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Postprandial energy expenditure of protein is affected by its phosphorus content

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ABSTRACT

Postprandial energy expenditure (PEE) is largely dependent on ATP production, which is may be affected by phosphorus (P) availability. Proteins are known to have high levels of P and induce high levels of PEE. This study aimed at assessing the effect of P in PEE of normal and high protein meals. A single-blind randomized crossover study was conducted with two groups of 12 healthy lean male subjects who received iso-caloric (554 Kcal) meals. Group1: normal protein (NPr) meal with or without P (500 mg) and group 2: high protein (HPr) meal with or without P (500 mg), on two visits separated by a minimum of 1-week washout period. Energy expenditure and substrate oxidation were measured at baseline and every 30 min for 4 h after meal ingestion using a ventilated hood for indirect calorimetry. NPr and HPr meals had similar postprandial energy expenditure and this was significantly increased ($P = 0.005$) by P ingestion. Our work shows that PEE of protein meal is highly affected by P content of the meal.

1. Introduction

Postprandial energy expenditure (PEE) covers the cost of digestion, absorption, transport, and storage of the ingested food and is related to ATP production (Scott and Devore, 2005). PEE is the least reproducible component of total daily energy expenditure because it is affected by many external (e.g. food composition, etc.) and internal (e.g. hormonal status, etc.) factors (Acheson et al., 2011; Scott and Devore, 2005; Tentolouris et al., 2008). Among macronutrients, PEE of proteins is known to be the highest (Acheson et al., 2011; Karst et al., 1984; Scott and Devore, 2005; Smeets et al., 2008) and this is paralleled by the fact that the body has no storage capacity of excess protein and thus, free amino acids are either assimilated into protein or oxidized. Accordingly, protein turnover was suggested to account for most (~68%) of the thermogenic effect of proteins (Paddon-Jones et al., 2008) as the energy costs of protein synthesis and protein breakdown are estimated to be 3.6 and 0.7 KJ/g, respectively (Veldhorst et al., 2009). However, the wide variations in PEE between proteins even for those of high biological values (Acheson et al., 2011; Karst et al., 1984) raise a question on whether other factors are involved in PEE of proteins. This encouraged us to assume a role for P, since most proteins contain high levels of bioavailable P.

ATP production is affected by the availability of P (Morris et al., 1978) and is important for several processes that are known to influence eating behavior and energy expenditure (Obeid et al., 2010). Ingestion of low P containing carbohydrates, e.g. refined cereals, caloric sweeteners, potatoes etc., is accompanied by a stimulation in insulin release which is known to increase the peripheral P uptake and the phosphorylation of many compounds. This is believed to compromise the availability of P to fulfill the requirements of all processes, including ATP production which can act as a phosphate donor (Karczmar et al., 1989). Data on the impact of P ingestion on the postprandial energy metabolism of protein manipulated meals are limited. The aim, therefore, of the present study was to investigate the effect of P ingestion on PEE and substrate oxidation in healthy lean male subjects. This entailed consuming two iso-caloric meals, a normal protein (NPr) and a high protein (HPr) meal, using egg white protein that has high biological value and is almost devoid of P (Kalantar-Zadeh et al., 2010), as the main source of protein.

2. Methods

2.1. Study design and participants

The study was a single-blind, randomized, and placebo-controlled crossover clinical trial conducted between September 2015 and June

Abbreviations: PEE, postprandial energy expenditure; %CO, percentage of carbohydrate oxidation; %FO, percentage of fat oxidation

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2016 at the Department of Nutrition and Food Sciences at the American University of Beirut (Beirut, Lebanon). Healthy lean male subjects with an age range between 19 and 35 years and with a normal body mass index (BMI: 18.5–24.9 kg/m^2) were recruited. Consented subjects were referred for a fasting blood test to determine their plasma glucose, creatinine and estimated glomerular filtration rate (Est. GFR) at the American University of Beirut Medical Center (Beirut, Lebanon). The study protocol was conducted according to Declaration of Helsinki principles and approved by the Institutional Review Board at the American University of Beirut. All participants provided written informed consent prior to inclusion in the study. The trial is registered with Clinical Trial.gov, NCT02482142.

2.2. Experimental protocol

The study was composed of two meal groups (NPr and HPr), with 12 healthy lean male subjects each. In the NPr group, subjects were asked to ingest a low-P normal protein meal (NPr) with or without 500 mg of P, while in the HPr group, subjects were asked to ingest a low-P high protein meal (HPr) with or without 500 mg of P. The two test meals were iso-caloric (554 Kcal) and comprised of a toast of white wheat bread, butter and a protein shake that was prepared using egg white powder. The composition of the test meals is summarized in Table 1. Subjects were asked to avoid any intense physical activity and the use of nutritional supplements 3 days prior to the study. Thereafter, subjects were requested to come to the research unit at around 8:00 a.m. after an overnight fast (> 8 h) on two visits separated by a minimum of 1-week washout period. Subjects were examined in a quiet room with a stable room temperature (22 °C). Anthropometric measurements including weight, height, waist and hip circumference were taken at baseline. Subjects were then asked to rest for around 30 min on a couch in a semi-upright sitting position (45 degrees), then resting energy expenditure (REE), substrate oxidation namely % fat oxidation (FO) and % carbohydrate oxidation (CO) and respiratory quotient (RQ) were measured for 30 min using a ventilated hood and canopy system (Cosmed Quark Cpet Unit, Italy) for indirect calorimetry measurement. Carbohydrate and fat oxidation were calculated using the following equations of Frayn (1983):

$$c = 4.55\ VCO_2(l/min) - 3.21\ VO_2 - 2.87n;$$
$$f = 1.67\ VO_2(l/min) - 1.67\ VCO_2 - 1.92n$$

[c and f are the amounts (in grams) of carbohydrate and fat oxidized per minute, respectively], and [n is the amount (in grams) of urinary nitrogen excreted per day and this was assumed to be 13 g/day] as per Charrière et al. (2016).

Respiratory quotient was calculated according to the Weir equation ($RQ = VCO_2/VO_2$) (Weir, 1949). Baseline metabolic measurements [REE, CO(%), FO(%) and RQ] were taken for one hour and value represent the average of two measurements. Subjects were then given the appropriate meal (NPr or HPr) with 500 mg of P tablets (potassium phosphate) on one visit or with placebo (cellulose) tablets (NutraScience Labs, Farmingdale, NY, USA) on the other visit in a random order. Subjects were asked to consume the meal within 15 min after which postprandial thermogenesis (PT), FO(%), CO(%) and RQ were measured for the next 4 h on 30 min basis (15 min break followed by 15 min measurements). Subjects were asked to void their urine before the initiation of any measurement. Urine was collected within and at the end of the experimental period in each visit and the total volume was recorded. Urine aliquots were then stored in a freezer at −20 °C for later use. Urine specimens were thawed at room temperature and were then centrifuged for 5 min at 3500 rpm at 20 °C using Eppendorf Centrifuge 5810 R (Hamburg, Germany). Thereafter, urinary levels of phosphorus, creatinine, and urea nitrogen (UUN) were measured using the Vitros 350 analyzer (Ortho-Clinical Diagnostics, Raritan, NJ, USA).

Table 1
Composition of the test meals.

	NPr meal	HPr meal
Carbohydrate (%)	52.61	28.61
Protein (%)	15.20	50.43
Fat (%)	32.17	20.95
Phosphorus (mg)	109	143
Total Calories (Kcal)	554	554

Abbreviations: NPr, normal-protein; HPr, high-protein.

2.3. Statistical analysis

A sample size of 10 subjects was determined by the application of power analysis to detect a 5% difference in the mean, with a standard deviation of 3.3% for the population, at a 0.05 significance level and a desired statistical power of 90% (Charrière et al., 2016). Data are presented as mean ± SD and metabolic measurements are expressed as absolute values. A two-sample *t*-test was used to analyze differences in baseline characteristics between the 2 groups. Area under the curve (AUC) was calculated using GraphPad Prism version 7.00 (GraphPad Software, La Jolla California USA) and paired *t*-tests were used to analyze differences within each group (NPr or HPr) following phosphorus ingestion. A General linear model (GLM) with time as random effect was performed to determine the effect of time, phosphorus, protein (NPr or HPr) and their interactions. The level of statistical significance was set at $P < 0.05$. Statistical analyses were conducted using the Minitab 17 software program.

3. Results and discussion

All subjects had similar baseline anthropometric characteristics, fasting plasma glucose, creatinine, and GFR. Additionally, they had comparable baseline metabolic measurements including resting energy expenditure (REE), FO(%)CO(%), and RQ (Table 2) and were in line with others (Veldhorst et al., 2009).

Postprandial energy expenditure of all groups increased from time 30 min onwards and did not return to baseline levels by the end of the experimental period. The pattern of PT varied between groups and a statistically significant difference was detected according to phosphorus and not protein content of the meal (Fig. 1A). This was further associated with a significant difference in AUC of the phosphorus supplemented (NPr + P and HPr + P) groups as compared to non-supplemented groups, while no differences in the AUC between NPr and HPr groups were detected (Table 3). In line, we have recently reported that phosphorus ingestion with a high carbohydrate meal was able to increase PT

Table 2
Baseline characteristics of lean subjects consuming normal (NPr) or high (HPr) protein meals.

Characteristics	NPr (12 subjects)	HPr (12 subjects)
Age (years)	23.1 ± 5.0	22.3 ± 4.5
Weight (kg)	73.7 ± 6.6	73.8 ± 8.3
Height (cm)	178.1 ± 5.6	180.2 ± 5.1
BMI (kg/m^2)	23.3 ± 2.4	22.7 ± 2.0
Waist circumference (cm)	77.8 ± 6.6	79.0 ± 6.2
Hip circumference (cm)	98.4 ± 5.5	98.6 ± 6.4
Fasting plasma glucose (mg/dl)	89.7 ± 8.4	88.8 ± 6.7
Creatinine (mg/dl)	0.92 ± 0.13	0.88 ± 0.09
Est. glomerular filtration rate (ml/min/1.73 m^2)	114.9 ± 16.2	121.0 ± 9.9
Baseline resting energy expenditure (kcal/min)*	1.226 ± 0.124	1.200 ± 0.097
Baseline carbohydrate oxidation (%)*	17.4 ± 9.6	20.4 ± 12.9
Baseline fat oxidation (%)*	63.6 ± 10.7	60.5 ± 12.8
Baseline respiratory quotient*	0.76 ± 0.04	0.78 ± 0.05

* The baseline metabolic measurements are the average of the two visits. Variables are expressed as Mean ± SD.

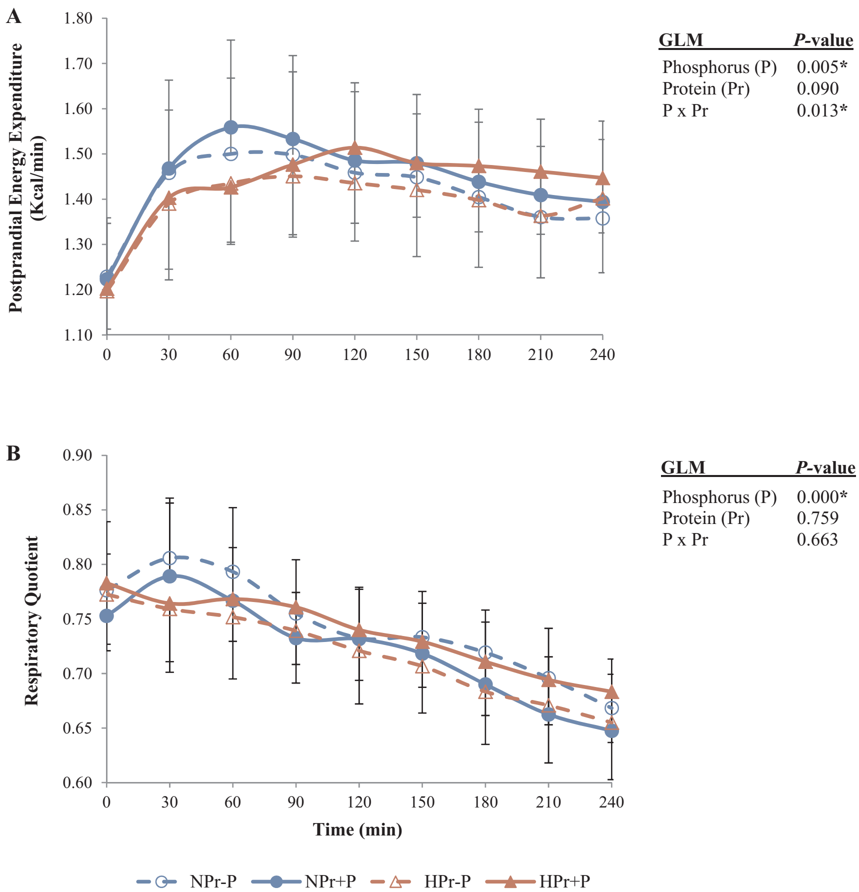

Fig. 1. Postprandial thermogenesis and substrate oxidation. (A) Postprandial thermogenesis (Kcal/min), (B) carbohydrate oxidation (%), (C) fat oxidation (%) and (D) the respiratory quotient of lean subjects consuming the normal protein and high protein meals. Measurements presented in empty circles and dashed lines ( ) are registered for placebo intake among lean subjects consuming the normal protein meal; the ones of full circles and full lines ( ) are for phosphorus intake among lean subjects consuming the normal protein meal; the ones of empty triangles and dashed lines ( ) are for placebo intake among lean subjects consuming the high protein meal; the ones of full triangles and full lines ( ) are for phosphorus intake among lean subjects consuming the high protein meal. General Linear Model (GLM) with time as random effect was performed to determine the effect of time, phosphorus, protein (normal or high) and their interactions. Values are presented as Mean ± SD at different time point compared to baseline values. * The level of statistical significance was set at $P < 0.05$.

Table 3
Area Under the Curve (AUC) of postprandial energy expenditure (EE) and respiratory quotient (RQ) of lean subjects consuming normal or high protein meals with or without phosphorus.

AUC	NPr		HPr	
	-P	+P	-P	+P
REE (Kcal/min)	342.7 ± 36.6	350.5 ± 39.1[a]	335.8 ± 30.9	346.8 ± 31.4[a]
RQ	178.8 ± 9.87	173.8 ± 11.3	172.4 ± 11.1	177.1 ± 10.5

*Significant? 2-Sample T-Test: NPr-P vs HPr-P.
NPr-P: Normal protein without phosphorus. NPr+P: Normal protein with phosphorus. HPr-P: High protein without phosphorus. HPr+P: High protein with phosphorus.
Values are presented as Mean ± SD. The level of statistical significance was set at $P < 0.05$.
[a] Significant Paired T-Test NPr-P vs NPr+P or HPr-P vs HPr+P.

of lean and obese subjects (Assaad et al., 2019). Further supporting our suggestion that postprandial energy expenditure is affected by phosphorus content of the meal.

Moreover, the impact of P addition on PEE was found to vary with the protein content of the meal as indicated by the significant differences in the phosphorus x protein interactions ($P = 0.013$). PT was mainly apparent from time 60 min in the NPr + P group, while that of the HPr + P was apparent from time 120 min and onward. The varied pattern of changes in PT between the NPr and HPr meals may relate to the difference in their carbohydrate contents (NPr: ~53% of energy as carbohydrate; HPr: ~29% of energy from carbohydrate). Carbohydrate has the capacity to stimulate insulin release which in turn is known to enhance peripheral P uptake (Karczmar et al., 1989; Khattab et al., 2015) and thus favoring ATP production and PEE. In the case of the HPr meal, a dampened insulin release would be expected to extend P availability, which may ultimately be used to support several energy-requiring metabolic processes including glycogen (Mattar et al., 2010; Xie et al., 2000) and protein syntheses (Bender, 2012). Protein absorption rates (e.g. slow, fast such as casein and whey) were reported to exert different effects on postprandial protein metabolism (synthesis, oxidation), hormonal profile, and energy expenditure (Acheson et al., 2011; Boirie et al., 1997). A slow protein absorption rate was reported to extended protein synthesis and increase protein deposition (Boirie et al., 1997). The capacity of P to enhance energy expenditure (Jaedig et al., 1994; Kaciuba-Uściłko et al., 1993) may help in explaining several observations including, first the high thermic effect of casein compared to egg white (Karst et al., 1984). Casein is known to be rich in P unlike egg white, which has negligible amounts of P. Second, there is an inverse relationship between the consumption of dairy products and body weight, given that milk is a high source of P (Wagner et al., 2007; Yanovski et al., 2009). Furthermore, the above findings may relate to the capacity of P ingestion with the meal in decreasing appetite (Ayoub et al., 2015) and subsequent energy intake (Obeid et al., 2010) especially that appetite is known to be inversely related to PT (Crovetti et al., 1998; Jaedig et al., 1994; Kaciuba-Uściłko et al., 1993; Lejeune et al., 2006).

Respiratory quotient of the NPr treatments (NPr-P and NPr + P) increased slightly in the first 30 min and decreased gradually thereafter, while that of the HPr treatments (HPr-P and HPr + P) decreased from the time of meal ingestion (Fig. 1B). The pattern of RQ changes in non-supplemented groups seems to parallel that of the supplement groups and significant difference according to phosphorus content of the meal. This difference was not detected when AUC was calculated (Table 3) and implies that RQ was minimally affected by P content of the meal. The similarities in urinary nitrogen excretion over the experimental period among the groups points towards a resemblance in the

Table 4
Urinary parameters of lean subjects consuming NPr or HPr meals with or without phosphorus (P).

Variable	NPr		HPr		*P*-value*		
	Placebo	Phosphorus	Placebo	Phosphorus	P	Pr	P × Pr
Urine (ml)	460.8 ± 237.8	354.2 ± 168.9	391.7 ± 228.9	414.2 ± 168.3	0.478	0.939	0.278
Total P (mg)	167.7 ± 115.0	236.6 ± 88.7	120.8 ± 53.6	246.9 ± 86.5	0.000**	0.479	0.271
Total UUN (mg)	3468 ± 859	3287 ± 851	3002 ± 714	3305 ± 1223	0.821	0.409	0.373
Total Creatinine (mg)	517.7 ± 141.5	475.3 ± 148.2	453.0 ± 75.4	444.0 ± 83.2	0.450	0.162	0.623
P/Cr	0.31 ± 0.17	0.50 ± 0.17	0.27 ± 0.13	0.56 ± 0.18	0.000**	0.806	0.271

Abbreviations: P, phosphorus; UUN, urine urea nitrogen; Cr, creatinine. Variables are expressed as Mean ± SD.
Urine was collected over the 4 h experimental period.
* *P*-values for repeated measures analysis of variance (ANOVA).
** The level of statistical significance was set at $P < 0.05$.

contribution of protein oxidation to total energy expenditure among these groups.

Looking at substrate's oxidation, postprandial CO(%) and FO(%) of the non-supplemented groups were found to parallel that of supplemented groups, while a significant difference was found according to phosphorus content of the meal (Supplementary Fig. 1A and 1B). However, the failure of AUC to produce any significant differences among the groups implies that the detected differences by the general linear model were related to the minimal variations in baseline values (Supplementary table 1). The higher carbohydrate content of the NPr compared to the HPr meals may have been responsible for the early ramp in RQ, due to increased CHO oxidation, of the NPr meals. While, the high rate of fat oxidation at time 30 min following the HPr meals was in line with others (Labayen et al., 2004; Lejeune et al., 2006).

Urine analysis shows that P ingestion with both meals was associated with a significant increase in urinary P ($P = 0.000$) and P/Cr excretions ($P = 0.000$), but failed to affect urea nitrogen (UUN) or creatinine excretions (Table 4). The failure to detect changes in UUN excretion may have been compounded by the known fluctuation in postprandial protein metabolism (Boirie et al., 1997).

Postprandial P status is highly affected by insulin release and P content of the meal (Khattab et al., 2015). The ability of insulin to increase peripheral uptake of P and reduce serum P (Kalaitzidis et al., 2005) is paralleled by its capacity to enhance energy requiring anabolic processes. As a consequence, it is hypotheses that a competition for P arises and this compromises the activity of several processes including that of ATP production (Obeid, 2013). As a result, the P content of the meal becomes vital for lessening the impact of such competition and optimizing the activity of metabolic processes. On the other hand, several observations suggest a role of P in energy metabolism. For example, the ingestion of P-containing supplements after a meal (Kaciuba-Uściłko et al., 1993) or with orange juice (Jaedig et al., 1994) was reported to increase resting metabolic rate (RMR) of obese women. Additionally, P supplementation with main meals for 12 weeks was shown to significantly decrease body weight and waist circumference of overweight and obese adults (Ayoub et al., 2015). It is worth noting that the ability of meal supplementation with P to enhance energy expenditure (Jaedig et al., 1994; Kaciuba-Uściłko et al., 1993) and suppress appetite (Ayoub et al., 2015; Obeid et al., 2010) resembles that of that of high protein diets, which is known to increase energy expenditure (Acheson et al., 2011; Leidy et al., 2015; Paddon-Jones et al., 2008; Yang et al., 2014) and decrease appetite (Halton and Hu, 2004; Latner and Schwartz, 1999; Weigle et al., 2005). This resemblance was e motivation for this work to try to discern the role of P from that of protein.

In the present study, the dose of P was within the nutritionally required range and no invasive measurements were taken to minimize the impact of stress on energy metabolism. However, an assessment of blood metabolites and hormones would have provided an insight into the mechanism(s) related to the effect of P.

In conclusion, our results show that PEE of protein is highly affected by P content of the meal. Moreover, the fact that both glucose and amino acids have similar energy cost for transport (Bannink et al., 2006) implies that the observed sustenance of elevated energy expenditure of the P supplemented meals beyond the absorptive phase is highly related to enhanced anabolic processes. Further research should be considered to examine the exact mechanism of action and the long-term effects of P on the human body.

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Declarations of interest

None.

Author contributions

OO was involved in the concept. OO and AO decided on the experimental design. LA performed data collection. OO, AO, and LA performed data analysis, manuscript preparation, and revision.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jtherbio.2018.10.013.

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