat mass ($r=0.39$, $p<0.01$) and AEE ($r=-0.46$, $p<0.01$). After controlling for habitual physical activity status, the association between AEE and GE remained, but not that for percent fat mass and GE. BMI and RMR were not associated with GE. In summary, faster GE is considered to be a marker of a habitually active lifestyle in males, and is associated with a higher AEE and lower percent fat mass. The possibility that GE contributes to a gross physiological regulation (or dysregulation) of food intake with physical activity level deserves further investigation.

Keywords: body composition; energy expenditure, gastric emptying; physical activity.

INTRODUCTION

Gastric emptying (GE) has a fundamental role in the digestion of nutrients and is a major determinant of postprandial glycaemia[1] and gastric symptoms[2,3]. In addition, altered GE has been implicated in the pathogenesis of overconsumption leading to weight gain and obesity[4-10]. Over the last 30 years a number of studies have investigated this possible linkage but with conflicting outcomes indicating that the role of GE in obesity is still unclear. Accelerated[6-8], similar[11-13], and delayed[10,14-16] emptying rates have been reported when comparing obese with lean individuals. This inconsistency has generally been attributed to methodological differences and limitations (e.g. meal size, gender)[17]. Another possibility is that inconclusive findings may be due to the influence of additional unmeasured or uncontrolled factors, for example habitual physical activity level, body composition (fat mass (FM) and fat free mass (FFM)) and energy expenditure (EE).

When considering metabolic health, the importance of body composition[18] and physical activity level[19] is becoming increasingly apparent. Furthermore, body composition, but not BMI has been shown to be associated with daily energy intake in obese adults[20]. However to date, BMI or ideal body weight has been the major criterion for distinguishing obese and non-obese groups in GE studies[6-8,10-15]. To the best of our knowledge, only two studies have reported directly on body composition (FM and/or FFM)[8,13]. Vasquez-Roque et al.[13] characterised gastric functions in normal weight, overweight and obese individuals categorised by BMI and reported lean mass. Although no significant differences were found between groups, increased body weight was associated with faster GE. In another cross sectional study, Mathus-Vliegen et al.[8] reported a faster solid emptying in taller subjects with a greater FFM, and in subjects with more intra-abdominal fat. These findings suggest a possible relationship between body composition and GE, yet further studies are clearly needed to establish this hypothesis further. Despite numerous studies examining the role of GE in obesity, body composition has received very little attention.

Differences in physical activity and EE may also influence GE. Exercise is known to improve leptin sensitivity via reducing fat mass[21,22] which some evidence in animals suggests may interact with gut hormones such as cholecystokinin (CCK) and vagal afferent fibres to influence gastric motility[23]. It is acknowledged that habitual activity, EE and body composition are interrelated. Indeed, a higher activity EE (AEE) can also arise in obese individuals due to the greater energy cost of activities associated with increased body weight[24]. However, the influence of resting EE or AEE on GE is unknown. Evidence that GE is faster in marathon runners[25] compared to inactive individuals arises from a single quarter-century old study by Carrio et al.[25] They identified faster GE in ten marathon runners compared to ten inactive individuals but body surface area was the only proxy characteristic of body composition reported and EE was not measured.

Given the growing interest in targeting the gastrointestinal (GI) tract for the treatment of obesity and diabetes[4,26-28], it is pertinent that a better understanding of factors influencing GE is established. In addition, given the role of the GI tract in satiation and satiety[16,26,29,30], understanding associations between physical activity and GE may provide potential mechanistic insight into processes contributing to appetite regulation with exercise. The aims of the present study were to examine and compare GE in habitually active and inactive individuals across a continuum of body compositions (including lean and obese) and to determine associations amongst habitual exercise, body composition, EE and GE.

MATERIALS AND METHODS

Participants

Forty-four males were studied. Inclusion criteria were: male, aged 18-55 yrs, BMI 18-40 kg/m^2, weight stable (±4 kg over last 6 months), no history of GI disorder, non-diabetic, no medical conditions and not taking medication known to influence body composition, EE, GE or appetite, willing to consume study test meal, not a heavy smoker (<10 per day) and either inactive (undertaking ≤1 structured exercise session per week and not engaged in strenuous work) or active (undertaking ≥4 structured exercise sessions per week) over the last 6 months. One exercise session was defined as at least 40 minutes of moderate to high intensity activity[(31)]. Based on our previous work[(32)], a sample size of 22 participants per group was identified as sufficient to detect a 10% difference between groups for three out of the four GE outcome measures (t_{lag}, $t_{1/2}$, t_{asc}). This equated to the ability to detect a mean difference of 13 minutes in GE half time ($t_{1/2}$) between groups at 90% power and significance level of 0.5%. The study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures were approved by the Queensland University of Technology Research Ethics Committee. All participants provided written informed consent.

Study Design

After a 12-hour overnight fast, and having avoided alcohol and strenuous exercise for 24 hours, participants attended the laboratory on two separate test days one week apart. Participants were instructed to maintain their typical diet prior to the testing days, in order to be tested in their habitual state. At the first testing session, body composition and resting metabolic rate (RMR) were measured. At the second test session, GE was assessed. Between the two testing sessions, as described further below, participants wore an accelerometer to assess physical activity.

Anthropometry and Body Composition

Height was measured without shoes to the nearest 0.5 cm and weight to the nearest 0.01 kg. Body composition (FM and FFM) was measured using air displacement plethysmography (BodPod™), (Life Measurement, Inc., Concord, CA, USA).

Resting Metabolic Rate

RMR was measured by indirect calorimetry using a ventilated hood system (TrueOne 2400 Metabolic Cart, ParvoMedics, Utah, USA). The participant lay supine in a thermoneutral environment and oxygen uptake, with carbon dioxide production and the respiratory quotient (RQ) being measured over 30 minutes. Resting heart rate was measured continuously (Polar Electro Oy, Kempele, Finland). RMR was calculated using the Weir formula[(33)], as the average resting EE over the 10 minutes with the lowest coefficient of variation (CV)[(34)]. The CV for resting EE was less than 5% for all participants (mean (SD) CV, Active: 3.3 (0.9)%; Inactive: 3.1 (0.8)%).

Physical Activity and Energy Expenditure

Physical activity was monitored using a tri-axial GT3X accelerometer (Actigraph, Pensacola, FL, USA) over seven days prior to the GE test day, a duration estimated to result in 90% reliability[(35)]. Participants were instructed to wear the device on the waist, in line with the right hip during waking hours and to remove it only during contact with water (e.g. showering). Data were processed using ActiLife software (version 6.4.5). Tri-axial vector magnitude (VM3) counts were summed over 60 second epochs and levels of activity were defined as counts per minute according to validated recommendations[(36)]. Data were checked for spurious values (counts per minute of >15,000). A non-wear period was defined as at least 90 minutes of consecutive zero counts without interruption[(37)]. Wear time exceeding 600 minutes was considered a valid day[(38)] and a valid dataset considered a combination of at least three weekdays and one weekend day[(39,40)]. Time spent in moderate and vigorous (combining vigorous and very vigorous) activity was also calculated. Activity count data were converted to AEE using the 'Freedson VM3 combination ('11)' option in Actilife software (version 6.4.5). Total energy expenditure (TEE) was subsequently calculated in Microsoft EXCEL using the following formula:

$$TEE = (AEE + REE) \times 1.11$$

where AEE = activity energy expenditure, REE = resting energy expenditure, and the thermic effect of food is fixed at 10% of TEE[(41)].

Gastric Emptying (GE)

GE parameters were calculated using the ^{13}C-octanoic acid breath test (^{13}C-OBT)[(42)], using an identical procedure to that described previously[(32)]. In brief, the egg yolk of a standardized pancake breakfast meal [1676 kJ (400 kcal); 15g (15%) PRO, 17g (37%) Fat, 48g (48%) CHO)] was labelled with 100mg ^{13}C-octanoic acid (Cambridge Isotope Laboratories, Andover, USA). Participants consumed the meal with a 250ml water drink within 10 minutes. Breath samples were collected in 10ml glass Exetainer tubes (Labco, Buckinghamshire, UK) prior to the breakfast, immediately after, and subsequently every 15 minutes for 5 hours. Participants remained in sedentary activities (reading or working on a computer) and were supervised in the laboratory throughout the test morning.

^{13}C breath test analysis

^{13}C enrichment of breath samples was measured by isotope ratio mass spectrometry (Hydra 20-20, Sercon, Cheshire, UK). Data were analysed according to Ghoos et al.[(42)] To calculate the percent of ^{13}C dose recovered, enrichment values were multiplied by the estimated total CO_2 production (VCO_2) for each individual. Following the procedure outlined by Ghoos et al.[(42)], resting VCO_2 was predicted from body surface area according to Shreeve et al.[(43)]. Body surface area was calculated according to Haycock et al.[(44)]. To determine the influence of the predicted VCO_2 value on results, identical analyses were undertaken using a constant value of measured VCO_2 calculated during the RMR measurement. The conventional uncorrected time based parameters (lag time (t_{lag}) and half time ($t_{1/2}$)) proposed by Ghoos et al.[(42)] and the parameters latency time (t_{lat}) and ascension time (t_{asc}) proposed by Schommartz et al.[(45)] were calculated. The r^2 coefficient between the modelled and raw data was accepted if $r^2 > 0.9$.

Statistical Analysis

All parameters were tested for normality by the Shapiro–Wilk test. Data are expressed as mean ± standard deviation (SD) for normally distributed values and as medians (25th, 75th percentiles) for non-normally distributed values. Differences between groups were assessed by t-test and Mann-Whitney U test. Independent t-tests were used to compare groups split by median values for body composition. Pearson or Spearman correlations where appropriate were used to determine relationships between GE and key variables. Associations were further explored using partial correlations after controlling for group. To identify potential predictors of GE, variables of interest were included in multiple linear regression analysis with GE $t_{1/2}$ and t_{lag} as the dependent variables. The variance inflation factor (VIF) was checked for multicollinearity. Statistical analysis was performed using PASW Statistics 18.0 (SPSS Inc., Chicago, IL, USA) and Graph Pad Prism version 6.0 for Mac (GraphPad Software, San Diego, CA, USA). Statistical significance was set at $p<0.05$.

RESULTS

Participant Characteristics

All participants completed all components of the study (n=22 per group), except for the accelerometery assessment, where there was invalid data for three participants in the inactive group. In the combined cohort, the range of percent FM and BMI were 9-42% and 21-36kg/m^2 respectively. Eight individuals were classified as obese by BMI (n=7 inactive), 14 overweight (n=9 inactive) and 22 normal weight (n=6 inactive). Descriptive characteristics for active and inactive groups are shown in **Table 1**. Participants in the active group reported taking part in various types of physical activity including aerobic exercise, resistance training, field sports and combinations of different modes of exercise. As expected, significant differences were found between the two groups for a number of characteristics. Measured RMR values were within 1% (inactive) and 5% (active) of predicted values[(46)].

[Table 1 About Here]

Gastric Emptying

Comparison of GE in Active and Inactive groups

GE was significantly faster in the active group for all parameters **(Table 2)**. GE outcome measures were identical regardless of the VCO_2 value - predicted or directly measured - used (data not shown).

[Table 2 About Here]

GE $t_{1/2}$ in Groups Split by Median Body Composition and BMI

In order to compare our findings with prior studies comparing GE in overweight/obese with normal weight individuals classified by BMI, we compared GE $t_{1/2}$ between groups split by median BMI (25kg/m^2) and body composition values (**Figure 1)**. There were no significant differences between low and high BMI groups, but GE was significantly faster in the high FFM group and lower percent FM group (**Figure 1)**.

[Figure 1 About Here]

Cumulative Percent Dose Recovered

There were no significant differences in the cumulative percent dose recovered between groups, except for a small significant difference when divided by median percent FM (FM>20%, 43%; FM<20%, 41%; $p<0.05$). Adjusting for RQ did not influence the outcomes for any comparisons between active and inactive groups or groups in **Figure 1**.

Relationships between Variables and Determinants of GE

Simple Correlation Analysis between Variables

When the data from the two groups were pooled (n = 44), age was positively correlated with t_{lag} ($r=0.32$, $p<0.05$). Although BMI was not associated with GE**,** body composition was associated with several parameters. t_{lag} was associated with percent FM ($r=0.50$, $p<0.01$), absolute FM ($r=0.46$, $p<0.01$) and absolute FFM ($r=-0.32$, $p<0.05$); while $t_{1/2}$ was associated with percent FM ($r=0.39$, $p<0.01$), absolute FM ($r=0.35$, p,<0.05), and absolute FFM ($r=-0.29$, $p=0.05$).

RMR was not associated with GE. However, AEE was negatively correlated with t_{asc} ($r=-0.32$, $p<0.05$), t_{lat} ($r=-0.37$, $p<0.05$) and $t_{1/2}$ ($r=-0.46$, $p<0.01$, **Figure 2**). Average time spent in vigorous activity per day was also negatively correlated with t_{asc} ($r=-0.35$, $p<0.05$), t_{lat} ($r=-0.50$, $p<0.01$), t_{lag} ($r=-0.53$, $p<0.01$) and $t_{1/2}$ ($r=-0.46$, $p<0.01$). Similar negative correlations were observed between average time in moderate activity per day and GE variables (t_{lag}, $r=-0.42$, $p<0.01$; $t_{1/2}$, $r=-0.41$, $p<0.01$). These correlations collectively indicate that a higher amount of time spent and energy expended in physical activity were associated with a faster GE.

[Figure 2 About Here]

Partial correlations controlling for activity

Partial correlations of relevant variables with GE in the pooled data (n=44) were performed by controlling for group (**Table 3**). Significant associations between adiposity and GE were then no longer evident, whereas associations between age and t_{lag} and between AEE and TEE with GE remained significant (**Table 3**).

[Table 3 About Here]

Multiple regression analysis

When considering age, percent FM, activity and FFM as independent variables, activity status (active or inactive) was the only significant predictor of GE $t_{1/2}$ (Model adjusted R^2: 0.25, β=-0.51, $p<0.01$). In addition, AEE was a significant independent predictor of GE $t_{1/2}$ (β=-0.40, $p<0.01$). As there was no evidence of strong multicollinearity between AEE and activity status (VIF:1.2) these variables were included in the same model. Together, AEE and activity status accounted for the greatest variance of GE $t_{1/2}$ (model adjusted R^2, 0.34, $p<0.001$; activity: **β**, -.45, $p<0.01$; AEE: **β**,-0.28, p=0.05).

For t_{lag}, activity status and AEE together explained 31% of the variance (model adjusted R^2, 0.31, $p<0.001$; activity: **β**, -0.37, p=0.01; AEE: **β**, -0.33, p=0.03). Percent FM and FFM were not significant predictors of t_{lag}. However, including age increased the model adjusted R^2 to 0.38 ($p<0.01$).

DISCUSSION

Although GE has long been implicated in the pathogenesis of obesity, findings have been inconclusive, perhaps because of the influence of additional factors**,** such as habitual physical activity levels of participants. The findings from the present study provide evidence that GE is faster in habitually active compared to inactive males, that greater time spent in physical activity and AEE are associated with faster GE, and that body composition - but not BMI - is associated with GE. Although two studies that previously investigated GE in active and inactive individuals reported faster GE in active individuals[25,47], neither controlled for EE and body composition. The present study has involved a larger sample size, with a wider range of body compositions and activity modes, and has characterised EE, FM and FFM.

The results suggest that differences in physical activity level and associated differences in body composition (FM and FFM) and AEE between individuals may represent one explanation for the inconsistent outcomes of previous studies examining GE in obesity[6-8,10,13-15,48]. Recently Seimon et al.[48] comprehensively assessed GE and other postprandial responses in normal weight, overweight and obese males classified by BMI and reported no differences in GE of a nutrient drink between groups. However, body composition and EE were not reported. In the present study, the data from the two groups were pooled and split by median BMI (25 kg/m^2) and body composition values, in order to allow comparison with previous studies. GE did not differ significantly between groups split by BMI but was faster in males with a lower percent FM and higher FFM. Previous limited evidence has shown somewhat similar findings regarding relationships between body composition and GE[8]. In addition, we examined associations amongst EE and GE. While there was no association between resting EE and GE, a higher amount of time spent in physical activity and a higher AEE were associated with a faster GE. These data are compatible with a hypothesis that appetite signals arising from the GI tract may be more related to AEE than RMR[49]. Collectively the findings demonstrate that a higher AEE, lower percent FM and higher FFM (but not BMI or RMR) are associated with a faster GE in males.

Whereas a number of previously observed associations, including between adiposity and GE were no longer evident after controlling for activity status (active or inactive), the associations between AEE, age and GE remained. Further, the multiple regression analyses indicated that differences in body composition or BMI did not explain the faster GE observed in active individuals. Of the variables measured, habitual activity status and AEE accounted for the greatest variance in GE in males. These findings suggest that in the absence of differences in physical activity GE may not be altered in obese individuals. Interestingly, others have shown that associations between body composition and eating frequency are mediated by physical activity[41].

The present findings have a number of possible interpretations and implications in relation to appetite control and weight management. Interactions between EE and energy intake have long been of interest in the study of energy balance. Indeed, sixty years ago (in this journal), Edholm et al.[50] proposed that differences in food intake originate from differences in EE. Our findings of a faster GE in active individuals and in those with higher AEE are counterintuitive to the argument that a faster GE and hence reduced gastric distension contributes to overconsumption and obesity[6,9]. However, although a faster GE may lead to an earlier onset of the next meal through a reduced gastric distension, the influence of GE on intestinal factors must also be considered. The rate of GE plays an important role in the delivery of nutrients to the intestine[29] and hence in the release of intestinal satiation peptides[30,51] including CCK[52], glucagon-like peptide-1 (GLP-1)[53] and peptide YY (PYY)[13]. Meyer-Gerspach et al.[16] recently demonstrated slower GE rates in obese individuals along with reduced postprandial GLP-1 and PYY secretion, reduced ghrelin suppression and reduced satiation compared to normal-weight individuals. It was suggested the slower delivery of nutrients to the intestine could contribute to the blunted release of gut peptides and hence overconsumption[16]. Perhaps, the faster GE we observed in active individuals could lead to an earlier activation of intestinal satiety signals in response to food intake and mean that appetite is better regulated in response to intestinal satiety signalling between meals. Faster GE could be one contributing mechanism to an improved sensitivity of appetite control[31] and "gross" physiological regulatory control of energy intake[54], arising from increased activity EE and physical activity. In inactive individuals, in contrast, a slower GE could have a role in predisposing to weight gain and a 'dysregulation' of appetite with inactivity[55] through a delayed or reduced release of gut peptides signalling satiety from the intestine[10,16]; and mean that other factors such as sensory cues or social values may be more likely to influence food intake.

Although differences in GE between active and sedentary individuals could also be a consequence of different habitual dietary intakes[56], the causal nature of this association is not possible to determine from cross-sectional studies and requires additional longitudinal assessments. A slower GE might also be secondary to weight gain[14] with inactivity. However, our results suggest associations between body composition and GE are mediated by physical activity. Other mechanisms previously proposed to contribute to faster GE in active individuals have included enhanced parasympathetic tone[25] and gastric electroactivity[47]. In the present study, active males had a significantly lower resting heart rate consistent with higher levels of parasympathetic tone[57]. Hormonal factors may also have a mechanistic role. Fasting ghrelin[58], blood glucose[59] and insulin sensitivity[60] can influence GE and are known to change in response to exercise training[61,62]. Future characterisation of blood profiles may yield further information on the underlying mechanisms. In summary, while causal inferences cannot be drawn from the present study, the findings allow for an increased understanding of factors associated with GE. Additionally, they provide insight into processes potentially contributing to meal-to-meal appetite control and energy balance with habitual physical activity, and can be used to inform prospective studies examining the efficacy of targeting GE for weight management.

It is important to acknowledge some methodological issues in the present study. The ^{13}C-OBT has many advantages[42] and has been shown to be unaffected in various medical conditions[63,64]. However, unlike scintigraphy, the ^{13}C-OBT does not permit direct imaging of gastric function and emptying times are longer than those using scintigraphy. Although it is possible that various factors including VCO_2 predictions and RQ may influence the ^{13}C recovery, the present analyses suggest that these factors are unlikely to have affected the results. Moreover, reports of both faster and slower GE in obese individuals using both the ^{13}C-OBT[10,65] and scintigraphy[6,14,15] indicate that the method used is unlikely to bias the GE results. A limitation of accelerometers placed on the hip in detecting upper body exercise may have underestimated activity in active individuals. Nevertheless the Actigraph accelerometer has been demonstrated to reasonably correlate with EE measured by doubly labelled water[66]. Finally it should also be noted that males only were included so that gender and phase of menstrual cycle were not confounding factors.

In conclusion, our findings show that GE is faster in habitually active males and a greater time spent in physical activity and greater AEE are associated with faster GE. These results highlight the importance of considering body composition and physical activity level in studies examining GE (and parameters influenced by GE). Further investigations are needed to explore the possibility that GE contributes to a gross physiological regulation (or dysregulation) of appetite and food intake at different levels of physical activity. The potential therapeutic implications of physical activity for certain patient populations, such as those with gastroparesis who have been characterised by low energy expenditures[67] are also relevant for future work. These findings help improve understanding of factors that influence variability in GE and may have relevance to both researchers and clinicians working in gastroenterology, nutrition and obesity.

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Conflicts of Interest

The authors have no conflicts of interest to disclose.

Authorship

KMH, NMB, GJC and NAK contributed to the design of the study; KMH collected the data, analysed the data and drafted the manuscript; NMB, GJC and NAK contributed to data analysis and critical revision of the manuscript. All authors read and approved the final manuscript.

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Table 1. Participants' anthropometric, body composition, physical activity and energy expenditure characteristics (n=22 per group)

	Active (n=22)		Inactive (n=22)		*P*-value
	Mean	SD	Mean	SD	
Age (years)*	26.5	(23.0, 36.3)	27.5	(24.0, 34.3)	0.56
Height (m)	1.80	0.07	1.78	0.08	0.55
Weight (kg)	79.2	11.7	87.1	15.8	0.07
BMI (kg/m^2)*	23.7	(22.7, 27.0)	27.0	(23.7, 30.0)	**0.02**
BSA (m^2)	1.99	0.18	2.08	0.22	0.13
FM (%)*	11.6	(10.1, 18.6)	26.6	(20.0, 34.1)	**<0.001**
FFM (kg)	67.7	8.9	63.3	8.2	0.10
Resting HR (bpm)	52.7	8.5	64.1	9.3	**<0.001**
RMR (kcal/day)	1933	244	1970	340	0.68
Physical Activity[1]					
Steps per day*	8474	(7663, 10581)	7376	(5297, 8842)	**0.02**
AEE (kcal/day)	709	239	525	185	**<0.01**
TEE (kcal/day)	2890	430	2665	413	0.09

Data are means ± SD.
*Data are medians (25th, 75th percentile).
[1]Physical activity data refers to n =19 in Inactive group.
BMI, body mass index; BSA, body surface area; FM, fat mass; FFM, fat free mass; HR, heart rate; RMR, resting metabolic rate; AEE, activity energy expenditure; TEE, total energy expenditure.

Table 2. Gastric emptying parameters in Active and Inactive groups (n=22 per group)

	Active (n=22)		Inactive (n=22)		*P*-value
	Mean (SD)	Range	Mean (SD)	Range	
t_{lag} (min)	95 (13)	76-119	110 (16)	85-158	**<0.001**
$t_{1/2}$ (min)	157 (18)	125-195	179 (21)	139-231	**<0.001**
t_{lat} (min)*	27 (25, 34)	22-46	36 (23, 41)	20-60	**0.01**
t_{asc} (min)	127 (15)	101-162	143 (19)	110-179	**<0.01**

Data are means ± SD.
*Data are medians (25th, 75th percentile).
$t_{1/2}$, half time; t_{lag}, lag time; $t_{1/2s}$, t_{asc}, ascension time; t_{lat}, latency time.

Table 3. Partial correlations of age, body composition, resting metabolism and energy expenditure variables with GE t_{lag} and $t_{1/2}$ after controlling for activity group (Active or Inactive) (n=44)

	GE t_{lag}		**GE $t_{1/2}$**	
	r	*P*-value	r	*P*-value
Age	**0.41**	**<0.01**	0.19	0.21
BMI	0.03	0.86	-0.05	0.77
FM (%)	0.15	0.34	0.04	0.80
FFM (kg)	-0.21	0.17	-0.19	0.23
Waist circumference	0.07	0.64	-0.06	0.70
RMR	-0.22	0.15	-0.26	0.09
RHR	0.07	0.67	0.04	0.77
AEE[1]	**-0.35**	**0.03**	**-0.31**	**0.05**
TEE[1]	-0.30	0.06	**-0.31**	**0.05**

AEE, activity energy expenditure, FFM, fat free mass, RHR, resting heart rate, RQ, respiratory quotient, RMR, Resting metabolic rate, TEE, total energy expenditure, GE $t_{1/2}$, gastric emptying half time; GE t_{lag}, gastric emptying lag time.

[1]n=41.

Figure Legends

Figure 1. GE half time ($t_{1/2}$) for low and high BMI, FM and FFM groups based on median splits of 25kg/m^2 (BMI), 20% (%FM) and 67kg (FFM) of the pooled data for the whole cohort. Descriptive characteristics (mean±SD) were BMI (low: 23±1; high: 29±3 kg/m^2), % FM (low: 12±3; high: 28±6%), FFM (low: 58±4; high: 73±5kg). n=22 per group for all categories. Error bars indicate SD.

Figure 2. Scatter plot of the relation between activity energy expenditure (AEE) and GE half time ($t_{1/2}$) ($r=-0.46$, $p<0.01$). n=41.