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SYSTEMATIC REVIEW

Acute Exercise and Hormones Related to Appetite Regulation: A Meta-Analysis

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Abstract

Background Understanding of the impact of an acute bout of exercise on hormones involved in appetite regulation may provide insight into some of the mechanisms that regulate energy balance. In resting conditions, acylated ghrelin is known to stimulate food intake, while hormones such as peptide YY (PYY), pancreatic polypeptide (PP) and glucagon-like peptide 1 (GLP-1) are known to suppress food intake.

Objective The objective of this review was to determine the magnitude of exercise effects on levels of gastrointestinal hormones related to appetite, using systematic review and meta-analysis. Additionally, factors such as the exercise intensity, duration and mode, in addition to participant characteristics, were examined to determine their influence on these hormones.

Data Sources Major databases (PubMed, Scopus, Google Scholar, Science Direct, Academic Search Premier and EBSCOHost) were searched, through February 2013, for original studies, abstracts, theses and dissertations that examined responses of appetite hormones to acute exercise.

Study Selection Studies were included if they evaluated appetite hormone responses during and in the hours after an acute bout of exercise and reported area under the concentration–time curve (AUC) values for more than three datapoints. Studies reporting mean or pre/post-values only were excluded.

Study Appraisal and Synthesis Initially, 75 studies were identified. After evaluation of study quality and validity, using the Physiotherapy Evidence Database scale, data from 20 studies (28 trials) involving 241 participants (77.6 % men) had their data extracted for inclusion in the meta-analyses. A random-effects meta-analysis was conducted for acylated ghrelin ($n = 18$ studies, 25 trials) and PYY ($n = 8$ studies, 14 trials), with sub-group analyses and meta-regressions being conducted for moderator variables. Because the number of studies was limited, fixed-effects meta-analyses were performed on PP data ($n = 4$ studies, 5 trials) and GLP-1 data ($n = 5$ studies, 8 trials).

Results The results of the meta-analyses indicated that exercise had small to moderate effects on appetite hormone levels, suppressing acylated ghrelin (effect size [ES] Cohen's d value −0.20, 95 % confidence interval [CI] −0.373 to −0.027; median decrease 16.5 %) and increasing PYY (ES 0.24, 95 % CI 0.007 to 0.475; median increase 8.9 %), GLP-1 (ES 0.275, 95 % CI −0.031 to 0.581; median increase 13 %), and PP (ES 0.50, 95 % CI 0.11 to 0.89; median increase 15 %). No significant heterogeneity was detected in any meta-analysis (using Cochrane's Q and I^2); however, publication biases were detected for all analyses. No moderator variables were observed to moderate the variability among the studies assessing acylated ghrelin and PYY.

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Limitations The majority of the present literature is acute in nature; therefore, longer-term alterations in appetite hormone concentrations and their influence on food and beverage intake are unknown. Furthermore, our review was limited to English-language studies and studies reporting AUC data.

Conclusions An acute bout of exercise may influence appetite by suppressing levels of acylated ghrelin while simultaneously increasing levels of PYY, GLP-1 and PP, which may contribute to alterations in food and drink intake after acute exercise. Further longitudinal studies and exploration into mechanisms of action are required in order to determine the precise role these hormones play in long-term appetite responses to an exercise intervention.

1 Introduction

At the physiological and cellular level, human appetite and food intake are regulated by the neuroendocrine system [1]. Hormones secreted by the gastrointestinal tract work as regulators of appetite and food intake through mediation of hunger and satiety [1–3]. Of these hormones, ghrelin is the only known orexigenic peripheral peptide, being predominantly secreted by the gastric oxyntic cells and endocrine glands of the gastric mucosa [4]. Ghrelin exists in its total form, its acylated form and its des-acylated form [5]. While only about 10–20 % of circulating ghrelin is acylated ghrelin, this form is believed to be responsible for appetite stimulation [5–7]. Acylated ghrelin is of particular interest because it appears to be more susceptible to acute manipulation of energy balance through energy deficits or macronutrient ingestion [8]. Acylated ghrelin has only been able to be accurately measured since ~2005, and since then it has been examined extensively to elucidate its precise role in appetite regulation.

Acting in opposition to acylated ghrelin are a number of anorexigenic gastrointestinal hormones. These hormones include peptide YY (PYY; also known as peptide tyrosine-tyrosine), pancreatic polypeptide (PP), glucagon-like peptide 1 (GLP-1) and cholecystokinin. GLP-1 is formed by proteolytic cleavage in the L cells of the gut and is the most powerful incretin hormone [9, 10]. GLP-1 also influences glucose homeostasis, gastric emptying, insulin secretion and control of food intake [11]. PP is released by pancreatic F cells in response to vagal sensing, cholecystokinin and ghrelin concentrations, and sympathetic nervous system activation [12, 13]. Its major physiological role is to reduce food intake by delaying gastric emptying and motility via inhibition of pancreatic secretions and gallbladder motility [12, 13]. PYY is a member of the pancreatic polypeptide-fold family and is produced by intestinal L-cells [4, 14]. PYY has two major forms, $PYY_{1–36}$ and $PYY_{3–36}$, with $PYY_{3–36}$ being the predominant circulating form and playing a greater role in appetite regulation—in particular, satiety and meal termination [15]. Like other gastrointestinal satiety hormones (GLP-1, PP and cholecystokinin), PYY levels react to energy balance or macronutrient ingestion, increasing after a meal while causing a delay in gastric emptying, thereby prolonging feelings of satiety and fullness [14, 16, 17].

The effect of exercise on these hormones, and energy intake, has been studied extensively throughout the past decade [18–27]. Although there is no definitive consensus, there is evidence suggesting that concentrations of plasma acylated ghrelin are suppressed after strenuous endurance exercise, while concentrations of anorexigenic hormones (PYY, PP and GLP-1) are increased [26, 28, 29]. These hormonal changes have been shown to track with associated changes in appetite at rest (as assessed using subjective visual analogue scales for feelings such as hunger, fullness, satisfaction and prospective food consumption) and may provide a potential mechanism for alterations of appetite or food and beverage intake post-exercise [19, 29, 30].

The characteristics of the exercise interventions that have examined alterations in appetite hormones have varied considerably. Most studies have utilized moderate to vigorous cycling or running at intensities ranging from 50 to 75 % of maximal oxygen uptake ($\dot{V}O_{2max}$) and lasting approximately 1 h [21, 22, 25, 26, 29, 31, 32]. Other modes of activity such as swimming [33], resistance exercise [28, 34] and sprint interval training [35] have also been evaluated. Additionally, most of these studies have utilized relatively small sample sizes. Finally, previous reviews have been narrative in nature [18, 19].

With these factors in mind, a systematic review and meta-analysis of existing data is necessary to specifically examine how bouts of acute exercise impact hormonal markers related to appetite. This should provide quantification of the changes in appetite-regulating hormone levels after acute exercise, and could also determine which study characteristics may best explain these observed changes. Our purpose, therefore, was to conduct a meta-analysis of data from studies that have examined the effect of an acute bout of exercise on acylated ghrelin, PYY, PP and GLP-1 concentrations. These hormones were selected over others such as leptin because of the knowledge (as evidenced in the existing literature) that they respond to stimuli such as acute perturbations in energy balance. While there are other hormones involved in appetite regulation and energy balance, these particular hormones have the strongest literature base at the present time. Acylated ghrelin is particularly important because it is the only known peripheral hormone to stimulate feeding behaviour. Hormones related

to satiety, such as PYY, PP and GLP-1, are also important to examine because changes in these hormones may lead to alterations in gastrointestinal physiology that may contribute to alterations in feeding. Using sub-group analyses and meta-regression, we also hoped to identify which study characteristics could explain the variations in the results and best predict changes in hormone levels. The outcomes from this meta-analysis may serve to better inform researchers on what is known about the effects of exercise on appetite-regulating hormones, and may provide mechanistic insight into exercise-induced alterations in food and beverage intake.

2 Methods

2.1 Study Selection and Inclusion Criteria

Major research databases (PubMed, Scopus, EBSCOHost, Google Scholar, Academic Search Premier, ScienceDirect and SpringerLink) were searched from 1 July 2012 through 1 February 2013. Keyword searches were performed for 'exercise', 'physical activity', 'energy expenditure', 'energy intake', 'appetite', 'hunger', 'food intake', 'acylated ghrelin', 'peptide YY', 'PYY', 'glucagon-like peptide 1', 'GLP-1', 'pancreatic polypeptide' and 'PP'. (Details of the search strategy are provided in the Electronic Supplementary Material.) Potential studies were identified by examining the abstracts, and full-text copies were obtained if they met the initial criteria of evaluating appetite hormone changes in response to an acute exercise bout. Guidelines from the PRISMA (Preferred Reporting in Systematic Reviews and Meta-Analyses) Statement were followed in the preparation of this paper, including use of the PRISMA checklist for reporting systematic reviews and meta-analyses (provided in the Electronic Supplementary Material) [36].

Participants in the studies were required to be non-smoking adults (lean, overweight and/or obese), with no history of chronic disease and no contraindications to exercise. The selection criteria were not limited by study duration or observation time post-exercise. Study selection was also not limited by a set intensity or duration of the exercise bout, nor was inclusion constrained by exercise modality. Food intake during the observation period was permitted, and we have reported data on food intake responses to acute exercise bouts elsewhere [37]. For the majority of the studies that evaluated food intake ($n = 18$), the observation period ended prior to meal consumption ($n = 10$). For the remaining studies, meals were either standardized between conditions ($n = 2$), were a combination of standardized and ad libitum meals ($n = 2$) or were offered ad libitum ($n = 4$). The final six studies were examined to determine if changes in food intake at the ad libitum meals may have altered the hormone data, but no differences in food intake at individual meals were reported between the exercise and control trials [20–23, 38, 39].

All studies were required to have a control condition for inclusion and were required to employ trial order randomization. The control condition was required to be the same as the exercise condition with regard to the protocol, minus the exercise bout. Studies were included if they had more than three datapoints over time and utilized area under the concentration–time curve (AUC) data for analysis, since this is the tool most widely used for examination of hormonal changes over time. In this situation, the AUC of a particular hormone is calculated by plotting hormonal concentrations against time (the observation period), using the trapezoidal rule. Although the AUC is a common metric for reporting hormone concentrations by time, the methods of calculating the AUC can be variable, depending on the sampling rate, whether the AUC considers total or above/below baseline values only, and how researchers divide their 'data bins' during analysis. This has led to a large variation in AUC values between studies for similar periods of time (Table 1).

Since the interventions were exercise bouts, investigators were not blinded. Studies were included if they were published in peer-reviewed journals or were available in conference proceedings, theses or dissertations. We chose a broad range of sources for study inclusion to minimize the risk of publication bias, which can occur if only published studies are included (given that studies with larger effect size (ES) values are more likely to be published in the peer-reviewed literature) [36].

2.2 Exclusion Criteria

Studies were excluded from further analysis if they did not measure or report AUC data for acylated ghrelin, PYY, GLP-1 and/or PP in response to an exercise bout. Studies were also excluded if they lacked a control trial. In the event that a study reported hormone data in graphical form and/or did not report a standard deviation, the corresponding author was contacted to request the raw data for synthesis. Studies that examined environmental factors had their data extracted for control and normal/neutral exercise conditions only [25, 40].

2.3 Data Synthesis

Once studies were obtained, they were assessed for quality and validity independently by two authors (MS and ML), using established criteria (Physiotherapy Evidence Database [PEDro], http://www.pedro.org.au/english/downloads/

Table 1 Effects of acute exercise on hormone area under the concentration–time curve (AUC) data

Study[a]	Participants, BMI (mean ± SD)	Intervention	Sampling medium, AUC duration and analytical method	Hormone AUC (pg·mL^{-1}; mean ± SD)[b]				PEDro Score
				Acylated ghrelin	PYY	GLP-1	PP	
Broom et al. [29]	9 men, BMI 22.2 ± 2.1 kg/m^2	60 min treadmill running @ 72 % $\dot{V}O_{2max}$	Venous 9 h ELISA	CON: 1,401 ± 1,564 EX: 917 ± 1,026*	NM	NM	NM	6
Martins et al. [24]	6 men and women combined, BMI 22.0 ± 3.2 kg/m^2	60 min cycling @ 65 % HR_{max}	Venous 3.5 h RIA	NM	CON: 21,070 ± 4,652 EX: 21,384 ± 5,998	CON: 24,761 ± 6,270 EX: 29,306 ± 7,199	CON: 11,570 ± 7,144 EX: 19,770 ± 10,149	6
Broom et al. [28]	11 men, BMI 23.1 ± 1.3 kg/m^2	60 min treadmill running @ 70 % $\dot{V}O_{2max}$	Venous 8 h ELISA	CON: 811 ± 852 EX: 736 ± 895	CON: 1,411 ± 365 EX: 1,750 ± 564*	NM	NM	6
Broom et al. [28][c]	11 men, BMI 23.1 ± 1.3 kg/m^2	90 min resistance training: 10 exercises, 3 sets, 12 reps @ 80 % 12-RM	Venous 8 h ELISA	CON: 811 ± 852 EX: 696 ± 650	CON: 1,411 ± 365 EX: 1,381 ± 322	NM	NM	6
Shorten et al. [25]	11 men, BMI 24.1 ± 2.3 kg/m^2	40 min treadmill running @ 70 % $\dot{V}O_{2max}$	Capillary 1.7 h Multiplex	CON: 144 ± 112 EX: 109 ± 40	CON: 183 ± 72 EX: 195 ± 73	NM	CON: 192 ± 107 EX: 229 ± 114	7
Ueda et al. [26]	7 obese men, BMI 30.0 ± 3.1 kg/m^2	60 min cycling @ 50 % $\dot{V}O_{2max}$	Venous 2 h ELISA	CON: 31,651 ± 20,151 EX: 33,380 ± 21,514	CON: 729 ± 92 EX: 788 ± 84*	CON: 317 ± 117 EX: 353 ± 107*	NM	7
Ueda et al. [26][c]	7 men, BMI 22.4 ± 2.4 kg/m^2	60 min cycling @ 50 % $\dot{V}O_{2max}$	Venous 2 h ELISA	CON: 19,979 ± 4,391 EX: 23,202 ± 4,682	CON: 853 ± 205 EX: 951 ± 216*	CON: 435 ± 256 EX: 584 ± 344*	NM	7
Ueda et al. [50]	10 men, BMI 22.5 ± 1 kg/m^2	30 min cycling @ 50 % $\dot{V}O_{2max}$	Venous 1 h ELISA	NM	PYY (3–36) measured	CON: 265 ± 94 EX: 314 ± 95*	NM	7
Ueda et al. [50][c]	10 men, BMI 22.5 ± 1 kg/m^2	30 min cycling @ 75 % $\dot{V}O_{2max}$	Venous 1 h ELISA	NM	PYY (3–36) measured	CON: 265 ± 94 EX: 313 ± 89*	NM	7
King et al. [21]	9 men, BMI 23.6 ± 1.2 kg/m^2	90 min treadmill running @ 70 % $\dot{V}O_{2max}$	Venous 10 h ELISA	CON: 934 ± 387 EX: 697 ± 345*	NM	NM	NM	6
King et al. [20]	14 men, BMI 23.4 ± 2.2 kg/m^2	60 min self-paced 'brisk walking' (7.0 ± 0.4 km/h; 45 ± 7.5 % $\dot{V}O_{2max}$)	Venous 8 h ELISA	CON: 395 ± 55 EX: 390 ± 129	NM	NM	NM	6
Unick et al. [27]	19 pre-menopausal, overweight women, BMI 32.5 ± 4.3 kg/m^2	~45 min treadmill walking @ 70–75 % HR_{max}	Venous 3 h ELISA	CON: 13,053 ± 5,901 EX: 12,721 ± 6,677	NM	CON: 423 ± 103 EX: 402 ± 99*	NM	6.5

Table 1 continued

Study[a]	Participants, BMI (mean ± SD)	Intervention	Sampling medium, AUC duration and analytical method	Hormone AUC (pg·mL^{-1}; mean ± SD)[b]				PEDro Score
				Acylated ghrelin	PYY	GLP-1	PP	
Balaguera-Cortes et al. [34]	10 men, BMI 23.7 ± 2.0 kg/m^2	45 min treadmill running @ 70 % $\dot{V}O_{2max}$	Capillary 2 h Multiplex	CON: 109 ± 43 EX: 111 ± 68	CON: 109 ± 27 EX: 112 ± 38	NM	CON: 103 ± 85 EX: 226 ± 152	6
Balaguera-Cortes et al. [34][c]	10 men, BMI 23.7 ± 2.0 kg/m^2	45 min RT: 3 sets of 12 reps or to failure of 8 exercises, 1 min between sets	Capillary 2 h Multiplex	CON: 109 ± 43 EX: 87 ± 47*	CON: 109 ± 27 EX: 105 ± 32	NM	CON: 103 ± 85 EX: 115 ± 86	6
King et al. [22]	12 men, BMI 22.8 ± 1.4 kg/m^2	90 min treadmill running @ 70 % $\dot{V}O_{2max}$	Venous 9 h ELISA	CON: 1,055 ± 956 EX: 961 ± 880*	PYY (3–36) measured	NM	NM	6
King et al. [33]	14 men, BMI 23.2 ± 2.2 kg/m^2	60 min intermittent swimming [6 × (7 min swim/3 min rest)]	Venous 3 h ELISA	CON: 505 ± 651 EX: 473 ± 696*	NM	NM	NM	6
Vatansever-Ozen et al. [66]	10 men, BMI 22.0 ± 0.4 kg/m^2	105 min treadmill running @ 50 % $\dot{V}O_{2max}$ + 15 min @ 70 % $\dot{V}O_{2max}$	Venous 4 h ELISA	CON: 1,077 ± 63 EX: 1,004 ± 92*	NM	NM	NM	5.5
Becker et al. [31]	8 men, BMI 24 ± 0.9 kg/m^2	60 min cycling @ 70 % $\dot{V}O_{2max}$	Venous 2 h ELISA	CON: 96.8 ± 53.2 EX: 60.7 ± 24.3*	NM	NM	NM	5.5
Kelly et al. [40]	10 men, BMI 23.9 ± 2.1 kg/m^2	45 min treadmill running @ 70 % $\dot{V}O_{2max}$	Capillary 2 h Multiplex	CON: 152 ± 62 EX: 145 ± 67	CON: 189 ± 35 EX: 197 ± 56	NM	CON: 193 ± 98 EX: 206 ± 88	7
Larson-Meyer et al. [32]	9 female runners, BMI 19.8 ± 1.0 kg/m^2	60 min treadmill running @ 70 % $\dot{V}O_{2max}$	Venous 2 h RIA	CON: 8,071 ± 11,319 EX: 21,466 ± 17,292*	CON: 17,040 ± 12,780 EX: 17,484 ± 19,892	CON: 5,392 ± 6,088 EX: 6,323 ± 5,660	NM	4.5
Larson-Meyer et al. [32][c]	10 female walkers, BMI 22.1 ± 3.4 kg/m^2	60 min treadmill walking @ 70 % $\dot{V}O_{2max}$	Venous 2 h RIA	CON: 7,744 ± 9,660 EX: 9,734 ± 13,885	CON: 13,952 ± 15,232 EX: 28,008 ± 26,948	CON: 6,940 ± 6,814 EX: 8,293 ± 8,936	NM	4.5
Wasse et al. [39]	10 men, BMI 24.8 ± 2.4 kg/m^2	60 min treadmill running @ 70 % $\dot{V}O_{2max}$	Venous 7 h ELISA	CON: 755 ± 541 EX: 644 ± 388*	CON: 848 ± 224 EX: 912 ± 292*	NM	NM	6.5
Wasse et al. [49]	11 men, BMI 23.4 ± 2.3 kg/m^2	60 min treadmill running @ 70 % running $\dot{V}O_{2max}$	Venous 4 h ELISA	CON: 606 ± 378 EX: 455 ± 355*	NM	NM	NM	5.5
Wasse et al. [49][c]	11 men, BMI 23.4 ± 2.3 kg/m^2	60 min cycling @ 70 % cycling $\dot{V}O_{2max}$	Venous 4 h ELISA	CON: 606 ± 378 EX: 448 ± 315	NM	NM	NM	5.5

Table 1 continued

Study[a]	Participants, BMI (mean ± SD)	Intervention	Sampling medium, AUC duration and analytical method	Hormone AUC ($pg \cdot mL^{-1}$; mean ± SD)[b]				PEDro Score
				Acylated ghrelin	PYY	GLP-1	PP	
Deighton et al. [35]	12 men, BMI 24.2 ± 2.9 kg/m^2	60 min cycling @ 65 % $\dot{V}O_{2max}$	Venous 6.25 h ELISA	CON: 344 ± 146 EX: 277 ± 112*	CON: 915 ± 310 EX: 1,028 ± 352	NM	NM	5.5
Deighton et al. [35][c]	12 men, BMI 24.2 ± 2.9 kg/m^2	30 min sprint-interval exercise: 3.5 min warm-up, 6 × 30 s Wingate tests (4 min rest between), 3.5 min warm-down	Venous 6.25 h ELISA	CON: 344 ± 146 EX: 237 ± 116*	CON: 915 ± 310 EX: 991 ± 269	NM	NM	5.5
Hagobian et al. [48]	11 men, BMI 26 ± 4 kg/m^2	Cycling @ 70 % $\dot{V}O_{2max}$ until 30 % EDEE (82 ± 13 min)	Venous 2 h ELISA	CON: 83,213 ± 118,573 EX: 123,914 ± 146,227	PYY (3–36) measured	NM	NM	6
Hagobian et al. [48][c]	10 women, BMI 24 ± 2 kg/m^2	Cycling @ 70 % $\dot{V}O_{2max}$ until 30 % EDEE (84 ± 17 min)	Venous 2 h ELISA	CON: 53,740 ± 81,063 EX: 35,121 ± 25,128	PYY (3–36) measured	NM	NM	6

12-RM 12 repetitions maximum, *BMI* body mass index, *CON* resting control trial, *EDEE* estimated daily energy expenditure, *ELISA* enzyme-linked immunosorbent assay, *EX* exercise trial, *GLP-1* glucagon-like peptide 1, HR_{max} maximum heart rate, *NM* not measured, *PEDro* Physiotherapy Evidence Database scale, *PP* pancreatic polypeptide, *PYY* peptide YY, *reps* repetitions, *RIA* radioimmunoassay, *RT* resistance training, $\dot{V}O_{2max}$ maximum oxygen uptake

* Significantly different from control (as reported within studies; $p < 0.05$)

[a] Studies are arranged alphabetically by author and year of publication

[b] Values reported as $pmol \cdot L^{-1}$ were converted to $pg \cdot mL^{-1}$ by multiplying by 3.38 for acylated ghrelin, 4 for PYY, 3.3 for GLP-1 and 2.39 for PP

[c] These data are for the second condition/participant population in this study, which included more than one condition/participant population

pedro-scale/ [41]), with a third reviewer (BD) being consulted if there was a discrepancy between scores. Inter-rater reliability and agreement were reported as Cohen's kappa [42]. The following data were extracted by one author (MS) into a computerized spreadsheet: the name of the first author and the year of the study publication, hormone AUC data (pmol·L^{-1}/h, pg·mL^{-1}/h) for exercise and control conditions, gross exercise energy expenditure (ExEE), sample size, participant characteristics, blood analytical methods, information about meals provided, and exercise intervention information.

In studies that reported hormone values in pmol·L^{-1}, values were converted to pg·mL^{-1} as follows: multiplied by 4 for PYY, 3.38 for acylated ghrelin, 3.297 for GLP-1 and 2.39 for PP. Standard error of the mean (SEM) values were converted to standard deviation values. All descriptive data are reported as ranges with median values.

2.4 Meta-Analysis Procedures

Upon data extraction, all data were entered into software designed specifically for meta-analyses (Comprehensive Meta-Analysis, version 2; Biostat, Englewood, NJ, USA). The input data included the sample sizes, AUCs for the control and exercise conditions (with their respective standard deviations) and mean differences between control and exercise trials. The software calculated the standardized difference in means to determine the ES, expressed as Cohen's d, for each study; additionally, Hedge's g was used to account for potential bias due to the small sample sizes in the reviewed studies. There were no differences between Hedge's g and Cohen's d, so we report Cohen's d ES values only. Overall ES values for acylated ghrelin and PYY were calculated using a random-effects model that accounts for true variation in effects occurring from study to study, as well as random error within a single study. The random-effects model was chosen over a fixed-effects model because experimental factors such as ExEE and intensity varied considerably among studies, and a random-effects model better accounts for these variations during analysis [43]. Because of the smaller number of studies ($n < 10$), we utilized fixed-effects modelling for analysis of GLP-1 and PP; however, the data yielded no difference between fixed- or random-effects modelling for those hormones.

In accordance with Cohen [44], we interpreted ES values of <0.2 as trivial, 0.2–0.3 as small, 0.4–0.8 as moderate and >0.8 as large. A negative ES indicates that exercise was associated with *decreased* hormone levels, while a positive ES indicates that hormone levels *increased* with exercise.

Heterogeneity was calculated as Cochrane's Q and the I^2 index. Values of 25, 50 and 75 % were used for the I^2 analysis and correspond to low, moderate and high heterogeneity, respectively [45]. For Cochrane's Q, significant heterogeneity is considered to exist when the Q value exceeds the degree of freedom (d_f) of the estimate [46]. Sensitivity analyses were conducted by excluding one study at a time to examine if the results were driven by any one study.

To assess whether differences in experimental design could explain the variation in ES values between the studies evaluating acylated ghrelin and PYY, we performed sub-group meta-analyses and/or meta-regressions (method-of-moments model), as has been performed previously [37]. This analysis included meta-regressions of continuous data, such as the energy expenditure of exercise, exercise duration, exercise intensity, body mass index (BMI) and length of AUC observation time. Sub-group meta-analyses were conducted for categorical data, such as the exercise mode, fed state, sex (men, women or both) and hormone analytical method (enzyme-linked immunosorbent assay [ELISA], radioimmunoassay [RIA] or Multiplex).

Publication bias was assessed utilizing funnel plots, as described previously [37] (see Figs. S1–4 in the Electronic Supplementary Material). If there is no publication bias, studies should be distributed evenly around the mean ES because of random sampling error. The trim-and-fill correction described by Duval and Tweedie [47] was used to assess bias. This technique allows for computation and inclusion of potentially missing studies to create symmetry about the overall mean ES.

Statistical significance was set at $p < 0.05$ in a Z test analysis. The Z tests were utilized to examine if ES values were significantly different from zero.

3 Results

3.1 Overview

Figure 1 presents the decision tree of study selection. In total, 75 studies were initially identified. After filtering, 20 studies met the inclusion criteria for the meta-analyses. All studies had been published (or accepted for publication) in peer-reviewed scientific journals. In summary, the experimental trials within the studies were conducted over the course of several hours and generally began with participants either ingesting a standardized breakfast meal (providing either an absolute amount of energy or a relative amount of carbohydrate, set as g·kg^{-1} body mass) followed by a bout of exercise, or a bout of exercise in the fasted state. Throughout the trials, blood samples were taken at regular intervals.

The studies on acute exercise and changes in appetite-related hormones are summarized in Table 1. Multiple studies utilized more than one category of participants (lean versus obese, runners versus walkers, and men versus women) [26, 32, 48] or different modes or intensities of

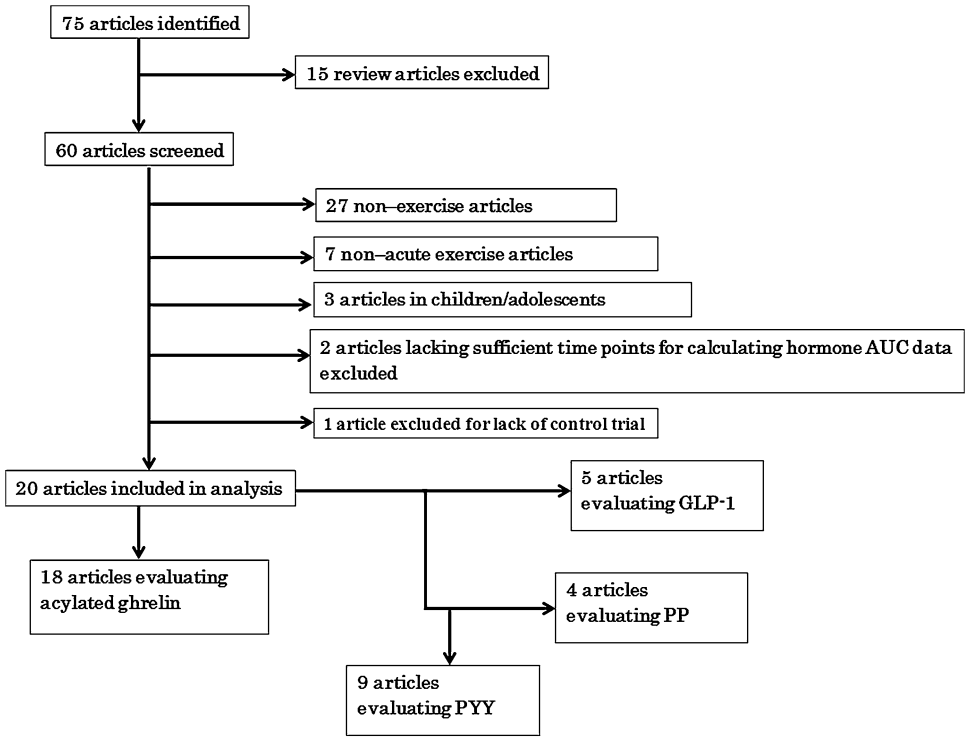

Fig. 1 Flowchart of study selection. *AUC* area under the concentration–time curve, *GLP-1* glucagon-like peptide 1, *PP* pancreatic polypeptide, *PYY* peptide YY

exercise [28, 34, 49, 50]. Therefore, those studies are reported as two trials. When accounting for differences, this raised the total number of trials to 28, each with an exercise condition and a control condition. In summary, 18 studies (25 trials) reported acylated ghrelin AUC data, nine studies (14 trials) reported PYY AUC data, five studies (eight trials) reported GLP-1 AUC data and four studies (five trials) reported AUC data for PP. Eleven of the 28 trials utilized cycling as the mode of exercise, 11 utilized running, three utilized walking, two utilized resistance training and one utilized swimming. Fourteen of the 28 trials were conducted 1–3.5 h postprandially (median 1.75 h), while the remaining 14 trials were conducted after an 8 to 10 h fast. The energy value of the pre-exercise meal ranged from 882 to 3,423 kJ (median 2,345 kJ). The mean PEDro score for the 20 studies was 6.08 ± 0.63. Cohen's kappa, indicative of the level of agreement between reviewers (where −1.0 is perfect disagreement, 0 is random agreement/disagreement and 1.0 is perfect agreement [42]), was equal to 0.91. All studies were generally of high quality; however, the PEDro scores may not accurately reflect this, because of the inherent problem of blinding, which accounts for three of the 11 items on the PEDro checklist [41].

3.2 Participant Demographics and Exercise Intervention Characteristics

The majority of participants ($n = 241$) were men ($n = 187$; 77.6 %), with BMI values ranging from 19.8 to 32.5 $kg \cdot m^{-2}$ (median 23.4 $kg \cdot m^{-2}$) and $\dot{V}O_{2max}$ values between 34 and 63 $mL \cdot kg^{-1} \cdot min^{-1}$ (median 56.9 $mL \cdot kg^{-1} \cdot min^{-1}$). Aerobic exercise interventions ranged from 30 to 120 min (median 60 min) at an intensity of 45–75 % $\dot{V}O_{2max}$ (median 70 % $\dot{V}O_{2max}$). Resistance exercise interventions were 45 and 90 min long, at intensities of ~80 % of 10–12 repetitions maximum (the protocols are summarized in Table 1). Gross energy expended during the exercise bouts ranged from 600 to 6,500 kJ (median 2,730 kJ). There was a median of 11 participants per study (range 7–21). The length of all trials ($n = 28$) was between 1.5 and 9 h (median 4 h).

3.3 Alterations in Appetite Hormone Levels in Response to Exercise

A median 16.5 % decrease was observed in the median acylated ghrelin AUC values ($n = 25$) over a median 4 h period (control 811 ± 541 vs. exercise 696 ± 388 $pg \cdot mL^{-1}$). Conversely, the median PYY AUC values ($n = 14$) increased by 8.9 % over the same median 4 h period (control 884 ± 267 vs. exercise 970 ± 280 $pg \cdot mL^{-1}$). The median GLP-1 AUC values ($n = 8$) increased by 13.1 % over a median 2 h period (control 428 ± 186 vs. exercise 493 ± 225 $pg \cdot mL^{-1}$). Finally, median PP AUC values ($n = 5$) increased by 15 % over a median 2 h period (control 192 ± 98 vs. exercise 226 ± 114 $pg \cdot mL^{-1}$).

3.4 Meta-Analysis

Individual study statistics and results for each model are provided in Tables S1–4 in the Electronic Supplementary Material.

3.4.1 Effect Size and Moderator Variables for Acylated Ghrelin AUC Analysis

The results of the meta-analysis indicated a small mean effect of exercise in suppressing acylated ghrelin levels (ES −0.20, 95 % confidence interval [CI] −0.373 to −0.027; $n = 25$; Fig. 2), which was statistically different from zero ($p = 0.024$). No significant heterogeneity among these studies was detected ($I^2 = 0$ %; $Q = 20.04$, $d_f = 24$, $p = 0.695$). Sensitivity analysis showed minor shifts only, and these shifts did not impact the overall significance of the mean effect.

Data from the analyses of moderator variables are presented in Table 2. None of the moderator variables were found to influence the variability among studies examining the acylated ghrelin AUC.

Inspection of the funnel plot (see Fig. S1 in the Electronic Supplementary Material) of standard error by the ES showed a shift to the left of the mean, suggesting the presence of publication bias. Using the trim-and-fill correction, one further study that did not report exercise-induced suppression of acylated ghrelin is needed in order to bring symmetry about the mean. This study would moderate the ES to −0.17 (95 % CI −0.34 to −0.003) while not altering the statistical significance, but it would need to have a moderate or greater ES (≥0.5).

3.4.2 Effect Size and Moderator Variables for Peptide YY AUC Analysis

The meta-analysis revealed a small mean effect for exercise to increase PYY AUC values (ES 0.24, 95 % CI 0.007 to 0.475; $n = 14$; Fig. 3), and this was significantly different from zero ($p = 0.044$). There was minimal heterogeneity among these studies ($I^2 = 0$ %; $Q = 4.629$, $d_f = 13$, $p = 0.982$). Sensitivity analysis showed that a study by Broom et al. [28] influenced the results toward positive values. The removal of this trial decreased the ES to 0.20 (95 % CI −0.04 to 0.447) and also negated its significance ($p = 0.102$).

Data from the analyses of moderator variables are presented in Table 2. None of the moderator variables that were examined reached statistical significance.

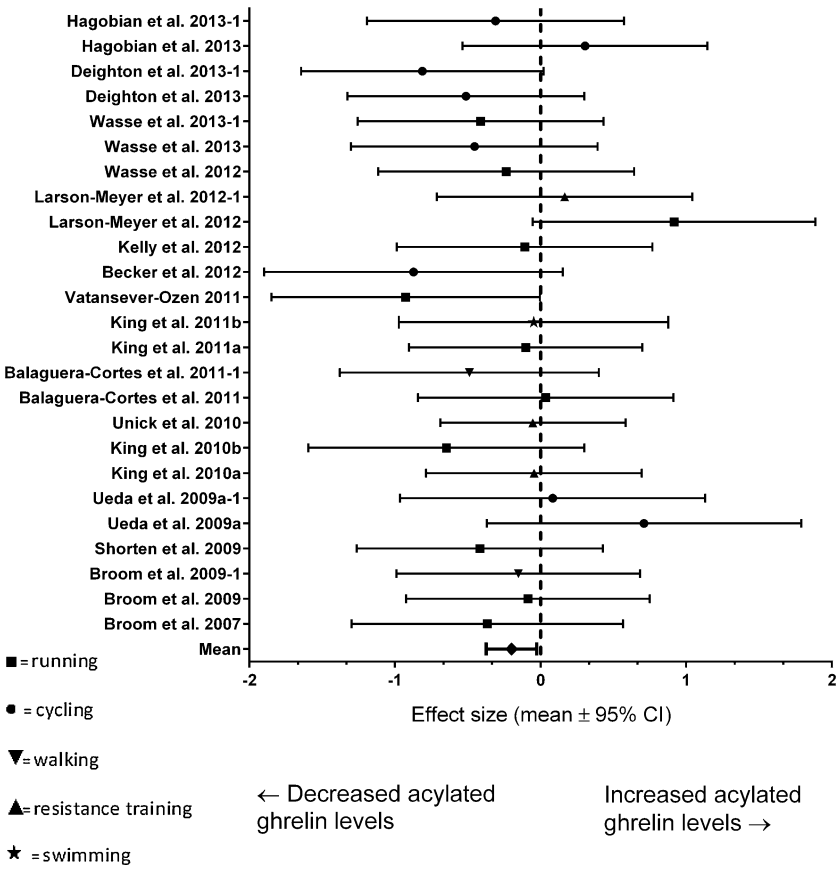

Fig. 2 Forest plot of effect sizes (means ± 95 % confidence intervals [CIs]) for studies evaluating the acylated ghrelin area under the concentration–time curve (AUC)

Table 2 Summary of moderator variable analysis for acylated ghrelin and peptide YY meta-analyses by sub-group and meta-regression

Moderator variable	p value[a]	Comparison
Acylated ghrelin ($n = 25$)		
Exercise mode	0.871	Cycling ($n = 8$; ES −0.27, 95 % CI −0.63 to 0.094)
		Running ($n = 11$; ES −0.22, 95 % CI −0.48 to 0.047)
		Resistance training ($n = 2$; ES −0.31, 95 % CI −0.92 to 0.3)
		Walking ($n = 3$; ES 0.001, 95 % CI −0.42 to 0.42)
		Swimming ($n = 1$; ES −0.05, 95 % CI −0.97 to 0.88)
Sex	0.124	Men ($n = 21$; ES −0.27, 95 % CI −0.46 to −0.077)
		Women ($n = 4$; ES 0.12, 95 % CI −0.34 to 0.58)
Fed state	0.418	Fed ($n = 9$; ES −0.08, 95 % CI −0.46 to 0.30)
		Fasted ($n = 16$; ES −0.26, 95 % CI −0.47 to −0.04)
Analytical method	0.139	ELISA ($n = 19$; ES −0.26, 95 % CI −0.45 to −0.06)
		RIA ($n = 2$; ES 0.51, 95 % CI −0.22 to 1.24)
		Multiplex ($n = 4$; ES −0.25, 95 % CI −0.68 to 0.19)
ExEE	0.289	Meta-regression of ExEE vs. ES (slope −0.00007, 95 % CI −0.0002 to 0.00006)
ExDur	0.712	Meta-regression of ExDur vs. ES (slope −0.00169, 95 % CI −0.01067 to 0.00729)
ExInt	0.133	Meta-regression of ExInt vs. ES (slope −0.00713, 95 % CI −0.0164 to 0.00217)
BMI	0.948	Meta-regression of BMI vs. ES (slope −0.00206, 95 % CI −0.0603 to 0.0645)
AUC time	0.231	Meta-regression of AUC time vs. ES (slope −0.038, 95 % CI −0.100 to 0.02423)
$\dot{V}O_{2max}$	0.168	Meta-regression of $\dot{V}O_{2max}$ vs. ES (slope −0.01313, 95 % CI −0.0318 to 0.0055)
Peptide YY ($n = 14$)		
Exercise mode	0.535	Cycling ($n = 5$; ES 0.31, 95 % CI −0.08 to 0.71)
		Running ($n = 6$; ES 0.24, 95 % CI −0.12 to 0.60)
		Resistance training ($n = 2$; ES −0.11, 95 % CI −0.72 to 0.50)
		Walking ($n = 1$; ES 0.64, 95 % CI −0.26 to 1.54)
Sex	0.862	Men ($n = 11$; ES 0.24, 95 % CI −0.02 to 0.51)
		Women ($n = 2$; ES 0.34, 95 % CI −0.30 to 0.99)
		Both ($n = 1$; ES 0.06, 95 % CI −0.74 to 0.86)
Fed state	0.515	Fed ($n = 7$; ES 0.32, 95 % CI −0.02 to 0.66)
		Fasted ($n = 7$; ES 0.17, 95 % CI −0.16 to 0.49)
Analytical method	0.629	ELISA ($n = 7$; ES 0.34, 95 % CI 0.009 to 0.68)
		RIA ($n = 3$; ES 0.23, 95 % CI −0.27 to 0.73)
		Multiplex ($n = 4$; ES 0.075, 95 % CI −0.36 to 0.51)
ExEE	0.435	Meta-regression of ExEE vs. ES (slope 0.0001, 95 % CI −0.00016 to 0.00036)
ExDur	0.97	Meta-regression of ExDur vs. ES (slope −0.0003, 95 % CI −0.0168 to 0.162)
ExInt	0.682	Meta-regression of ExInt vs. ES (slope −0.0072, 95 % CI −0.0414 to 0.0271)
BMI	0.513	Meta-regression of BMI vs. ES (slope 0.0415, 95 % CI −0.0830 to 0.1661)
AUC time	0.388	Meta-regression of AUC time vs. ES (slope 0.0463, 95 % CI −0.0588 to 0.151)
$\dot{V}O_{2max}$	0.298	Meta-regression of $\dot{V}O_{2max}$ vs. ES (slope −0.01451, 95 % CI −0.0419 to 0.0129)

AUC area under the concentration–time curve, *BMI* body mass index, *CI* confidence interval, *ELISA* enzyme-linked immunosorbent assay, *ES* effect size, *ExDur* exercise duration, *ExEE* exercise energy expenditure, *ExInt* exercise intensity, *RIA* radioimmunoassay, $\dot{V}O_{2max}$ maximal oxygen uptake

[a] Test for statistical difference between moderator sub-group or significance of meta-regression (see text for explanations)

Inspection of the funnel plot (see Fig. S2 in the Electronic Supplementary Material) of standard error by the ES showed a distribution of ES values to the right of the mean, suggesting publication bias for studies revealing an exercise-mediated increase in PYY. Using the trim-and-fill correction, one study with an ES between −0.3 and −0.4 would be necessary to bring symmetry about the mean. This would modify the ES accordingly (ES 0.20, 95 % CI −0.023 to 0.43) and would negate its significance.

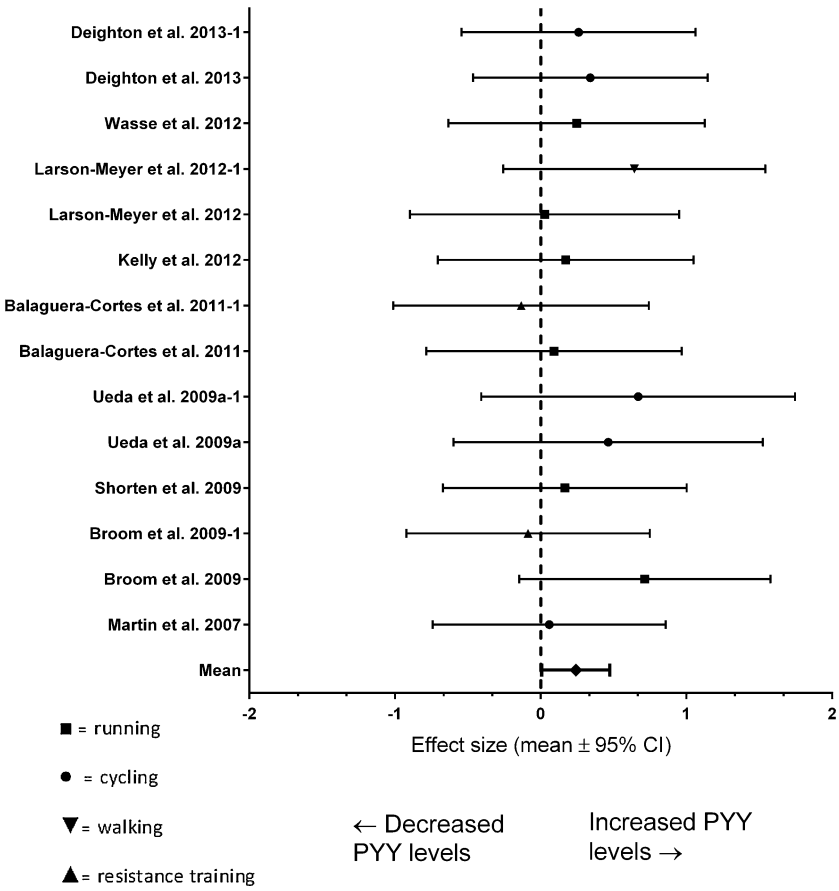

Fig. 3 Forest plot of effect sizes (means ± 95 % confidence intervals [CIs]) for studies evaluating the peptide YY (PYY) area under the concentration–time curve (AUC)

3.4.3 Effect Size for Glucagon-Like Peptide 1 AUC Analysis

The meta-analysis for GLP-1 revealed a small mean effect for exercise to increase AUC values (ES 0.28, 95 % CI −0.031 to 0.581; $n = 8$; Fig. 4), although this only trended towards a significant difference from zero ($p = 0.078$). There was minimal heterogeneity among these studies ($I^2 = 0$ %; $Q = 3.934$, $d_f = 7$, $p = 0.787$). Several studies moderated the results; for example, removing the study with the largest positive ES [24] would decrease the mean ES to 0.21 (95 % CI −0.118 to 0.54) and would also moderate the significance ($p = 0.209$). Conversely, removing the only study with a negative ES [27] would increase the mean ES to 0.417 (95 % CI 0.069 to 0.766) and would also create a significant difference from zero ($p = 0.019$).

Inspection of the funnel plot (see Fig. S3 in the Electronic Supplementary Material) of standard error by the ES showed a distribution to the right of the mean, suggesting the presence of publication bias. Given the small number of studies, this would be expected. Using the trim-and-fill correction, four studies that do not report exercise-mediated increases in GLP-1 (with moderate ES values of approximately −0.5) would be needed to create symmetry about the mean. If these studies were to be found, they would nullify the ES (ES 0.033, 95 % CI −0.22 to 0.286).

3.4.4 Effect Size for Pancreatic Polypeptide AUC Analysis

The meta-analysis revealed a moderate mean effect for exercise to increase PP AUC values (ES 0.50, 95 % CI 0.113 to 0.893; $n = 5$; Fig. 5), which was significantly different from zero ($p = 0.011$). Minimal heterogeneity was detected among these studies ($I^2 = 0$ %; $Q = 3.567$, $d_f = 4$, $p = 0.468$). Two studies with the largest ES values [24, 34] increased the mean ES, and the removal of either study would decrease the ES to 0.39 (95 % CI −0.04 to 0.83) while moderating the significance ($p = 0.078$).

Inspection of the funnel plot (see Fig. S4 in the Electronic Supplementary Material) of standard error by the ES showed a distribution to the right of the mean, suggesting the presence of publication bias. As with GLP-1, this would be expected because of the small number of studies. Using the trim-and-fill correction, one study that did not report an exercise-induced increase in PP (with an ES of approximately −0.25) would be necessary to create symmetry about the mean. This would moderate the ES to 0.40 (95 % CI 0.0376 to 0.757) but would not alter its significance.

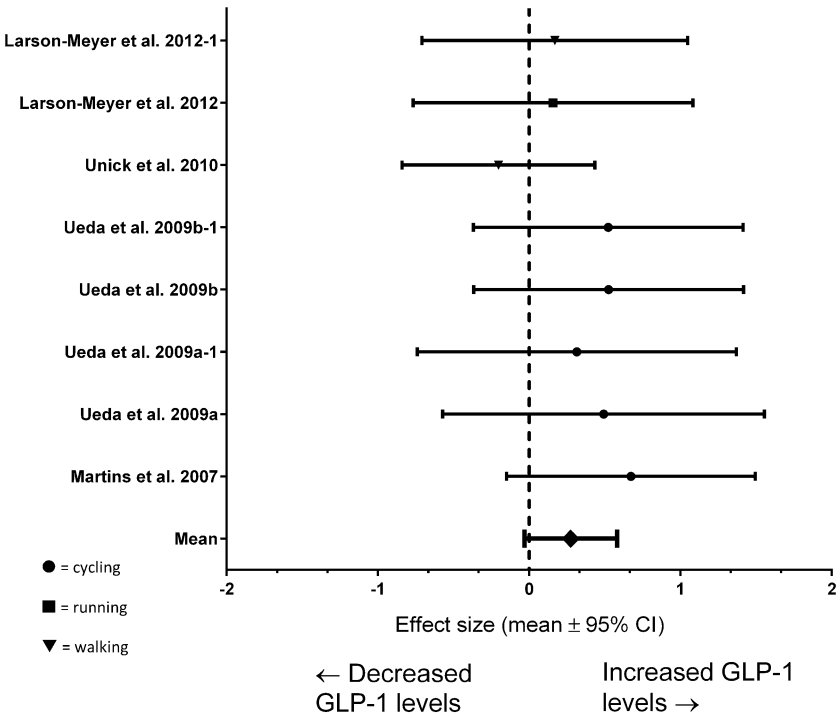

Fig. 4 Forest plot of effect sizes (means ± 95 % confidence intervals [CIs]) for studies evaluating the glucagon-like peptide 1 (GLP-1) area under the concentration–time curve (AUC)

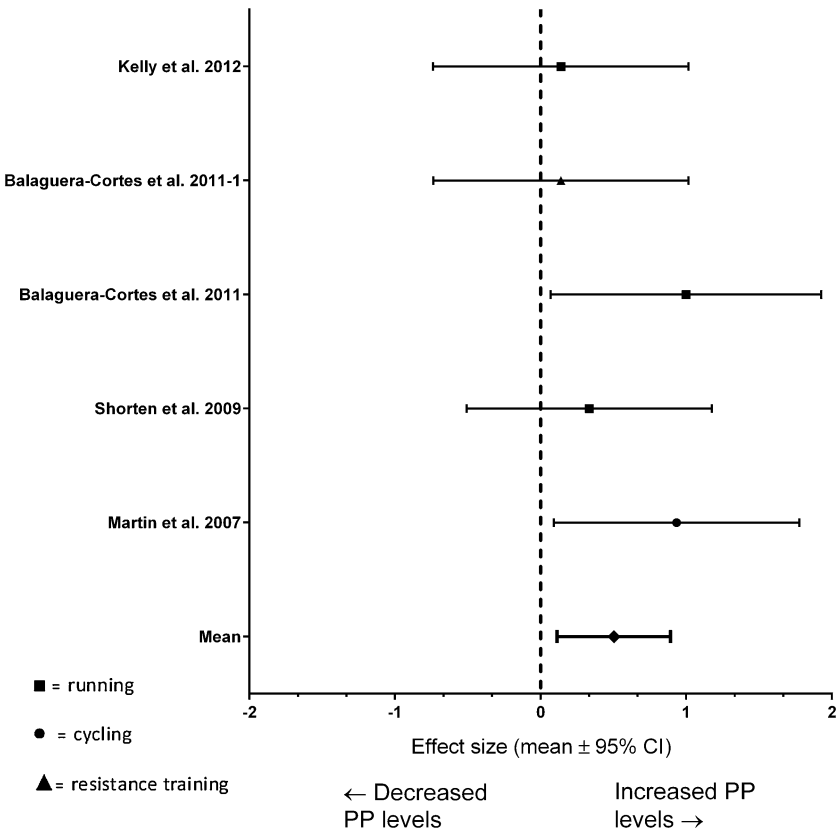

Fig. 5 Forest plot of effect sizes (means ± 95 % confidence intervals [CIs]) for studies evaluating the pancreatic polypeptide (PP) area under the concentration–time curve (AUC)

4 Discussion

Examination of the impact of acute exercise on levels of hormones related to appetite regulation may provide mechanistic insight into exercise-induced changes in appetite and food and beverage intake. The purpose of this review was to perform a meta-analysis to determine the efficacy of acute exercise bouts to alter hormonal mediators of appetite in the hours after exercise. We observed small to moderate changes in opposing directions for acylated ghrelin and three anorexigenic hormones (PYY, GLP-1 and PP). These results are consistent with the majority of the present literature, as well as the known actions of these hormones on appetite regulation. Acylated ghrelin is a peripheral hormone that is well known to stimulate hunger and food intake, while PYY, GLP-1 and PP all inhibit food intake through various mechanisms of action. Our results indicate that exercise alters levels of hormones known to influence feeding behaviour in directions that could be expected to contribute to prospective changes in food and beverage intake post-exercise, such as a transient suppression of hunger or increased feeding latency [51, 52].

It has been extensively speculated that exercise intensity is a factor that may influence exercise-induced suppression of acylated ghrelin [21, 29, 49]. Aside from one study [35], research has been conducted at intensities of <75 % $\dot{V}O_{2max}$. Deighton et al. [35] provided evidence that supra-maximal exercise may be more potent for suppressing acylated ghrelin than vigorous endurance exercise (~68 % $\dot{V}O_{2max}$), although the supra-maximal sprint exercise led to higher hunger levels later in the observation period (albeit with no changes in energy intake). Given that sprint interval training has attracted considerable interest as a time-saving alternative to endurance exercise [53], further information on hormonal and energy intake responses to sprint- and high-intensity interval training is warranted, particularly in special populations (i.e. overweight/obese individuals and patients with diabetes).

Differences in appetite and energy intake may exist between men and women [54, 55]. In a complex crossover study, Hagobian and colleagues evaluated sex differences in hormones and energy intake in response to control conditions, 4 days of exercise with energy replacement (to maintain energy balance) and 4 days of exercise without energy replacement (energy deficit) [54]. In response to a meal tolerance test after 4 days in balance or deficit, the authors found that women had markedly higher acylated ghrelin concentrations compared with baseline, while no significant difference existed in men [54]. However, a more recent paper found that men and women did not differ in their hormonal or appetite responses to an exercise bout matched for relative energy expenditure (~30 % of estimated daily EE); indeed, both sexes had significant reductions in relative energy intake post-exercise and no observable changes in appetite hormone (acylated ghrelin and $PYY_{3–36}$) levels [48]. Although initial reports questioned whether long-term exercise was as effective for weight loss in women as compared with men [56, 57], recent literature has indicated that when energy expenditure is matched between sexes, differences in weight loss or energy intake do not occur [58, 59]—appetite hormone responses and their roles remain to be more thoroughly elucidated.

With respect to other individual differences, BMI showed no effect on the variation among the studies. The majority of the participants were young, lean individuals, and only two of the studies evaluated overweight and obese individuals. Other than the fact that obese individuals appear to have a blunted postprandial ghrelin response [60], relatively little is known about how exercise can influence concentrations of acylated ghrelin in obese individuals. The same holds true for anorexigenic appetite hormones, although it is known that obese individuals have lower circulating PYY levels and appear to have a deficiency in postprandial secretion [61]. Although the evidence is equivocal at present, GLP-1 impairment and postprandial deficiency can also occur in obese individuals [62]. In support of our findings that exercise increased PYY and GLP-1 concentrations, exercise may help improve both absolute concentrations and sensitivity to PYY and GLP-1, potentially assisting obese individuals to terminate meals more rapidly and maintain a longer inter-meal interval. However, this remains to be determined.

The effect of the exercise mode on appetite regulation is of considerable interest because variety in exercise training is a potentially important predictor of compliance [63, 64]. It has been speculated that studies involving exercise that induces greater metabolic and mechanical demands (potentially causing muscle damage and greater muscle loading, e.g. running) tend to more potently suppress levels of hunger and acylated ghrelin [21, 29]. One would expect acylated ghrelin suppression to be lesser after running-type exercise because of altered splanchnic blood flow, which inhibits ghrelin secretion [28, 29]; however, research indicates that altered blood flow also occurs after vigorous cycling [65]. Additionally, increased secretion of PYY and GLP-1 from the small intestine and PP from the pancreas could attenuate gastric motility, theoretically decreasing the desire to eat [32]. The only study that directly compared running and cycling reported no differences in acylated ghrelin levels between the exercise modes at the same relative intensity [49]. The two studies that evaluated resistance training found suppressed acylated ghrelin levels, while endurance exercise either increased or did not alter PYY concentrations in the same studies [28, 34].

At this point, it would be applicable to examine the findings of the present meta-analysis in the context of the results of our previous meta-analysis on energy intake after acute exercise [37]. Twelve of the studies in the present meta-analysis were also included in the previous analysis [20–22, 25–27, 32–34, 40, 66]. In that meta-analysis, we evaluated food intake responses to acute bouts of exercise and observed minimal changes in absolute energy intake (ES 0.14, mean difference ~200 kJ) and large deficits in relative energy intake (ES −1.35, mean difference ~2,000 kJ) [37]. These results suggested that in response to acute exercise, individuals did not compensate for the expended energy by increasing their food intake 2–10 h afterward. The changes in appetite hormones observed in the present analysis may suggest that exercise-induced hormone changes may mediate food and beverage intake responses post-exercise, potentially through prolonged feeding latency, which may be caused by increased levels of PYY, GLP-1 and PP. While it is difficult to quantify whether the changes in hormones would predict changes in energy intake, Larson-Meyer et al. [32] reported that the changes in PYY and GLP-1 levels were associated with reductions in food intake after running and walking in women.

It must be stressed that hormones involved in appetite regulation are one aspect of a complex system that regulates human feeding behaviour. A number of other variables related to exercise have been shown to influence food and beverage intake. For example, environmental conditions can mediate hormone levels, food and beverage intake, and appetite sensations [25, 39, 40, 67]. It has also been shown that exercise alters the receptivity of brain regions involved in food reward when subjects are shown images of food after exercise [68], which could influence the central drive to eat. Others have reported that exercise can increase food reward and the desire for high-fat, sweet foods in some overweight/obese men and women, which may contribute to attenuations in weight loss through post-exercise consumption of high-energy foods [69]. Recent work has also indicated that substrate oxidation of carbohydrate during exercise (1,675 kJ @ 70 % of the maximum heart rate) accounts for 37 % of the variance in post-exercise energy intake in overweight and obese women [70]. The same authors have also published work showing that the resting metabolic rate is a significant predictor of meal size and energy intake, being predominantly driven by fat-free mass [71]. Thus, the relationships between exercise, appetite hormones, substrate oxidation during exercise and changes in resting metabolic rate and fat-free mass in response to exercise training offer potential opportunities to explore the mechanisms of how exercise may influence food intake and energy balance. Finally, it is possible that chronic exercise alters the sensitivity to these hormones, much as exercise is known to improve insulin sensitivity [72]. Studies have reported that fasting acylated ghrelin levels increase [73] or do not change [74] after 12 weeks of aerobic or resistance exercise in overweight and obese individuals. Those studies also reported no alterations in fasting levels of PYY, GLP-1 and PP [73, 74], and another study reported no alterations in cholecystokinin after a similar aerobic exercise program [75]. However, it has been demonstrated that 12 weeks of training led to a greater amplitude of change and suppression of acylated ghrelin postprandially, while levels of GLP-1 and PYY trended towards greater increases 1.5–2 h post-meal [73].

Determining an index of clinically meaningful changes in appetite hormone levels is challenging, as fasting levels tend to vary considerably among individuals [2, 3]. However, we examined the literature on appetite hormone responses to nutrient intake to obtain an estimate of what may be meaningful. It has been reported that acylated and total ghrelin concentrations are suppressed by 25–60 % 1–2 h post-meal ingestion (~1,600–2,500 kJ of varying macronutrient composition), while GLP-1 and PYY concentrations increase by 20–40 % [30, 76, 77]. Bearing this in mind, the exercise stimuli from the studies included in the present meta-analysis had comparatively smaller effects on appetite hormone levels (i.e. alterations of ~9–17 %), suggesting that while these hormones are sensitive to changes in energy expenditure, they respond comparatively more strongly to energy intake.

There are, of course, several limitations in our meta-analysis that warrant mention. Firstly, all studies cited herein were acute in nature, and little knowledge exists regarding hormonal responses and their contributions to feeding behaviour after exercise training, as mentioned above. Secondly, the studies were limited to English-language works. Thirdly, while the present hormones that were examined are well-known mediators of appetite regulation, other hormones and non-physiological factors are involved. For example, cholecystokinin plays a major role in appetite regulation but, to our knowledge, only two studies have evaluated cholecystokinin responses to acute exercise, both in response to incremental exercise to exhaustion, and both studies reported increased cholecystokinin responses immediately post-exercise and up to 2 h afterward [78, 79]. Fourthly, we cannot exclude the possibility that we were unable to obtain all potentially relevant studies. Finally, at this point in time, any link between alterations in appetite hormone levels and changes in actual energy intake post-exercise is still tenuous and speculative, although the results of our two meta-analyses suggest that a potential relationship does exist.

5 Conclusions

This meta-analysis found that exercise may influence appetite by suppressing levels of acylated ghrelin (by ~16.5 %), while simultaneously increasing levels of PYY, GLP-1 and PP (by 9, 13 and 15 %, respectively) for 2–9 h post-exercise. Given the known functions of these hormones, such changes could provide a potential explanation for alterations in food and beverage intake post-exercise. However, many questions still remain, and a few are mentioned below:

1. How does exercise above 'maximal' intensities influence hormones?
2. How does intermittent- or game-type exercise influence hormones and appetite?
3. Do individuals of high fitness have different hormonal responses compared with individuals of lesser levels of fitness?
4. What role does body composition play in hormonal responses to exercise?
5. What impacts do long-term exercise interventions, with and without weight loss, have on appetite hormones?
6. What are the precise mechanisms of action of exercise on appetite regulation?

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