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ORIGINAL CONTRIBUTION

# Acute feeding with almonds compared to a carbohydrate-based snack improves appetite-regulating hormones with no e□ect on self-reported appetite sensations: a randomised controlled trial

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## Abstract

**Purpose** Early satiety has been identified as one of the mechanisms that may explain the beneficial e ects of nuts for reducing obesity. This study compared postprandial changes in appetite-regulating hormones and self-reported appetite ratings after consuming almonds (AL, 15% of energy requirement) or an isocaloric carbohydrate-rich snack bar (SB).

**Methods** This is a sub-analysis of baseline assessments of a larger parallel-arm randomised controlled trial in overweight and obese (Body Mass Index 27.5–34.9 kg/m$^2$) adults (25–65 years). After an overnight fast, 140 participants consumed a randomly allocated snack (AL [$n = 68$] or SB [$n = 72$]). Appetite-regulating hormones and self-reported appetite sensations, measured using visual analogue scales, were assessed immediately before snack food consumption, and at 30, 60, 90 and 120 min following snack consumption. A sub-set of participants (AL, $n = 49$; SB, $n = 48$) then consumed a meal challenge bu et ad libitum to assess subsequent energy intake. An additional appetite rating assessment was administered post bu et at 150 min.

**Results** Postprandial C-peptide area under the curve (AUC) response was 47% smaller with AL compared to SB ($p < 0.001$). Glucose-dependent insulinotropic polypeptide, glucagon and pancreatic polypeptide AUC responses were larger with AL compared to SB (18%, $p = 0.005$; 39% $p < 0.001$; 45% $p < 0.001$ respectively). Cholecystokinin, ghrelin, glucagon-like peptide-1, leptin and polypeptide YY AUCs were not di erent between groups. Self-reported appetite ratings and energy intake following the bu et did not di er between groups.

**Conclusion** More favourable appetite-regulating hormone responses to AL did not translate into better self-reported appetite or reduced short-term energy consumption. Future studies should investigate implications for longer term appetite regulation.

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**Keywords** Nuts · Almonds · Appetite · Satiety · Gastrointestinal peptides

## Introduction

The high prevalence of overweight and obesity is a major public health concern [1]. Obesity is characterised by an excess of body fat that impairs both physical and psychosocial health and well-being [2]. Long-term regulation of body weight is controlled by balancing energy intake with

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weight [8–16]. A recent meta-analysis reported no increase in body weight with diets that included nuts compared to nut-free diets, but did report reductions in waist circumference with consumption of almonds [17]. In another recent meta-analysis, a higher intake of nuts was associated with reductions in body weight and body fat [16].

It has been suggested that humans compensate for the energy from nuts by reducing intake of other foods at subsequent eating occasions [18]. This may be due to the satiating e ects of nuts, which possibly results from their high protein, fibre, and unsaturated fatty acid content, in conjunction with their low glycaemic load [19–21]. Additionally, nuts are associated with higher postprandial thermogenesis, which may raise resting energy expenditure with long-term consumption and help to balance the energy from nuts [9, 22]. Finally, it has been suggested that the available energy from nuts is less than predicted by the Atwater factor due to incomplete lipid release for absorption, therefore, contributing less energy than expected [19, 23].

Adaptive responses resulting from nut consumption may reflect e ects on hormones involved in appetite control [24]. Recent studies have suggested that nut consumption may influence appetite through the modulation of gastrointestinal and pancreatic peptides including glucagon-like peptide-1 (GLP-1) [13, 25, 26], glucose-dependent insulinotropic polypeptide (GIP) [25, 26], ghrelin [25, 27], peptide YY (PYY) and pancreatic polypeptide (PP) [26]. However, not all studies have reported the beneficial e ects of nut consumption on appetite-regulating hormones [28–31], possibly reflecting the complexity of adaptive responses and di ering study designs.

The purpose of this study was to compare the e ects of eating almonds or a carbohydrate-based snack on appetite-regulating hormones, self-reported appetite ratings, and short-term energy intake. We hypothesised that almonds would have favourable e ects on appetite-regulating hormones and self-reported appetite ratings, reducing subsequent energy intake compared to the carbohydrate-based snack, and thus providing insight into the association of nut consumption with a reduced risk of obesity.

## Materials and methods

### Ethics approvals and trial registration

Ethics approval was obtained from the University of South Australia Human Research Ethics Committee (201,436) and the trial was registered with the Australian and New Zealand Clinical Trials Registry (ATCRN12618001861246).

### Study setting, design and participants

Data reported here were obtained from a parallel-arm randomised controlled trial that was conducted between January 15, 2019 and March 10, 2021 in the research facilities of the Alliance for Research in Exercise, Nutrition and Activity Centre (ARENA) at the University of South Australia, Adelaide. Written informed consent was obtained from participants prior to participation. The intervention trial examined whether the inclusion of almonds or carbohydrate-rich snacks in an otherwise nut-free energy-restricted diet would promote weight loss and protect against weight regain. Energy requirements were calculated using the Schofield equation and physical activity captured via the International Physical Activity Questionnaire [32]. Energy recommendations for weight loss were set at 30% less than requirements. Participants then incorporated 15% of their energy-restricted diet as unsalted whole, natural Californian almonds with skins or a carbohydrate-rich snack (oven-baked fruit cereal bar and rice crackers), 6 days/week for 9 months. The full protocol for the larger study has been published [33]. This paper reports on outcomes from acute baseline appetite testing-.0clt(oec-.025 Tc0cSe.1(eppl.3(enda)]TJ0 Tc0 Tw(-)Tj.005 Tc.012 T

PP, PYY as well as C-peptide and glucagon were assessed using a multiplex analysis system (LUMINEX MAGPIX, Millipore, Merck). CCK was assessed using ELISA (Ray Biotech). All samples for the same participant were run in the same assay.

Participants were asked to rate their subjective appetite sensations by answering four questions at the time of each

### Appetite-regulating hormones

C-peptide AUC response was significantly smaller in AL compared to SB (46.9%, $P < 0.001$) (Table 2). Timepoint comparisons indicated a lower C-peptide concentration at 30, 60, 90 and 120 min ($P < 0.001$ for all time points) in AL compared to SB (Fig. 2).

The AL GIP AUC response was significantly larger than the response for SB (17.8%, $P = 0.005$) (Table 2). Higher concentrations occurred at time points 60 ($P = 0.010$), 90 ($P = 0.003$) and 120 min ($P = 0.005$) in AL compared to SB (Fig. 2).

The AL glucagon AUC response was significantly larger than the response for SB (38.7%, $P < 0.001$) (Table 2). Timepoint comparisons indicated a higher glucagon concentration at 30, 60, 90 and 120 min ($P < 0.001$ for all time points) in AL compared to SB (Fig. 2).

PP AUC response was significantly larger in AL compared to SB (44.5%, $P < 0.001$) (Table 2). Higher concentrations occurred at time point 30, 60, 90 and 120 min ($P < 0.001$ for all) in AL compared to SB (Fig. 2).

AUC for CCK, ghrelin, GLP-1, leptin and PYY did not di er between groups. Timepoint comparisons indicated a higher GLP-1 concentration at 60 ($P = 0.015$), 90 ($P < 0.001$) and 120 min ($P = 0.024$) in AL compared to SB (Supplementary Fig. 1).

### Subjective appetite ratings

There was no evidence of a difference in self-reported appetite sensations (feelings of hunger, fullness, satisfaction and prospective food consumption [prospective eating]), obtained via VAS, to the di erent test snacks. In both groups, hunger and prospective eating decreased post snack and steadily increased over the remainder of the testing period. Similarly, fullness and satisfaction increased in both groups post snack and decreased over the remainder of the testing period (Fig. 3).

### Meal challenge bu□et energy intake

There was no significant di erence in total energy intake (AL 2887 [194] kJ, SB 3185 [196] kJ; $p = 0.286$) or energy from core (AL 2120 [118] kJ, SB 2150 [119] kJ; $p = 0.860$) or discretionary foods (AL 767 [132] kJ, SB 1035 [133] kJagt6(b)4(di er)2.4(enY<8n2 SB -020.22.4(e ).5(w)(AL 767 [132] k

$p = 0.003$). Following the bu et meal (time point 150 min), the VAS responses for hunger, fullness, satisfaction, and pro

satiety [46] and may promote weight loss via increased thermogenesis, energy expenditure, and fatty acid oxidation [46]. The increased GIP and glucagon response in the AL group is likely due to the low carbohydrate and high fat and protein content of almonds. In keeping with our findings, Kendall et al. reported greater increases in GIP in an acute crossover feeding trial with pistachios compared to

testing. Longer term appetite assessment is also needed to further understand the e ects of nuts, appetite and weight management.

## Conclusion

Foods that promote satiety help to regulate energy balance and may assist with weight management. Future studies should consider test food dose and composition carefully as the volume of food, its sensory qualities, and the acceptance of the food respective of usual meal patterns, may be important in eliciting a feeling of fullness and satisfaction.

Appetite hormone responses may be skewed in obesity, so testing in a healthy weight population may provide addi-

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