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The effects of collagen peptide supplementation on appetite and post-exercise energy intake in females: a randomised controlled trial

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Research Article

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Abbreviations:
CCK, cholecystokinin; CON, placebo control; CP, collagen peptides; GI, gastrointestinal; GLP-1, glucagon like peptide 1; PYY, peptide YY; RPE, rating of perceived exertion; sDPP-4, soluble dipeptidyl peptidase-4

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Abstract

This study examined whether supplementation with collagen peptides (CP) affects appetite and post-exercise energy intake in healthy active females. In this randomised, double-blind cross-over study, fifteen healthy females (23 (SD 3) years) consumed 15 g/d of CP or a taste matched non-energy control (CON) for 7 d. On day 7, participants cycled for 45 min at ~55 % W_{max}, before consuming the final supplement. Sixty-min post supplementation an *ad libitum* meal was provided, and energy intake recorded. Subjective appetite sensations were measured daily for 6 d (pre- and 30 min post-supplement) and pre (0 min) to 280 min post-exercise on day 7. Blood glucose and hormone concentrations (total ghrelin, glucagon-like peptide-1 (GLP-1), and peptide YY (PYY), cholecystokinin (CCK), dipeptidyl peptidase-4 (sDPP-4), leptin, and insulin) were measured fasted at baseline (day 0), then pre-breakfast (0 min), post-exercise (100 min), post-supplement (115, 130, 145, 160 min) and post-meal (220, 280 min) on day 7. *Ad libitum* energy intake was ~10 % (~41 kcal) lower in the CP trial ($P = 0{\cdot}037$). There was no difference in gastrointestinal symptoms or subjective appetite sensations throughout the trial ($P \geq 0{\cdot}412$). Total plasma GLP-1 (AUC, CON: 6369 (SD 2330); CP: 9064 (SD 3021) pmol/l; $P < 0{\cdot}001$) and insulin (+80 % at peak) were higher after CP ($P < 0{\cdot}001$). Plasma ghrelin and leptin were lower in CP (condition effect; $P \leq 0{\cdot}032$). PYY, CCK and glucose were not different between CP and placebo ($P \geq 0{\cdot}100$). CP supplementation following exercise increased GLP-1 and insulin concentrations and reduced *ad libitum* energy intake at a subsequent meal in physically active females.

Increasing dietary protein intake, either acutely as a single bolus (e.g. 15–70 g) or over several days, may reduce appetite, as measured by decreased energy intake[1–4], increased subjective feelings of fullness and reduced hunger[4–6] and/or increased levels of satiety hormones such as glucagon peptide-1 (GLP-1)[7,8]. This has sparked an interest in the effects of different protein sources and supplements (e.g. whey, soy) on appetite suppression and weight management, particularly in combination with exercise to manage obesity-related disease[4,9,10]. Several mechanisms have been proposed to explain how dietary protein supplements affect appetite, including delayed gastric emptying[11], increased thermogenesis[12], interactions with satiety hormones such as GLP-1, peptide tyrosine tyrosine (PYY) and insulin[13] and sensory characteristics of drinks (e.g. thickness or creaminess)[14–16].

To date, research on protein supplements and appetite regulation has largely focused on complete proteins such as milk[17,18], whey[19], soya[1] and casein[20], with limited research on protein supplements with different amino acid profiles. Collagen peptides (CP) are low in branched chain amino acids, but rich in hydroxyproline, glycine and proline[21]. In animals, gelatin hydrolysates were shown to stimulate secretion of insulin and GLP-1[22], and in humans, circulating glycine is strongly associated with reduced hunger and energy intake[23], suggesting CP supplements also have the potential to affect appetite. Recently, Duarte *et al.*[24] compared the effects of hydrolysed collagen (40 g) and whey protein (40 g) on biomarkers of appetite in young females and reported greater circulating leptin after collagen supplementation, but no difference in subjective appetite or energy intake 130 min following supplementation. However, this study did not measure the effects of collagen supplementation *v.* placebo control or the

effects on several key regulatory hormones such as GLP-1 and ghrelin. Earlier studies showed that meals rich in gelatin, a form of hydrolysed collagen (10–25 % of total energy), may be more satiating than other proteins such as whey, casein and soy[6,25]; however, Akhavan *et al.*[26] reported no effect of gelatin (6 g) intake on subsequent energy intake in healthy males. Thus, it remains unclear how short term or acute supplementation with CP can affect appetite regulation in humans.

Several recent studies have shown that CP supplements may hold benefits for athletic performance and recovery. Indeed, studies have shown that ingesting collagen supplements (5–30 g) in the days before and/or daily after exercise bouts can enhance recovery[27], increase muscle mass[28] improve body composition[29–32] reduce muscle and joint pain[33,34] and enhance sleep quality[35]. Collagen supplementation may also improve tendon structure and/or function[36–39] and therefore reduce the risk of musculoskeletal injury and/or accelerate rehabilitation[40,41]. Consequently, collagen supplements are increasingly recommended to athletes for health and performance benefits. There are currently no studies examining their effects on appetite regulation after exercise. It is feasible that CP could modify the appetite response to exercise, which could have implications for recovery and weight management in athletes and recreational exercisers.

The primary aim of this 'proof of concept' study was to determine if, compared to a non-energy placebo, consuming CP (15 g/d) influences subjective appetite, hormone response and *ad libitum* energy intake after a bout of exercise in healthy active females. A secondary aim was to examine subjective appetite and fasted hormone response in the 6 d leading up to the main exercise trials. We hypothesised that CP supplementation would attenuate appetite and reduce energy intake at a subsequent meal following exercise.

Methods

Participants

Seventeen volunteers were initially recruited and screened to take part in this study; however, two were excluded, one due to food allergies and one due to an inability to obtain blood samples at familiarisation. Therefore, fifteen healthy, physically active females completed the study (descriptive data are displayed in Table 1). Eleven participants were contraceptive users (*n* 6 combined monophasic pills; *n* 1 progesterone only pills and *n* 2 implant and *n* 2 Mirena coil), and their experimental trials were completed in the same contraceptive phase. Four females were regularly menstruating, and their experimental trials were completed in the early follicular phase. For study inclusion, participants had to be female, aged 18–30 years, and considered either Tier 1 or Tier 2 according to the guidelines set forth in the participant classification framework[42]. Briefly, Tier 1 classifies participants as recreationally active; they meet the WHO minimum activity guidelines and may perform multiple sports or activities. Tier 2 participants are classified as trained/developmental; they train/exercise regularly and identify with a specific sport. Participants were also required to not be taking any supplements or medications that may interfere with appetite and free from health conditions or food allergies that could affect study outcomes. Eligibility was checked with a health and exercise screening questionnaire at study entry. This study was conducted at Loughborough University according to the guidelines laid down in the Declaration of Helsinki. Institutional ethical approval was granted by Loughborough University Ethics Sub-Committee (Ethics Code: 12 570), and participants gave verbal and written informed consent. The study protocol was pre-registered on the Open Science Framework (Title: Amino Acids and Post-Exercise Appetite; registration DOI: https://osf.io/5v6np).

Table 1. Baseline characteristics of study participants (*n* 15) (Mean values and standard deviations)

Variable	Mean ± SD
Age (years)	23 ± 3
Height (m)	1·62 ± 0·08
Body mass (kg)	60·5 ± 10·1
Body mass index (kg/m^2)	22·9 ± 3·1
$\dot{V}O_{2peak}$ (ml/kg/min)	40·8 ± 7·7
IPAQ Score (Total MET-min pw)	3580 ± 1591

International Physical Activity Questionnaire (IPAQ; shortened version).

Preliminary visit and $\dot{V}O_{2peak}$ test

Participants reported to the laboratory after an overnight fast (water intake was allowed) and completed the International Physical Activity Questionnaire[43,44] before a venous blood sample was collected by venepuncture. Participants were then allowed the opportunity to consume food before completing a $\dot{V}O_{2peak}$ test on a cycle ergometer (Lode Corival), starting at 50 W and increasing by 25 W step increments every 3 min until volitional exhaustion. Participants self-selected their food intake prior to the test. An expired breath sample was collected into a Douglas bag in the final 60 s to determine $\dot{V}O_{2peak}$. O_2 and CO_2 content (Servomex 1400 Gas Analyzer; Servomex), volume (Harvard Dry Gas Meter, Harvard Apparatus), temperature and ambient air were collected simultaneously with expired gas samples to correct $\dot{V}O_2$ and $\dot{V}CO_2$ values.

Pre-trial standardisation

In the 6 d leading up to the main exercise trial, participants were free to consume their habitual diet and perform their usual exercise regimen, the latter of which was self-recorded and tracked with physical activity monitors, as described below. They were encouraged to replicate their diet and physical activity as closely as possible at the same time points prior to their repeat trial. For the 24 h prior to the first exercise trial (day 7), participants were required to complete a physical activity and diet diary (food and fluid intake), which was replicated before the subsequent trial. A standardised evening meal was provided (lasagne (beef or vegetable depending on individual preference); Tesco; UK) and cereal bar (Go Ahead Yoghurt Break; United Biscuits; UK). In the 24 h before trials, participants also abstained from alcohol and exercise.

Protocol

The study was a randomised, placebo-controlled, double blind, repeated-measures design. As used previously[35], a minimum 7-day wash out was scheduled between trials; all trials were completed between October 2023 and March 2024. Each experimental trial was 7 d in total (see schematic Figure 1). On the 6 d prior to the exercise bout on day 7, participants consumed either 15 g/d of bovine CP (Rousselot BV) or a placebo (water),

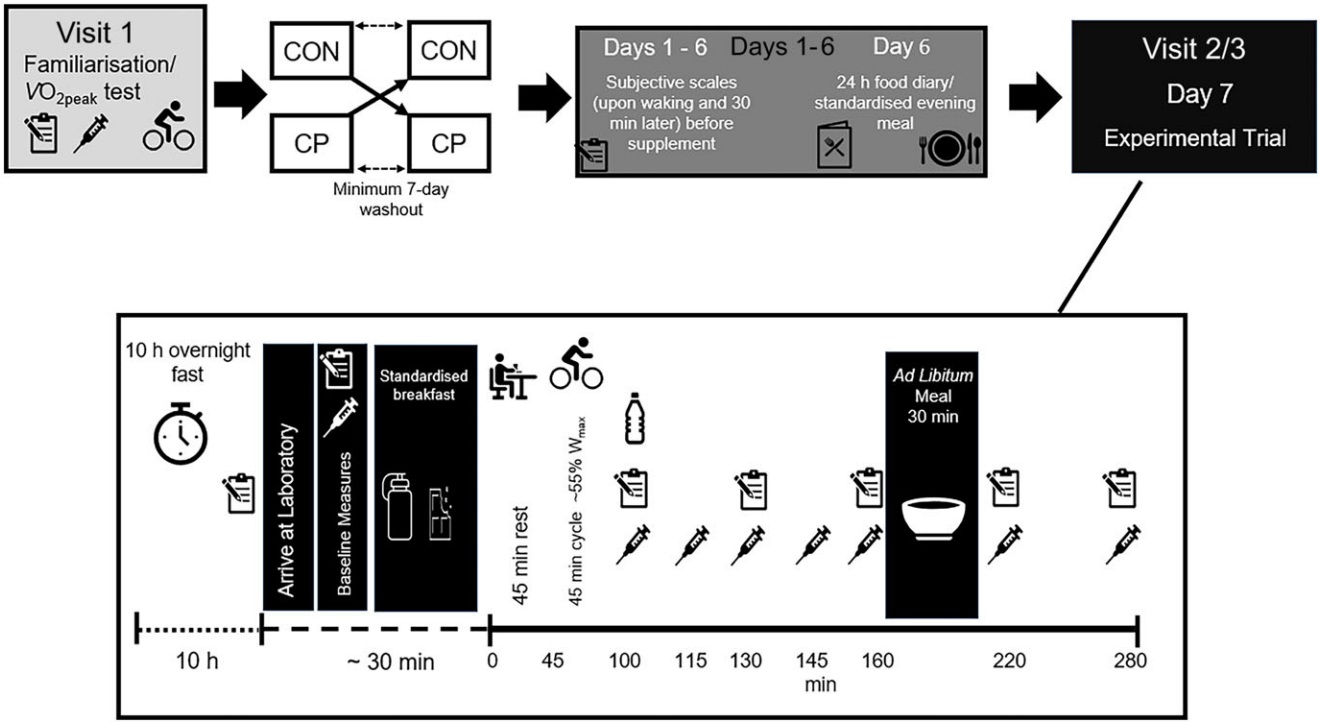

Figure 1. Schematic outline of study protocol. Clipboard symbols represent time-points when subjective appetite measures were taken; syringe symbols blood sampling time-points, and the water bottle symbol when fluid was consumed. CON, control supplement; CP, collagen peptides supplement (15 g/d).

which was taste matched with flavourings (Cherry Flavour Natural), upon waking. This dose was chosen as similar amounts have been shown to beneficially affect markers of exercise recovery[40,45,46] and elevate hydroxyproline concentrations[47] without causing gastrointestinal (GI) distress; thus, these amounts are typically recommended to physically active populations for health and performance benefits. Drinks were 150 ml in volume and provided in identical opaque bottles that were randomly allocated using simple randomisation generated by online software. Randomisation was performed by a researcher not responsible for data collection.

On the mornings of the exercise trials (day 7), participants arrived at the laboratory after an overnight fast (10 h) but consumed 300 ml of water 90 min before arrival. Participants were asked to verbally and in writing confirm if they had missed any dose of their supplements in the days leading up to the main trial and whether they followed the diet and exercise restrictions. After 10 min of seated rest, a flexible 20-gauge cannula was inserted into an antecubital vein, and a baseline blood sample was drawn. The same breakfast was provided to participants for both trials (Nature Valley Honey and Oat bar; Middlesex, UK; 196 kcal), which they had 10 min to consume, alongside water. This was followed by 45-min rest. Participants then cycled for 45 min at ~55 % W_{max} before resting for the remainder of the trial. 100 ml of water was provided after 15 min of cycling, and participants had 15 min to drink all the water. 10 min after the exercise, participants consumed their final supplement, and an *ad libitum* pasta meal was provided 60 min later. The meal consisted of pasta, tomato sauce and olive oil (all Tesco, UK) and provided 10·0 % protein; 57·2 % carbohydrate; 30·1 % fat; 2·7 % fibre. The energy density of the meal was 1·40 kcal/g. Participants ate their meal in a separate room where mobile phones and other distractions were not permitted; they had 30 min in which to eat and were asked to eat 'until they were comfortably full'. Energy intake was quantified by weighing the food pre and post consumption, and water intake was matched between trials. Eating rate was quantified by dividing energy intake (kcal) by time eating (seconds).

Measures

A GI-focused visual analogue scale (VAS) was used to assess nausea, headache, GI discomfort and urge to defecate, which were anchored at 0 mm by 'no nausea', 'no headache', 'not at all' and 'no urge to defecate', respectively; at 100 mm was 'worst possible nausea', 'worst possible headache', 'a lot', and 'extreme urge to defecate', respectively. An appetite focused VAS was used to assess hunger, fullness, desire to eat, thirst and prospective food consumption, which were anchored at 0 mm by 'not hungry at all', 'not full at all', 'no desire at all', 'not thirsty at all' and 'nothing at all', respectively; at 100 mm was 'extremely hungry', 'extremely full ', 'extreme desire', 'extremely thirsty' and 'a large amount', respectively[48]. The GI and appetite VAS scales were measured with pen and paper upon waking and then 30 min post supplement ingestion on the 6 d pre-experimental trial and then upon waking on the morning of the experimental trial and at 0, 90 min, 130 min, 160 min (pre *ad libitum* meal), 190 min (immediately after the *ad libitum* meal), 220 and 280 min. Composite appetite scores were calculated (hunger + (100-fullness)+desire to eat +prospective food consumption)/4)[49,50]. Heart rate (Polar H10; Polar Electro Oy; Kempele, Finland) and rating of perceived exertion (RPE; 6–20 scale[51]) were measured every 15 min during exercise. Venous blood (20 ml) was obtained at –15 min (before breakfast), 100 min, 115 min, 130 min, 145 min, 160 min, 220 min and 280 min, and the cannula was kept patent by flushing with sterile saline after sampling. Body mass (minimal clothing; Adam CFW-150, Milton Keynes, UK) was measured before baseline measurements. Laboratory humidity and temperature were measured at –30 min, 45 min, 100 min, 145 min and 280 min. On the 6 d prior to the

experimental trial, participants wore a physical activity monitor on their non-dominant wrist (Motionwatch Rugged; Cambridgeshire; UK) and were asked to record any physical activity in a diary. They recorded training type, duration (min) and self-reported the exercise intensity as either low, moderate or severe. The breakdown of total time spent sedentary and participating in low, moderate or vigorous physical activity for the 6 d prior to the trial was calculated from the monitors. Data were collected from midnight on day 1 of the trial and finished at midnight of day 7. Data from the physical activity tracker was used to quantify time spent sedentary ($\leq$ 178·49 counts per minute (CPM)) or doing moderate (178·5–562·49 CPM) to vigorous ($\geq$ 562·5 CPM) activity using threshold as per Landry *et al.*,[52] for the 6 d before each experimental trial. Physical activity data are presented for thirteen participants, as on two occasions the watches failed.

Sample processing and analysis

We were unable to obtain blood samples for one person, so blood data are presented for *n* 14. From these fourteen participants, there were four time points where blood samples could not be obtained. At each time point, 20 ml of blood was collected, and 5 ml was dispensed into an EDTA tube (1·6 mg EDTA/ml; Sarstedt), 4·5 ml was dispensed into a serum tube (clotting activator; Sarstedt) and left to clot for ~15 min, and 9 ml was dispensed into chilled EDTA protease inhibitor tubes (dipotassium EDTA < 100 %; tacrine < 20 % and BD P800). All tubes were centrifuged (2500 × *g*, 10 min, 4°C), before being aliquoted and stored at −80°C until analysis. Approximately 0·1 ml of blood was used for immediate determination of blood glucose concentrations (StatStrip Xpress 2 Glucose and Ketone Hospital Meter; Nova Biomedical; Waltham, MA; USA).

ELISA was used to determine ghrelin (Human Ghrelin (Total); Merck Millipore; Billerica, MA; USA), GLP-1 (GLP-1 Total; Merck Millipore; Billerica, MA; USA), PYY (Human PYY (Total); Merck Millipore; Billerica, MA; USA), leptin (Human Leptin ELISA kit; Merck Millipore; Billerica, MA; USA), insulin (insulin ELISA kit; Diametra; Spello; Italy), dipeptidyl peptidase-4 (sDPP-4) (Human sCD26 ELISA kit; Invitrogen; Vienna; Austria) and cholecystokinin (CCK) (CCK Raybiotech; Norcross, GA; USA). Serum samples were used to measure sDPP-4 and CCK, EDTA samples were used to analyse insulin, and leptin and EDTA protease inhibited samples were used to measure ghrelin, GLP-1 and PYY. Several of the samples for CCK were above the limits of detection, even after serial dilutions, and therefore, data are presented for *n* 9 participants. All inter-plate CV were < 15 %, and intra-plate CV were < 7 % for all analysis.

Statistical analyses

We performed a sample size calculation for our primary outcome, energy intake at the *ad libitum* meal. All other outcomes were considered secondary outcomes. Our calculation was based on previous studies examining the effects of whey protein[53] and milk[17] on post-exercise energy intake, which observed a ~700 KJ (~167 kcal) difference in energy intake between intervention and control conditions. Using a 700 KJ mean difference (and 700 SD units) and the following parameters for the power analysis: two tailed, α0·05, power 0·80, it was estimated that ten participants were required to detect a statistically significant difference in a paired *t* test (G * Power 3.1.9.2.)[54,55]

Data analysis was performed with jamovi (The jamovi project (2024). *jamovi* (Version 2.5) (Computer Software). Retrieved from https://www.jamovi.org). All data were checked for normality by inspecting histograms of the residuals; homogeneity of variance was checked by plotting the residuals against the predicted values. Energy intake, eating rate and physical activity levels were analysed with paired *t* tests; appetite and GI VAS, RPE, HR and all blood outcomes were analysed with linear mixed models. On the main exercise trial day, blood outcomes were measured with experimental condition (CP and CON) and time point modelled as fixed factors and participant as a random factor. In comparing blood samples taken fasted at familiarisation and at day 7, only one fixed factor was modelled (CP, CON and familiarisation). Random effects for participant intercepts were included in the models, which were estimated with restricted maximum likelihood. If there were significant main interaction effects, *post hoc* tests were performed using Holm–Bonferroni adjustments. Total area under the curve was calculated for all appetite and blood variables using the time series response analyser[56]. Correlation analysis was performed to assess relationships between variables showing statistically significant between condition differences; details and results are provided in the online Supplementary Material. All data are presented as mean (standard deviation). Statistical significance is accepted as $P < 0{\cdot}05$. Cohen's d_z effect sizes are presented for our primary outcome and any *post hoc* paired interaction effects; differences were quantified and interpreted as 'small' (0·20–0·49), 'medium' ($\geq$ 0·50–0·79) and 'large' ($\geq$ 0·80).

Results

There was 100 % compliance with supplement intake and the pre-trial dietary and exercise tracking and standardisation, as confirmed in writing and verbally on arrival to the laboratory. All diet diaries were inspected for accuracy and replication, with all participants following the guidance provided. Body mass was 60·4 (SD 9·9) in the CP trial and 60·5 (SD 10·2) in the CON trial. Body mass presented in Table 1 was recorded at familiarisation.

Physical activity

There was no difference in physical activity between the two conditions: sedentary, including sleep (CP: 5234 (SD 388) min; CON 5278 (SD 44) min; $P = 0{\cdot}746$), low activity (CP: 1932 (SD 230) min; CON 1966 (SD 259) min; $P = 0{\cdot}713$), moderate activity (CP: 810 (SD 238) min; CON 794 (SD 223) min; $P = 0{\cdot}695$) or vigorous activity (CP: 641 (SD 139) min; CON 546 (SD 164) min; $P = 0{\cdot}172$).

Subjective visual analogue scale (appetite and gastrointestinal) for 6 d pre-experimental trial

The data presented are the average for the 6 mornings for pre and 30 min post supplementation and are displayed in online Supplementary Table S1. There were no interaction effects for any variable on the appetite VAS ($P \geq 0{\cdot}299$). Hunger, thirst, desire to eat, prospective food consumption and composite appetite score increased 30 min post supplementation compared with waking (online Supplementary Table S1). Fullness had a condition effect ($P = 0{\cdot}027$), where fullness was higher in CON compared with CP.

Online Supplementary Table S2 shows the results from the GI VAS (as described above) for the 6 d for pre and 30 min post supplementation prior to attending the laboratory. Headache incidence, GI discomfort and urge to defecate did not differ between conditions ($P > 0{\cdot}05$). However, there was a condition

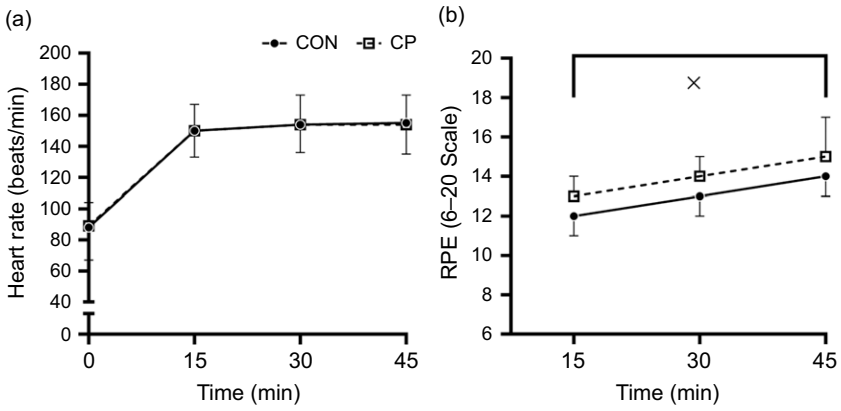

Figure 2. Heart rate (bpm; (a)) and rating of perceived exertion (RPE; 6–20 scale; (b)) measured throughout exercise for control (CON) and collagen peptides (CP) trials. ˣ denotes significant condition effects. Data presented for *n* 15.

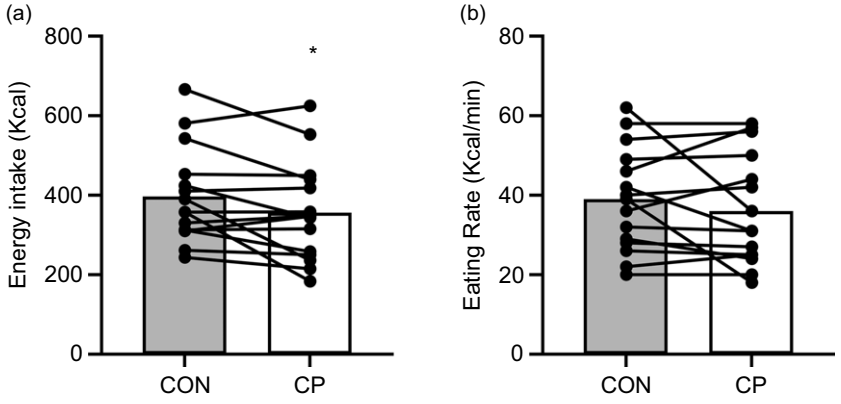

Figure 3. Energy intake (kcal; (a)) and eating rate (kcal/min; (b)) from the *ad libitum* meal on day 7, for control (CON) and collagen peptides (CP) trials. Bars represent means and lines individual responses. * denotes $P < 0{\cdot}05$. Data presented for *n* 15.

effect for nausea ($P = 0{\cdot}026$), which was marginally higher in CON compared with CP.

Exercise

There were no differences in heart rate during exercise between supplements (condition $P = 0{\cdot}981$; time × condition interaction $P = 0{\cdot}979$; Figure 2(a)), but heart rate did increase with the onset of exercise (time $P < 0{\cdot}001$). RPE increased over time ($P < 0{\cdot}001$), and there was a difference between conditions ($P = 0{\cdot}035$; d_z: 0·555) with RPE being higher in the CP trial but there was no time × condition interaction ($P = 0{\cdot}682$; Figure 2(b)).

Ad Libitum Pasta meal

Total energy intake was ~10 % (~41 kcal) lower in the CP trial compared with CON ($P = 0{\cdot}037$; $d_z = 0{\cdot}593$; Figure 3(a)). The reduction in energy intake remained when accounting for body mass (CP 6·1 (SD 2·7) kcal/kg; CON 6·8 (SD 2·7) kcal/kg; $P = 0{\cdot}022$; d_z: 0·666). However, there was no difference in eating rate ($P = 0{\cdot}263$; Figure 3(b)).

Subjective appetite ratings

There were time ($P < 0{\cdot}001$) effects, but no condition ($P \geq 0{\cdot}376$) or time × condition interaction effects ($P \geq 0{\cdot}279$) for hunger (Figure 4(a)), fullness (Figure 4(c)), desire to eat (Figure 4(e)), prospective food consumption (Figure 4(g)), thirst (Figure 4(i)) or composite appetite score (Figure 5(a)).

There was no difference in total area under the curve for hunger ($P = 0{\cdot}788$; Figure 4(b)), fullness ($P = 0{\cdot}744$; Figure 4(d)), desire to eat ($P = 0{\cdot}987$; Figure 4(f)), prospective food consumption ($P = 0{\cdot}932$; Figure 4(h)) thirst ($P = 0{\cdot}674$; Figure 4(j)) and composite appetite score ($P = 0{\cdot}833$; Figure 5(b)).

Gastrointestinal scales

There was no difference in nausea or GI discomfort between conditions (condition $P \geq 0{\cdot}587$; time × condition interaction $P \geq 0{\cdot}412$; online Supplementary Figure S1), but both nausea and GI discomfort changed over time ($P \leq 0{\cdot}008$). There was no difference in headache incidence over time ($P = 0{\cdot}579$), and there was no time × condition interaction ($P = 0{\cdot}833$; online Supplementary Figure S1), but there was a condition effect ($P = 0{\cdot}010$; $d_z = 0{\cdot}671$), indicating incidence was lower in the CP trial compared with CON. There was no difference between CP and CON and no time effects for urge to defecate (condition $P = 0{\cdot}767$; time × condition interaction $P = 0{\cdot}530$; time $P = 0{\cdot}785$; online Supplementary Figure S1).

Blood analysis at familiarisation

Samples were taken fasted before any supplementation at a familiarisation visit (PRE). We compared these values to those taken on day 7 of the main trials in the fasted state (0 min) after both CON and CP ingestion, to assess any baseline, fasted differences. There were no differences in ghrelin ($P = 0{\cdot}753$), insulin ($P = 0{\cdot}317$), PYY ($P = 0{\cdot}054$), leptin ($P = 0{\cdot}595$) or sDPP4 ($P = 0{\cdot}173$). CCK were significantly higher in CON compared with pre-supplementation (PRE) ($P = 0{\cdot}023$), but there was no

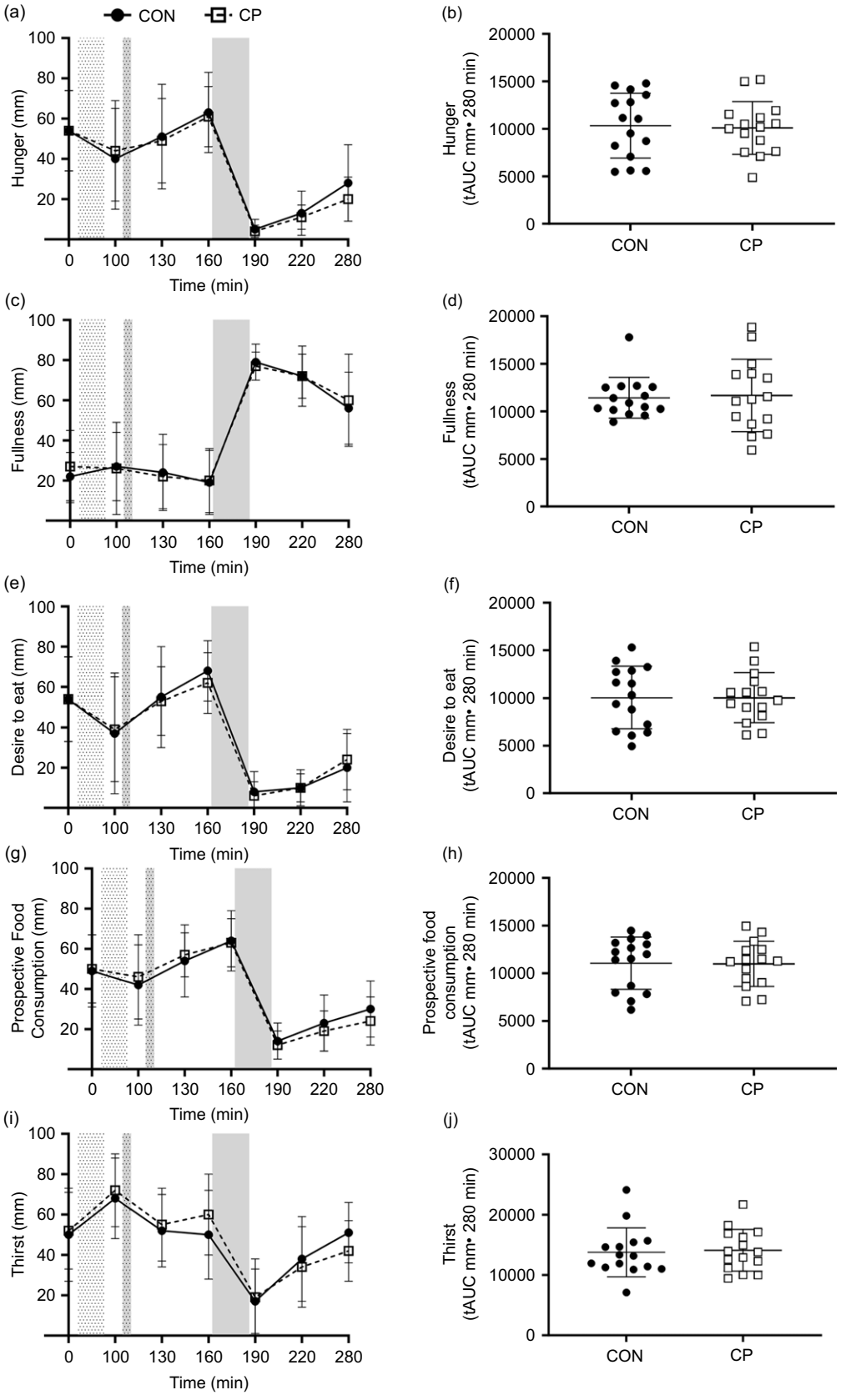

Figure 4. Appetite ratings (mm; hunger, (a); Fullness, (c); desire to eat, (e); prospective food consumption, (g); thirst, (i)) and tAUC (hunger, (b); Fullness, (d); desire to eat, (f); prospective food consumption, (h); thirst, (j)) for the control (CON) and collagen peptides (CP) on day 7. The first shaded area denotes exercise (45–90 min), the second supplement ingestion (100 min) and the third, the *ad libitum* meal (160–190 min). Data presented for *n* 15.

difference with CP consumption. GLP-1 was significantly lower in CON ($P = 0{\cdot}007$) compared with pre-supplementation but not different with CP consumption. Data are presented in online Supplementary Table S3.

Blood analysis on day 7

There were time effects for blood glucose concentrations ($P < 0{\cdot}001$); specifically, 0 min (fasted) was significantly lower

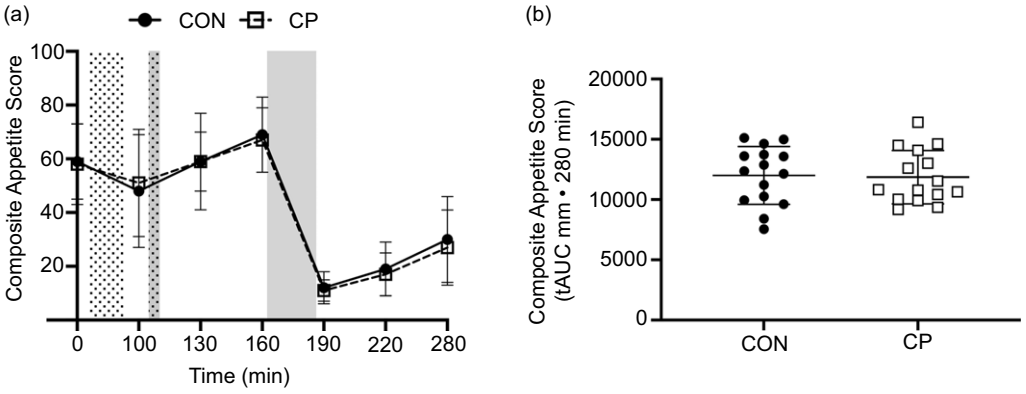

Figure 5. Composite appetite score (a) and tAUC (b) for the control (CON) and collagen peptides (CP) on day 7. The first shaded area denotes exercise (45–90 min), the second supplement ingestion (100 min) and the third, the *ad libitum* meal (160–190 min). Data presented for *n* 15.

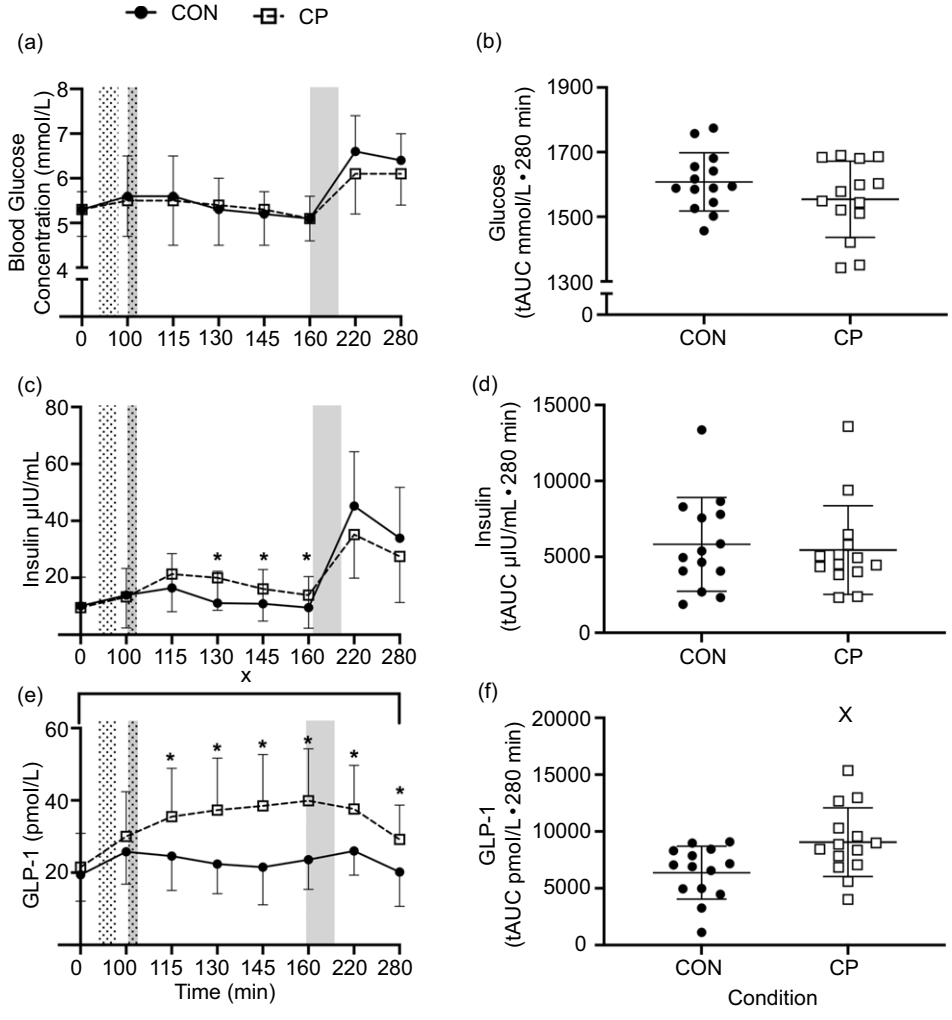

Figure 6. Blood glucose (a), plasma insulin (c), plasma glucagon peptide 1 (GLP-1) (e), and tAUC (blood glucose, (b); plasma insulin, (d); plasma GLP-1, (f)) for the control (CON) and collagen peptides (CP) on day 7. The first shaded area denotes exercise (45–90 min), the second supplement ingestion (100 min) and the third the *ad libitum* meal (160–190 min). *denotes *post hoc* significant time × condition effects ($P < 0{\cdot}05$); ˣ denotes significant condition effects. Data presented for *n* 14.

than post-meal (220 min: $P < 0{\cdot}001$; and 280 min: $P < 0{\cdot}001$; Figure 6(a)). There were no condition ($P = 0{\cdot}213$) or time × condition interaction effects ($P = 0{\cdot}648$).

There were time ($P < 0{\cdot}001$), condition ($P < 0{\cdot}001$; d_z: 3·87), and time × condition interaction effects ($P < 0{\cdot}001$; Figure 6(e)) for plasma total GLP-1 concentrations. Plasma GLP-1 was significantly higher in CP *v.* CON at all time points except 0 min and 100 min (115 min $P < 0{\cdot}001$; d_z: 1·36; 130 min $P < 0{\cdot}001$; d_z:1·86; 145 min $P < 0{\cdot}001$; d_z: 2·12; 160 min $P < 0{\cdot}001$; d_z: 2·17; 220 min $P < 0{\cdot}001$; d_z:1·146; 280 min $P < 0{\cdot}001$; d_z: 1·12). There was a time ($P < 0{\cdot}001$) and a time × condition interaction ($P < 0{\cdot}001$; Figure 6(c)) for plasma insulin concentrations but no effect of condition ($P = 0{\cdot}448$). Insulin concentrations were higher in CP *v.* CON at 130 min ($P < 0{\cdot}001$; d_z: 0·791), 145 min ($P < 0{\cdot}001$; d_z: 0·467) and 160 min ($P < 0{\cdot}001$; d_z: 0·390).

Both leptin (Figure 7(a)) and total ghrelin (Figure 7(c)) had effects of time ($P \leq 0{\cdot}001$) and effects of condition ($P \leq 0{\cdot}032$; d_z: $\geq 0{\cdot}577$) with lower concentrations of both in the CP trial

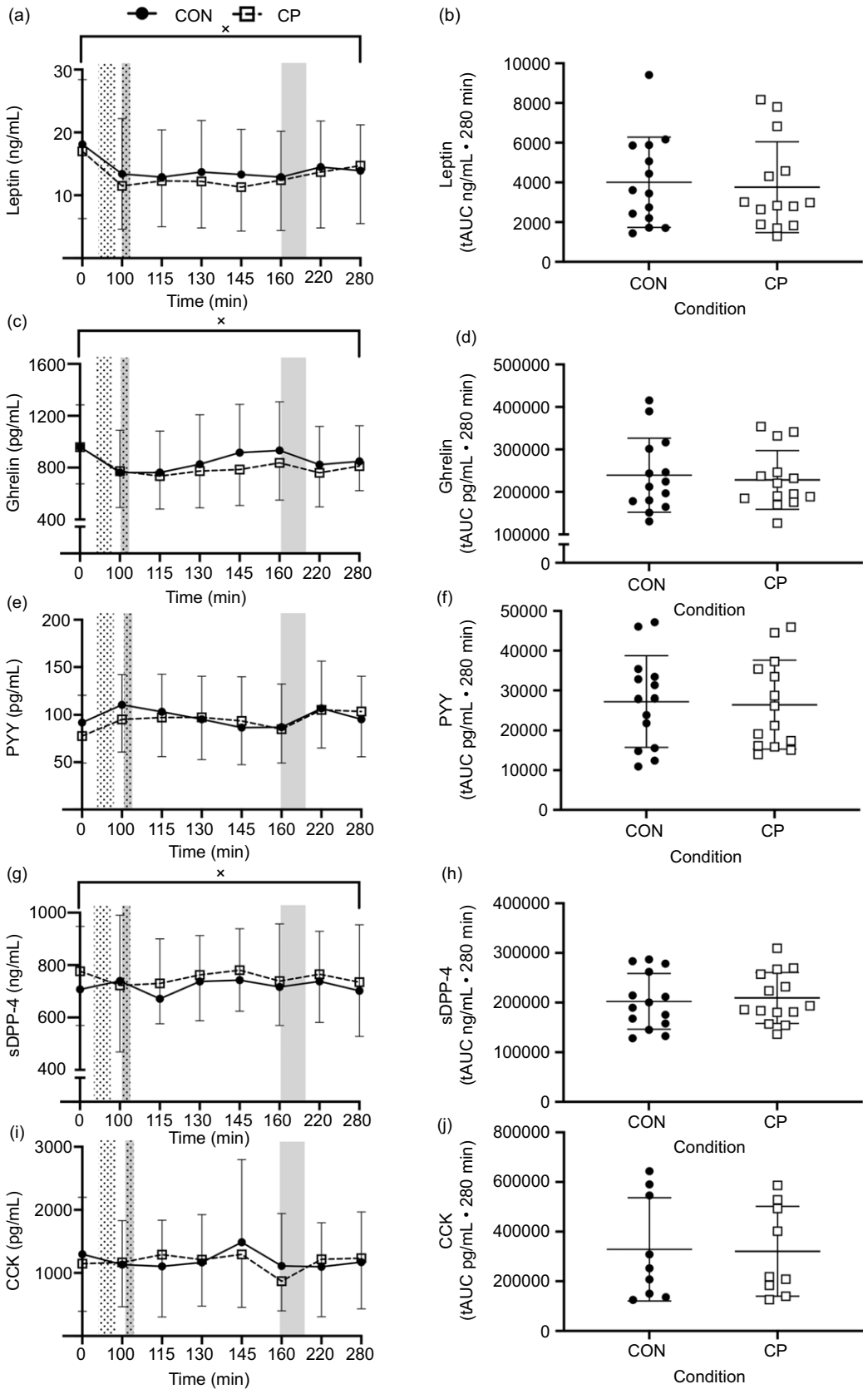

Figure 7. Plasma leptin (a), plasma ghrelin (c), plasma PYY (e) plasma sDPP-4 (g) and cholecystokinin (CCK) (i) concentrations and tAUC (plasma leptin, (b); plasma ghrelin, (d); plasma PYY, (f); plasma sDPP-4, (h); and plasma CCK; (j) for the control (CON) and collagen peptides (CP) on day 7. The first shaded area denotes exercise (45–90 min), the second supplement ingestion (100 min) and the third the *ad libitum* meal (160–190 min).ˣ denotes significant condition effects. Data presented for *n* 14. CCK, CON, control; PYY.

compared to CON. There were no time × condition interactions ($P \geq 0{\cdot}256$). PYY changed over time ($P < 0{\cdot}001$), but there was no condition ($P = 0{\cdot}743$) or time × condition interaction ($P = 0{\cdot}100$; Figure 7(e)). There were no time ($P = 0{\cdot}499$) or time × condition interactions ($P = 0{\cdot}870$; Figure 7(g)) for sDPP-4, but there was a condition effect ($P = 0{\cdot}036$; d_z: 0·566), with higher concentrations

in the CP trial. There were no time ($P = 0{\cdot}523$), condition ($P = 0{\cdot}494$), or time × condition interaction ($P = 0{\cdot}768$; Figure 7(i)) for CCK.

In the CP trial, total area under the curve for GLP-1 was significantly higher than CON ($P < 0{\cdot}001$; d_z: 0·999; Figure 6(f)). There was no difference in total area under the curve for glucose ($P = 0{\cdot}121$; Figure 6(b)), insulin ($P = 0{\cdot}421$; Figure 6(d)), leptin ($P = 0{\cdot}496$; Figure 7(b)), ghrelin ($P = 0{\cdot}237$; Figure 7(d)), PYY ($P = 0{\cdot}466$; Figure 7(f)), sDPP-4 ($P = 0{\cdot}346$; Figure 7(h)) and CCK ($P = 0{\cdot}623$; Figure 7(j)).

Discussion

The main findings of this study were that post-exercise intake of CP (15 g) reduced *ad libitum* energy intake (~41 kcal; medium effect size) in a subsequent meal compared to a flavour matched placebo (water). Whilst there were no treatment differences in subjective appetite and GI symptoms, ghrelin was lower and GLP-1 and insulin concentrations higher in the CP trial, which may partly explain the reduction in energy intake. However, 6 d of CP intake did not significantly affect morning subjective appetite sensations or fasting blood glucose and appetite-related hormone concentrations. This is the first study to report that consuming CP after exercise increases GLP-1 and insulin concentrations and may attenuate energy intake at a subsequent meal in healthy females.

Consuming CP 10 min after exercise reduced energy intake at an *ad libitum* meal 60 min later by 10 % (~41 kcal). These findings are consistent with previous studies examining whey protein supplementation[19,53] or milk[17,18], which have also been shown to reduce *ad libitum* energy intake following exercise. The decreased energy intake in those studies was greater than in the present study (~100 kcal), possibly owing to the larger doses of protein provided (21–30 g) and/or energy content of the supplements. Differences in protein source, supplement energy content, exercise type, meal composition or sex could also explain the differences in energy intake between studies. Only one previous study[24] has examined the effects of CP on appetite, and while they found no differences in energy intake between acute intake of CP and whey protein, direct comparisons with our study are not possible as no inert placebo was included. However, given previous studies in exercise contexts[19,53] have suggested whey protein reduces energy intake relative to a placebo/control, it is interesting that CP appears to also demonstrate this capacity. As in previous studies[7,57,58], we chose to compare CP to a non-energy matched placebo in a proof-of-concept design, as no previous study has established whether CP intake could affect appetite and hormone responses. This was especially important for our primary outcome (energy intake) and biochemical outcomes, as protein supplements have not been consistently shown to alter either compared to water or energy matched controls[4,18,53,57,59,60]. Indeed, future studies are required to examine whether CP has a greater influence on *ad libitum* energy intake than an energy matched control.

There were no differences in subjective ratings of appetite and satiety between the two supplements post-exercise on day 7. The lack of difference post-supplementation is somewhat surprising given that energy intake was reduced in the post-exercise meal. However, subjective appetite/satiety ratings and *ad libitum* energy intake do not necessarily correlate; indeed, several similarly designed studies reported no differences in appetite and satiety with post-exercise milk[46,47,51] or whey protein[19] ingestion, despite reduced *ad libitum* energy intake at a subsequent meal. In our study, the CP supplement was relatively low in energy (~60 kcal), which could partly explain the lack of change in subjective appetite. In addition, the aforementioned studies, and ours, were perhaps not adequately powered to detect small but statistically significant changes in subjective appetite/satiety. There were also no significant between supplement differences in fasted appetite or satiety in the 6 d pre-exercise. A condition effect showed that fullness was marginally higher with CON compared to CP (online Supplementary Table S1) but as fullness was rated higher before the supplement was consumed, it is unclear if this is directly related to the supplement that was consumed 24 h prior, or random chance. Regardless, our findings suggest that the reduction in energy intake is unlikely to be explained by changes in subjective appetite sensations.

One possible explanation for the reduced energy intake in the CP trial is the decrease in total ghrelin and increase in total GLP-1 and insulin. The moderate and strong correlations ($r \geq -0{\cdot}477$) between the difference in GLP-1 and insulin post-supplement on day 7, with the difference in energy intake between conditions, lend some support to this (online Supplementary Material). As ghrelin stimulates hunger[61], suppression of this hormone could have contributed to the reduced energy intake in the *ad libitum* meal with CP. However, there was no interaction effect, and a weak correlation with the difference in energy intake (online Supplementary Material) and the effect size was markedly lower than that for GLP-1. Interestingly, GLP-1, which stimulates insulin secretion and delays gastric emptying[62], remained significantly higher than the control in the 2 h after the *ad libitum* meal, suggesting the initial increase following CP intake cannot be attributed to energy intake. We are unaware of any previous studies examining CP on these hormones in humans, but *in vitro* studies suggest that CP[63] or collagen hydrolysates[64] increase GLP-1 partly though inhibiting DPP-4. However, in our study, sDPP-4 was slightly elevated in the CP trial (condition effect; $d_z = 0{\cdot}566$) suggesting other mechanisms likely explain the increased GLP-1. The increase could be partly attributed to the insulinotropic effects of specific peptides, or glycine, which has previously been shown to stimulate GLP-1[64–66]. Notwithstanding, our findings are in line with human studies involving gelatin supplementation whereby a single 20 g dose of gelatin (in both individuals with obesity and lean individuals) was shown to increase GLP-1 and insulin concentrations but not total PYY or total ghrelin[67]. In another study, diets rich in gelatin (10 % or 25 % of energy intake) increased GLP-1 and decreased ghrelin after meals and to a greater extent than after an energy matched casein protein diet[6]. Collectively, these studies and our findings suggest that CP may stimulate the satiety promoting hormones GLP-1 and insulin, which may lower energy intake. The mechanisms to explain these effects warrant further research.

Whilst there were no effects on PYY or CCK, leptin was lower in the CP trial (condition effect). Although not statistically significant, there was a strong positive correlation for the difference in leptin post-supplement and difference in energy intake (online Supplementary Material). This contrasts with a previous study[24] that reported higher leptin concentrations in the 120 min after consuming 40 g of CP compared with 40 g of whey protein. The discrepancy in findings with our study could be due to differences in dose and/or the comparator. Although leptin is thought to suppress food intake, *ad libitum* energy intake in our study was reduced by CP, suggesting leptin did not significantly influence appetite. This could be because the increase was small and not physiologically meaningful, or because leptin has a limited effect on acute hunger and satiety. Indeed, acute post-prandial changes in

leptin are not strongly associated with satiety or subsequent energy intake[68–70], likely in part because leptin concentrations do not tend to change until several hours post food intake, with fluctuations more associated with longer-term regulation of energy balance[71].

The study has limitations. First, our participants were young, physically active females, so our findings may not translate to males, the elderly or individuals with obesity. Second, during the exercise, there was no difference in HR between the trials, but RPE was higher (condition effect) during the CP trial, suggesting participants perceived the exercise as harder. As a higher RPE is associated with greater energy intake in some[72,73], but not all studies[74], it is feasible that the greater RPE could have increased appetite and energy intake in the CP trial. However, the influence on our results was likely limited, given the difference in RPE was minor (mean difference of 0·2–0·6), and energy intake was lower in the CP trial. There was also no correlation between the difference in RPE and energy intake between the two conditions (online Supplementary Material). Third, we did not quantify systemic changes in collagen-specific amino acids (glycine, proline and hydroxyproline) or other peptides during the trials. However, we are confident that these amino acids were elevated in the CP trial, as several previous studies have shown similar doses rapidly and markedly elevate systemic and urinary concentrations of these amino acids[46,47,75]. We also did not assess the effectiveness of the supplement blinding. Finally, we studied the satiating effects of CP over a relatively short period (7 d) with the energy intake assessment restricted to one *ad libitum* meal. Future research is warranted to examine the acute (e.g. single dose) and longer-term effects (e.g. 4 weeks) of CP intake and on appetite, energy intake and weight management in individuals with obesity and athletic populations, in the absence of exercise. In addition, future studies should assess the dose–response of CP intake on appetite and compare its effects on other dietary protein sources.

In conclusion, our findings demonstrate that 7 d of CP supplementation, with the final dose consumed post-exercise on day 7, increases circulating insulin and GLP-1 concentrations and reduces *ad libitum* energy intake in physically active females. Our findings suggest that if consuming CP after exercise for health or recovery benefits, there may be a compensatory decrease in energy intake at the next meal. However, the 10 % (41 kcal) reduction in energy intake after CP was relatively small, so may not be clinically meaningful. Future research should explore the underlying mechanisms and to examine the effects of longer-term CP supplementation, at different doses, on appetite and weight management in other populations.

Supplementary material. For supplementary materials referred to in this article, please visit https://doi.org/10.1017/S0007114525103851

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T.C., L.J.J., S.J.B, N.V. and J.P. conceptualised the study and acquired the funding. T.C., K.R. and L.J.J. approved the final study diet. T.C., K.R., J.T., E.H., M.F., A.T., S.J.B., L.J.J. and D.S. contributed to the methodology/use of software and data collection, with K.R. and T.C. leading project administration. T.C., K.R., J.T. and E.H., performed data analysis. T.C., K.R., J.T., E.H., M.F., A.T., S.J.B., L.J.J. and D.S. contributed to the writing and editing of the manuscript. N.V. and J.P. provided the supplements.

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