Eur J Appl Physiol (2006) 97: 122–126
DOI 10.1007/s00421-006-0169-x

ORIGINAL ARTICLE

A. Z. Jamurtas · V. Theocharis · G. Koukoulis
N. Stakias · I. G. Fatouros · D. Kouretas
Y. Koutedakis

The effects of acute exercise on serum adiponectin and resistin levels and their relation to insulin sensitivity in overweight males

Accepted: 21 February 2006 / Published online: 9 March 2006

Abstract The purpose of this study was to investigate the effects of a submaximal aerobic exercise bout on adiponectin and resistin levels as well as insulin sensitivity, until 48 h post-exercise in healthy overweight males. Nine subjects performed an exercise bout at an intensity corresponding to approximately 65% of their maximal oxygen consumption for 45 min. Adiponectin, resistin, cortisol, insulin, glucose and insulin sensitivity were measured prior to exercise, immediately after exercise as well as 24 and 48 h after exercise. Data were analyzed using repeated measures ANOVA while Pearson's correlations were performed to identify possible relationship among the assessed variables. There were no significant differences for adiponectin (μg ml^{-1}) [pre, 3.61(0.73); post, 3.15(0.43); 24 h, 3.15(0.81); 48 h, 3.37(0.76)] or resistin (ng ml^{-1}) [pre, 0.19(0.03); post, 0.13(0.03); 24 h, 0.23(0.04); 48 h, 0.23(0.03)] across time. Insulin sensitivity increased and insulin concentration decreased significantly only immediately after exercise. Furthermore, no significant correlations were observed among the variables assessed except for the expected between insulin level and insulin sensitivity. These results indicate that a submaximal aerobic workout does not result in significant changes in adiponectin and resistin up to 48 h post-exercise. Furthermore, it appears that adiponectin or resistin is not associated with insulin sensitivity.

Keywords Lipids · Metabolism · Fat · Carbohydrates · Training · Obesity · Diabetes

A. Z. Jamurtas (✉) · V. Theocharis · Y. Koutedakis
Department of Physical Education and Sport Sciences,
University of Thessaly, Karies, 42100, Trikala, Greece
E-mail: ajamurt@pe.uth.gr
Tel.: +30-243-1047054
Fax: +30-243-1047042

G. Koukoulis · N. Stakias
School of Medicine, University of Thessaly, Larissa, Greece

I. G. Fatouros
Department of Physical Education and Sports Science,
Democritus University of Thrace, Komotini, Greece

D. Kouretas
Department of Biochemistry and Biotechnology,
University of Thessaly, 42100, Larisa, Greece

Y. Koutedakis
School of Sport, Performing Arts and Leisure,
Wolverhampton University, Walshall, UK

Introduction

Adiponectin is a cytokine protein released by human adipose tissue, which modulates various biological functions and its levels exhibit an inverse relationship with insulin resistance (Hotta et al. 2000; Maeda et al. 2001; Weyer et al. 2001). In addition, modifications in body weight have been shown to influence the levels of adiponectin (Esposito et al. 2003; Hulver et al. 2002). These findings indicate that adiponectin may improve insulin sensitivity. Exercise has been shown to increase insulin sensitivity up to 48 h after an exercise session (reviewed in Borghouts and Keizer 2000). Research on the effects of acute exercise on adiponectin levels has produced conflicting results. There are studies which have reported no effect of acute exercise on adiponectin levels in healthy, normal weight individuals (Ferguson et al. 2004; Kraemer et al. 2003; Punyadeera et al. 2005). On the other hand, Jurimae et al. (2005) reported a significant decrease immediately following exercise and significant increase 30 min post-exercise in healthy normal weight individuals, whereas Kriketos et al. (2004) reported that very short exercise training (approximately 1 week) resulted in increased levels of adiponectin and insulin sensitivity in obese individuals without a concomitant body weight loss. It is worth mentioning, that none of the acute studies have examined the probable prolonged effect of exercise on adiponectin levels and its relationship with insulin sensitivity.

Resistin is a newly discovered hormone that is produced by white and brown adipose tissue (Adeghate

2004). Resistin is greatly increased in obesity and it is thought to regulate glucose homeostasis and act as a physiological antagonist to hepatic insulin action (Wolf 2004). Eventhough there are reports in the literature that relate resistin with obesity and diabetes (Steppan et al. 2001), there is a lack of information as to what the effects of acute exercise on resistin levels.

Therefore, the purpose of this study was to examine the effects of acute exercise on serum levels of adiponectin, resistin, and insulin sensitivity over a period of 48 h post-exercise in overweight individuals. Since it is well known that in obesity large decreases in insulin resistance are evident after acute and chronic exercise (Henriksen 2002) and considering the proposed role of adiponectin and resistin in insulin resistance we thought that by employing overweight individuals the probability to find significant alterations in the two hormones increases.

Methods

Subject characteristics

Nine young overweight males volunteered to participate in this study. A written informed consent was provided by all participants after the volunteers were informed of all risks, discomforts and benefits involved in the study. The procedures were in accordance with the Helsinki declaration of 1975 and approval was received for this study from Institutional Review Board. Inclusion criteria included (1) BMI levels in between 27 and 32 kg m^{-2}, (2) not participating in a structured exercise program for the last 6 months, (3) having stable weight (smaller deviation of 2 kg) over the past 6 months, and (4) not using tobacco products and alcohol consumption 2 days prior to data collection.

Experimental design

Body mass was measured to the nearest 0.5 kg (Seca 714, Seca Vogel & Halke GmbH & Co. KG, Hamburg, Germany) with subjects lightly dressed and barefooted. Standing height was measured to the nearest 0.5 cm (Seca 714, Seca Vogel & Halke GmbH & Co. KG, Hamburg, Germany). Waist-to-hip ratio and percentage body fat was calculated according to the published guidelines (ACSM guidelines 2000), i.e., from seven skinfold measures (average of two measurements of each site) using a Harpenden (HSK-BI, British Indicators, Ltd, West Sussex, UK) skinfold caliper. Subjects visited the lab where a maximal oxygen consumption (VO_{2max}) test on a cycle ergometer (Monark 834E, Sweden) was performed. The protocol began at 50 W and was increased by 25 W every 2 min until VO_{2max} was reached. Criteria used to determine VO_{2max} were (1) subject exhaustion, (2) a <2 ml kg^{-1} min^{-1} increase in VO_{2max} with an increase in work rate, (3) a respiratory exchange ratio greater than or equal to 1.1, (4) a heart rate within 10 bpm of the theoretical maximum heart rate (220-age). Respiratory gas variables were measured using a metabolic cart (Vmax29; Sensormedics, USA), which was calibrated before each test using standard gases of known concentration. Exercise heart rate was monitored by telemetry (Polar Tester, S610^{TM}, Electro Oy, Finland).

Exercise protocol

At least 5 days after the VO_{2max} test and within 14 days, subjects returned to the lab in the morning to perform a sub-maximal workout on the cycle ergometer. Subjects refrained from exercise 48 h prior to this visit and reported in a fasted state (8–10 h). Subjects exercised for 45 min at an intensity corresponding to 65% of VO_{2max} which was calculated from results obtained of the subjects' maximal exercise test. Expired gas samples were taken every 10 min to ensure the prescribed exercise intensity. Subjects consumed 400 ml of water prior to the beginning of exercise and consumed water ad libitum during exercise.

Dietary analysis

Subjects were instructed to follow their usual eating habits during the days of data collection. They were also asked to record their diet the day prior to exercise bout and for the next 2 days. Each subject had been provided with a written set of guidelines for monitoring dietary consumption and a record sheet for recording food intake. Diet records were analyzed using the computerized nutritional analysis system Science Fit Diet 200A (Sciencefit, Greece).

Blood collection and analysis

Post-exercise changes in plasma volume were computed based on hematocrit and hemoglobin as described (Dill and Costill 1974). The hematologic parameters were measured in a Sysmex K-1000 (TOA Electronics, Japan) autoanalyzer. Blood samples (10 ml) were collected prior to exercise, immediately after exercise as well as 24, and 48 h post-exercise. Blood was allowed to clot at room temperature for 30 min and centrifuged at 1,500 *g* to collect serum. The collected serum was stored in multiple aliquots for assessment of glucose, insulin, adiponectin, resistin, and cortisol. Insulin sensitivity was assessed utilizing the Homeostasis Model Assessment (HOMA-IR) method and calculated using the following formula: fasting plasma glucose (mg dl^{-1}) $\times$ fasting plasma insulin (μU ml^{-1}) $\times$ 405^{-1} (Matthews et al. 1985). Glucose was analyzed using the kit from Elitech Diagnostics (Sees, France). Insulin, adiponectin, resistin, and cortisol were analyzed with commercially available

ELISA kits (Phoenix Co., USA). The intra-assay coefficient of variation for glucose, insulin adiponectin, resistin, and cortisol were 5.1, 4.2, 5.4, 4.5, and 2.3%, respectively.

Statistical analysis

Data are presented as mean (SEM). The distribution of all dependent variables was examined by the Kolmogorov–Smirnov test and was found not to differ significantly from normal. Data were analyzed through ANOVA for repeated measurements.

When a significant main effect was observed, post hoc analysis was performed with planned contrasts to determine the location of the significant differences. Linear correlation analysis among the assessed variables was carried out by Pearson's product moment correlation coefficient. All statistics were performed utilizing the SPSS (version 10, Chicago, IL, USA). The level of significance was set at $P < 0.05$.

Results

The physical characteristics of the participants are shown in Table 1. The range of the VO_{2max} of the subjects was in between 29.1 and 39.1 ml kg^{-1} min^{-1} whereas the average value was 32.8 ml kg^{-1} min^{-1}. This value puts this group of individuals in the 10th percentile in the classification for maximal aerobic power for their age (ACSM 2000). The average cycling intensity was 64.1% of their VO_{2max}. Dietary analysis prior to exercise bout and for the next 2 days appears on Table 2.

Insulin level decreased and insulin sensitivity increased significantly post-exercise whereas glucose levels remained unaltered (Fig. 1). Exercise did not result in significant changes in adiponectin, resistin or cortisol concentrations (Fig. 2). None of the examined variables appeared significantly modified 24 and 48 h post-exercise. Table 3 shows that we did not find any significant correlation among variables except for between insulin levels and HOMA-IR ($r = 0.957$)

Table 1 Physical characteristics of the participants

Variable	Mean	SEM
Age (years)	31.6	1.7
Weight (kg)	95.6	4.1
Height (cm)	182	4
BMI (kg m^{-2})	28.9	0.5
Waist (cm)	110.0	1.1
Hip (cm)	102.6	0.9
W/H	1.08	0.01
Fat (%)	22.8	1.1
VO_{2max} (ml kg^{-1} min^{-1})	32.8	1.2

BMI body mass index, *W*/*H* waist-to-hip ratio

Discussion

The main finding of the present study is that acute exercise did not affect the levels of either adiponectin or resistin as well as there were no any significant correlation between these two hormones with insulin sensitivity either pre- or post-exercise.

Previous studies that examined the effects of acute exercise on adiponectin responses reported that trained rowers experienced a significant decrease in the levels of adiponectin immediately after an acute bout of maximal exercise that was followed by a significant increase 30 min post-exercise (Jurimae et al. 2005). Kriketos et al. (2004) reported a significant increase in adiponectin levels in obese sedentary males following a short-term exercise training program (approximately 1 week). On the contrary, Ferguson et al. (2004) reported no effect of a submaximal aerobic exercise bout on adiponectin concentration in young healthy normal weight individuals. Similarly, Kraemer et al. (2003) indicated that there was no significant increase in adiponectin levels in young healthy males following a heavy continuous or intermittent running exercise. Finally, Punyadeera et al. (2005) reported no significant changes in adiponectin levels during exercise or recovery in healthy normal-weight individuals.

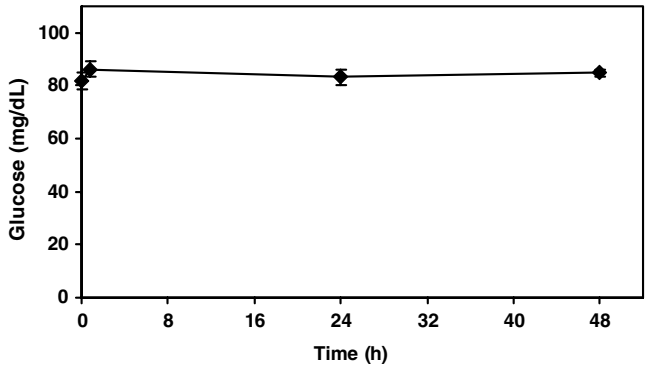
Training studies have shown that exercise might not be a significant factor towards adiponectin modifications (Marcell et al. 2005; Ryan et al. 2003). It seems that modifications in body weight or body composition might be responsible for alterations in adiponectin levels (Esposito et al. 2003; Hulver et al. 2002; Monzillo et al. 2003). Recent reports indicate that in young obese men, adiponectin levels are increased following an improvement of the body composition and this is more important than the way training is performed (Hara et al. 2005). The present study examined the acute effects of exercise where there was no evidence of body weight change and this could explain the lack of modifications in adiponectin levels.

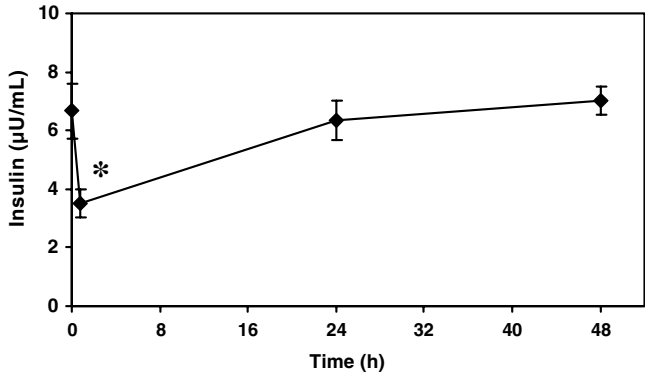
Resistin is an adipocyte-released hormone that has been positively correlated with several features of body composition and insulin resistance (Steppan et al. 2001). Nevertheless, the physiological role of resistin on obesity and insulin resistance is unclear, with some studies reporting a significant association between

Table 2 Dietary analysis prior to exercise bout and for the next two consecutive days

	Pre-exercise	24 h	48 h
Energy intake (MJ/day)	11.8 (1.3)	12.4 (1.9)	12.2 (1.7)
CHO (%)	49.6 (4.3)	47.5 (4.8)	45.2 (6.2)
Fat (%)	38.9 (2.9)	38.9 (2.3)	39.4 (3.4)
Protein (%)	13.5 (1.3)	13.6 (2.4)	15.4 (1.8)

Data presented as mean (*SEM*)
CHO carbohydrates





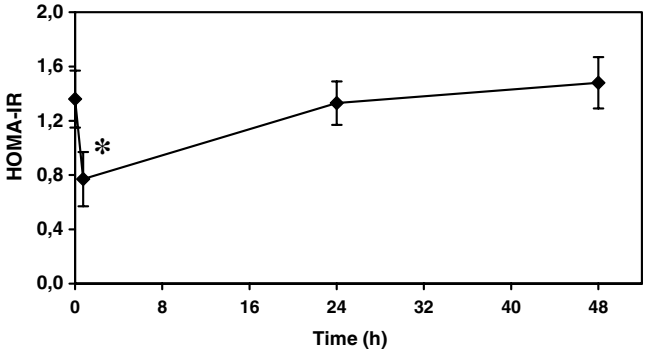
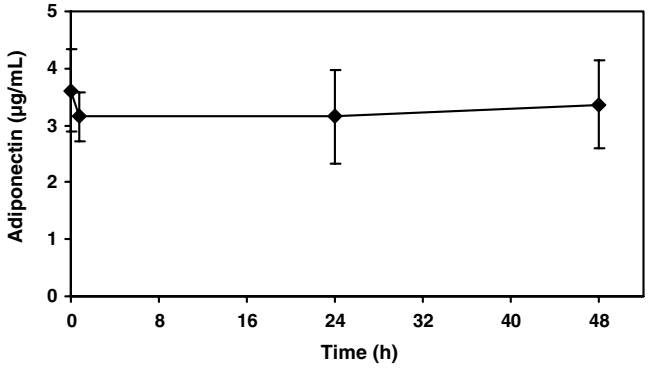
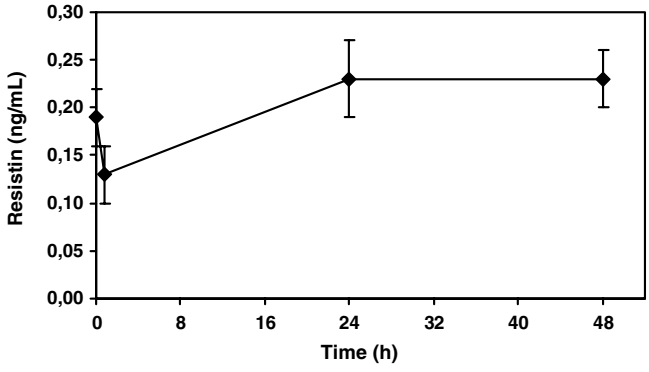


Fig. 1 The effect of exercise on glucose (**a**), insulin (**b**), and HOMA (**c**) levels. Data presented as mean (*SEM*). Asterisk indicates value significantly different compared to resting values





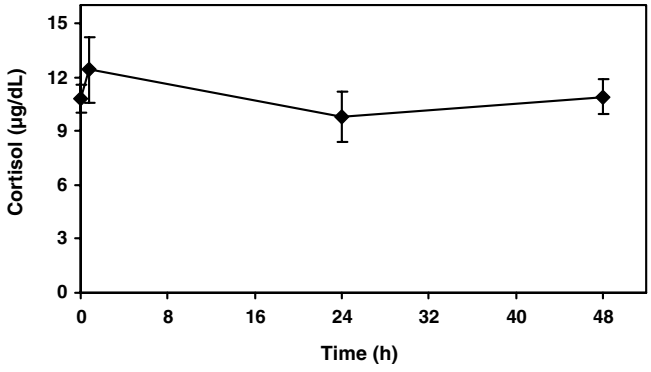


Fig. 2 The effect of exercise on adiponectin (**a**), resistin (**b**) and cortisol (**c**) levels. Data presented as mean (SEM)

resistin levels, obesity, and insulin resistance (Fujinami et al. 2004), whereas others do not (Lee et al. 2003). To our knowledge, this is the first study that examined the effects of acute exercise on resistin levels and reported relatively stable levels of this hormone after exercise. This finding is in accordance with the available chronic exercise studies that did not find alterations in the levels of the hormone after aerobic training for several months (Giannopoulou et al. 2005; Monzillo et al. 2003).

Table 3 Pearson's correlation coefficients at rest among insulin sensitivity, adiponectin, resistin, cortisol, and variables related to body composition

	HOMA-IR	Adiponectin	Resistin	Cortisol	Glucose	Insulin	W/H	Fat
HOMA-IR								
Adiponectin	0.357							
Resistin	−0.164	0.058						
Cortisol	0.463	−0.170	0.498					
Glucose	0.507	0.313	0.043	0.058				
Insulin	0.957*	0.286	−0.164	0.510	0.241			
W/H	0.237	−0.443	0.355	0.400	0.217	0.214		
Fat	0.061	−0.358	−0.628	−0.642	−0.118	0.115	0.184	
B*MI*	−0.351	0.313	−0.201	−0.454	−0.346	−0.233	0.296	0.575

BMI body mass index, *HOMA-IR* homeostasis model assessment, *W/H* waist-to-hip ratio

*Significant at $P < 0.001$

In conclusion, it appears that acute aerobic exercise does not result in immediate or prolonged alteration in adiponectin or resistin levels in healthy overweight individuals. Therefore, these two hormones may not be responsible for the well-known effect of acute exercise to decrease insulin resistance.

Acknowledgement This work was supported by a grant from the Gatorade Sports Science Institute.

References

Adeghate E (2004) An update on the biology and physiology of resistin. Cell Mol Life Sci 61(19–20):2485–2496

Borghouts LB, Keizer HA (2000) Exercise and insulin sensitivity: a review. Int J Sports Med 21:1–12

Dill DB, Costill DL (1974) Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. J Appl Physiol 37:247–248

Esposito K, Pontillo A, Di Palo C, Giugliano G, Masella M, Marfella R, Giugliano D (2003) Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. JAMA 289(14):1799–804

Ferguson MA, White LJ, McCoy S, Kim HW, Petty T, Wilsey J (2004) Plasma adiponectin response to acute exercise in healthy subjects. Eur J Appl Physiol 91(2–3):324–329

Fujinami A, Obayashi H, Ohta K, Ichimura T, Nishimura M, Matsui H, Kawahara Y, Yamazaki M, Ogata M, Hasegawa G, Nakamura N, Yoshikawa T, Nakano K, Ohta M (2004) Enzyme-linked immunosorbent assay for circulating human resistin: resistin concentration in normal subjects and patients with type 2 diabetes. Clin Chim Acta 339:57–63

Giannopoulou I, Fernhall B, Carhart R, Weinstock RS, Baynard T, Figueroa A, Kanaley JA (2005) Effects of diet and/or exercise on the adipocytokine and inflammatory cytokine levels of postmenopausal women with type 2 diabetes. Metabolism 54:866–875

Hara T, Fujiwara H, Nakao H, Mimura T, Yoshikawa T, Fujimoto S (2005) Body composition is related to increase in plasma adiponectin levels rather than training in young obese men. Eur J Appl Physiol 94(5–6):520–526

Henriksen EJ (2002) Invited review: effects of acute exercise and exercise training on insulin resistance. J Appl Physiol. 93(2):788–796

Hotta K, Funahashi T, Arita Y, Takahashi M, Matsuda M, Okamoto Y, Iwahashi H, Kuriyama H, Ouchi N, Maeda K, Nishida M, Kihara S, Sakai N, Nakajima T, Hasegawa K, Muraguchi M, Ohmoto Y, Nakamura T, Yamashita S, Hanafusa T, Matsuzawa Y (2000) Plasma concentration of a novel, adipose specific protein, adiponectin, in type 2 d, adipose-specific protein, adiponectin, in type 2 diabetic patients. Arterioscler Thromb Vasc Biol 20:1595–1599

Hulver MW, Zheng D, Tanner CJ, Houmard JA, Kraus WE, Slentz CA, Sinha MK, Pories WJ, MacDonald KG, Dohm GL (2002) Adiponectin is not altered with exercise training despite enhanced insulin action. Am J Physiol Endocrinol Metab 283(4):E861–E865

Jurimae J, Purge P, Jurimae T (2005) Adiponectin is altered after maximal exercise in highly trained male rowers. Eur J Appl Physiol 93(4):502–505

Kraemer RR, Aboudehen KS, Carruth AK, Durand RT, Acevedo EO, Hebert EP, Johnson LG, Castracane VD (2003) Adiponectin responses to continuous and progressively intense intermittent exercise. Med Sci Sports Exerc 35(8):1320–1325

Kriketos AD, Gan SK, Poynten AM, Furler SM, Chisholm DJ, Campbell LV (2004) Exercise increases adiponectin levels and insulin sensitivity in humans. Diabetes Care 27(2):629–630

Lee JH, Chan JL, Yiannakouris N, Kontogianni M, Estrada E, Seip R, Orlova C, Mantzoros CS (2003) Circulating resistin levels are not associated with obesity or insulin resistance in humans and are not regulated by fasting or leptin administration: cross-sectional and interventional studies in normal, insulin resistant, and diabetic subjects. J Clin Endocrinol Metab 88:4848–4856

Maeda N, Takahashi M, Funahashi T, Kihara S, Nishizawa H, Kishida K, Nagaretani H, Matsuda M, Komuro R, Ouchi N, Kuriyama H, Hotta K, Nakamura T, Shimomura I, Matsuzawa Y (2001) PPAR-gamma ligants increase expression and plasma concentrations of adiponectin, an adipose-derived protein. Diabetes 50:2094–2099

Marcell TJ, McAuley KA, Traustadottir T, Reaven PD (2005) Exercise training is not associated with improved levels of C-reactive protein or adiponectin. Metabolism 54(4):533–541

Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC (1985) Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 28(7):412–419

Monzillo LU, Hamdy O, Horton ES, Ledbury S, Mullooly C, Jarema C, Porter S, Ovalle K, Moussa A, Mantzoros CS (2003) Effect of lifestyle modification on adipokine levels in obese subjects with insulin resistance. Obes Res 11:1048–1054

Punyadeera C, Zorenc AH, Koopman R, McAinch AJ, Smit E, Manders R, Keizer HA, Cameron-Smith D, van Loon LJ (2005) The effects of exercise and adipose tissue lipolysis on plasma adiponectin concentration and adiponectin receptor expression in human skeletal muscle. Eur J Endocrinol 152(3):427–436

Ryan AS, Nicklas BJ, Berman DM, Elahi D (2003) Adiponectin levels do not change with moderate dietary induced weight loss and exercise in obese postmenopausal women. Int J Obes Relat Metab Disord 27(9):1066–1071

Steppan CM, Bailey ST, Bhat S, Brown EJ, Banerjee RR, Wright CM, Patel HR, Ahima RS, Lazar MA (2001) The hormone resistin links obesity to diabetes. Nature 409(6818):307–312

Weyer C, Funahashi T, Tanaka S, Hotta K, Matsuzawa Y, Pratley RE, Tataranni PA (2001) Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. J Clin Endocrinol Metab 86:1930–1935

Wolf G (2004) Insulin resistance and obesity: resistin, a hormone secreted by adipose tissue. Nutr Rev 62 (10):389–394

Yamauchi T, Kamon J, Waki H, Terauchi Y, Kubota N, Hara K, Mori Y, Ide T, Murakami K, Tsuboyama-Kasaoka N, Ezaki O, Akanuma Y, Gavrilova O, Vinson C, Reitman ML, Kagechika H, Shudo K, Yoda M, Nakano Y, Tobe K, Nagai R, Kimura S, Tomita M, Froguel P, Kadowaki T (2001) The fat-derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity. Nat Med 7:941–946

Yang WS, Lee WJ, Funahashi T, Tanaka S, Matsuzawa Y, Chao CL, Chen CL, Tai TY, Chuang LM (2001) Weight reduction increase plasma levels of an adipose-derived anti-inflammatory protein, adiponectin. J Clin Endocrinol Metab 86:3815–3819