Original Article

Effects of Short-term Resistance Training on Serum Leptin Levels in Obese Adolescents

Patrick W.C. Lau[1], Zhaowei Kong[2], Choung-rak Choi[3], Clare C.W. Yu[4], Dorothy F.Y. Chan[5], Rita Y.T. Sung[5], Beeto W.C. Leung[6]

[1]*Department of Physical Education, Hong Kong Baptist University, HONG KONG*
[2]*Faculty of Education, University of Macau, MACAU*
[3]*Sports Management, Division of Sport Science, College of Natural Science, Konkuk University, KOREA*
[4]*Institute of Human Performance, University of Hong Kong, HONG KONG*
[5]*Department of Pediatrics, The Chinese University of Hong Kong, HONG KONG*
[6]*Department of Psychology, University of Hong Kong, HONG KONG*

The purpose of this study was to examine the effects of a short-term resistance training program on serum leptin concentrations in obese adolescents. Eighteen Chinese obese adolescents participated in the supervised weight management program. Resistance training was conducted three times a week on alternate days for 6 weeks. Body composition [body fat mass (FM) and body lean mass (LM)] determined by dual-energy X-ray absorptiometry, muscle strength and leptin, insulin and glucose were measured before and after training. Subjects demonstrated significantly improved strength of leg, chest and bicep under conditions of stability in weight, FM and LM. There were positive correlations between leptin and body mass index, FM, %FM, waist girth and hip girth, and negative correlations between leptin and %LM at the resting state before and after 6 weeks of resistance training. No significant relationship was found between leptin concentration and the parameters of muscular strength or concentrations of insulin and glucose. Compared to pre-training values, serum leptin levels were not significantly altered, while relative leptin (leptin/FM) decreased significantly after the short-term resistance training. These results indicate that short-term resistance training enhances muscle strength but does not alter leptin levels in obese adolescents with weight and FM stability. The results also suggest that 6 weeks of resistance training decreases the requirement of leptin per unit of FM and improves leptin sensitivity in obese adolescents. [*J Exerc Sci Fit* • Vol 8 • No 1 • 54–60 • 2010]

Keywords: adolescent obesity, leptin, resistance training

Introduction

Obesity is a chronic disease and is posing a growing threat to public health in developed and developing countries. There are 25% of children who are overweight and 11% who are obese in the USA, 28.9% who are overweight and 12–36% who are obese in China, 26.5% who are overweight in Taiwan, 15.6% who are overweight in Thailand, 15.1% who are overweight in Korea, and 11% who are overweight in Japan (Gill 2006; Lou 2002). Worldwide, there are 155 million children aged 5–17 who are overweight (International Obesity Taskforce 2008), while in Hong Kong, 21.3% of primary school children are overweight (Department of Health 2008). Obese children and adolescents exhibit a significantly higher risk of a broad range of illnesses when they become adults than do adolescents of normal weight (Freedman et al. 2005). Therefore, early interventions

Corresponding Author
Choung-rak Choi, Sports Management, Division of Sport Science, College of Natural Science, Konkuk University, Danwol-dong, Chungju-city, Chungbuk, KOREA.
E-mail: spoman@kku.ac.kr

in childhood and adolescence may decrease the risk of obesity-associated complications that are apparent in adults.

Leptin, the product of the *ob* gene and secreted from adipose tissue, not only assists in the regulation of body weight and energy intake and expenditure (Zhang & Scarpace 2006), but also plays a multifunctional role related to the neuroendocrine system (Tam et al. 2009), immunity (Demas & Sakaria 2005), and reproduction (Zieba et al. 2005). Leptin concentrations are associated with adipose content, and changes in fat mass (FM) resulting from weight loss are known to be associated with a reduction in circulating leptin (Pérusse et al. 1997; Trevaskis & Butler 2005).

From the literature, most studies have investigated the effects of aerobic activity on serum leptin. It is generally agreed that serum leptin remains relatively unchanged or is reduced at extreme exercise conditions after a single bout of aerobic exercise (Sari et al. 2007; Pérusse et al. 1997). High intensity physical activity may lead to a decrease in leptin concentrations because of weight reduction and fat loss in obese men (Pasman et al. 1998; Pérusse et al. 1997), women (Polak et al. 2006), and children (Faigenbaum et al. 2002; Gutin et al. 1999), while generally acute exercise or relatively short-term training (<12 weeks) have no effect on leptin unless the training is associated with fat loss (Bouassida et al. 2008; Kraemer et al. 2002). These studies reported either no effect on leptin concentrations with short-term training, unless the training was associated with fat loss, or a reduction in leptin levels beyond that accounted for by reduction in FM in long-term training.

Though children and youth have traditionally been encouraged to participate in aerobic exercise, resistance training has proven to be a safe and effective method of conditioning for all youth regardless of body size (Malina 2006). Several studies have reported that resistance training programs lead to positive changes in body composition in obese children and adolescents (Shabi et al. 2006; Yu et al. 2005). Also, there is some evidence to indicate that aerobic training concurrent with a very-low-calorie-diet could cause a greater loss in fat-free mass than would occur with a very-low-calorie-diet alone (Hammer et al. 1989). Research in preventing and treating pediatric chronic diseases including obesity is limited and there are very few studies that have examined the effects of resistance exercise on leptin. Studies have shown that 6-week resistance training had no effects on leptin levels in healthy men (Ara et al. 2006) or in type 2 diabetic men and women (Kanaley et al. 2001), but 16 weeks of resistance training increased fat-free mass and decreased leptin concentrations in obese postmenopausal females (Ryan et al. 2000).

For obese children, few studies have examined the effect of resistance training on leptin. Therefore, the purpose of this study was to investigate leptin response to resistance training. It is hypothesized that short-term resistance training cannot alter leptin levels in obese adolescents with weight and FM stability.

Methods

Participants

Eighteen Chinese overweight adolescents (5 girls, 13 boys), mean age 12.45±1.77 years, were recruited from a pediatric obesity clinic of a hospital. All subjects were obese according to the international age-related cut-off points for childhood obesity (Cole et al. 2000), and they had no previous resistance training experience. Both the subjects and their parents were informed about the nature of the project, and accepted for participation voluntarily. Informed consent was obtained from all the adolescents and their parents before the initial training.

Exercise prescription

Based on the previous finding of significant effects on body composition from a study on combined dietary and strength training intervention in obese children (Sung et al. 2002), the present study adopted 6 weeks as an appropriate duration of intervention. The 6-week summer vacation would also allow for total commitment from the participants to take part in resistance training without school activities making a demand on their time, and avoid having external factors like school extracurricular activities impacting on the results of the study intervention.

The training program comprised 1-hour sessions conducted three times a week on alternate days for 6 weeks. Under the direction of professional fitness instructors, all resistance training sessions took place in a weight training room. In addition to warm-up and cool-down exercises, 10 resistance training stations with machines were performed by the participants in each 1-hour session (Table 1). Three-set circuit training format was employed and three instructors provided individual help to the participants. A 3- to 5-minute break between sets of training was allowed for rest. The resistance exercises were tailor-made to each individual adolescent according to their fitness test results prior to the training.

One repetition maximum (1RM) was established in each movement by asking the participants to lift a load

Table 1. List of exercises, repetition, and relative loads of the resistance exercise protocol

Station	Set	Repetition of 75–85% 1RM
Chest press	3	5–8 reps
Lat pull down	3	5–8 reps
Shoulder press	2	5–8 reps
Leg press	2	5–8 reps
Leg extension	2	5–8 reps
Leg curl	2	5–8 reps
Heel raise	2	5–8 reps
Bicep curl	3	5–8 reps
Tricep extension	3	5–8 reps
Adjusted push-up	1	5–8 reps

1RM = 1 repetition maximum.

through a full range of motion. From our previous study on test–retest strength in 13 adolescents (5 boys, 8 girls) within 7 days (unpublished data), the correlation coefficients for leg strength, chest strength and bicep strength are near to or above 0.99. The information of 1RM served as the prescription basis for different exercise stations. Quantitatively, the resistance training level was set at 70–85% of 1RM (low repetition, heavy load training) depending on individual performance and progression. All training intensity (set, weight, repetitions) and stations were adjusted and modified according to each individual's progress during the training.

Body composition and blood samples

Body weight was measured to the nearest 0.1 kg using an electronic body weight scale (Seca 707; Seca GmbH & Co. KG., Hamburg, Germany). Height was measured to the nearest 0.5 cm on a Harpenden Statiometer (Holtain Ltd., Pembrokeshire, Wales, UK). Body composition was assessed by dual-energy X-ray absorptiometry (DEXA) using the Hologic QDR-4500 (Hologic Inc., Waltham, MA, USA). The total body scan provided values for bone mineral content, non-bone lean tissue, and FM in the whole body and in the arms, legs, trunk and head separately. Fat-free mass was defined as the sum of non-bone lean tissue and bone mineral content. Waist and hip circumferences were obtained in duplicate with a tape measure, and the waist-to-hip ratio was determined.

Resting blood samples were collected at the same times in the morning after a 12-hour overnight fast at baseline and 48 hours after the last training session of the 6-week resistance training intervention. Blood was collected in vacutainers, immediately placed on ice and centrifuged (at 4°C and 1,500*g* for 15 minutes). Leptin concentrations were measured in duplicate using a human leptin enzyme-linked immunosorbent assay (Diagnostic Systems Laboratories Inc., Webster, TX, USA) with an 8% intra-assay precision and a sensitivity of $0.5\,ng \cdot mL^{-1}$. Insulin concentrations were determined in duplicate using a radioimmunoassay (Pharmacia Diagnostics AB, Uppsala, Sweden). Both the inter-assay and intra-assay coefficients of variation for insulin were <5%. The lowest value of the insulin radioimmunoassay was $<15\,ng \cdot mL^{-1}$. Blood glucose was measured by the glucose oxidase method (Lau et al. 2004).

Statistical analysis

Results were expressed as mean ± standard deviation. Comparison of means between the protocols was performed by analysis of variance and Student's paired *t* tests for all dependent variables. To examine which factors were associated with leptin concentration (cross-sectional analyses), we calculated Pearson's correlation coefficients for leptin levels and the variables of body composition pre- and post-training. The level of significance was set at $p < 0.05$. All analyses were conducted using SPSS version 10.0 (SPSS Inc., Chicago, IL, USA).

Results

After short-term resistance training, the subjects demonstrated increases in height ($p < 0.01$) and waist and hip circumferences ($p < 0.05$); however, the effect was small (effect size < 0.2). A decreasing trend was found for serum leptin concentrations. There were no changes in body composition such as FM, lean mass and %FM after the 6-week resistance training, while leg, chest and bicep strength increased significantly (Table 2).

Compared to pre-training levels, relative leptin (leptin/FM) decreased significantly ($p < 0.05$) after the resistance training; the effect size was 0.67, which means "over than moderate" (Cohen 1992) (Figure).

Leptin concentrations were highly correlated with body mass index, FM and %FM ($r = 0.70$–0.91, $p < 0.01$), and moderately correlated with waist and hip circumferences. No significant relationship was found between leptin concentration and the parameters of muscular strength or insulin and glucose concentrations (Table 3).

Discussion

Six weeks of resistance training resulted in significant increases in leg, chest and bicep muscular strength, but the present study showed that short-term resistance

Table 2. Effects of a short-term resistance training intervention on leptin and strength

	Exercise		*p*	Effect size
	Pre	Post		*d* value
Height (cm)	154.1 ± 8.9	155.0 ± 8.8*	0.00	0.10
Weight (kg)	72.7 ± 15.3	73.3 ± 15.1	0.07	0.04
BMI ($kg \cdot m^{-2}$)	30.5 ± 4.9	30.4 ± 4.8	0.92	0.02
Leg strength (kg)	197.4 ± 44.1	285.8 ± 64.9*	0.00	1.59
Chest strength (kg)	54.5 ± 19.9	87.9 ± 23.2*	0.00	1.55
Biceps strength (kg)	4.7 ± 1.6	6.2 ± 1.7*	0.00	0.91
Waist circumference (cm)	93.0 ± 10.8	93.6 ± 11.5	0.40	0.05
Hip circumference (cm)	105.1 ± 10.5	107.0 ± 11.6†	0.05	0.17
WHR	0.9 ± 0.1	0.9 ± 0.1	0.63	0.00
BMC (kg)	15.8 ± 2.9	16.2 ± 3.0*	0.00	0.14
Lean mass (kg)	43.2 ± 8.9	43.4 ± 8.4	0.66	0.02
FM (kg)	29.4 ± 8.9	29.5 ± 8.8	0.61	0.01
%LM	58.6 ± 5.4	58.6 ± 5.7	0.98	0.00
%FM	39.3 ± 5.6	39.2 ± 5.9	0.91	0.02
Leptin ($ng \cdot mL^{-1}$)	34.8 ± 15.0	30.3 ± 11.8	0.09	0.33
Lep/FM ($ng \cdot mL^{-1} \cdot kg^{-1}$)	1.2 ± 0.3	1.0 ± 0.3†	0.04	0.67
Glucose ($mmol \cdot L^{-1}$)	4.6 ± 0.4	4.8 ± 0.4*	0.00	0.50
Insulin ($ng \cdot mL^{-1}$)	140.7 ± 39.8	129.2 ± 58.7	0.26	0.23

*$p < 0.01$; †$p < 0.05$. BMI = body mass index; WHR = waist-to-hip ratio; BMC = bone mineral content; FM = fat mass; %LM = percentage of lean mass; %FM = percentage of fat mass; Lep/FM = leptin concentration/fat mass.

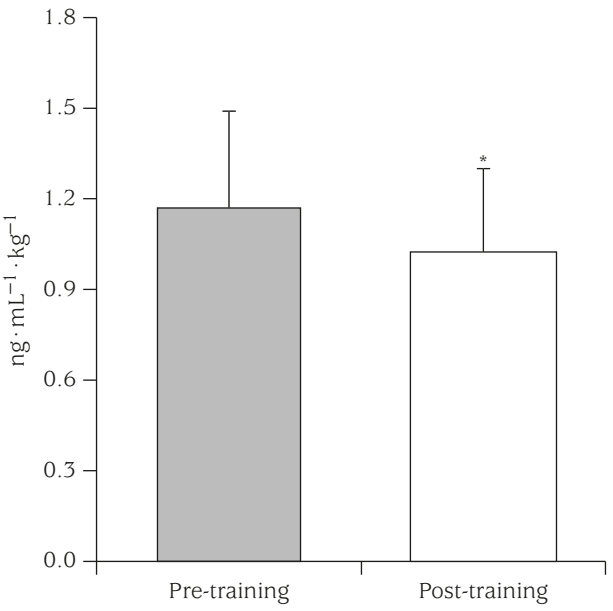

Fig. Decrease in leptin/fat mass after 6-week resistance training in 13 obese adolescents. *$p < 0.05$.

training does not significantly alter absolute serum leptin concentrations despite a decreasing trend in obese adolescents with stability of weight and FM. This is the first study to evaluate circulating leptin responses in obese adolescents following resistance training without weight loss.

Table 3. Relationship between serum leptin levels and body composition pre- and post-resistance training in obese adolescents

	Pre-training		Post-training	
	r	*p*	*r*	*p*
BMI	0.76*	0.00	0.74*	0.00
BMC	−0.05	0.85	−0.02	0.94
FM	0.80*	0.00	0.70*	0.00
LM	0.03	0.90	0.08	0.75
%FM	0.91*	0.00	0.80*	0.00
%LM	−0.91*	0.00	−0.79*	0.00
Waist circumference	0.52†	0.03	0.56†	0.02
Hip circumference	0.68*	0.00	0.54†	0.02
WHR	−0.15	0.57	0.19	0.46

*$p < 0.01$; †$p < 0.05$. BMI = body mass index; BMC = bone mineral content; FM = fat mass; LM = lean mass; %FM = percentage of fat mass; %LM = percentage of lean mass; WHR = waist-to-hip ratio.

Leptin is believed to play a crucial role in energy balance (Campfield et al. 1995). Many previous studies have discussed the effects of regular physical activity on leptin concentrations (Ara et al. 2006; Polak et al. 2006; Fatouros et al. 2005; Pasman et al. 1998; Pérusse et al. 1997), but uncertainty remains as to how circulating leptin reacts to exercise training since previous studies have produced inconsistent results. It is likely

that differences in the populations investigated (men, women, trained, untrained), the training protocols used (format, intensity, volume, duration), and participants' status and energy balance might be contributing to these conflicting results on the relationship between leptin levels and exercise (Bouassida et al. 2008; Kraemer et al. 2002).

The present investigation, which evaluated the effects of resistance training, differs from previous studies that have been on leptin in obese adolescents' weight loss. Resistance training may be advantageous during periods of severe energy restriction as it has been shown to have a low metabolic cost, to create a smaller energy deficit than aerobic training and, most importantly, to preserve fat-free mass. Among the limited number of studies, the impact of acute resistance exercise and several weeks of strength training on leptin in adults are controversial (Rahmani-Nia & Hodjati 2008; Ara et al. 2006; Fatouros et al. 2005; Zefeiridis et al. 2003; Nindl et al. 2002; Kanaley et al. 2001). In accordance with our results, Ara et al. (2006) and Kanaley et al. (2001) did not observe significant changes in serum leptin levels following 6 weeks of resistance training in young or middle-aged men. However, changes in body FM were different with strength training in these two studies: one found no reduction (Kannaley et al. 2001) while the other observed a 7% reduction in FM. Two studies have examined the effects of chronic resistance training on leptin. Ryan et al. (2000) studied the effects of 16 weeks of resistance training in obese postmenopausal females and found that not every one lost weight. Leptin concentrations declined by 36% in the group who lost weight, while the resistance training increased fat-free mass. Fatouros et al. (2005), in a randomized controlled trial, investigated leptin responses in 50 inactive old men following a 1-year exercise training and detraining. Leptin changes were strongly associated with resting metabolic rate and anthropometric changes in body mass index and waist-to-hip ratio. Percentage of leptin decrease was associated with percentage of body mass index decrease and percentage of resting metabolic rate increase. Based on the aforementioned findings, we speculate that 6 weeks of resistance training may be the crossroads to change resting leptin concentrations. Whether or not there is a significant change in circulating leptin depends, just as with aerobic exercise, on the subjects investigated, resistance training protocols used, and participants' status and energy balance. The significant effect of resistance training on reducing leptin secretion will not occur until training accumulation reaches a certain point.

It is well known that leptin concentration increases with weight gain (Aas et al. 2009) and decreases with weight loss (Gutin et al. 1999). The mechanism responsible for the maintenance of serum leptin in obese adolescents is possibly unchanged resting insulin. Secretion of leptin by an enlarged store of adipose tissue may cause insulin resistance because of the insulin-antagonizing effects of leptin (Cohen et al. 1996). Unlike Ahmadizad et al. (2007) who reported that 12 weeks of resistance or endurance training increased insulin sensitivity in healthy males without weight loss, we did not find any improvement in insulin sensitivity in the present study.

The observation that there is no significant change in circulating leptin levels but significant reduction in leptin per unit of FM between pre- and post-training suggests that the lower concentrations of leptin normalized to FM observed after 6 weeks of resistance training are independent of fat loss. Such a discrepancy has been reported in a study of 12 obese males after 3 months of dynamic strength training (Klimcakova et al. 2006). It is reasonable to conclude that the reduction in leptin/FM is not due to the enhancement of androgen induced by heavy resistance–low repletion training and/or maturation even though serum testosterone concentrations were not measured in the present study. Data from previous investigations have shown that higher intensity exercises, running or resistance can increase testosterone concentrations in adults (Gorostiaga et al. 2004). Maturation and/or training-induced gains in muscular strength should not be attributed to increased androgen and suppressed leptin secretion. The gains in muscular strength during childhood have been attributed primarily to neuromuscular adaptations as opposed to hypertrophic factors (Sale 1989). Although not assessed in this study, increases in motor unit activation and improvements in motor skill coordination have been suggested as possible mechanisms by which participants increase their muscular strength in response to resistance training (Ozmun et al. 1994).

A possible explanation for the decrease in circulating leptin per unit of FM could be its enhanced removal from the blood, and the lack of effect on FM might be a regulation at the level of leptin secretion or protein turnover (Klimcakova et al. 2006). Relatively low circulating leptin might result from the pre-training-induced higher sensitivity of leptin. Leptin suppression with resting energy expenditure elevation is not due to exercise but rather the energy deficit induced by the excess post oxygen consumption of acute resistance training. Human obesity is associated with apparent resistance to leptin

with high circulating concentrations of leptin in obese subjects (Venner et al. 2006; Pérusse et al. 1997). Lower leptin per unit of FM with similar fatness means that 6 weeks of negative diet balance and physical activity can decrease the requirement of leptin per unit of FM. The mechanism by which short-term resistance training influences relative leptin concentrations is open to further investigation.

There were some limitations to the present study. First, there was no control group without any intervention or diet alone. Therefore, we cannot exclude the possibility that diet alone might have similar effects on serum leptin levels and body composition in obese adolescents. Second, because the sample size was small, the changes in absolute serum leptin in this study were not statistically significant. Further studies with larger sample sizes are needed on changes in serum leptin, strength and body composition induced by resistance training and hypocaloric diet.

In summary, to the best of our knowledge, this is the first prospective study to examine changes in circulating leptin due to short-term resistance training in obese adolescents. We have shown that 6 weeks of resistance training is effective in elevating muscular strength but does not change resting serum leptin levels, while it does decrease circulating leptin per unit of FM in obese adolescents in conditions of weight and FM stability. These results suggest that 6 weeks of resistance training decreases the requirement of leptin per unit of FM and improves leptin sensitivity in obese adolescents. A longer and more sustained exercise intervention program with different intervention modes should be explored.

Acknowledgments

We thank Dr. Amy F.Y. Lo (a pediatrian in the Department of Paediatrics, The Chinese University of Hong Kong, Hong Kong SAR) for her invaluable contribution to the subject recruitment process.

References

Aas AM, Hanssen KF, Berg JP, Thorsby PM, Birkeland KI (2009). Insulin-stimulated increase in serum leptin levels precedes and correlates with weight gain during insulin therapy in type 2 diabetes. *J Clin Endocrinol Metab* 94:2900–6.

Ahmadizad S, Haghighi AH, Hamedinia MR (2007). Effects of resistance versus endurance training on serum adiponectin and insulin resistance index. *Eur J Endocrinol* 157:625–31.

Ara I, Prerez-Gomez J, Vicente-Rodriguez G, Chavarren J, Dorado C, Calbet JÁ (2006). Serum free testosterone, leptin and soluble leptin receptor changes in a 6-week strength-training programme. *Br J Nutr* 96:1053–9.

Bouassida A, Chamari K, Zaouali M, Feki Y, Zbidi A, Tabka Z (2008). Review on leptin and adiponectin responses and adaptations to acute and chronic exercise. *Br J Sports Med* Oct 16. [Epub ahead of print]

Campfield LA, Smith FJ, Guisez Y, Devos R, Burn P (1995). Recombinant mouse OB protein: evidence for a peripheral signal liking adiposity and central networks. *Science* 269:546–9.

Cohen B, Novick D, Rubinstein M (1996). Modulation of insulin activities by leptin. *Science* 274:1185–8.

Cohen J (1992). A power primer. *Psychol Bull* 112:155–9.

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 320:1240–3.

Demas GE, Sakaria S (2005). Leptin regulates energetic tradeoffs between body fat and humoural immunity. *Proc Biol Sci* 272:1845–50.

Department of Health (2008). *Childhood Obesity.* Available from http://www.dh.gov.hk/ [Date accessed: June 20, 2008]

Faigenbaum AD, Milliken LA, Loud RL, Burak BT, Doherty CL, Westcott WL (2002). Comparison of 1 and 2 days per week of strength training in children. *Res Q Exerc Sport* 73:416–24.

Fatouros IG, Tournis S, Leontsini D, Jamurtas AZ, Sxina M, Thomakos P, Manousaki M, Douroudos I, Taxildaris K, Mitrakou A (2005). Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related. *J Clin Endocrinol Metab* 90:5970–7.

Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS (2005). The relation of childhood BMI to adult adiposity: The Bogalusa Heart Study. *Pediatrics* 115:22–7.

Gill T (2006). Epidemiology and health impact of obesity: an Asia Pacific perspective. *Asia Pac J Clin Nutr* 15 Suppl:3–14.

Gorostiaga EM, Izquierdo M, Ruesta M, Iribarren J, Gonzalez-Badillo JJ, Ibanez J (2004). Strength training effects on physical performance and serum hormones in young soccer players. *Eur J Appl Physiol* 91:698–707.

Gutin B, Ramsey L, Barbeau P, Cannady W, Ferguson M, Litaker M, Owens S (1999). Plasma leptin concentrations in obese children: changes during 4-month periods with and without physical training. *Am J Clin Nutr* 69:388–94.

Hammer RL, Barrier CA, Roundy ES, Bradford JM, Fisher AG (1989). Calorie-restricted low-fat diet and exercise in obese women. *Am J Clin Nutr* 49:77–85.

International Obesity Taskforce (2008). *Childhood Obesity*. Available from http://www.iotf.org/childhoodobesity.asp [Date accessed: June 18, 2008]

Kanaley JA, Fenicchia LM, Miller CS, Ploutz-Synder LL, Weinstock RS, Carhart R, Azevedo JL Jr (2001). Resting leptin responses to acute and chronic resistance training in type 2 diabetic males and females. *Int J Obes* 25:1474–80.

Klimcakova E, Polak J, Moro C, Hejnova J, Majercik M, Viguerie N, Berlan M, Langin D, Stich V (2006). Dynamic strength training improves insulin sensitivity without altering plasma levels and gene expression of adipokines in subcutaneous adipose tissue in obese men. *J Clin Endocrinol Metab* 91:5107–12.

Kraemer RR, Chu H, Castracane VD (2002). Leptin and exercise. *Exp Biol Med* 227:701–8.

Lau WC, Yu CW, Lee A, Sung YT (2004). The physiological and psychological effects of resistance training on Chinese obese adolescents. *J Exerc Sci Fit* 2:115–20.

Lou Z (2002). Obesity: a warning to Chinese children. *Beijing Rev* 45: 14–6.

Malina RM (2006). Weight training in youth-growth, maturation and safety: an evidence-based review. *Clin J Sport Med* 16:478–87.

Nindl BC, Kraemer WJ, Arciero PJ, Samatallee N, Leone CD, Mayo MF, Hafeman DL (2002). Leptin concentrations experience a delayed reduction after resistance exercise in men. *Med Sci Sports Exerc* 34: 608–13.

Ozmun JC, Mikesky AE, Surburg PR (1994). Neuromuscular adaptations following prepubescent strength training. *Med Sci Sports Exerc* 26: 510–4.

Pasman WJ, Westerterp-Plantenga MS, Saris WH (1998). The effects of exercise training on leptin levels in obese males. *Am J Physiol* 274: E280–6.

Pérusse L, Collier G, Gagnon J, Leon AS, Rao DC, Skinner JS, Wilmore JH, Nadeau A, Zimmet PZ, Bouchard C (1997). Acute and chronic effects of exercise on leptin levels in humans. *J Appl Physiol* 83:5–10.

Polak J, Klimcakova E, Moro C, Viguerie N, Berlan M, Hejnova J, Richterova B, Kraus I, Langin D, Stich V (2006). Effect of aerobic training on plasma levels and subcutaneous abdominal adipose tissue gene expression of adiponectin, leptin, interleukin 6, and tumor necrosis factor α in obese women. *Metabolism* 55:1375–81.

Rahmani-Nia F, Hodjati Z (2008). Acute effects of aerobic and resistance exercises on serum leptin and risk factors for coronary heart disease in obese females. *Sport Sci Health* 2:118–28.

Ryan AS, Pratley RE, Goldberg AP (2000). Changes in plasma leptin and insulin action with resistive training in postmenopausal females. *Int J Obes Relat Metab Disord* 24:27–32.

Sale D (1989). Strength training in children. In: Gisolfi CV (Ed.), *Perspectives in Exercise Science and Sports Medicine*. Benchmark Press, Indianapolis, Indiana, pp. 165–216.

Sari R, Balci MK, Balci N, Karayalcin U (2007). Acute effect of exercise on plasma leptin level and insulin resistance in obese women with stable caloric intake. *Endocr Res* 32:9–17.

Shabi G, Cruz M, Ball G, Weigensberg M, Salem G, Crespo N, Goran M (2006). Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. *Med Sci Sports Exerc* 38:1208–15.

Sung RYT, Yu CW, Chang SKY, Mo SW, Woo KS, Lam CWK (2002). Effects of dietary intervention and strength training on blood lipid level in obese children. *Arch Dis Child* 86:407–10.

Tam J, Fukumura D, Jain RK (2009). A mathematical model of murine metabolic regulation by leptin: energy balance and defense of a stable body weight. *Cell Metab* 9:52–63.

Trevaskis JL, Butler AA (2005). Double leptin and melanocortin-4 receptor gene mutations have an additive effect on fat mass and are associated with reduced effects of leptin on weight loss and food intake. *Endocrinology* 146:4257–65.

Venner AA, Lyon ME, Doyle-Baker PK (2006). Leptin: a potential biomarker for childhood obesity? *Clin Biochem* 39:1047–56.

Yu C, Sung R, So R, Lui K, Lau W, Lam P, Lau E (2005). Effects of strength training on body composition and bone mineral content in children who are obese. *J Strength Cond Res* 19:667–72.

Zefeiridis A, Smilios I, Conisidine V, Tokmakidis SP (2003). Serum responses after acute resistance exercise protocols. *J Appl Physiol* 94:591–7.

Zhang Y, Scarpace PJ (2006). The role of leptin in leptin resistance and obesity. *Physiol Behav* 88:249–56.

Zieba D, Amstalden M, Williams G (2005). Regulatory roles of leptin in reproduction and metabolism: a comparative review. *Domest Anim Endocrinol* 29:166–85.