Eur J Appl Physiol (2011) 111:57–66
DOI 10.1007/s00421-010-1628-y

ORIGINAL ARTICLE

Effects of Tai Chi on adiponectin and glucose homeostasis in individuals with cardiovascular risk factors

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Accepted: 17 August 2010 / Published online: 1 September 2010

Abstract The aim of this study was to evaluate the acute effect of a single bout of Tai Chi (TC) exercise on adiponectin and glucose homeostasis in individuals with cardiovascular risk factors. Twenty-six individuals (mean age 60.2 years) with at least one cardiovascular risk factor who had been practicing Yang's style TC exercise for at least 3 months were recruited from a regional hospital in Taiwan. A one-group repeated measured quasi-experimental design was used. Participants completed a 60-min Yang's style TC exercise routine including warm up, stretching exercises, and TC followed by a 30-min resting period. After a 1-week washout period, the same group of participants underwent a control condition in which they were instructed to remain seated for 90 min at the study location. Blood samples were collected both before and after the TC intervention or the sitting condition. The difference between pre-post measurements for adiponectin was 0.58 ± 1.42 μg/ml in the TC trial and -0.46 ± 0.99 μg/ml in the sitting trial. The differences between the two trials were statistically significant ($P = 0.004$). The changes from pretrial to posttrial were significantly greater for glycerol ($P < 0.001$), cholesterol ($P = 0.046$), and LDL-C ($P = 0.038$) in the TC trial compared with those in the sitting trial. Conversely, the changes were significantly lesser for HOMA-IR ($P = 0.004$), log (HOMA-IR) ($P = 0.001$), and glucose ($P = 0.003$) in TC trial compared with those in the sitting trial. In conclusion, a single bout of TC exercise had a significant positive effect on blood adiponectin concentrations in individuals with cardiovascular risk factors.

Keywords Tai Ji · Exercise · Cardiovascular disease · Insulin resistance · HOMA-IR

Communicated by Keith George.

R.-Y. Chang and M. Koo made equal contributions to this paper.

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Introduction

Adiponectin (also called ARCP30, AdipoQ, apM1, and GBP28) is a 247-amino acid peptide hormone produced specifically by adipose tissue (Trujillo and Scherer 2005). Studies have shown that blood adiponectin concentration is inversely associated with several metabolic disorders, including obesity, type 2 diabetes mellitus (Weyer et al. 2001), cardiovascular disease (Schulze et al. 2005), and metabolic syndrome (Salmenniemi et al. 2004). Adiponectin level is directly correlated with high-density lipoprotein level and inversely correlated with triglycerides, percent body fat, and serum leptin (Kazumi et al. 2004). There are several mechanisms linking adiponectin with glucose and plasma lipid profiles. Adiponectin stimulates fatty acid oxidation and glucose uptake by skeletal myocytes or adipocytes (Yamauchi et al. 2002). Adiponectin can stimulate

insulin secretion by pancreatic β cells and suppress gluconeogenesis and hepatic glucose output (Gu et al. 2006). High-molecular-weight adiponectin suppresses secretion of apolipoprotein B and E from hepatocytes and stimulates synthesis and secretion of apolipoprotein A-I (Neumeier et al. 2007). Therefore, adiponectin can increase HDL formation and reverse cholesterol transport from peripheral tissue to the liver (Matsuura et al. 2007). In addition, circulating adiponectin levels have been observed to be inversely associated with markers of endothelial dysfunction and systemic inflammation, such as tumor necrotic factor-α (TNF-α) and C-reactive protein (CRP) in diabetic patients and individuals at risk for diabetes (Shetty et al. 2004).

The beneficial effects of exercise on insulin sensitivity is well accepted (Hawley 2004). However, it is not clear whether the beneficial effects are mediated through changes in adiponectin levels and whether exercise can modify adiponectin levels in the absence of weight loss or dietary interventions. In addition, a systematic review of the effects of exercise on adiponectin identified seven acute exposure exercise studies and conflicting data have been observed (Simpson and Singh 2008). Some studies have shown no effect of acute exercise on adiponectin levels in normal or overweight individuals (Ferguson et al. 2004; Kraemer et al. 2003; Numao et al. 2008) but other studies have observed significant changes in adiponectin levels (Jürimäe et al. 2005; Jürimäe et al. 2006). Most randomized controlled studies of acute exposure exercise were small and no clear conclusions can yet be made regarding whether aerobic or resistance exercise and whether moderate- or high-intensity exercise has the greatest impact on adiponectin levels.

Tai Chi (TC) is a traditional Chinese martial art and can be classified as moderate-intensity endurance exercise. The therapeutic effects of TC include rehabilitation of congestive heart failure and coronary artery disease (Channer et al. 1996), improving aerobic and cardiorespiratory capacity (Yeh et al. 2004), balance control and fall prevention in the elderly (Li et al. 2004), and reducing blood pressure and serum cholesterol (Taylor-Piliae et al. 2006). The effects of 8 weeks of TC were investigated on 12 elderly subjects with type 2 diabetes. A significant decrease in blood glucose and a significant increase in high- and low-affinity insulin receptor numbers and low-affinity insulin receptor binding capacity were observed. In addition, a single bout of TC exercise was found to increase blood glucose, high- and low-affinity insulin receptor numbers, and their binding capacity (Wang 2008). Furthermore, an attenuated parasympathetic nervous activity has been suggested to contribute to the development of insulin resistance (Lindmark et al. 2003), and TC exercise has been shown to be associated with an acute effect of increasing parasympathetic nervous activity as shown by an increased in normalized high-frequency power and a decreased in normalized low-frequency power of heart rate variability (Chang et al. 2008). However, to our knowledge, the acute effects of TC exercise on adiponectin have not been reported in the literature. This study is designed to evaluate the acute effects of TC exercise on adiponectin and glucose homeostasis in individuals with cardiovascular risk factors.

Patients and methods

Participants

Twenty-six participants (age 60.2 ± 8.4 years) with at least one cardiovascular risk factor who had been practicing Yang's style TC for at least 3 months participated in this study. The cardiovascular risk factors were defined as cigarette smoking, hypertension (systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg), dyslipidemia (serum total cholesterol ≥200 mg/dl, triglyceride ≥150 mg/dl, LDL cholesterol ≥130 mg/dl, HDL cholesterol <40 mg/dl in men, and <50 mg/dl in women), diabetes mellitus (fasting glucose ≥126 mg/dl, 2 h plasma glucose after a 75 g oral glucose load ≥200 mg/dl), obesity (body mass index >30 kg/m^2), and abdominal obesity (waist circumference >90 cm in men and >80 cm in women), family history of premature coronary artery disease (male first-degree relative <55 years old, female first-degree relative <65 years old), and age (men ≥45 years old, women ≥55 years old) (De Backer et al. 2003; Grundy et al. 1999; NCEP 2001). Of 26 participants, 11 took medications including antihypertensive agents, sulfonylurea, biguinide, fibrate, or statin. The medications, dose, and administration time for each participant remained the same throughout the study. None of the participants took thiazolidinediones. All participants gave written informed consent and the study was approved by the institutional review board of Chia-Yi Christian Hospital, Taiwan.

Anthropometric measures

Body weight and height were determined with a physician's scale. Body mass index (BMI) was calculated by dividing body weight in kilograms by height in meters squared. Body fat (%) and visceral fat level were measured with a fat analyzer scale (Omron HBF-352, Japan). Circumferences of waists and hips were measured and the waist-to-hip ratio was calculated. The characteristics of the participants are shown in Table 1.

Activity and diet before test

All participants were instructed to maintain their usual dietary habits, physical activity patterns, and TC practice

Table 1 Participant characteristics

	Total $N = 26$*	Male $n = 14$	Female $n = 12$	P value
Age (year)	60.2 ± 8.4	63.1 ± 8.5	56.8 ± 7.3	0.055
Body weight (cm)	63.9 ± 8.9	67.5 ± 8.3	59.6 ± 8.0	0.022
Body height (kg)	160.6 ± 7.9	165.3 ± 7.3	155.0 ± 4.0	<0.001
Body mass index (kg/m^2)	24.8 ± 3.0	24.7 ± 2.7	25.0 ± 3.4	0.838
Waist circumference (cm)	83.3 ± 6.8	86.0 ± 5.8	80.0 ± 6.6	0.020
Hip circumference (cm)	97.4 ± 6.0	96.9 ± 4.7	98.0 ± 7.4	0.630
Waist to hip circumference ratio	0.86 ± 0.06	0.89 ± 0.05	0.82 ± 0.04	0.001
Body fat (%)	29.5 ± 5.4	25.8 ± 3.3	34.7 ± 2.9	<0.001
Visceral fat (%)	10.4 ± 4.1	12.4 ± 3.5	7.7 ± 3.1	0.003
Years of TC practice	5.3 ± 5.8	3.9 ± 5.8	6.9 ± 5.6	0.199
Times of TC practice/week	4.1 ± 2.6	3.4 ± 2.7	4.9 ± 2.5	0.156
Mean heart rate during TC	90 ± 16	92 ± 16	85 ± 17	0.357
Maximum heart rate during TC	118 ± 21	120 ± 20	112 ± 23	0.409
Percentage of maximum heart rate	73.8 ± 13.6	77.2 ± 13.2	67.6 ± 12.9	0.136
Smoking	1	1	0	1.000
Hypertension	9	5	4	1.000
Type 2 diabetes mellitus	7	5	2	0.391
Hyperlipidemia	19	10	9	1.000
10-year CHD Framingham risk score	6.2 ± 5.4	9.9 ± 4.7	1.9 ± 1.6	<0.001

Data are mean ± SD or count where appropriate

* $n = 26$ except for body fat ($n = 25$), visceral fat ($n = 25$), mean heart rate during TC ($n = 20$), maximum heart rate during TC ($n = 20$), and percentage of maximum heart rate ($n = 20$)

TC Tai Chi, *CHD* coronary heart disease

during the entire experiment. They were directed to avoid a high-fat diet and alcohol 3 days before each trial. Standard dinner meals were provided to the participants on the day before each trial (containing 54, 29, and 17% energy from carbohydrates, fat, and protein, respectively).

Experimental design

A quasi-experimental, one-group, repeated-measures design was used. All participants underwent the intervention followed by a 1-week washout period. Participants arrived at the study location at 8:00 a.m. After resting for 10 min, a pre-test fasting (>12 h) blood sample (10 ml) was obtained from the participants in sitting position. The participants were asked to consume 400 ml of water prior TC exercise. At 8:30 a.m., the participants went through a 60-min TC exercise routine followed by 30 min of rest. The 60-min TC exercise included a 30-min warm up, stretching exercises, and a balance program with a crystal music CD playing in the background, followed by a 30-min Yang's style TC routine with classic Chinese music CD playing in the background. The TC exercise routine was conducted under the instruction of a professional Yang's style TC teacher. After the TC exercise routine, the participants rested for 30 min and post-test blood samples were collected.

At the end of the one-week washout period, the same group of study participants underwent a control condition in which they were instructed to remain seated on a chair for 90 min at the study location. Participants arrived at the study location at 8:00 a.m. After resting for 10 min, a pre-test fasting (>12 h) blood sample (10 ml) was obtained from the participants in sitting position. The participants were asked to consume 400 ml of water during their sitting session. The second blood sample was collected at the end of the 90-min rest.

Exercise intensity

Heart rate (HR) was monitored for each subject during TC, using Medilog Holter recorders (Oxford Instruments Medical, Abingdon, UK) attached with leads II, V2, and V5. The percentage of maximum age predicted heart rate (%HR max) was used to determine exercise intensity. The %HR max was calculated by maximum HR during 60-min TC routine/(220 − age).

Blood analysis

Serum total adiponectin concentrations were measured by radioimmunosorbent assay (LINCO Research, Missouri,

USA) with an intra-assay variability of 3.59%. The adiponectin radioimmunosorbent assay utilizes ^{125}I-labeled murine adiponectin and a multispecies adiponectin rabbit antiserum to determine the level of adiponectin in serum by the double-antibody/PEG technique. Serum glucose concentrations were analyzed by means of a glucose oxidase method (GOD-PAP, RANDOX Laboratories Ltd., Co. Antrim, UK) with an autoanalyzer (Chiron Express Plus, Chiron Diagnostic East Walpole, MA, USA). The intra-assay variability of serum glucose concentrations was 1.48% and the minimum detection level was 2.88 mg/dl. Serum levels of insulin were analyzed by electrochemiluminescence immunoassay (ECLIA) (Roche Elecsys 2010, Germany) with an intra-assay variability of 1.5%. Serum FFA concentrations were analyzed by enzymatic colorimetric methods (RANDOX Laboratories Ltd., Co. Antrim, UK) with an intra-assay variability of 0.42% and the minimum detection level was 0.04 mmol/l. Glycerol concentrations were analyzed by enzymatic colorimetric methods (RANDOX Laboratories Ltd., Co. Antrim, UK) with an intra-assay variability of 1.67% and the minimum detection level was 15.91 μmol/l. Insulin sensitivity was assessed utilizing the Homeostasis Model Assessment (HOMA) method. The HOMA-IR index was calculated using the following formula: fasting plasma glucose (mg dl^{-1}) × fasting plasma insulin (μU ml^{-1}) × 405^{-1} (Matthews et al. 1985). Serum lipid levels (the levels of total cholesterol, triglycerides, and high-density lipoprotein cholesterol) were analyzed by RANDOX automated assays (RANDOX Laboratories Ltd., Co. Antrim, UK) using an autoanalyzer (Chiron Express Plus, Chiron Diagnostic East Walpole, MA, USA). RANDOX total cholesterol kits were liquid ready-to-use Trinder-based (CHOD-PAP) colorimetric end-point assays. The intra-assay variability of cholesterol was 3.73, 1.71, 3.84% at 64.68, 178.9, 291.24 mg/dl, respectively. The intra-assay variability of triglyceride was 3.29, 1.55, 1.77% at 27.42, 122.07, 496.27 mg/dl, respectively. RANDOX direct HDL cholesterol kits were liquid ready-to-use two-point kinetic assays. The intra-assay variability of HDL cholesterol were 1.20, 1.53% at 37.07, 57.93 mg/dl, respectively. Low-density lipoprotein cholesterol was calculated by using the equation of Friedewald (Friedewald et al. 1972). The minimum detection levels for cholesterol, triglycerides, HDL cholesterol and LDL cholesterol were 3.35, 11.5, 1.43, and 7.35 mg/dl, respectively.

Statistical analysis

Data entry and analysis was done using SPSS for Windows version 13.0 (SPSS Inc., Chicago, IL, USA). Mean and standard deviations (SD) were calculated for continuous variables, and frequencies, and percentages for categorical variables. Paired t tests was used to compare the differences between pre- and post values of the parameters between TC and sitting trials. Pearson's correlation coefficients were used to reflect the linear relationship between adiponectin and the other variables. Level of significance was set at $\alpha = 0.05$.

Results

The characteristics of the participants are shown in Table 1. Twenty-eight participants enrolled in the study, but only 26 participants completed both the intervention and control routine. The two drop-outs were participants who were too busy to return for the control trial. The participants had practiced Yang's TC for a mean (±SD) duration of 5.3 ± 5.8 years (median 2.0 years) with an average frequency of 4.1 ± 2.6 times per week (median 4.5 times per week). The maximum and mean heart rates during the TC exercise were 119 ± 22/min and 91 ± 16/min, respectively. The percentage of maximum age predicted heart rate ranged from 54 to 95% (mean 73.8 ± 13.6%), which indicated that TC can be classified as moderate- to high-intensity exercise (ACSM 1998).

Responses of a single bout of TC exercise on adiponectin, glucose homeostasis, and lipid metabolism are summarized in Table 2. The difference between pre-post measurements for adiponectin was 0.58 ± 1.42 μg/ml in the TC trial and −0.46 ± 0.99 μg/ml in the sitting trial and the differences between the two trials were significantly different ($P = 0.004$). The changes from pretrial to posttrial were significantly greater for glycerol ($P < 0.001$), cholesterol ($P = 0.046$), and LDL-C ($P = 0.038$) in the TC trial compared with those in the sitting trial. Conversely, the changes from pretrial to posttrial were significantly lesser for HOMA-IR ($P = 0.004$), log (HOMA-IR) ($P = 0.001$), and glucose ($P = 0.003$) in TC trial compared with those in the sitting trial. The changes from pretrial to posttrial for insulin, free fatty acid, triglyceride, HDL-C, and ratios of LDL-C to HDL-C were not significantly different between the two trials.

When the data were analyzed separately for males and females, we observed that most of the significant changes in the whole group of participants were also seen in males. There were significant changes in adiponectin ($P = 0.017$), HOMA-IR ($P = 0.031$), log (HOMA-IR) ($P = 0.017$), glucose ($P = 0.024$), glycerol ($P = 0.005$), and triglycerides ($P = 0.031$) between the TC and sitting trial (Table 3). However, in females, only log (HOMA-IR) ($P = 0.018$), glycerol ($P = 0.013$), cholesterol ($P = 0.046$), LDL-C ($P = 0.023$), and ratios of LDL-C to HDL-C ($P = 0.032$) were significantly different between the TC and sitting trial (Table 4).

Table 2 Blood adiponectin, glucose homeostasis and lipid metabolism before and after TC and sitting trial ($N = 26$)

Variable	Time	TC trial	Sitting trial	Difference between pre and post within TC trial	Difference between pre and post within sitting trial	*P* value
Adiponectin (μg/ml)	Pre	9.36 ± 5.41	9.19 ± 5.45	0.58 ± 1.42	−0.46 ± 0.99	0.004
	Post	9.94 ± 5.51	8.73 ± 5.34			
HOMA-IR	Pre	3.62 ± 1.99	3.49 ± 1.87	−0.83 ± 1.19	0.03 ± 0.78	0.004
	Post	2.79 ± 1.35	3.51 ± 1.73			
Log (HOMA-IR)	Pre	0.50 ± 0.22	0.47 ± 0.28	−0.10 ± 0.15	0.02 ± 0.10	0.001
	Post	0.41 ± 0.17	0.49 ± 0.24			
Glucose (mg/dl)	Pre	122.22 ± 35.48	112.84 ± 38.24	−32.44 ± 22.36	−8.6 ± 31.04	0.003
	Post	89.79 ± 23.66	104.20 ± 21.63			
Insulin (μU/ml)	Pre	12.11 ± 5.59	13.04 ± 7.30	0.49 ± 2.45	0.78 ± 1.88	0.608
	Post	12.60 ± 4.58	13.81 ± 6.72			
Glycerol (mmol/l)	Pre	100.06 ± 25.79	96.34 ± 39.05	19.43 ± 34.27	−12.81 ± 22.49	<0.001
	Post	119.49 ± 46.32	83.53 ± 32.82			
Free fatty acid (mmol/l)	Pre	0.65 ± 0.16	0.71 ± 0.39	0.15 ± 0.19	0.07 ± 0.36	0.316
	Post	0.80 ± 0.21	0.78 ± 0.28			
Cholesterol (mg/dl)	Pre	201.16 ± 46.03	199.56 ± 43.20	8.78 ± 18.09	0.76 ± 7.81	0.046
	Post	209.93 ± 48.10	200.32 ± 43.35			
Triglyceride (mg/dl)	Pre	129.62 ± 56.01	124.35 ± 51.54	2.51 ± 11.04	−2.59 ± 8.16	0.055
	Post	132.13 ± 58.25	121.76 ± 49.61			
HDL-C (mg/dl)*	Pre	61.83 ± 20.74	58.93 ± 15.46	−1.95 ± 15.25	−0.44 ± 4.60	0.640
	Post	59.89 ± 16.97	58.49 ± 15.48			
LDL-C (mg/dl)*	Pre	113.40 ± 36.39	115.76 ± 33.23	10.22 ± 18.67	1.71 ± 9.29	0.038
	Post	123.62 ± 39.59	117.47 ± 32.61			
LDL-C/HDL-C*	Pre	1.95 ± 0.65	2.01 ± 0.54	0.293 ± 1.108	0.060 ± 0.282	0.305
	Post	2.25 ± 1.38	2.08 ± 0.60			

Data are mean ± SD

HOMA-IR homeostasis model assessment-insulin resistance, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

* $N = 25$ for the variables LDL-C, HDL-C, LDL-C/HDL-C

The changes in serum adiponectin levels were not significantly correlated with HOMA-IR, log(HOMA-IR) glucose, insulin, glycerol, free fatty acid, cholesterol, triglyceride, HDL-C, or LDL-C (Table 5).

Discussion

To our knowledge, this is the first study to explore the effect of TC exercise on adiponectin response in individuals with cardiovascular risk factors. Adiponectin has been reported to play a significant role in metabolic disorders, such as cardiovascular disease and type 2 diabetes due to its anti-inflammatory (Ajuwon and Spurlock 2005) and insulin sensitizing properties (Lara-Castro et al. 2006). Our results showed that a single bout of TC was effective in improving adiponectin concentration. Previous studies on the effects of acute exercise on adiponectin levels have shown discrepancies. A systematic review of the effects of exercise on adiponectin identified seven acute bout studies on adiponectin (Simpson and Singh 2008). Two of the seven studies reported an increase in circulating adiponectin after exercise (Jürimäe et al. 2006; Kraemer et al. 2003). In the three studies that showed no change in adiponectin, the exercise protocol was set at low to moderate intensities (Ferguson et al. 2004; Punyadeera et al. 2005; Jamurtas et al. 2006). However, studies on highly trained male athletes found different results. Jürimäe et al. (2005) reported a significant reduction in plasma volume adjusted adiponectin levels immediately after high-intensity rowing exercise. After the first 30 min of recovery, adiponectin was significantly increased above the resting value with or without correction for plasma volume. Jürimäe et al. (2006) also compared the response of adiponectin between rowers of different levels of performance. They concluded that training might modify adiponectin response to short-duration exercise depending on the performance level of athletes. Therefore, authors from the

Table 3 Blood adiponectin, glucose homeostasis, and lipid metabolism before and after TC and sitting trial in males ($n = 14$)

Variable	Time	TC trial	Sitting trial	Difference between pre and post within TC trial	Difference between Pre and Post within sitting trial	*P* value
Adiponectin (μg/ml)	Pre	7.80 ± 4.37	7.63 ± 4.55	0.76 ± 1.60	−0.51 ± 0.82	0.017
	Post	8.56 ± 4.67	7.12 ± 4.86			
HOMA-IR	Pre	3.62 ± 2.14	3.61 ± 2.11	−0.93 ± 1.10	−0.25 ± 0.85	0.031
	Post	2.69 ± 1.62	3.37 ± 1.81			
Log (HOMA-IR)	Pre	0.50 ± 0.23	0.48 ± 0.30	−0.12 ± 0.16	−0.01 ± 0.12	0.017
	Post	0.38 ± 0.20	0.46 ± 0.26			
Glucose (mg/dl)	Pre	134.12 ± 35.19	122.79 ± 47.06	−40.32 ± 5.88	−14.86 ± 39.12	0.024
	Post	93.80 ± 29.25	107.93 ± 27.15			
Insulin (μU/ml)	Pre	10.92 ± 5.57	12.36 ± 7.41	0.53 ± 2.85	0.47 ± 2.07	0.952
	Post	11.45 ± 4.50	12.83 ± 6.93			
Glycerol (mmol/l)	Pre	97.36 ± 23.40	92.30 ± 44.07	19.44 ± 33.99	−14.68 ± 21.76	0.005
	Post	116.81 ± 45.77	77.63 ± 31.81			
Free fatty acid (mmol/l)	Pre	0.68 ± 0.12	0.76 ± 0.37	0.14 ± 0.18	0.03 ± 0.39	0.288
	Post	0.81 ± 0.19	0.79 ± 0.26			
Cholesterol (mg/dl)	Pre	186.53 ± 43.15	183.75 ± 38.94	5.07 ± 10.56	2.48 ± 8.79	0.537
	Post	191.60 ± 42.27	186.23 ± 40.76			
Triglyceride (mg/dl)	Pre	135.08 ± 67.81	129.19 ± 62.96	1.69 ± 8.87	−4.63 ± 7.37	0.031
	Post	136.77 ± 69.61	124.56 ± 59.18			
HDL-C (mg/dl)*	Pre	56.90 ± 24.59	52.06 ± 11.69	−5.09 ± 19.84	−1.73 ± 3.61	0.548
	Post	51.80 ± 14.62	50.33 ± 10.07			
LDL-C (mg/dl)*	Pre	102.62 ± 35.17	105.85 ± 32.90	9.83 ± 17.63	5.13 ± 8.35	0.420
	Post	112.45 ± 40.92	110.98 ± 34.83			
LDL-C/HDL-C*	Pre	2.01 ± 0.78	2.09 ± 0.64	0.45 ± 1.47	0.17 ± 0.24	0.498
	Post	2.46 ± 1.81	2.26 ± 0.71			

Data are mean ± SD

HOMA-IR homeostasis model assessment-insulin resistance, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

* $N = 25$ for the variables LDL-C, HDL-C, LDL-C/HDL-C

systematic review concluded that additional studies are needed to clarify the relationship between adiponectin and exercise, particularly, modes other than aerobic exercise (Simpson and Singh 2008).

The discrepancy of the acute effects of exercise on adiponectin level may be attributed to differences in timing in blood sampling, variation in the exercise intensity and duration, differences in the participant populations, and whether the participants had previously engaged in regular exercise training. In the present study, all participants were avid practitioners of TC with a median duration of 2 years of TC practice. During the 60-min of TC exercise, participants performed sustained anaerobic, resistance, and endurance exercises which may increase adiponectin level and improve insulin resistance. In addition, levels of circulating adiponectin may be affected a change in plasma volume. Kraemer et al. (2003) conducted running exercises at an intensity of 79% of maximal oxygen consumption for 30 min in six healthy men. A significant increase in adiponectin was observed but the increase may be attributed to normal plasma volume shifts. We conducted a substudy on 13 participants of our study to compare their plasma volume after TC and after sitting. The results indicated an increase of 0.71% with TC exercise and an increase of 0.96% in control condition. We used these figures to adjust for the data in the Table 2 and we observed similar statistical results compared with the original unadjusted data. The measurement values shown in all tables were unadjusted.

Our study also found that a single bout of TC significantly associated with a decrease in serum HOMA-IR, log (HOMA-IR), and glucose while significantly associated with an increase in glycerol, cholesterol, and LDL-C. Together, these findings suggested that acute TC exercise could increase insulin sensitivity in individuals with cardiovascular risk factors. Numao et al. (2008) also reported similar findings in the changes in glucose and glycerol in a study of acute aerobic exercise in healthy men. Exercise

Table 4 Blood adiponectin, glucose homeostasis, and lipid metabolism before and after TC and sitting trial in females ($n = 12$)

Variable	Time	TC trial	Sitting trial	Difference between pre and post within TC trial	Difference between pre and post within sitting trial	*P* value
Adiponectin (μg/ml)	Pre	11.19 ± 6.10	11.01 ± 6.03	0.37 ± 1.22	−0.40 ± 1.18	0.122
	Post	11.56 ± 6.16	10.60 ± 5.63			
HOMA-IR	Pre	3.61 ± 1.88	3.33 ± 1.60	−0.70 ± 1.35	0.37 ± 0.52	0.058
	Post	2.91 ± 0.95	3.70 ± 1.68			
Log (HOMA-IR)	Pre	0.51 ± 0.22	0.46 ± 0.26	−0.06 ± 0.13	0.06 ± 0.06	0.018
	Post	0.44 ± 0.14	0.52 ± 0.22			
Glucose (mg/dl)	Pre	107.09 ± 31.01	100.18 ± 17.76	−22.40 ± 25.98	−0.73 ± 14.12	0.067
	Post	84.68 ± 13.42	99.45 ± 10.97			
Insulin (μU/ml)	Pre	13.63 ± 5.48	13.90 ± 7.41	0.43 ± 1.96	1.17 ± 1.62	0.197
	Post	14.06 ± 4.46	15.07 ± 6.55			
Glycerol (mmol/l)	Pre	103.22 ± 29.05	101.05 ± 33.56	19.41 ± 36.11	−10.64 ± 25.05	0.013
	Post	122.63 ± 48.78	90.41 ± 36.17			
Free fatty acid (mmol/l)	Pre	0.61 ± 0.20	0.66 ± 0.41	0.16 ± 0.20	0.12 ± 0.32	0.724
	Post	0.78 ± 0.24	0.78 ± 0.31			
Cholesterol (mg/dl)	Pre	219.78 ± 44.53	219.69 ± 41.36	13.49 ± 24.44	−1.43 ± 6.04	0.046
	Post	233.27 ± 46.48	218.26 ± 41.45			
Triglyceride (mg/dl)	Pre	122.67 ± 38.17	118.19 ± 33.90	3.55 ± 13.73	0.01 ± 8.72	0.477
	Post	126.23 ± 42.13	118.20 ± 36.48			
HDL-C (mg/dl)*	Pre	68.12 ± 13.02	67.68 ± 15.69	2.06 ± 3.88	1.20 ± 5.33	0.718
	Post	70.18 ± 14.31	68.88 ± 15.21			
LDL-C (mg/dl)*	Pre	127.13 ± 34.64	128.37 ± 30.49	10.72 ± 20.78	−2.64 ± 8.90	0.023
	Post	137.85 ± 34.46	125.74 ± 29.02			
LDL-C/HDL-C*	Pre	1.89 ± 0.46	1.92 ± 0.37	0.09 ± 0.25	−0.08 ± 0.28	0.032
	Post	1.98 ± 0.42	1.84 ± 0.30			

Data are mean ± SD

HOMA-IR homeostasis model assessment-insulin resistance, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

* $N = 25$ for the variables LDL-C, HDL-C, LDL-C/HDL-C

was associated with a significant trial and time interaction in plasma glucose and serum glycerol. Plasma glucose concentration decreased progressively while serum glycerol concentration increased progressively with exercise. Previous studies have been shown that total adiponectin concentration was acutely regulated by free fatty acids ($r = 0.66$, $P < 0.05$) (Bernstein et al. 2004) and insulin ($r = -0.39$, $P = 0.042$) (Yu et al. 2002) after administration of adipimox and thiazolidinediones, respectively. However, a dramatic increase in free fatty acid concentration and a decrease in insulin concentration during aerobic exercise did not significantly change the total adiponectin during exercise (Numao et al. 2008). In our study, adiponectin, cholesterol, LDL-C, and glycerol showed an increase after TC exercise whereas HOMA-IR, log (HOMA-IR), and glucose showed a decrease. Adiponectin did not show any significant correlations with other variables.

TC, along with Qi Gong, acupuncture, herbology, and Tui Na, forms the primary components of traditional Chinese medicine. It is considered different from other physical exercises in four main aspects: (1) the slow movement between its different postures, (2) the need for full concentration during practice, (3) the incorporation of physical movements with breathing techniques, and (4) the need for complete relaxation during practice (Chu 2004). Previous studies reported that TC exercise could decrease sympathetic activity in older adults. Lu and Kuo (2003) reported that TC exercise enhanced vagal modulation and tilt the sympathovagal balance toward deceased sympathetic modulation in older persons and the effect persisted for 60 min after TC performance. Motivala et al. (2006) also found that the practice of TC exercise could induce acute decreases of sympathetic nervous system activity as measured by preejection period in healthy older adults. In our previous study, we also found that TC exercise could lead

Table 5 Pearson correlation analysis of blood adiponectin with other glucose homeostasis and lipid metabolism parameters

	Adiponectin ($n = 26$)	
	r	P value
HOMA-IR	−0.086	0.677
Log (HOMA-IR)	−0.235	0.247
Glucose	−0.016	0.940
Insulin	−0.283	0.161
Glycerol	−0.020	0.923
Free fatty acid	−0.022	0.913
Cholestero1	0.029	0.887
Triglyceride	0.112	0.587
HDL-C	−0.206	0.312
LDL-C	0.182	0.374

HOMA-IR homeostasis model assessment-insulin resistance, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

to increased normalized high-frequency power and decreased normalized low-frequency power 30 min after exercise in patients with coronary artery disease (Chang et al. 2008). Animal studies indicated that, independently of alterations in adiposity, sympathetic nervous system physiologically regulates adiponectin production and the resultant serum adiponectin levels (Imai et al. 2006). Therefore, it is plausible that the increase in adiponectin levels after TC exercise is mediated through modulation of the sympathetic nervous system. The discrepancy of the acute effects of exercise on adiponectin levels may also be attributed to the variations in the autonomic balance associated with different exercise modality. Further study on the correlations between adiponectin and autonomic function may help to elucidate the mechanism.

For the blood lipid profiles, the increase in mean LDL-C in participants with TC exercise was significantly greater than that in participants in the control trial ($P = 0.038$). However, the extent of the changes in LDL-C did not lead to significant differences in the mean changes of the ratios of LDL-C to HDL-C ($P = 0.305$). Since this is the first study to explore the effect of a single bout of TC exercise on blood lipid responses, no previous data are available for comparison purposes. The effect of long-term TC was associated with either beneficial or no effect on LDL-C. A study on 53 patients with dyslipidemia found that a 12-month TC program was associated with a reduction in LDL-C (Lan et al. 2008). Tsai et al. (2003) also reported that 12 weeks of TC exercise significantly reduced LDL-C in a randomized controlled trial of 76 patients with borderline hypertension. However, the results of another randomized controlled trial of 14 weeks of TC practice on 20 women with type 2 diabetes did not reveal a significant difference in LDL-C (Zhang and Fu 2008). Yu et al. (2002) reported that adiponectin was directly correlated with HDL-C ($r = 0.59$, $P < 0.001$) and inversely correlated with triglyceride ($r = -0.61$, $P < 0.001$) in thiazolidinediones-treated lean, obese, and diabetic subjects. However, no significant correlations were observed between adiponectin and HDL-C or triglyceride in our study.

Adiponectin exhibits a sexual dimorphism with higher levels in females than males. There were no differences before puberty in the levels of total adiponectin between males and females. However, after puberty, total adiponectin levels were significantly reduced in males but remained unchanged in females. The reduction is likely to be attributed to the suppression effect of testosterone (Andersen et al. 2007). In our study, we also observed a higher adiponectin levels in females than in males (Tables 3, 4).

When the data were analyzed separately for males and females, most of the significant changes in the whole group of participants were seen only in males. The exact mechanism of the differences in responses to TC exercise between the males and females are unclear. No existing acute bout studies have compared the sex differences of adiponectin responses to exercise (Simpson and Singh 2008). One plausible explanation to the differences in responses to TC exercise between males and females is the exercise-induced suppression of testosterone. Since testosterone can lead to a reduction of adiponectin levels (Andersen et al. 2007), adiponectin levels will increase if testosterone levels can be suppressed by exercise. Rämson et al. (2009) reported that 30-min postexercise testosterone concentrations in male rowers were significantly decreased with high training volumes compared with low training volumes. The issue whether TC exercise can similarly suppress testosterone levels and thus affect adiponectin levels in males and females differently requires further investigation.

Several limitations of this study should be noted. First, the participants mostly had practiced TC for several years. Additional studies will be required to explore whether the observed acute response of TC on adiponectin and the HOMA-IR will also appear in individuals who do not routinely practice TC exercise. Second, without the use of a crossover design, the possibility of an order effect in which the order of the trials affects subsequent responses cannot be completely ruled out. However, we have implemented a 1-week washout period to minimize such effect. In addition, none of the baseline measures for all of the variables were significantly different between the two trials (P values not shown). Third, the control condition used in the present study was non-exercise, sedentary, seated condition rather than other form of sham exercise such as gentle stretching. Therefore, specific effects of TC such as those attributed to its breathing and mindfulness components could not be evaluated. Fourth, Numao et al. (2010), in their study on

high-intensity exercise in middle-aged abdominally obese men, concluded that the change in total adiponectin was mainly due to a change in middle-plus low-molecular-weight (MLMW) adiponectin concentration. However, adiponectin oligomer levels were not measured in our study. Therefore, we cannot conclude whether the changes in total adiponectin levels in TC exercise was also due to a change in MLMW adiponectin.

Altogether, our study has shown that a single bout of Yang's style TC enhanced adiponectin concentration and reduced insulin resistance at the acute phase of exercise in individuals with cardiovascular risk factors. The long-term effect of TC on adiponectin and glucose homeostasis needs further investigation.

Acknowledgments We acknowledge the support provided by the staff of the Division of Cardiology and Cardiovascular Surgery, and grant support by Department of Medical Research of Chia-Yi Christian hospital, Taiwan. We also acknowledge Mr. Fu-Chu Lin for his voluntary contribution as the TC instructor during the study.

Conflict of interest None.

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