Lead Article

Effect of physical exercise and training on gastrointestinal hormones in populations with different weight statuses

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Several types of hormones exert control over appetite in humans. This narrative review explores the effects of exercise and training on the concentrations of gastrointestinal hormones in healthy and obese individuals. It focuses on the major hormones of appetite regulation: ghrelin, glucagon-like peptide 1, peptide YY, cholecystokinin, leptin, and oxyntomodulin. In normal-weight and overweight individuals, responses to most of these hormones depend on the intensity of exercise and training. However, findings in obese individuals are limited in number and, to some degree, contradictory. Although some gastrointestinal hormones have been studied extensively (eg, leptin), most have not been investigated systematically. Further research is required to confirm the effectiveness of exercise and training on gut hormones and to better understand the effect of gut hormones on appetite and hunger suppression in individuals with obesity. Investigations to elucidate the impact of various forms of exercise that have recently engaged the public interest, eg, high-intensity interval training or concurrent aerobic and resistance training, are warranted.

INTRODUCTION

The epidemic of overweight and obesity is one of the major health concerns of the 21st century. It has transformed from a relatively minor public health issue that primarily affected the most affluent societies to an important, globalized threat to public health. The French National Epidemiological Survey of Overweight and Obesity (ObiEpi), conducted from 1997 to 2012, showed that obesity in France has increased steadily, regardless of sex, although the progression slowed slightly between 2009 and 2012.[1] In 2012, the percentages of men and women with obesity in France were 4.3% and 15.7%, respectively.[1] The results of ObiEpi indicate that, at the national level, 7 million French people are obese, which represents almost 15% of the population according to World Health Organization (WHO) data published in 2014.[1] These trends in France are reflected in a multitude of populations in other countries as well.

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Linked to complex interactions between biological, psychological, behavioral, and environmental determinants, obesity is considered a pathological condition by the WHO. Furthermore, the scientific literature confirms that obesity contributes to significant increases in the prevalence of many comorbidities, including cardiovascular diseases,[2] osteoarticular complications,[3] metabolic dysfunctions,[4] and some types of cancer.[5]

Obesity can be defined as the result of an inequity in energy balance. Energy intake via food intake increases to the detriment of energy expenditure.[6] Energy expenditure consists of 3 components, namely basic metabolism, postprandial thermogenesis, and physical activity. Physical activity is the most variable and is known to be decreased in obese individuals,[6] while food intake in this population is typically increased.[7,8]

How food intake is defined and characterized is an important and complex subject. More specifically, food intake comprises 3 phases. The first, the ingestive phase, is characterized by the sensation of hunger. The second, the prandial phase, corresponds to food intake, when satiation takes place. The last phase, the postprandial phase, is characterized by the state of satiety. The regulation of food intake is part of a complex system that involves hormonal signaling from throughout the body, including the gastrointestinal system and fat cells. In general, food intake is mainly under the control of the hypothalamus, which integrates the nervous and hormonal signals of eating behavior and caloric intake. Hormones affecting the brain centers are synthesized and released from peripheral tissues, including the intestine and adipose cells (adipocytes). These hormones can be divided into 2 categories, anorectic (appetite suppressing) and orexigen (appetite stimulating). The main hormones regulating appetite and satiety are ghrelin, glucagon-like peptide 1, peptide YY, pancreatic polypeptide, cholecystokinin, and leptin.

The pathology of obesity is accompanied by altered secretion of the hormones of appetite, which leads to uncontrolled food intake.[9] Moreover, as already noted, physical activity plays a leading role in the management of energy balance in both lean and obese individuals.[10–14] Despite the undisputed role of physical activity in the management of obesity, very few studies have examined the modulatory effects of physical activity on the hormones that control and induce food intake. To this end, this review examines the influence of acute (a single exercise session) and chronic (multiple exercise sessions) on gastrointestinal tract (ie, gut) hormones in lean and obese people. The aims are as follows: (1) to examine whether the evidence supports a causal relationship between chronic exercise and gut hormone alterations in obese individuals; (2) to determine whether the secretion of the hormones that regulate appetite, which induce food intake and, therefore, energy intake, are modulated by the pathology of obesity; and (3) to assess evidence that physical activity and its different modalities (continuous, intermittent forms) lead to orexigenic or anorectic effects. The main hormones dictating appetite and food intake in healthy and obese individuals, as well as the various obesity-caused alterations in the secretion of these hormones, are examined. Moreover, the effects of physical activity, exercise, and training on the concentrations of these hormones in healthy and obese individuals are identified.

Literature search

Three electronic databases were searched: PubMed, ISI Web of Knowledge, and SPORTDiscus. The following key terms (and synonyms for which the MeSH database searched) were included and combined: "obesity," "gut hormones," "gastrointestinal hormones," "training," "physical activity," "exercise," "ghrelin," "glucagon-like peptide-1," "peptide YY," "pancreatic polypeptide," "cholecystokinin," and "leptin." The search identified 3474 and 2035 records in PubMed and SPORTDiscus, respectively. Only peer-reviewed articles written in English were included. Specifically, only studies that investigated the effect of acute and chronic exercise on gut hormones in lean and obese people were included. In addition, the reference lists and citations (Google Scholar) of the identified studies were examined to identify further relevant research papers. The final screening by investigators was based on the relevance of the identified items to the assessment of gut hormones.

EFFECTS OF ACUTE AND CHRONIC EXERCISE ON ENERGY-REGULATING HORMONES IN LEAN AND OBESE INDIVIDUALS

Ghrelin response

Ghrelin, also known as the hormone of hunger, is a peptide and a stomach-derived orexigenic hormone produced by endocrine cells of the gastric mucosa and recognized as the endogenous ligand of the orphan growth hormone secretagogue receptor. It plays a major role in food intake and appetite regulation.

According to Shiiya et al,[15] the circulating concentration of ghrelin is inversely associated with body mass index. This is confirmed by McLaughlin et al,[16] who reported reduced ghrelin levels in individuals with obesity and high BMI.[16] This is proposed to be caused by hyperinsulinemia and a deficiency of insulin sensitivity.[17,18] In the postprandial period, suppression of ghrelin was attenuated

in obese individuals vs lean individuals, resulting in higher energy consumption.[19] This may contribute to altered satiety signaling in obese patients and the establishment of a positive energy balance and weight gain.

Several researchers observed an interaction between exercise and ghrelin, a regulator of appetite and energy homeostasis.[20,21] As far as can be determined, only 2 studies have focused specifically on ghrelin concentrations in healthy individuals after aerobic exercise (60 minutes at 74% of maximum oxygen consumption (VO_{2max}) and 60 minutes at 65% of the maximum heart rate).[22,23] No changes in ghrelin levels were observed in either study, but other studies have reported decreases in ghrelin concentrations in response to aerobic-related exercise[24–26] or resistance exercise.[27–29] Toshinai et al[25] examined the response of ghrelin in healthy men after 40 minutes of exercise with progressive intensity (4 stages of 10 minutes of progressive intensity) and found ghrelin was suppressed in an intensity-dependent manner. In this same study, changes in ghrelin levels were also associated with changes in adrenaline ($r = 0.533$) and norepinephrine ($r = 0.603$). The authors proposed that the sympathetic nervous system induced a reduction in the gastric blood supply, which resulted in a decrease in the release of ghrelin into the bloodstream. Hence, it appears that aerobic exercise of moderate, maximal, and progressive intensity affects the secretion of ghrelin via responses mediated by catecholamine (epinephrine and norepinephrine).

To study the impact of training on ghrelin levels, Leidy et al[30] performed 24-hour blood sampling in a group of normal-weight women before and after exercise (45 minutes of moderate exercise, 5 times per week) over a 12-week period that included a concomitant dietary intervention. Study participants showed a 4% reduction in body weight and an increase in ghrelin concentrations during the day, but the authors were unable to determine whether the observed changes came from training, the dietary intervention, or the associated weight loss.

Morpurgo et al[31] observed that, despite a 5% weight loss in morbidly obese men and women who completed an aerobic exercise training program combined with dietary restriction, circulating levels of ghrelin (either on an empty stomach or after meal ingestion) remained unchanged. Numerous investigators report increased concentrations of ghrelin after moderate-intensity exercise and training[23,30,32–38] (Table 1[23,32,34–48]), while others found no changes.[39–41,44,46] These findings raise questions about whether the magnitude of changes in ghrelin levels depends on the intensity and the amount of exercise, since most studies focused only on aerobic exercise and no study reported data for anaerobic exercise.

The intensity of exercise could play a role in ghrelin secretion via catecholamine-related mechanisms,[14] but to date, no study has investigated the response of ghrelin following highly intensive exercise or training known to increase catecholamine secretions in both lean individuals and individuals with obesity. In fact, Larson-Meyer et al[49] found increased levels of acylated ghrelin after moderate-intensity training in trained women, while other investigators observed decreased or even unchanged levels of acylated ghrelin after similar types of training[14,50–52] (Table 2[22,49–81]). These differences can be explained mainly by the different training protocols used in these studies (training duration, type of training [eg, running, walking or cycling], intensity of training, etc). Clearly, some aspects of the ghrelin response to select forms of exercise need further investigation.

Glucagon-like peptide 1 response

Glucagon-like peptide-1 (GLP-1) is an intestinal hormone secreted in response to food intake. It stimulates insulin secretion by the β cells and reduces glucagon secretion by α cells in response to a meal, resulting in a decrease in hepatic glucose production. Hence, this physiological action of endogenous GLP-1 is glucose dependent.

According to several authors (Adam and Westerterp-Plantenga,[82] Carroll et al,[83] and Verdich et al,[84] obesity is associated with an attenuated postprandial response of GLP-1 (anorectic hormone) to stimuli. In fact, according to Verdich et al,[84], the postprandial response of GLP-1 30 minutes after consumption of a control meal is significantly attenuated in obese individuals compared with normal-weight individuals, thereby delaying satiety and leading to excessive food intake. In another study, GLP-1 levels were increased in normal-weight individuals 10 minutes after ingestion of a standard liquid meal but were decreased in obese individuals 20 minutes after ingestion of the same meal[83] (Table 3[23,48,49,52,53,73,76–78,85–95]). Conversely, in the preprandial period, Adam and Westerterp-Plantega[82] reported that levels of GLP-1 in obese and normal-weight adults were similar.

Collectively, these findings suggest that the feeling of satiety during the 20 minutes following the ingestion of a meal is inhibited in individuals with obesity. This could perhaps explain an uncontrolled increase in food intake that would favor an energy imbalance in favor of inputs. However, the available studies are limited, and further work is needed to test this assumption.

Very low-volume sprint interval exercise resulted in increased GLP-1 levels in overweight men and women.[76] Likewise, acute exercise at moderate intensity

Table 1 **Studies examining the effect of chronic exercise on ghrelin concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Kang et al (2018)[43]	Obese middle-aged women	N = 26 46–54 y	5 times/wk for 12 wk	↑ Ghrelin concentrations
Gibbons et al (2017)[40]	Overweight/obese individuals: 16 completed 12 wk of aerobic exercise and 16 were age- and BMI-matched nonexercising controls	N = 32 37–49 y	12-wk exercise intervention, 5 exercise sessions per week	No change in acylated ghrelin
Martins et al (2017)[46]	Sedentary obese individuals BMI = 33.3 ± 2.9	N = 46 (30 women, 16 men) 25–44 y	A 12-wk isocaloric program of MICT or HIIT, or a short-duration HIIT (½HIIT)	No change in acylated ghrelin
Arikan & Serpek (2016)[39]	Women: BMI = 22.0 ± 0.6 Men: BMI = 22.6 ± 0.8	N = 35 18–24 y	60-min cycling exercises on 4 d of the week for 8 wk at 50%–70% of previously determined heart rate	No change in ghrelin
Ueda et al (2013)[48]	Healthy middle-aged women BMI = 27.6 ± 0.4 VO_{2peak} = 23.5 ± 0.9	N = 20 Age not reported	12 wk of exercise training at 65% of HR_{max}	↑ Ghrelin
Guelfi et al (2013)[41]	Overweight/obese men BMI = 30.8 ± 4.2	N = 33 42–56 y	12 wk of training (3 d/wk). 3 groups: aerobic (n = 12), 40–60 min at 70%–80% of HR_{max}; resistance (n = 13), 3–4 sets of 8–10 reps at 75%–85% of 1RM; control (n = 8)	No change in acylated ghrelin after aerobic or resistance training program
Gueugnon et al (2012)[42]	Obese adolescents BMI *z* score = 4.1	N = 32 (10 boys, 22 girls) 14–15 y	Physical exercise 5 times/wk during the following 7 mo	↑ Ghrelin
Martins et al (2010)[34]	Sedentary overweight men and women BMI = 31.3 ± 2.3 VO_{2max} = 32.9 ± 6.6	N = 15 28–46 y	12 wk of training (5 d/wk): treadmill walking or running at 75% of HR_{max} until energy deficit of 500 kcal is reached	↑ Ghrelin
King (2010)[44]	Men Body weight = 76.2 ± 1.0 kg	N = 69 22–23 y	90 min of resistance exercise and 60 min of swimming; 60 min of brisk walking; 90 min of treadmill running	No change in acylated ghrelin
Hagobian et al (2009)[35]	Overweight male volunteers BMI = 25.7 ± 2.3 VO_{2peak} = 44.9 ± 4.8 Overweight female volunteers BMI = 28.0 ± 3.5 VO_{2peak} = 34.9 ± 5.2	N = 18 15–29 y	Treadmill running 50%–65% of VO_{2peak} until 30% of TDEE in deficit or balance conditions (crossover)	Females: ↑ acylated ghrelin
Konopko-Zubrzycka et al (2009)[36]	Obese men and women BMI ≥ 40	N = 33 20–60 y	6 mo of physical exercise (45-min walk, 5 times/wk)	↑ Ghrelin
Kelishadi et al (2008)[37]	Obese children	N = 100 7–9 y	Physical training for 6 mo	↑ Ghrelin
Mizia-Stek et al (2008)[47]	Obese premenopausal women BMI = 36.5 ± 5.0	N = 37 29–52 y	3-mo weight-reduction treatment: diet of 1000 kcal/d and physical exercise	↑ Ghrelin
Martins et al (2007)[23]	Healthy, normal-weight volunteers	N = 12 (6 men, 6 women) 21–31 y	Cycled for 60 min at 65% of HR_{max} or rested	No change in acylated ghrelin
Santosa et al (2007)[38]	Hyperlipidemic women BMI = 28–39	N = 35 35–60 y	6-mo weight-loss trial	↑ Ghrelin
Foster-Schubert et al (2005)[32]	Women BMI = 24–25	N = 173 50–75 y	12-mo, moderate-intensity aerobic exercise intervention: minimum of 45-min of moderate-intensity aerobic exercise, 5 d/wk for 12 mo	↑ Ghrelin
Leidy et al (2004)[45]	Normal-weight young women BMI = 20.9 ± 1.5	N = 22 17–25 y	3-mo of energy-deficit diet plus exercise. Aerobic exercise 5 times/wk at 70%–80% of HR_{max}	↑ Ghrelin

Abbreviations and symbols: BMI, body mass index; HIIT, high-intensity interval training; HR_{max}, maximal heart rate; MICT, moderate-intensity continuous training; TDEE, total daily energy expenditure; VO_{2max}, maximum oxygen consumption; VO_{2peak}, peak oxygen uptake; 1RM, 1 rep maximum; ↑, increased; ↓, decreased.
[a]BMI shown as kg/m^2; VO_{2peak} shown as mL/kg/min.

Table 2 **Studies examining the effect of acute exercise on ghrelin concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Studies in exercise-trained women				
Howe et al (2016)[53]	Highly trained women BW = 58.4 ± 6.4 kg VO_{2max} = 55.2 ± 4.3	N = 15 18–40 y	Moderate-intensity (60% of VO_{2max}) and high-intensity (85% of VO_{2max}) treadmill running	Acylated ghrelin ↓
Tiryaki-Sonmez et al (2013)[51]	Female runners BMI = 28.3 ± 1.8	N = 9 20–24 y	60 min of running at 53% of VO_{2max}	No change in acylated ghrelin
Larson-Meyer et al (2012)[49]	Female runners BMI = 32.7 ± 0.8	N = 9 18–40 y	60-min run or walk at 70% of VO_{2max}	Acylated ghrelin ↑ post exercise vs rest
Gholipour et al (2011)[50]	Female volunteers BMI = 32.7 ± 0.8	N = 9 20–22 y	36-min treadmill run: 10 min, 10 min, 5 min, and 2 min at 65% of VO_{2max}, separated by 3 min at 3 km/h	Acylated ghrelin ↓
Unick et al (2010)[52]	Female volunteers BMI = 32.5 ± 4.8	N = 19 20–37 y	Walking at 70%–75% of age-predicted HR_{max} until 3.0 kcal/kg of body weight is expended (average energy expenditure: 354 ± 72 kcal; average duration: 42 ± 8 min)	No change in acylated ghrelin
Douglas et al (2017)[77]	Healthy lean women BMI = 22.4 ± 1.5 Overweight/obese women BMI = 29.2 ± 2.9	N = 47 22–58 y	60 min of treadmill exercise at 60% VO_{2peak})	No change in total ghrelin
Studies in exercise-trained men				
Broom et al (2017)[54]	Healthy men BMI = 23.6 ± 1.9	N = 18 19–26 y	Running at 75% of VO_{2peak}	Acylated ghrelin ↓
Laursen et al (2017)[55]	Male volunteers BMI = 79.4 ± 13.5	N = 11 21–29 y	3 separate 1-h cycling bouts at 60% of W_{max} in hot, cold, and room temperature conditions (33°C, 7°C, and 20°C)	No difference in total ghrelin or acylated ghrelin
Kojima et al (2016)[56]	Male college endurance runners BMI = 19.3 ± 0.4 VO_{2max} = 67.1 ± 1.0	N = 23 17–23 y	20-km outdoor run or a control trial with an identical period of rest	Acylated ghrelin ↓
Bailey et al (2015)[57]	Male volunteers BMI = 23.5 ± 2.0	N = 12 19–24 y	MIE normoxia; MIE hypoxia; HIIE normoxia; and HIIE hypoxia	Plasma acylated ghrelin was lower in hypoxia than in normoxia post exercise
Douglas et al (2015)[58]	Male volunteers BMI = 23.0 ± 1.9	N = 15 19–23 y	60 min of continuous moderate- to high-intensity treadmill running	No change in acylated ghrelin
King et al (2015)[59]	Male volunteers BMI = 22.6 ± 1.8	N = 9 20–24 y	90 min of moderate-intensity treadmill running	No change in acylated ghrelin
Sim et al (2014)[60]	Overweight men BMI = 27.7 ± 1.6	N = 17 22–38 y	HIIE	No change in acylated ghrelin
Kawano et al (2013)[61]	Male volunteers BMI = 22.1 ± 2.0 VO_{2max} = 47.0 ± 6.2	N = 15 22–27 y	Rope skipping 3 times for 10 min with 5 min of rest at 64.8% ± 6.9% of VO_{2max}; cycling 3 times for 10 min with 5 min of rest at 63.9% ± 7.5% of VO_{2max}	Acylated ghrelin ↓ up to 30 min post exercise ($P < 0.0167$)
Wasse et al (2013)[62]	Male volunteers BMI = 23.4 ± 2.4 VO_{2max} (running) = 57.8 ± 9.9 VO_{2max} (cycling) = 50.0 ± 9.5	N = 12 20–25 y	Exercise trials: running and cycling for 60 min at 70% of VO_{2max}	Acylated ghrelin ↓ post exercise ($P < 0.05$)
Deighton et al (2013)[63]	Male volunteers BMI = 24.2 ± 2.9 VO_{2max} = 46.3 ± 10.2	N = 12 20–26 y	Control, endurance exercise, and sprint interval exercise	Acylated ghrelin was suppressed during exercise but more so during sprint interval exercise
Becker et al (2012)[64]	Male volunteers BMI = 24 ± 0.9 VO_{2max} = 54.9 ± 2.6	N = 82 6–30 y	Cycling for 60 min at 70% of VO_{2max}	Acylated ghrelin ↓ post exercise vs control ($P = 0.04$)

(continued)

Table 2 **Continued**

Reference	Population[a]	No. and age of participants	Intervention	Results
Kelly et al (2012)[65]	Male volunteers BMI = 23.94 ± 2.1 VO_{2peak} = 59.8 ± 8.6	N = 10 20–23 y	Treadmill running for 45 min at 70% of VO_{2peak} in hydrated or dehydrated state	Post exercise, acylated ghrelin ↓ in dehydrated state vs control ($P = 0.045$) and hydrated state ($P = 0.014$)
Wasse et al (2012)[66]	Male volunteers BMI = 24.8 ± 2.4 VO_{2max} = 56.9 ± 6.5	N = 10 21–27 y	Treadmill running for 60 min at 70% of VO_{2max} at normoxic (20.9% O_2) or hypoxic (12.7% O_2) state	Acylated ghrelin ↓ post exercise ($P = 0.01$)
King et al (2010)[67]	Healthy men BMI ≤ 29.9	N = 9 18–27 y	90-min run at 68.8 ± 0.8% of VO_{2max}	No difference in acylated ghrelin between groups
Balaguera et al (2011)[68]	Male volunteers BMI = 23.7 ± 2.0 VO_{2peak} = 58.1 ± 7.3	N = 10 19–23 y	Treadmill running for 45 min at 70% of VO_{2peak}	Acylated ghrelin ↓ post exercise vs rest ($P = 0.05$)
King et al (2011)[69]	Male volunteers BMI = 22.8 ± 0.4 VO_{2max} = 57.3 ± 1.2	N = 12 22–25 y	Treadmill running at 70% of VO_{2max} for 90 min in an exercise energy deficit, a food energy deficit, or control	Acylated ghrelin ↓ post exercise ($P < 0.05$)
Vatansever-Ozen et al (2011)[70]	Elite male soccer players BMI = 22.03 ± 0.44 VO_{2max} = 62.74 ± 5.0	N = 10 19–21 y	Treadmill running for 105 min at 50% of VO_{2max}, then 15 min at 70% of VO_{2max}	Acylated ghrelin ↓ 120, 180, and 240 min post exercise ($P < 0.05$)
King et al (2010)[67]	Male volunteers BMI = 23.6 ± 0.4 VO_{2max} = 60.5 ± 1.5	N = 9 21–23 y	Treadmill running for 90 min at 68.8% of VO_{2max}	Acylated ghrelin ↓ during exercise trial ($P < 0.0045$); trend ↓ post exercise (NS)
King et al (2010)[71]	Male volunteers BMI = 23.4 ± 0.6 VO_{2max} = 55.9 ± 1.8	N = 14 21–23 y	Brisk walking for 60 min at 45.2% ± 2% of VO_{2max}	No difference in acylated ghrelin between trials
Broom et al (2009)[72]	Male volunteers BMI = 23.1 ± 0.4 VO_{2max} = 62.1 ± 1.8	N = 11 19–22 y	Treadmill running for 60 min at 70% of VO_{2max}	Acylated ghrelin ↓ post exercise ($P < 0.05$)
Ueda et al (2009)[73]	Obese and age-matched volunteers of normal weight	N = 14 18–27 y	Cycling exercise at 50% of VO_{2max}	No change in plasma ghrelin
Broom et al (2007)[74]	Men BMI = 23.1 ± 0.4 VO_{2max} = 62.1 ± 1.8	N = 91 9–22 y	Running for 60 min at 72% of VO_{2max}; rest for 8 h post exercise; test meal 3 h post exercise	Acylated ghrelin was ↓ 3 h post exercise vs rest ($P < 0.05$)
Zoladz et al (2005)[75]	Male volunteers BMI = 22.42 ± 0.49 VO_{2max} = 51.6 ± 1.5	N = 82 2–24 y	Incremental cycling in fed or fasted state until exhaustion or 150 Watts (59 ± 2% of VO_{2max})	No change in total ghrelin
Studies with exercise-trained men and women combined				
Holliday & Blannin (2017)[76]	Overweight men and women BMI = 27.7 ± 1.7	N = 8 (4 men, 4 women) 22–46 y	Very low volume sprint interval exercise (4×30 s of "flat-out" cycling on an ergometer)	Acylated ghrelin ↓
Martins et al (2015)[78]	Overweight/obese volunteers BMI = 32.3 ± 2.7 VO_{2max} = 30.5 ± 4.9	N = 12 (5 men, 7 women) 24–44 y	Acute isocaloric bouts of HIIC and MICC, or short-duration HIIC	Acylated ghrelin plasma levels were lower in MICC and HIIC groups, but not in S-HIIC group, vs control group
Metcalfe et al (2015)[79]	Male and female volunteers BMI = 23 ± 3 VO_{2max} = 51 ± 11	N = 11 (5 men, 6 women) 20–26 y	10-min of reduced-exertion high-intensity interval training as a cycling session	Acylated ghrelin ↓
Hagobian et al (2012)[80]	Healthy male and female volunteers	N = 21 19–24 y	Exercise on a cycle ergometer at 70% of VO_{2peak}	No change in total ghrelin
Russell et al (2009)[81]	Male and female endurance runners Male, BMI = 21.9 ± 1.5 VO_{2max} = 63.7 ± 6.3 Female, BMI = 21.0 ± 1.1 VO_{2max} = 53.2 ± 5.4	N = 21 18–36 y	8-d session: running on 7 d for 90 min at 63% of VO_{2max} + a 10-km time trial on 1 d	Total ghrelin ↑ immediately post exercise ($P < 0.0001$)

(continued)

Table 2 **Continued**

Reference	Population[a]	No. and age of participants	Intervention	Results
Burns et al (2007)[22]	Male and female volunteers Male, BMI = 23.4 ± 1.0 VO_{2max} = 63.2 ± 2.5 Females, BMI = 22.5 ± 0.8 VO_{2max} = 52.1 ± 2.4	N = 18 23–27 y	Treadmill running for 60 min at 73.5% of VO_{2max}	No difference in total ghrelin post exercise vs control trial

Abbreviations and symbols: BMI, body mass index; BW, body weight; HIIC, high-intensity intermittent cycling; HIIE, high-intensity intermittent exercise; HR_{max}, maximal heart rate; MICC, moderate-intensity continuous cycling; MIE, moderate-intensity exercise; NS, nonsignificant; O_2, oxygen; VO_{2max}, maximum oxygen consumption; VO_{2peak}, peak oxygen uptake; W_{max}, maximal power; ↑, increased; ↓, decreased.

[a]BMI shown in kg/m^2; VO_{2max} and VO_{2peak} shown in mL/kg/min.

(≈ 60% of VO_{2max}) resulted in higher GLP-1 levels in female runners.[52,82] In addition, a recent meta-analysis emphasized that exercise increases GLP-1 levels in normal-weight individuals.[96] Therefore, it can be surmised that lean persons would have a reduced desire to eat after this type of exercise. These results remain to be confirmed in individuals with obesity and across different modalities of exercise.

It is well known that weight loss induced by calorie restriction reduces GLP-1 levels, but weight loss induced by physical activity has been reported to induce a reverse effect. Indeed, Martins et al[34] were the first to examine the effect of 12 weeks of aerobic training on fasting GLP-1 levels and postprandial phase in individuals with obesity (energy expenditure = 500 kcal on the treadmill, 5 times per week). This type of training had no impact on the fasting GLP-1 concentration but tended to increase it in the postprandial phase. This would help explain why aerobic training results in reduced body mass. In fact, in some studies, moderate-intensity training and aerobic training resulted in increased levels of GLP-1 in trained men and women.[48,53,85] Interestingly, results from studies that explored the effect high-intensity training (intermittent type) found no changes in this hormone.[57,86,87] Again, further research seems warranted.

Peptide YY response

Glucagon-like peptide 1 is cosecreted with peptide YY, an anorectic hormone. The secretion of peptide YY is proportional to the quantity of dietary fats ingested during food intake.[97] The highest postprandial concentration of peptide YY occurs approximately 2 hours after meal ingestion and is correlated with the size and type of the meal.[98,99] Fat intake is the most potent stimulant of peptide YY secretion, while carbohydrate intake has a limited effect on secretion in individuals with or without obesity.[100] In several studies, individuals with obesity had attenuated postprandial peptide YY responses.[101–103] This could lead to uncontrolled food intake, which always favors a positive energy balance. The study of Zwirska-Korczala et al[103] supports this idea, as peptide YY secretion was decreased in women who were obese or morbidly obese compared with normal-weight women.

Most studies examining the effects of chronic exercise (moderate intensity) found no changes in peptide YY concentrations,[34,40–42,46,48,104,105] while 2 studies recorded an increase in peptide YY levels after long-term exercise interventions (> 32 weeks) in overweight or obese individuals[104,106] (Table 4[34,40–42,46,48,104–106]).

In contrast to studies involving dietary interventions in obese individuals, studies examining acute exercise in normal-weight individuals reported an increase in peptide YY levels in both men and women.[48,49,53,57,58,61,63,69,72,73,85,86,89,90,107,108] These findings are not, however, universal, as others report no changes in levels of peptide YY[44,56,59,60] (Table 5[23,44,48,49,53,56–61,63,66,69,72,73,77–81,85,86,89–91,95,107–109]). The increase in peptide YY levels is important because it could induce the suppression of hunger, thereby potentially reducing the postexercise compensation (ie, increased food intake) for energy expended. However, this remains to be verified in individuals with obesity.

To the best of knowledge, no studies have evaluated the impact of acute exercise on peptide YY responses in overweight or obese individuals. Jones et al[104] observed an increase in fasting peptide YY concentrations and a significant decrease in body fat after 32 weeks of exercise and training in overweight adolescents. In addition, Martins et al[34] reported a trend toward increased postprandial peptide YY levels in obese men and women after 12 weeks of training (aerobic training at 75% of maximum heart rate). More recently, Guelfi et al[41] indicated that aerobic or resistance training does not significantly alter fasting and postprandial peptide YY levels in individuals with obesity. Thus, the contribution of peptide YY to improving satiety following exercise and/or training in individuals with obesity remains uncertain and warrants further study. Again, the impact of

Table 3 **Studies examining the effect of acute exercise on glucagon-like peptide 1 (GLP-1) concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Studies in exercise-trained women				
Hallworth et al (2017)[85]	Healthy, active women BMI = 23.5 ± 2.8 VO_{2max} = 40.7 ± 5.4	N = 9 Age = 22–39 y	(1) Moderate-intensity continuous training (30 min, 65% of VO_{2max}); (2) sprint interval training (6 × 30 s of "all-out" cycling sprints with 4-min recovery)	GLP-1 increased
Howe et al (2016)[53]	Highly trained women BW = 58.4 ± 6.4 kg VO_{2max} = 55.2 ± 4.3	N = 15 Age = 18–40 y	Moderate-intensity (60% of VO_{2max}) and high-intensity (85% of VO_{2max}) treadmill running	GLP-1 increased
Ueda et al (2013)[48]	Healthy middle-aged women BMI = 27.6 ± 0.4 VO_{2peak} = 23.5 ± 0.9	N = 20 Age not reported	Aerobic exercise at 65% of HR_{max}	GLP-1 increased
Larson-Meyer et al (2012)[49]	Female runners BMI = 19.8 ± 1.0 VO_{2max} = 49.7 ± 3.0	N = 9 Age = 21–27 y	Run or walk for 60 min at 70% of VO_{2max}	GLP-1 increased post exercise vs rest
Unick et al (2010)[52]	Female volunteers BMI = 32.50 ± 4.83	N = 19 Age = 20–39 y	Walking at 70%–75% of age-predicted HR_{max} until 3.0 kcal/kg of body weight is expended (average energy expenditure, 354 ± 72 kcal; average duration, 42 ± 8 min)	No change in GLP-1
Studies in exercise-trained boys and men				
Yang et al (2018)[88]	Adolescent boys BMI > 30	N = 35 Age = 13–16 y	"Living High–Training Low"	Plasma GLP-1 increased
Hazell et al (2017)[89]	Male volunteers	N = 10 Age not reported	30 min of cycling at 65%–85% of VO_{2max})	No significant change in GLP-1
Hunschede et al (2017)[87]	Normal-weight and overweight/obese boys	N = 22 (11 normal weight, 11 overweight/obese) Age = 10–18 y	High-intensity exercise at 70% of VO_{2peak}	No significant change in GLP-1
Bailey et al (2015)[57]	Male volunteers BMI = 23.5 ± 2.0	N = 12 Age = 19–27 y	(1) MIE normoxia; (2) MIE hypoxia; (3) HIIE-normoxia; and (4) HIIE hypoxia	No differences in GLP-1 were observed between conditions
Beaulieu et al (2014)[86]	Male volunteers BM = 76.9 ± 9.7	N = 8 Age = 22–28 y	Sprint interval exercise training	No significant change in GLP-1
Ueda et al (2009)[90]	Male volunteers BMI = 22.5 ± 1.0 VO_{2max} = 45.9 ± 8.5	N = 10 Age = 19–28 y	Cycling for 30 min at 75% or 50% of VO_{2max} or rest	GLP-1 increased
Ueda et al (2009)[73]	Obese men and age-matched normal-weight men	N = 14 (7 obese, 7 normal weight) Age = 18–27 y	Cycling exercise at 50% of VO_{2max}	Plasma GLP-1 was increased by exercise
Studies with exercise-trained men and women combined				
Hazell et al (2017)[91]	Healthy adults	N = 21 (11 women, 10 men) Age = 22–39 y	Moderate-intensity continuous exercise and sprint interval exercise at 40%–75% of VO_{2max} for 30–60 min	GLP-1 increased
Holliday & Blannin (2017)[76]	Overweight volunteers BMI = 27.7 ± 1.7	N = 8 (4 men, 4 women) Age = 22–46 y	Very low volume sprint interval exercise (4 × 30 s of "flat-out" cycling on an ergometer)	GLP-1 increased
Douglas et al (2017)[77]	Healthy lean women BMI = 22.4 ± 1.5 Overweight/obese females BMI = 29.2 ± 2.9	N = 47 Age = 22–58 y	60 min of treadmill exercise 60% VO_{2peak})	GLP-1 increased
Martins et al (2015)[78]	Overweight/obese volunteers BMI = 32.3 ± 2.7 VO_{2max} = 30.5 ± 4.9	N = 12 (5 men, 7 women) Age = 24–44 y	Acute isocaloric bouts of HIIC and MICC, or short-duration HIIC	GLP-1 increased significantly during all exercise bouts

(continued)

Table 3 **Continued**

Reference	Population[a]	No. and age of participants	Intervention	Results
Chanoine et al (2008)[92]	Normal-weight and overweight adolescent boys	N = 34 (17 normal weight, 17 overweight) Age = 15–16 y	5 consecutive days of supervised aerobic exercise (1 h/d). Blood samples taken at baseline and 36 h after end of intervention in fasted state and postprandially for period of 4 h	No significant change in fasting plasma GLP-1 or 4-h AUC, but significant increase in GLP-1 response in first 30 min postprandially
Martins et al (2007)[23]	Healthy, normal-weight volunteers	N = 12 (6 men, 6 women) Age = 21–31 y	60 min of cycling at 65% of HR_{max} or rested	GLP-1 increased
Martins et al (2007)[93]	Healthy sedentary volunteers BMI = 22.7 ± 2.3	N = 29 (15 men, 14 women) Age = 18–42 y	1 h or intermittent cycling at 65% of HR_{max} (1 h after a 500-kcal breakfast) vs resting. Blood samples taken in fasted state and postprandially for a period of 3 h	Significant increase in postprandial GLP-1
O'Connor et al (2006)[94]	Endurance-trained men	N = 6 Age = 30–41 y	2-h treadmill run at 60% of VO_{2max} vs resting	Significant increase in fasting GLP-1 during exercise and resting
O'Connor et al (1995)[95]	Male and female marathon runners	N = 26 (23 men, 3 women) Age = 19–61 y	Marathon running (average time = 239 min)	Significant increases in fasting plasma GLP-1

Abbreviations and symbols: BM, body mass; BMI, body mass index; BW, body weight; HIIC, high-intensity intermittent cycling; HIIE, high-intensity intermittent exercise; HR_{max}, maximal heart rate; MICC, moderate-intensity continuous cycling; MIE, moderate-intensity exercise; VO_{2max}, maximum oxygen consumption; VO_{2peak}, peak oxygen uptake; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2; VO_{2max} and VO_{2peak} shown in mL/kg/min.

anaerobic exercise and training in lean individuals or those with obesity requires further investigation.

Pancreatic polypeptide response

Another anorectic hormone, pancreatic polypeptide, is secreted following meal consumption and affects dietary caloric intake.[110] However, studies on pancreatic polypeptide levels in individuals with obesity have shown conflicting results. Some investigators find no difference between normal-weight and obese individuals,[111] while other studies have demonstrated lower levels of pancreatic polypeptide in individuals with obesity.[112] If future studies demonstrate that pancreatic polypeptide levels are decreased in individuals with obesity, this could explain, at least in part, the development of overweight or even point to a causative factor in their condition. Further studies are needed to understand the role of this hormone in the pathology of obesity.

A limited amount of literature on the impact of exercise and training on pancreatic polypeptide levels in healthy or obese individuals is available. Studies examining the effect of acute exercise on pancreatic polypeptide levels found increases in fasting pancreatic polypeptide levels,[113] postprandial pancreatic polypeptide levels,[23,114] plasma pancreatic polypeptide levels[95,115,116] and postexercise pancreatic polypeptide levels.[117] Furthermore, the meta-analysis of Schubert et al[96] found that a session of exercise in healthy individuals induces an increase in pancreatic polypeptide concentrations. Such increases of pancreatic polypeptide levels could potentially explain the reduction in appetite reported during the hours following exercise (Table 6[109,118,119]). Some research studies examining this training-related effect on pancreatic polypeptide levels reported increased levels after moderate chronic exercise,[96,118] while others found no changes[119,120] (Table 7[23,95,113–117]).

Cholecystokinin response

Cholecystokinin is an anorexigenic hormone secreted by the duodenal and jejunal mucosa when highly acidic food enters the small intestine. Studies have shown that reduced levels of cholecystokinin may contribute to a reduced feeling of fullness and make it more difficult for some obese people to lose weight.[121] Cholecystokinin production is impaired (reduced) in individuals with obesity who are experiencing body weight reduction. Indeed, according to Sumithran et al,[122] cholecystokinin concentrations were reduced in individuals with obesity who lost 14% of their initial weight after 8 weeks of a hypocaloric diet and 2 weeks of stabilization. Similarly, in men with obesity who underwent a low-calorie dietary intervention for

Table 4 **Studies examining the effect of chronic exercise on peptide YY (PYY) concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Gibbons et al (2017)[40]	Overweight/obese individuals; 16 completed 12 wk of aerobic exercise and 16 were age- and BMI-matched nonexercising controls	N = 32 Age = 37–49 y	12-wk exercise intervention:-5 exercise sessions per week	No change in PYY
Martins et al (2017)[46]	Sedentary obese individuals BMI = 33.3 ± 2.9	N = 46 (30 women, 16 men) Age = 25–44 y	12 wk of isocaloric programs of MICT or HIIT, or a short-duration HIIT (½HIIT)	No change in PYY
Guelfi et al (2013)[41]	Overweight/obese men BMI = 30.8 ± 4.2	N = 33 Age = 42–56 y	12-wk training (3 d/wk). 3 groups: (1) aerobic (n = 12), 40–60 min at 70%–80% of HR_{max}; (2) resistance (n = 13) 3–4 sets of 8–10 reps at 75%–85% of 1RM; (3) control (n = 8)	No change in PYY after 12 wk of aerobic or resistance training program
Ueda et al (2013)[48]	Healthy middle-aged women BMI = 27.6 ± 0.4 VO_{2peak} = 23.5 ± 0.9	N = 20 Age = 49.1 ± 0.8 y	12 wk of exercise training at 65% of HR_{max}	No change in PYY
Gueugnon et al (2012)[42]	Obese adolescents BMI *z* score = 4.1	N = 32 (10 boys, 22 girls) Age = 14–15 y	Physical exercise 5 times/wk during the following 7 mo	No change in PYY
Martins et al (2010)[34]	Sedentary overweight men and women BMI = 31.3 ± 2.3 VO_{2max} = 32.9 ± 6.6	N = 15 Age = 28–46 y	12-wk exercise intervention	Higher postprandial PYY
Jones et al (2009)[104]	Overweight male and female adolescents BMI = 31.8 ± 5.2	N = 12 Age = 14–16 y	32 wk of exercise training	Significant increase in fasting plasma PYY
Kelly et al (2009)[105]	Older obese men and women with impaired glucose tolerance BMI = 34.4 ± 1.7	N = 19 Age = 65–69 y	12 wk of moderate-intensity aerobic exercise (treadmill/cycle ergometer) at ≈ 75% of VO_{2max} combined with eucaloric or hypocaloric diet	No change in PYY
Roth et al (2005)[106]	73 obese children and 45 age-matched normal-weight children	N = 118 Age = 9–13 y	1-y diet and exercise intervention	Significant increase in fasting plasma PYY

Abbreviations and symbols: BMI, body mass index; HIIT, high-intensity interval training; HR_{max}, maximal heart rate; MICT, moderate-intensity continuous training; VO_{2max}, maximum oxygen consumption; VO_{2peak}, peak oxygen uptake; 1RM, 1 rep maximum; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2; VO_{2max} and VO_{2peak} shown in mL/kg/min.

8 weeks and lost approximately 15% of their body weight, postprandial cholecystokinin concentrations decreased significantly compared with baseline values.[123] Both of these studies suggest that cholecystokinin levels are reduced as a result of rapid weight loss, which may serve as a rebound mechanism by promoting appetite and, ultimately, weight gain after rapid weight loss.

As with studies of pancreatic polypeptide, studies of the effect of exercise and training on cholecystokinin levels are very limited and controversial. For example, in several studies, plasma cholecystokinin levels[124,125] and cholecystokinin content of the intestine[126] increased significantly following intensive training or hypoxia. Conversely, cholecystokinin levels were decreased in female runners after high-intensity training[127] but were unchanged in another exercise study[126] (Table 8[124,125,127,128]). Interestingly, Martins et al[128] indicated that a 12-week training program in individuals with obesity induced a mean decrease in body weight of 3.5 kg (from 96.1 ± 11.0 to 92.6 ± 11.7 kg) but had no significant effect on fasting or postprandial cholecystokinin concentrations.

So far, studies on the impact of acute exercise on cholecystokinin levels seem to have been conducted only in normal-weight individuals (Table 9[115,124,129,130]). These studies reported an increase in cholecystokinin levels immediately after exercise and for up to 2 h after exercise.[115,124] These significant increases in cholecystokinin levels are associated with suppressed feelings of hunger during the hours following exercise.[96] The limited data in this area cannot be generalized to include the effect of exercise and training on cholecystokinin levels in individuals with obesity. Future studies in this area are urgently needed.

Table 5 **Studies examining the effect of acute exercise on peptide YY (PYY) concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Studies in exercise-trained women				
Hallworth et al (2017)[85]	Healthy active women BMI = 23.5 ± 2.8 VO_{2max} = 40.7 ± 5.4	N = 9 Age = 22–39 y	(1) MICT for 30 min at 65% VO_{2max}; (2) sprint interval training, 6 × 30 s of "all-out" cycling sprints with 4 min of recovery	Exercise ↑ PYY
Howe et al (2016)[53]	Highly trained women BW = 58.4 ± 6.4 kg VO_{2max} = 55.2 ± 4.3	N = 15 Age = 18–40 y	Moderate-intensity (60% of VO_{2max}) and high-intensity (85% of VO_{2max}) treadmill running	Exercise ↑ $PYY_{3–36}$
Ueda et al (2013)[48]	Healthy middle-aged women BMI = 27.6 ± 0.4 VO_{2peak} = 23.5 ± 0.9	N = 20 Age = 49.1 ± 0.8 y	Aerobic exercise at 65% of HR_{max}	Exercise ↑ PYY
Larson-Meyer et al (2012)[49]	Female runners BMI = 19.8 ± 1.0 VO_{2max} = 49.7 ± 3.0	N = 9 Age = 21–29 y	Run or walk for 60 min at 70% of VO_{2max}	PYY ($P < 0.01$) was ↑ post exercise vs rest
Studies in exercise-trained men				
Hazell et al (2017)[89]	Active young healthy male volunteers	N = 10 Age = NR	(1) MICT 8 × 30 min of cycling at 65% of VO_{2max}; (2) HICT 9 × 30 min of cycling at 85% of VO_{2max}; (3) sprint interval training: 6 × 30 s of 10 "all-out" cycling bouts with 4-min recovery periods; (4) control: no exercise	Total PYY ↑ only after HIE
Kojima et al (2016)[56]	College endurance runners Height = 171.2 ± 1.9 cm BW=56.3 ± 1.0 kg BMI = 19.3 ± 0.4 VO_{2max} = 67.1 ± 1.0	N = 23 Age = 19–21 y	20-km outdoor run or a control trial with an identical period of rest	No change in $PYY_{3–36}$
Bailey et al (2015)[57]	Men BMI = 23.5 ± 2.0	N = 12 Age = 19–24 y	(1) MIE normoxia; (2) MIE-hypoxia; (3) HIIE-normoxia; and (4) HIIE hypoxia	PYY was higher in HIIE than in MIE under hypoxic conditions during exercise
Douglas et al (2015)[58]	Men BMI = 23.0 ± 1.9	N = 15 Age = 19–23 y	60 min of continuous moderate-high intensity treadmill running	PYY was higher in the exercise vs the control trial
Beaulieu et al (2014)[86]	Men BMI = 76.9 ± 9.7	N = 8 Age = 22–28 y	Sprint interval exercise training	PYY ↑
King et al (2014)[59]	Young men BMI = 22.6 ± 1.8	N = 9 Age = 22 ± 1.2 y	90 min of moderate-intensity treadmill running	No change in PYY
Sim et al (2014)[60]	Overweight men BMI = 27.7 ± 1.6 BM = 89.8 ± 10.1 kg	N = 17 Age = 30 ± 8 y	HIIE	No change in PYY
Kawano et al (2013)[61]	Young men BMI = 22.1 ± 2.0 VO_{2max} = 47.0 ± 6.2	N = 15 Age = 24.4 ± 1.7 y	Rope skipping 3 times for 10 min with 5 min of rest at 64.8% ± 6.9% of VO_{2max}. Cycling 3 times for 10 min with 5 min of rest at 63.9% ± 7.5% of VO_{2max}	$PYY_{3–36}$ ↑ immediately post exercise ($P < 0.0167$)
Deighton et al (2013)[107]	Young men BMI = 23.7 ± 3.0 VO_{2max} = 52.4 ± 7.1	N = 12 Age = 22 ± 3 y	Cycling: steady-state 60 min at 59.5% ± 1.6% of VO_{2max}. High-intensity cycling: 10 times for 4-min intervals at 85.8% ± 4% of VO_{2max} with 2-min rests	$PYY_{3–36}$ ↑ post exercise in steady state and high-intensity cycling ($P = 0.002$ and $P = 0.015$, respectively)
Deighton et al (2013)[63]	Young men BMI = 24.2 ± 2.9 VO_{2max} = 46.3 ± 10.2	N = 12 Age = 23 ± 3 y	Control, endurance exercise, and sprint interval exercise	PYY ↑ during all exercise, but most consistently during endurance exercise
Wasse et al (2011)[66]	Young men BMI = 24.8 ± 2.4 VO_{2max} = 56.9 ± 6.5	N = 10 Age = 24 ± 3 y	Treadmill running for 60 min at 70% of VO_{2max} at normoxic (20.9% O_2) or hypoxic (12.7% O_2)	PYY ↑ ($P = 0.04$) in both conditions

(continued)

Table 5 **Continued**

Reference	Population[a]	No. and age of participants	Intervention	Results
King et al (2011)[69]	Young men BMI = 22.8 ± 0.4 VO_{2max} = 57.3 ± 1.2	N = 12 Age = 23.4 ± 1.0 y	Treadmill running at 70% of VO_{2max} for 90 min in exercise energy deficit, food deficit, or control condition	Energy deficit ↑ $PYY_{3–36}$ post exercise ($P < 0.05$)
King (2010)[44]	Young men BM = 76.2 ± 1.0 kg	N = 69 Age = 22.4 ± 0.3 y	(1) 90 min of resistance exercise and 60 min of swimming; (2) 60 min of brisk walking; (3) 90 min of treadmill running	No increase in $PYY_{3–36}$ post exercise
Broom et al (2009)[72]	Young men BMI = 23.1 ± 0.4 VO_{2max} = 62.1 ± 1.8	N = 11 Age = 21.1 ± 0.3 y	Treadmill running for 60 min at 70% of VO_{2max}	PYY ↑ post exercise ($P < 0.05$)
Shorten et al (2009)[108]	Young men BMI = 24.1 ± 2.3 VO_{2peak} = 53.8 ± 8.9	N = 11 Age = 20.8 ± 2.1 y	Treadmill running at 70% of VO_{2peak} for 40 min at neutral temperature (25°C) or in heat (36°C)	PYY ↑ post exercise ($P < 0.05$) in heat and in neutral conditions
Ueda et al (2009)[90]	Young men BMI = 22.5 ± 1.0 VO_{2max} = 45.9 ± 8.5	N = 10 Age = 23.4 ± 4.3 y	Cycling for 30 min at 75% or 50% of VO_{2max} or rest	Exercise ↑ ($P < 0.01$) $PYY_{3–36}$. $PYY_{3–36}$ ↑ in 75% vs 50% of VO_2max at 60 min post exercise ($P < 0.01$)
Ueda et al (2009)[73]	Obese and age-matched adults of normal weight	N = 14 (7 obese, 7 normal weight) Age = 26–34 y	Cycling exercise at 50% of VO_{2max}	Exercise ↑ plasma PYY
Studies with exercise-trained men and women combined				
Hazell et al (2017)[91]	Healthy adults	N = 21 (11 women, 10 men) Age = 22–39 y	MICT and sprint interval exercise at 40%–75% of VO_{2max} for durations of 30–60 min	PYY↑
Douglas et al (2017)[77]	Healthy lean women BMI = 22.4 ± 1.5 Overweight/obese women BMI = 29.2 ± 2.9	N = 47 Age = 22–58 y	60 min of treadmill exercise at 60% VO_{2peak})	PYY ↑
Martins et al (2015)[78]	Overweight/obese volunteers BMI = 32.3 ± 2.7 VO_{2max} = 30.5 ± 4.9	N = 12 (5 men, 7 women) Age = 24–44 y	Acute isocaloric bouts of HIIC and MICC, or short-duration HIIC	No significant differences in plasma $PYY_{3–36}$ levels
Metcalfe et al (2015)[79]	Men and women BMI = 23 ± 3 VO_{2max} = 51 ± 11	N = 11 (5 men, 6 women) Age = 20–26 y	10 min of reduced-exertion high-intensity interval training cycling session	No change in PYY
Kanaley et al (2014)[109]	Healthy obese men and women BMI = 30–45	N = 13 Age = 40–44 y	Walking for 1 h at 70%–75% of VO_{2peak}	No significant changes in PYY
Hagobian et al (2012)[80]	Healthy men and women	N = 21 Age = 19–24 y	Exercise on a cycle ergometer at 70% of VO_{2peak}	Total PYY ↑ 15 min after exercise or rest in women
Russell et al (2009)[81]	Endurance runners Male, BMI = 21.9 ± 1.5 VO_{2max} = 63.7 ± 6.3 Female, BMI = 21.0 ± 1.1 VO_{2max} = 53.2 ± 5.4	N = 21 Age = 18–36 y	8-d session: running on 7 d for 90 min at 63% of VO_{2max} + a 10-km time trial on 1 s	Total PYY ↑ immediately post exercise ($P < 0.0001$)
Martins et al (2007)[23]	Healthy normal-weight volunteers	N = 12 (6 men, 6 women) Age = 21–31 y	Cycling for 60 min at 65% of individual HR_{max} or rested	PYY ↑
O'Connor et al (1995)[95]	Marathon runners	N = 26 (23 men, 3 women) Age = 19–61 y	Marathon running (average time = 239 min)	PPY ↑ post race and 30 min post race ($P < 0.01$)

Abbreviations and symbols: BM, body mass; BMI, body mass index; BW, body weight; HICT, high-intensity continuous training; HIE, high-intensity exercise; HIIC, high-intensity intermittent cycling; HR_{max}, maximal heart rate; MICC, moderate-intensity continuous cycling; MICT, moderate-intensity continuous training; MIE, moderate-intensity exercise; NR, not reported; O_{2max}, maximum oxygen consumption; VO_{2peak}, peak oxygen uptake; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2; VO_{2max} and VO_{2peak} shown in mL/kg/min.

Leptin response

Leptin is an extensively studied anorectic adipocyte hormone. Secretion of leptin increases proportionally with the lipid content of a consumed meal. This hormone signals the hypothalamus receptors to reduce appetite and increase energy expenditure.[131] According to Akieda-Asai et al,[132] leptin potentiates

Table 6 **Studies examining the effect of acute exercise training on pancreatic polypeptide (PP) concentrations**

Reference	Population[a]	No. and age of participants	Intervention[b]	Results
Hurley et al (1991)[119]	Normal-weight sedentary men	N = 7 Age = college age	10-wk exercise program (20 min of jogging at 70% of VO_{2max}, 3 times/wk)	PP fasting and postprandial plasma levels ↑
Kanaley et al (2014)[109]	Healthy obese men and women BMI = 30–45	N = 13 Age = 40–44 y	Short-term aerobic exercise training (15 d)	PP ↑
Øktedalen et al (1983)[118]	Young men	N = 24 Age = 21–26 y	5-d training course with long-term physical exercise (35% of VO_{2max}), with or without caloric deficiency	Fasting serum PP ↑ during training in low-caloric group Serum PP ↓ to pretraining levels after 8 h of rest Postprandial PP increase was greater in low-caloric group than in group with caloric balance

Abbreviations and symbols: BMI, body mass index; VO_{2max}, maximum oxygen consumption; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2.
[b]VO_{2max} shown in mL/kg/min.

Table 7 **Studies examining the effect of chronic exercise on pancreatic polypeptide (PP) concentrations**

Reference	Population[a]	No. and age of participants	Intervention[b]	Results
Hilsted et al (1980)[113]	Male marathon runners	N = 6 Age not reported	3 h of exercise (cycle ergometer) at 40% at VO_{2max} vs resting	Fasting PP ↑
Greenberg et al (1986)[114]	Normal-weight male and female volunteers	N = 7 Age = 23–39 y	45 min of exercise (cycle ergometer) at 50% of VO_{2max}, 30 min after breakfast vs resting	Postprandial plasma PP ↑
O'Connor et al (1995)[95]	Male and female marathon runners	N = 26 (23 men, 3 women) Age = 19–61 y	Marathon running	Plasma PP ↑
Sliwowski et al (2001)[115]	Normal-weight men	N = 19 Age = 20–24 y	Treadmill run to exhaustion in fasting or fed state (5 min after a liquid meal)	Plasma PP ↑ after exercise, independently of feeding
Martins et al (2007)[23]	Healthy, normal-weight volunteers	N = 12 (6 men, 6 women) Age = 21–31 y	1 h or intermittent cycling at 65% of HR_{max} (1 h after breakfast) vs resting	Postprandial PP ↑
Mackelvie et al (2006)[117]	Normal-weight and overweight male adolescents Normal weight: BMI = 20.7 ± 0.5 Overweight: BMI = 32.4 ± 1.7	N = 17 Age = 15–16 y	1 h after a standardized breakfast, participants either cycled for 60 min at 65% of individual HR_{max} or rested	PP ↑ after exercise
Feurle et al (1980)[116]	Male long-distance runners or cyclists and male volunteers who did not exercise regularly	N = 18 (9 runners or cyclists, 9 nonathletes) Age = 25–35 y	Maximal bicycle ergometer test included 150 W and 75 W for athletes and nonathletes, respectively. After 8 min, the workload was gradually increased every 2 min until exhaustion. Maximal workload reached 379 W for athletes and 240 W for nonathletes	Plasma PP ↑

Abbreviations and symbols: BMI, body mass index; VO_{2max}, maximum oxygen consumption; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2.
[b]VO_{2max} shown in mL/kg/min.

the effects of cholecystokinin on inhibition of food intake, but this mechanism is disrupted by obesity. In a study of normal-weight and obese women, leptin levels were examined after 12 h of fasting and after ingestion of a standardized meal over a 2-h period.[133] Basal leptin levels were significantly higher in the obese group than in the normal-weight group and then progressively decreased at 15 minutes, 60 minutes, and 120 minutes postprandially. In contrast, in the normal-weight group, leptin levels

Table 8 **Studies examining the effect of chronic exercise training on cholecystokinin (CCK) concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Bailey et al (2001)[124]	Physically active men in normoxia or hypoxia BMI = 23.6 ± 1.6 VO_{2max} = 50 ± 9	N = 32 (14 normoxic, 18 hypoxic) Age = 19–25 y	Hypoxia and physical exercise with intermittent cycle training for 4 wk. Study performed in normobaric normoxia and normobaric hypoxia	Exercise in normoxic condition caused ↑ plasma CCK, but CCK was unchanged after normoxic exercise
Bailey et al (2000)[125]	Male mountaineers	N = 19 Age = 26–50 y	20 d stay at 5100 m (high altitude) to investigate possible role of altitude in the pathophysiology of anorexia, cachexia, and AMS, Participants were examined at rest and during a maximal exercise test at sea level before and after the expedition	Plasma CCK ↑ during day 2 in rest Plasma CCK ↑ after maximal exercise CCK response unchanged in 5 participants with anorexia on day 2 compared with those with normal appetite. In those with normal appetite, there was no relationship between increase in CCK and AMS score Resting CCK ↑ in those with AMS
Martins et al (2013)[128]	Overweight and obese healthy sedentary individuals BMI = 31.3 ± 3.3	N = 22 Age = 29–46 y	2-wk of supervised exercise	No change in plasma CCK
Hirschberg et al (1994)[127]	Female long-distance runners	N = 14 Age = 25–29 y	High-intensity training of 11 h/wk during their training season	Postprandial CCK ↓

Abbreviations and symbols: AMS, acute mountain sickness; BMI, body mass index; VO_{2max}, maximum oxygen consumption↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2; VO_{2max} shown in mL/kg/min.

Table 9 **Studies examining the effect of acute exercise training on cholecystokinin (CCK) concentrations**

Reference	Population	No. and age of participants	Intervention	Results
Bailey et al (2001)[124]	Physically active men in normoxia or hypoxia BMI = 23.6 ± 1.6 VO_{2max} = 50 ± 9	N = 32 (14 normoxic, 18 hypoxic) Age = 19–25 y	Hypoxia and physical exercise acute study with cycling test to exhaustion. Study performed in normobaric normoxia and normobaric hypoxia	Exercise in normoxic condition caused ↑ plasma CCK, but CCK was unchanged after normoxic exercise
Philipp et al (1992)[129]	Male and female marathon runners	N = 19 (11 male, 8 female) Age = 20–58 y	Long-distance running (marathon run)	CCK ↑ in pre-run CCK highest after the run
Ströhle et al (2006)[130]	Healthy untrained volunteers	N = 10 (2 women, 8 men) Age = 23–29 y	Study to assess antipanic effects (behavioral) of aerobic exercise (30 min at 70% of VO_{2max})	CCK-4 ↓
Sliwowski et al (2001)[115]	Normal-weight men	N = 19 Age = 20–24 y	Treadmill run to exhaustion in fasting or fed state (5 min after a liquid meal)	Plasma CCK ↑ after exercise, independently of feeding

Abbreviations and symbols: BMI, body mass index; VO_{2max}, maximum oxygen consumption.
[a]BMI shown in kg/m^2; VO_{2max} shown in mL/kg/min; ↑, increased; ↓, decreased.

remained relatively constant during the postprandial period. Thus, while leptin promotes satiety, individuals with obesity tend to develop resistance to leptin, which induces a regulatory disruption that can lead to uncontrolled food intake.

Most studies found decreased levels of leptin following exercise and training of low or moderate intensity,[128,134–155] while others found no change in leptin levels[156–164] (Table 10[124,128,134–152,154–164]). Most of this research focuses on aerobic/endurance training with or

Table 10 **Studies examining the effect of chronic exercise training on leptin concentrations**

Reference	Population[a]	No. and age of participants	Intervention[b]	Results
Fazelifar et al (2013)[134]	Obese boys BMI > 28	N = 24 Age = 11–13 y	12-wk (3 d/wk) concurrent training followed by a 4-wk detraining	Leptin ↓
Carter et al (2010)[156]	Overweight or obese insulin-resistant horses	N = 12 Age = 9–21 y	4 wk training at low intensity and 4 wk at higher intensity, followed by 2 wk of detraining	No change in leptin
Pérusse et al (1997)[157]	Sedentary adult men and women	N = 97 (51 men, 46 women) Age = 17–40 y	20 wk of endurance training on a computer-controlled cycle ergometer	No change in leptin after acute exercise After endurance training was completed, leptin ↓ in men but not in women
Dede et al (2015)[135]	Patients with type 2 diabetes mellitus	N = 60 Age not reported	Aerobic exercise	Leptin ↓
Nuri et al (2016)[158]	Male patients with colorectal cancer	N = 30 Age = 40–63 y	8 wk of aerobic exercise and followed by 1 wk of detraining	No change in plasma leptin
Murakami et al (2007)[136]	Obese nondiabetic individuals	N = 42 Age = 46–51 y	Weight reduction by a 12-wk calorie-restricted diet with or without aerobic exercise	Leptin ↓
Sari et al (2007)[137]	Obese women	N = 23 Age = 30–52 y	Exercise program (45-min walking sessions at 60%–80% of HR_{max}) every day for 4 wk (20 exercise sessions total)	No change in leptin after acute exercise Leptin ↓ after chronic exercise
Koutsari et al (2003)[138]	Healthy, postmenopausal women	N = 8 Age = 56–64 y	Daily MIE (walking on treadmill for 60 min) plus a short-term high-carbohydrate diet	Fasting and postprandial circulating leptin ↓ after daily MIE and consumption of a short-term high-carbohydrate diet
Ordonez et al (2013)[139]	Obese young women with Down syndrome	N = 20 Age = 21–29 y	10-wk aerobic training program (30–40 min on a treadmill) at a work intensity of 55%–65% of peak heart rate	Plasma leptin ↓
Azizi (2011)[140]	Untrained women	N = 24 Age = 25–34 y	Aerobic training program: running on treadmill at 65%–85% of individual HR_{max} for three 30-min sessions per week for 8 consecutive weeks	Serum leptin ↓
Reseland et al (2001)[141]	Men with metabolic syndrome	N = 186 Age = 42–48 y	Long-term reductions in food intake plus increased physical activity (60-min workouts of aerobics, circuit training, and fast walking and jogging, 3 times/wk)	Plasma leptin ↓ after both the diet and the exercise interventions
Houmard et al (2000)[159]	Young lean individuals (n = 16; 9 women, 7 men) Older individuals with relatively more adipose tissue (n = 14; 8 women, 6 men)	N = 30 Age, younger = 21–22 y Age, older = 57–60 y	Short-term aerobic training (60 min at 75% of VO_{2max} for 7 consecutive days)	No change in leptin
Gippini et al (1999)[160]	Nonprofessional body builders (n = 25) Mildly overweight sedentary individuals (n = 21) Normal–weight sedentary controls (n = 19)	N = 65 Age = 26–30 y	Resistance exercise	No change in leptin production
Kraemer et al (2001)[161]	Adolescent female distance runners	N = 7 Age = 14–16 y	Intense exercise for 7 wk	No change in leptin measured during resting or after maximal exercise
Kraemer et al (1999)[162]	Middle-aged obese women	N = 16 Age = 41–44 y	9-wk training program (3–4 d of exercise, including 20–30 min of step aerobics 2 d/wk and treadmill running or stationary cycling on additional days)	No change in leptin
Gomez-Merino et al (2002)[142]	Male soldiers	N = 26 Age = 19–23 y	3 wk of a military training program	Leptin ↓

(continued)

Table 10 **Continued**

Reference	Population[a]	No. and age of participants	Intervention[b]	Results
Unal et al (2005)[143]	Trained male athletes and healthy sedentary male volunteers	N = 46 (24 trained, 22 sedentary) Age = 16–23 y	Regular exercise	Leptin ↓
Unal et al (2005)[144]	Professional football players and healthy sedentary male volunteers	N = 27 (10 athletes, 17 nonathletes) Age = 17–20 y	Regular exercising	Leptin levels in football players ↓ more than in healthy nonathletes
Fatouros et al (2005)[145]	Inactive men BMI = 28.7–30.2	N = 50 Age = 65–78 y	Resistance training (3 d/wk for 6 mo; 3 sets of 10 exercises)	Plasma leptin ↓
Ishii et al (2001)[146]	Sedentary individuals with type 2 diabetes	N = 50 Age = 50–66 y	6 wk of aerobic training	Leptin ↓
Hickey et al (1997)[147]	Sedentary middle-aged men and women	N = 18 (9 men, 9 women) Age not reported	12 wk of aerobic training	Leptin ↓
Okazaki et al (1999)[148]	Obese and nonobese middle-aged sedentary women	N = 41 (15 obese, 26 nonobese) Age = 47–59 y	Mild aerobic exercise (50% of VO_{2max}) and personal diet counseling for 12 wk	Leptin ↓
Herrick et al (2016)[149]	Men and women	N = 10 (7 women, 3 men) Age = 22–60 y	6 mo of a diet/exercise weight loss program	Free leptin index ↓ at 3 mo and 6 m in men and at 6 mo in women
Ackel-D'Elia et al (2014)[150]	Adolescents	N = 132 Age = 15–19 y	Leisure physical activity, aerobic training, and aerobic plus resistance training as 6-mo interdisciplinary therapy	Leptin ↓ after aerobic training and aerobic plus resistance training
Lau et al (2010)[163]	Overweight adolescents	N = 18 (5 girls, 13 boys) Age = 10–15 y	Resistance training (3 times/wk on alternate days for 6 wk)	No change in serum leptin
Ko & Choi (2013)[151]	Sturdy men	N = 36 Age = 21–26 y	Aerobic exercise using a treadmill (60% of heart rate reserve), plus weight training (9 different exercises for the large muscles) 5 d/wk for 8 wk	Leptin ↓
Martins et al (2013)[128]	Overweight and obese healthy sedentary individuals BMI = 31.3 ± 3.3	N = 22 Age = 29–46 y	2 wk of supervised exercise	Fasting and postprandial leptin ↓
Kohrt et al (1996)[152]	Older women	N = 61 Age = 60–72 y	2-mo flexibility exercise program followed by a 9-mo exercise program (included walking, jogging, and stair climbing)	Serum leptin ↓
Miyatake et al (2004)[153]	Overweight men	N = 110 Age = 32–59 y	1-y aerobic exercise program (HR_{max}: 50%–65%) that included walking, aerobic dance, and swimming, and resistance training, ie, leg extension, leg flexion, sit-ups. Each session lasted 90 min	Leptin ↓
Pasman et al (1998)[154]	Obese men	N = 15 Age = 32–43 y	Weight loss program and endurance exercise (1-h sessions of moderate intensity, 3–4 times/wk) for 4 mo	Plasma leptin ↓ independently of changes in plasma insulin levels and percent body fat
Thong et al (2000)[164]	Obese men	N = 52 Age = 42–47 y	12 wk of weight loss program and exercise	No change in circulating leptin without weight loss. Circulating leptin ↓ with weight loss
Hayase et al (2002)[155]	Premenopausal and postmenopausal female volunteers	N = 18 (9 postmenopausal women, 9 volunteers) Age = 36–58 y	Aqua exercise (2 times/wk) plus resistance exercise (1 time/wk) for 10 wk	Plasma leptin ↓ in both groups

Abbreviations and symbols: BMI, body mass index; HR_{max}, maximal heart rate; MIE, moderate-intensity exercise; VO_{2max}, maximum oxygen consumption; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2.
[b]VO_{2max} shown in mL/kg/min.

without concurrent resistance training. Interestingly, the only study that investigated the effect of intensive training (endurance and interval training) found no changes in leptin levels, either at rest or after maximal exercise.[161] However, this study has limited generalizability because it investigated adolescent female runners.

Vatansever-Ozen et al[70] found a reduction in fasting leptin concentrations after aerobic exercise (105 min at 50% of VO_{2max}, followed by 15 min at 70% of VO_{2max}) in healthy participants. However, this change in leptin concentrations would likely increase the feeling of hunger in response to the aerobic exercise. Indeed, in individuals with obesity, Martins et al[128] points out that exercise-induced weight loss not only causes changes in fasting leptin levels but also results in changes in postprandial leptin levels that actually favor food intake. When aerobic training in obese individuals was investigated, leptin levels after a 12-week exercise intervention were significantly decreased on an empty stomach and after the ingestion of a standardized meal, leading to a weight loss of approximately 3.5 kg, from 96.1 ± 11.0 to 92.6 ± 11.7 kg (Table 11[75,115,165–187]). Thus, the reduction in leptin concentrations after chronic aerobic exercise may reflect an improved action of leptin, ie, sensitivity.

Oxyntomodulin response

Oxyntomodulin is a peptide hormone produced by the L cells in the small intestine. In humans, it leads to reduced food consumption and increased energy expenditure.[153,188] It is one of a group of gut hormones that decreases stomach acid and alters the emptying of stomach contents in rodents.[189] The effect of oxyntomodulin on energy expenditure in humans has not been explored thoroughly. Nonetheless, Wynne et al[190] suggest that oxyntomodulin does not alter resting energy expenditure but instead increases activity-related energy expenditure. This hypothesis needs further investigation.

Elevated oxyntomodulin levels can cause body weight loss in humans.[191,192] For example, Wynne et al[191] observed a weight loss of 2.3 kg in overweight and obese individuals who received subcutaneous administration of oxyntomodulin 3 times daily (400 nmol preprandially) over a 4-week period.

Interestingly, oxyntomodulin was recently shown to alter glucose metabolism in humans. Shankar et al[193] performed a randomized, double-blind, placebo-controlled crossover trial in which patients with type 2 diabetes mellitus were given oxyntomodulin as an infusion (3 pmol/kg/min). Following the infusion, patients showed improved insulin secretion and oxyntomodulin levels sufficient to induce glucose lowering.

Liu et al[194] explored the chronic effect of oxyntomodulin, administered as an infusion, in lean rats and assessed the antiobesity potential of oxyntomodulin in mice. They found improved energy expenditure in obese rodents, suggesting that long-acting oxyntomodulin analogs may have potential as a novel therapy to prevent and to treat obesity in humans. Nevertheless, far more work is necessary before these pharmaceutical interventions could be pursued on a regular clinical basis.

CONCLUSION

In summary, studies have reported decreases in ghrelin concentrations in response to acute aerobic exercise. Chronic aerobic exercise training or resistance programs resulted in unchanged or slightly increased ghrelin levels in obese individuals who lost weight. Levels of GLP-1 are found to increase in response to a single bout of exercise in normal-weight individuals, while results for the response to longer-term training are sparse and equivocal. For example, some studies have found aerobic or moderate-intensity training to induce slightly elevated levels of both ghrelin and GLP-1 in normal-weight individuals, while other studies have reported no changes in hormone levels in overweight or obese individuals. However, emergent data about low-volume sprint interval exercise show increased GLP-1 levels in overweight men and women. Results for peptide YY levels are similar, suggesting that low- to moderate-intensity exercise results in slightly higher or unchanged levels, while no data for intensive training in obese or overweight individuals are available. As for pancreatic polypeptide and cholecystokinin levels, changes in response to training are inconsistent, and studies have been conducted only in normal-weight and overweight individuals. Table 12 summarizes the research on the effects of exercise and training on the various hormones that affect appetite.

The response of most of these hormones to exercise and training is dependent on exercise intensity in normal-weight and overweight individuals, but findings in persons with obesity are limited in number and, to some degree, contradictory. Moreover, while some of these hormones, such as leptin, have been studied extensively, most have not been investigated in systematic, high-level work. Hence, further research is required to better understand the effect of these hormones on appetite and hunger suppression in individuals with obesity or morbid obesity to confirm the effectiveness of exercise and training. Additionally, research is needed to elucidate the impact of various forms of exercise that

Table 11 **Studies examining the effect of acute exercise on leptin concentrations**

Reference	Population[a]	No. and age of participants	Intervention	Results
Yi et al (2013)[165]	Male Sprague Dawley rats with type 2 diabetes (15-mo old; 450–470 g)	N = not reported Age = 15 mo	Acute exercise (two 90-min sessions with a 45-min interval between sessions)	Acute exercise activated the leptin signaling pathway leptin-AMPK-ACC
Guerra et al (2011)[166]	Healthy young men	N = 15 Age = 21–25 y	30-s sprint exercise (Wingate test)	Sprint is a leptin signaling mimetic in human skeletal muscle
Landt et al (1997)[167]	Men who fasted overnight and then pedaled a stationary ergometer for 2 h and 14 nonfasting ultramarathon runners	N = 14 Age = 28–32 y	Stationary ergometer for 2 h or prolonged exercise of an ultramarathon	Serum leptin concentrations ↓
Weltman et al (2000)[168]	Healthy young men	N = 7 Age = 25–29 y	30 min of exercise at various intensities and caloric expenditures	No change in leptin levels during exercise or during recovery (3.5 h)
Khodamoradi et al (2011)[169]	Healthy male volunteers	N = 15 Age = 22–31 y	3 resistance exercise trials at an intensity corresponding to 30%, 55%, and 80% of 1RM	Serum leptin concentrations ↓
Bouassida et al (2004)[170]	Physically active men and women	N = 17 (5 men, 12 women) Age = 21–25 y	45 s of supramaximal exercise at 120% of peak aerobic power	No change in plasma leptin concentrations
Torjman et al (1999)[171]	Healthy untrained men	N = 6 Age = 18–45 y	60 min of treadmill exercise at 50% of VO_{2max}	No change in leptin during a 4-h recovery period
Zoladz et al (2005)[75]	Men BMI = 22.42 ± 0.49 VO_{2max} = 51.6 ± 1.5	N = 8 Age = 22–24 y	2 incremental exercises: a maximal incremental test performed in fed state and a submaximal incremental test up to 150 W performed in fasted state	No change in leptin
Essig et al (2000)[172]	Trained men	N = 11 Age = 21–44 y	2 separate exercise tests: 800- and 1500-kcal treadmill runs	Plasma leptin ↓
Olive et al (2001)[173]	Trained men	N = 9 Age = 22–33 y	60 min of running at 70% of VO_{2max}	Leptin ↓ immediately after exercise and continued to ↓ during recovery, ie, at 24 h and 48 h after exercise
Kraemer et al (1999)[174]	Postmenopausal women	N = 30 Age = 46–56 y	30 min of exercise at 80% of VO_{2max}	Leptin ↓
Nindl et al (2002)[175]	Men	N = 10 Age = 20–22 y	50 sets of resistance exercise: 15 sets of squats, 15 sets of bench press, 10 sets of leg press, 10 lat pulldowns	Leptin ↓ 9 h, 12 h, and 13 h after exercise
Zafeiridis et al (2003)[176]	Young lean men	N = 10 Age = 19–27 y	Maximum strength, muscular hypertrophy, and resistance exercise protocols	Leptin concentrations ↓ 30-min into recovery after exercise protocols
Leal-Cerro et al (1998)[177]	Trained male athletes (marathon runners) and nonobese sedentary men	N = 51 (29 athletes, 22 sedentary men) Age = 35–39 y	42-km marathon	Leptin ↓
Karamouzis et al (2002)[178]	Long-distance swimmers	N = 16 Age = 18–45 y	12-km swim	Leptin ↓
Zaccaria et al (2002)[179]	Males	N = 45 Age = 32–50 y	3 competitive endurance races: a half-marathon run, a ski-alpinism race, and an ultramarathon race	Circulating serum leptin ↓ only after prolonged endurance exercises like the ski-alpinism and ultramarathon races

(continued)

Table 11 **Continued**

Reference	Population[a]	No. and age of participants	Intervention	Results
Sliwowski et al (2001)[115]	Normal-weight men	N = 19 Age = 20–24 y	Treadmill run to exhaustion in fasted or fed state (5 min after a liquid meal)	Leptin ↓ after meal
Ferguson et al (2004)[180]	Normal-weight male and female volunteers	N = 16 Age = 25–31 y	60-min exercise (cycle ergometer) at 65% of VO_{2max} vs resting	No change in plasma leptin
Jürimäe et al (2006)[181]	College-level rowers	N = 8 Age = 17–26 y	Rowing for 6.5 km at individual anaerobic threshold	No change in leptin immediately after exercise. Leptin ↓ after 30 min of recovery
Jürimäe et al (2009)[184]	Highly trained male rowers	N = 9 Age = 18–22 y	Rowing training session of about 2 h vs resting	Leptin was ↓ 30 min post exercise
Jürimäe et al (2007)[182]	Highly trained male rowers	N = 9 Age = 16–24 y	Single scull rowing performed below and above individual anaerobic threshold	No change in leptin
Jürimäe et al (2007)[183]	Elite male rowers	N = 8 Age = 19–26 y	Maximal 6000-m rowing ergometer test	Leptin ↓ immediately after exercise and remained significantly decreased after the first 30 min of recovery
Kyriazis et al (2007)[185]	Healthy young obese men	N = 15 Age = 23–27 y	Single exercise session of moderate intensity (58.4% of VO_2max) for 60 min	No change in plasma leptin
Racette et al (1997)[186]	Sedentary male volunteers (included lean and obese participants)	N = 5 Age = 36–41 y	Moderate-intensity cycle ergometer exercise (50% of HR_{max})	No change in plasma leptin or in leptin production
Tuominen et al (1997)[187]	Healthy men	N = 26 Age = 30–34 y	Glycogen depletion, tested using an insulin clamp 4 h after a 2-h treadmill exercise at an intensity of 75% of VO_{2max}	Serum leptin correlated directly with serum insulin, cortisol, and triglyceride and inversely with growth hormone concentrations. It is ↓ by glycogen-depleting exercise and is ↑ during hyperinsulinemic clamp

Abbreviations and symbols: AMPK-ACC, AMP-activated protein kinase–acetyl CoA carboxylase; BMI, body mass index; VO_{2max}, maximum oxygen consumption; 1RM, 1 rep maximum; ↑, increased; ↓, decreased.
[a]BMI shown in kg/m^2; VO_{2max} shown in mL/kg/min.

Table 12 **Assessment of the strength of the evidence for an effect of physical training on each hormone (eg, strong, limited, weak, indeterminate, or insufficient data)**

Hormone	Effect of physical training	
	Aerobic training[a]	Anaerobic training[b]
Ghrelin	Indeterminate	Insufficient data
Glucagon-like peptide 1	Insufficient data	Insufficient data
Peptide YY	Indeterminate	Insufficient data
Pancreatic polypeptide	Insufficient data	Insufficient data
Cholecystokinin	Insufficient data	Insufficient data
Leptin	Indeterminate	Insufficient data
Oxyntomodulin	Insufficient data	Insufficient data

[a]Includes endurance training (low to moderate intensity).
[b]Includes high-intensity interval training, strength training, and sprint training.

have recently engaged the public interest, such as high-intensity interval training (HIIT) or concurrent aerobic exercise and resistance training. Finally, much of the available research on exercise and hormones tends to assume that men and women have similar physiological responses.[195] A substantial body of evidence indicates this is not the case, and single-sex–based investigative work on exercise and gut hormones is needed.

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