Effects of Tai Chi on glucose homeostasis and insulin sensitivity in older adults with type 2 diabetes: a randomised double-blind sham-exercise-controlled trial

Tracey Tsang¹, Rhonda Orr¹, Paul Lam², Elizabeth Comino³, Maria Fiatarone Singh^{1,4}

Address correspondence to: T. Tsang. Tel: 612 9894 8003; Fax: 612 9680 8377. Email: ttsa6920@mail.usyd.edu.au

Abstract

Background a large proportion of adults with type 2 diabetes remain sedentary despite evidence of benefits from exercise for type 2 diabetes. Simplified Yang Tai Chi has been shown in one study to have no effect on insulin sensitivity in older adults. However, a modified Tai Chi form, Tai Chi for Diabetes (TCD) has recently been composed, claiming to improve diabetes control.

Methods subjects were randomised to Tai Chi or sham exercise, twice a week for 16 weeks. Primary outcomes were insulin resistance 72 h post-exercise (HOMA2-IR), and long-term glucose control (HbA_{1c}).

Results thirty-eight subjects (65 \pm 7.8 years, 79% women) were enrolled. Baseline BMI was 32.2 ± 6.3 kg/m², 84% had osteoarthritis, 76% hypertension, and 34% cardiac disease. There was one dropout, no adverse events, and median compliance was 100 (0 - 100)%. There were no effects of time or group assignment on insulin resistance or HbA_{1c} ($-0.07 \pm 0.4\%$ Tai Chi versus $0.12 \pm 0.3\%$ Sham; P = 0.13) at 16 weeks. Improvement in HbA_{1c} was related to decreased body fat (r = 0.484, P = 0.004) and improvement in insulin resistance was related to decreased body fat (r = 0.37, P = 0.03) and central adiposity (r = 0.38, P = 0.02), as well as increased fat-free mass (r = -0.46, P = 0.005).

Conclusions TCD did not improve glucose homeostasis or insulin sensitivity measured 72 h after the last bout of exercise. More intense forms of Tai Chi may be required to produce the body composition changes associated with metabolic benefits in type 2 diabetes.

Keywords: Tai Chi, diabetes mellitus, type 2, insulin resistance, elderly

Introduction

Type 2 diabetes (T2DM) affects over 100 million people worldwide and its prevalence is expected to increase to 300 million by 2025 [1]. Although moderate-to-vigorous aerobic and resistance exercises have been shown to improve all aspects of metabolic syndrome, a large proportion of adults with T2DM do not follow recommended physical activity guidelines [2]. A low-impact, low-intensity exercise such as Tai Chi (TC) may address poor compliance in this population and provide a beneficial alternative.

Though the standardised 24-form Yang TC style was recently shown in a well-designed RCT to have no effect on

insulin sensitivity, HbA_{1c}, body composition, lipids, or blood pressure in older adults [3] (of whom 14% had impaired fasting glucose or diabetes, and approximately 60% had hypertension, dyslipidemia, and/or obesity), a new form has recently been composed, Tai Chi for Diabetes (TCD), claiming to improve diabetes control via the energy flow the selected movements of the form would encourage [4]. However, no studies have yet investigated the effect of TCD on insulin resistance or glucose homeostasis.

This is the first RCT to investigate the effect of TCD on glucose homeostasis and insulin resistance in T2DM. We hypothesised that TCD would improve insulin resistance and

¹School of Exercise and Sport Science, Faculty of Health Sciences, University of Sydney, Australia

²Department of Family Medicine, University of New South Wales, Australia

³Senior Research Fellow, School of Public Health and Community Medicine, University of New South Wales, Australia

⁴Faculty of Medicine, University of Sydney, Sydney, Australia; Hebrew Rehabilitation Center for Aged and Jean Mayer USDA Human Nutrition Center on Aging at Tufts University, Boston, MA, USA

glucose homeostasis in older adults with diabetes relative to controls undertaking sham exercise.

Research design and methods

Study design

The study was a double-blind, randomised, sham-exercise-controlled trial.

Study population

Sedentary older adults with T2DM were recruited via community advertising. Screening included a telephone interview and medical examination. Eligibility included age ≥50 years, sedentary (<1 exercise session/week), and ambulatory without assistance. Exclusion criteria included cognitive impairment (Mini-Mental State Exam ≤24), current TC participation, nursing-home residence, limb amputation, any change in diabetic medication/dose within the past 3 months, and any unstable disease.

The study was approved by Human Research Ethics Committee of the Universities of Sydney and NSW, Australia, and written informed consent obtained (Clinical trials no.: ACTRN012605000715673).

Randomisation

Please see Appendix 1 in the supplementary data on the journal website (http://www.ageing.oupjournals.org.)

Interventions

Both groups completed two supervised 1-h sessions weekly for 16 weeks. Make-up sessions were allowed within a 4 week window. All classes were conducted by the same investigator extensively trained in both exercise forms. The TC group performed the TCD program [4], a 'hybrid' form of 12 movements from Sun and Yang styles. Each session commenced with whole-body warm-up exercises and concluded with cool-down exercises. Controls performed sham exercise (calisthenics and gentle stretching), previously shown to have no significant effects on physical or psychological outcomes [5, 6]. Most of the exercises were conducted seated, with 5-10 min of standing exercises holding onto the backs of chairs. No resistance other than opposing gravity, sustained isometric contractions, prolonged static stretches, or sustained rapid movements were performed, to minimise adaptations in strength, flexibility, and aerobic capacity. A sham-exercise-control was utilised as this design was able to control for effects of factors such as attention/social interaction (with group members and instructor), and participation in movement activities/classes, which would not have been possible with a usual care/non-exercise-control.

Outcomes

Outcome measures were conducted at baseline (before randomisation) and at follow-up (after 32 sessions).

Primary measures

Blinded 12 h fasting blood tests were performed by an independent laboratory. Blood tests were standardised to 72 h following the last exercise class to control for acute-bout influences on insulin resistance [7]. Please see Appendix 2 in the supplementary data on the journal website (http://www.ageing.oupjournals.org/)

Secondary outcomes and covariates

Body composition and nutritional status

Please see Appendix 3 in the supplementary data on the journal website (http://www.ageing.oupjournals.org/).

Habitual physical activity and quality of health

Please see Appendix 4 in the supplementary data on the journal website (http://www.ageing.oupjournals.org/).

Exercise intensity/compliance

Heart rate (HR) and rating of perceived exertion (RPE) was monitored for each subject during weeks 14–16, using a HR monitor (Polar Electro, OY, Finland), and the Borg scale (6–20), averaging HR and RPE every 15 min.

Percent compliance was calculated as: (# sessions attended/32) \times 100.

Adverse events

A weekly questionnaire probing possible adverse events and changes in health status was administered face-to-face or via telephone in both groups throughout the trial.

Sample size

Sample size was estimated from studies of aerobic and resistance training effects on HbA_{1 ϵ} in T2DM [8–10]. An absolute decrease in HbA_{1 ϵ} of 1 \pm 1% was estimated. Setting the power (1-beta) at 0.8, and an alpha value of 0.05, total estimated sample size required was 34. An estimated 15% dropout rate increased the sample size to 39.

Statistical methods

Statistical analyses were performed using Statview, version 5.0 (SAS Institute, Cary, NC). All data were visually inspected for normality of distribution. Non-normal data were log-transformed for use with parametric statistics. All values are reported as mean \pm standard deviation (SD); non-normal data reported as median (range). Groups were compared at baseline via *t*-tests for continuous variables, and chi-square tests for categorical variables. Changes over time and between groups were analysed using repeated analysis of variance (ANOVA). Variables that were different between groups and potentially related to the outcome of interest were used as covariates in analysis of covariance (ANCOVA) models of change scores. Ninety-five per cent

T. Tsang et al.

Table 1. Baseline subject characteristics

Characteristic	Tai chi (<i>n</i> = 18)	Control $(n = 20)$	Mean difference	Total $(n = 38)$	P value	Confidence interval	
Female (%) (CI)	88.9 (0.7–1.0)	70 (0.5–0.9)	—	78.9	0.15		
Body weight (kg)	87.52 (13.7)	80.70 (16.14)	6.82	83.93 (15.21)	0.17	-16.7, 3.1	
Waist circumference (cm)	106.10 (14.6)	98.35 (12.57)	7.74	102.66 (13.54)	0.09	-16.7, 1.2	
Body fat (%) ^b	42.95 (4.83)	` '		40.13 (7.33)	0.02^{a}	-10.2, -1.1	
Fat-free mass (kg) ^b	49.59 (6.94)	49.83 (8.27)	-0.24	49.71 (7.52)	1.00	-4.8, 5.29	
Fat mass (kg) ^b	37.93 (8.83)	31.05 (11.73)	6.88	34.49 (10.81)	0.05	-13.8, 0.01	
Ethnicity (% Caucasian) (CI)	94.4 (0.7-1.0)	85 (0.6-0.9)	_	89.5	0.34	_	
Duration of diagnosed diabetes	8.7 (0-25)	12.4 (0.7-50)	-3.73	10.64 (0-50)	0.31	$-0.3, 0.4^{\rm e}$	
(y)	, ,	,		,			
Falls, n (CI) ^c	8 (0.2-0.7)	6 (0.1-0.5)	_	14 (37)	0.15	_	
Chronic medical diagnoses (n):	6.9 (2.59)	6.1 (2.97)	0.79	6.5 (2.79)	0.39	-2.2, 0.2	
Osteoarthritis, n (CI)	16 (0.7-1.0)	16 (0.6-0.9)	_	32 (84.2)	0.45	_	
Coronary artery disease, n (CI)	7 (0.2–0.6)	6 (0.1-0.5)	_	13 (34.2)	0.56	_	
Arrhythmia, n (CI)	3 (0.1-0.4)	4 (0.1-0.4)	_	7 (18.4)	0.79	_	
Hypertension, n (CI)	13 (0.5-0.9)	16 (0.6-0.9)	_	29 (76.3)	0.57	_	
Dyslipidemia, n (CI)	11 (0.4-0.8)	14 (0.5-0.9)	_	25 (65.8)	0.56	_	
Metabolic syndrome (n) (CI)	14 (0.5-0.9)	17 (0.6-0.9)	_	31	0.57	_	
Total daily medications (n)	8.56 (4.0)	6.4 (3.7)	2.16	7.4 (4.0)	0.94	(-4.0, 0.6)	
Subjects taking any oral	10 (0.3-0.8)	16 (0.6-0.9)	_	26 (68.4)	0.11	_	
hypoglycaemic, n (CI)							
Insulin, n (CI)	4 (0.1, 0.5)	5 (0.1, 0.5)	_	9 (23.7)	0.84	_	
HbA _{1c} (%)	7.1 (0.9)	6.9 (0.9)	0.24	7.0 (0.9)	0.41	-0.8, 0.4	
Glucose (mmol/l)	7.5 (3.9-15.6)	8.4 (5.6-13.9)	-0.91	8.0 (3.9-15.6)	0.19	$-0.03, 0.2^{e}$	
Insulin (mu/l)	21.1 (8.5-60.8)	17.88 (4-35.2)	3.19	19.4 (4-60.8)	0.29	$-0.3, 0.1^{e}$	
Habitual physical activity ^d	94.7 (63.9)	149.7 (70.8)	-55.0	123.6 (72.3)	0.02^{a}	10.4, 99.6	
Daily energy intake (kcal)	1722.11 (547.29)	1542.77 (544.73)	179.3	1627.72 (546.10)	0.32	-539.1, 180.4	
Relative daily energy intake (kcal/kg/d)	20.0 (6.7)	19.8 (7.5)	0.2	19.9 (7.1)	0.93	-4.9, 4.5	

All data presented as mean (standard deviation) for normally distributed data, median (range) for non-normally distributed data, and (CI) for categorical

confidence intervals (CI) were calculated from the mean, SD, and n of each group. Relationships between variables of interest were analysed with simple and forward stepwise linear regression, or Spearman rank order correlation for non-normally distributed data. A P value of <0.05 was considered statistically significant. All available data were included irrespective of compliance. Intention to treat analysis (ITT) (last value carried forward) was performed on the primary outcomes (glucose homeostasis and insulin sensitivity) as a secondary analysis.

Results

Participant flow

Recruitment of 38 subjects occurred during March-July, 2004.

Participant characteristics

Baseline participant characteristics are shown in Table 1. Please see Appendix 5 in the supplementary data on the journal website (http://www.ageing.oupjournals.org/)

Compared to controls, subjects randomised to TC had greater total body fat (%BF) (P = 0.02), better cognition, lower social function, lower Physical Activity Scale for the Elderly (PASE) score, and greater daily dietary cholesterol intake (Table 1). There were no other significantly different variables at baseline (data not shown).

Compliance and patient satisfaction

The median time taken to complete the program was 16 (0.3-21.4) weeks. There was one dropout in the TC group who refused follow-up testing. Median compliance was 100 (6-100)% and 100 (0-100)% in TC and controls respectively (P=0.737).

^a Indicates a significant difference between Tai Chi and control groups (*P*≤0.05). Continuous variables analysed by *t*-test or Mann–Whitney U test for non-normally distributed data. Categorical variables analysed by chi square test.

^b Fat-free mass and percent body fat estimated by bioelectrical impedance [11]

 $^{^{}c}$ Number of subjects with ≥ 1 fall in the past year

^d The Physical Activity Scale for the Elderly was used to monitor habitual physical activity, based on the leisure time, household, and work-related activities performed in the previous seven days. A higher score reflects more physical activity performed [12].

^e Confidence intervals were generated for log values of non-normally distributed data.

Table 2. Mean differences

	Tai Chi group $(n = 17)$	Control group CI (Tai Chi) (n = 20)		CII (control)	Group : Time effect		× Time interaction	
Variable	Mean change	_	Mean change	_	f	P	f	P
Glucose Metabolism	_	—	—	—	_	—	_	—
HbA ₁₆ (%)	-0.07(0.4)	-0.6, 0.4	0.12(0.3)	-0.3, 0.5	0.33	0.57	2.43	0.13
HOMA2-IR ^b	0 (-1.9-2.0)	$-0.1, 0.1^{e}$	-0.1 (-2.6 - 1.2)	−0.1, 0.04 e	0.13	0.73	0.004	0.95
HOMA2%S ^b	-0.8 (-40.2 - 54.8)	$-0.1, 0.1^{e}$	5.0 (-63.5-75.1)	$-0.04, 0.1^{e}$	0.94	0.34	0.89	0.35
HOMA2%B ^b	-2.4 (-88.0 - 81.7)	$-0.1, 0.1^{e}$	4.9 (-71.1-28.7)	$-0.1, 0.1^{e}$	2.11	0.16	0.48	0.49
Body Composition	-	_	_	_		_	_	_
Weight (kg)	-1.0(2.9)	-7.3, 5.3	-0.12(1.7)	-7.7, 7.5	2.06	0.16	1.29	0.26
BMI^{c} (kg/m ²)	-0.39(1.1)	-2.8, 2.0	-0.07(0.7)	-3.4, 3.3	2.42	0.13	1.23	0.28
Waist circumference (cm)	0.49 (3.3)	-6.3, 7.3	0.34 (2.7)	-5.5, 6.2	0.72	0.40	0.02	0.88
Body fat (%)	-0.61(2.4)	-3.1, 2.6	-1.0(2.8)	-4.8, 3.7	6.70	0.01^{a}	0.16	0.70
Fat-free mass (kg)	-0.04(3.0)	-2.9, 4.1	0.62 (2.6)	-3.9, 4.0	0.001	0.97	0.47	0.50
Fat mass (kg)	-1.0(3.6)	-5.5, 3.6	-0.83(2.5)	-6.6, 4.9	2.96	0.095	0.02	0.90
Habitual physical activity ^d	17.65 (51.0)	-16.7,52.0	-35.26 (49.5)	-66.2, -4.3	1.11	0.30	9.96	0.003^{a}

Values are mean (SD) or median (range).

One subject (with pre-existing symptomatic spinal stenosis diagnosed after screening) in the TC group found the exercise intolerable secondary to pain and fatigue and completed two sessions.

Adverse events

There were no exercise-related adverse events, and no group differences in acute health problems. During the course of the study, diabetic medication was commenced by one TC, and ceased in one control participant.

Exercise intensity

The average HR during exercise was 83.3 ± 13.7 beats/min in TC, and 81.0 ± 11.8 beats/min in controls (P = 0.69). Mean RPE was 11 ± 2 and 10 ± 3 for TC and controls respectively (P = 0.28).

Primary outcomes

There were no significant changes in logHOMA2-IR or HbA_{1 ℓ} after the intervention, or in logHOMA2%B or logHOMA2%S (Table 2). Secondary ITT analysis using the last value carried forward for missing data points (n = 1) did not alter these results (data not shown), though logHOMA2%B tended to change significantly over time after ITT analysis (P = 0.07, f = 3.5).

ANCOVA models were constructed using the change scores for HbA_{1c} or HOMA2-IR as the dependent variables,

adjusted for baseline values of either HbA_{1c} or $\mathrm{HOMA2}$ -IR, respectively, as well as waist circumference and physical activity level. The group effects for HbA_{1c} (f=0.923, P=0.35) and $\mathrm{logHOMA2}$ -IR (f=0.004, P=0.95) remained non-significant.

Improvement in HbA_{1c} over time was significantly associated with decrease in %BF (r = 0.484, P = 0.004), explaining 23.5% of the variance in HbA_{1c} .

Improvements in logHOMA2-IR were greatest in those with the highest baseline physical activity level (r=-0.34, P=0.037). The improvement in logHOMA2-IR was significantly related to increased FFM (r=-0.46, P=0.005), and decreased %BF (r=0.37, P=0.03), waist circumference (r=0.38, P=0.019), and PASE score over the intervention period (r=0.36, P=0.03) (Figure 1). Changes in both waist circumference and PASE score independently contributed to change in logHOMA2-IR ($r^2=0.347$, P=0.001) in a forward stepwise regression model of the significant univariate predictors above.

Secondary outcomes

Body composition

Body weight did not change over time (P=0.16) or between groups (P=0.26) in unadjusted models, or in an ANCOVA model adjusted for baseline PASE score (P=0.15). Body fat decreased slightly but significantly over time (Table 2), but not differentially between sham exercise and TCD. There were no significant changes in waist circumference or FFM (Table 2).

 $^{^{}a} P < 0.05$

b Homeostasis Model Assessment Index 2 (HOMA2): an estimate of insulin resistance (HOMA2-IR), insulin sensitivity relative to normals (HOMA2%S), and beta-cell function relative to normals (HOMA2%B). Higher values indicate better insulin sensitivity (%S) and beta-cell function (%B), and worse insulin resistance (IR). HOMA2%B was calculated only in subjects who were not taking exogenous insulin [13].

^c Body Mass Index: an indicator of body fat calculated by Weight (kg)/Height² (m). Normal values range from 18.5–24.9 kg/m². Values ≥ 25 are considered overweight, and > 30 obese.

d PASE was used to determine a score for habitual physical activity, based on the leisure time, household, and work-related activities performed in the previous 7 days. A higher score reflects more energy expenditure [12].

^e CI were generated for log values of non-normally distributed data.

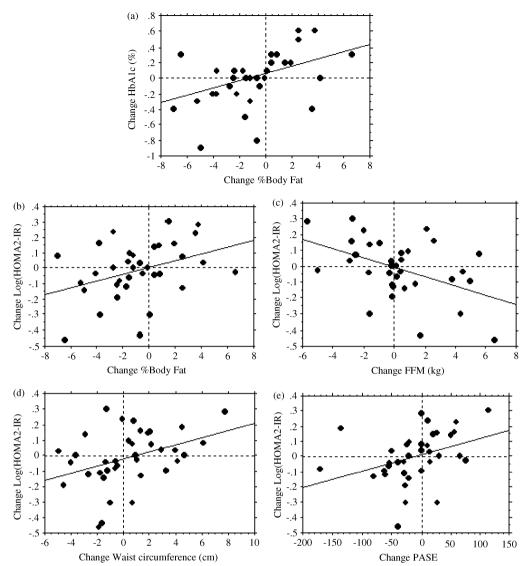


Figure I. Associations between change scores: (a) HbA_{1c} and %Body fat, linear regression analysis: $(r=0.37,\ P=0.03)$; (b) Log(HOMA2-IR) and %BF $(r=0.37,\ P=0.03)$; (c) Log(HOMA2-IR) and waist circumference $(r=0.38,\ P=0.02)$; (d) Log(HOMA2-IR) and PASE score $(r=0.36,\ P=0.03)$; (e) Log(HOMA2-IR).

Energy intake/expenditure

Physical activity levels increased in TC, (17.65 ± 50.96) and decreased in controls $(35.26 \pm 49.52, f = 9.963, P = 0.003)$. However, after adjusting for baseline differences in physical activity, %BF, and social function, this difference was attenuated and no longer significant (P = 0.195).

Daily fat intake decreased over the study period (-15.9% in TC, 0.85% in controls; f=7.761, P=0.0095), with no difference between groups, and similarly, daily caloric intake tended to decline over time (-6.022% in TC, -3.307 in controls; f=4.084, P=0.0529). There were still no group differences, after adjusting for baseline differences in daily fat, caloric intake and waist circumference (data not shown).

Discussion

This study has shown that practicing the TCD form twice a week for 16 weeks does not improve HbA_{1e} or insulin sensitivity 72 h post-exercise in older adults with T2DM. Our results extend those of Thomas *et al.* [3] who also reported no benefits of TC to diabetes control in primarily non-diabetic older adults after a year of thrice-weekly supervised TC practice plus additional unsupervised sessions.

A number of previous studies have demonstrated absolute improvements in HbA_{1c} of 0.74–1.21% after moderate to high-intensity aerobic or resistance training of similar duration to that used in the present study [8, 10, 14]. The mean absolute decrease in HbA_{1c} observed after TC in our study of 0.07%, was not significant statistically or clinically, or even close to changes previously reported after exercise

interventions. Another study examining low-to-moderate intensity exercise in patients aged over 65 years with T2DM also found no change in HbA_{1c} after 16 weeks of aerobic, strength and stretching exercises, for 80 min, three times a week [15]. However a recent non-controlled study observed an absolute decrease in HbA₁₆ of 0.46% after a 12-week, thrice-weekly TC program, where the Cheng style 37 Forms was practiced 1 h per class [16]. Subjects in this study were younger, and did not have as long-standing diabetes as our cohort. There was no mention of the medications taken by these subjects either. Though the statistically significant HbA_{1c} improvement reported by Yeh et al.'s study [16] was statistically significant, it was smaller than effects seen with other forms of robust exercise, and the absence of a control group precludes drawing definitive conclusions from this trial. The difference in results compared to our study may be due to the lack of a control group, the different TC style utilised, the increased frequency of training (3 versus 2 days/week), or the subject characteristics and diabetes duration. Thus, additional, well-designed research needs to be conducted to define the role, if any, of TC in the treatment of T2DM. Perhaps the TCD form may be beneficial for younger subjects, who have greater preservation of insulin secretory reserve, and who are on fewer medications for diabetes. Long-term glucose control was associated with changes in %BF in our trial, and this outcome changed minimally. This lack of a robust effect on body fat may partly explain the absence of improvement in HbA16. Improvements in glucose control have previously been reported following moderate to high-intensity exercise programs without significant weight change [7, 9, 14]. It is likely that the exercise intervention used in this study was not intense enough to induce significant changes to HbA_{1c} or body composition. Our intention was to study the form of TC that is advocated internationally for its benefits on diabetes, not to alter the form prior to testing this hypothesis. It is possible that the very small effect seen ($-0.07 \pm 0.4\%$ absolute change in HbA₁₆ after TC) would have been statistically significant with a larger sample size (n = 57 per group), but the clinical significance of a change of such small magnitude is unlikely. Similarly, insufficient intensity was also the most likely reason for the non-significant findings in the previous study of Yang style TC [3], despite the higher volume of exercise in that study. Reduced body fat is related to induction of a negative energy balance through either increased energy expenditure or decreased energy intake, or both. Diet-induced weight/fat loss improves insulin resistance [17], and exercise may enhance compliance to hypocaloric diets [7]. Although we found modestly reduced dietary fat and caloric intake in both groups, these may not have been great enough to induce robust body composition changes, hence glucose control and insulin resistance were unchanged. It should also be noted that our cohort had relatively good baseline diabetes control, and chronic use of medication. This, in addition to the subjects responding to the study's recruitment campaigns/advertisements, may signify a high motivation of this cohort to improve their diabetes control or health. However our results show that this potential bias had no effect on the primary outcome, as no improvements were observed. Some subjects may have been motivated to change their diet during the study (reflected in decreased reporting of fat/energy intake over time in both groups), and it would thus appear that the changes in body fat which were linked to improved metabolic control may have been secondary to dietary restriction rather than directly related to the exercise undertaken by either group.

Two factors must be considered when interpreting our insulin resistance results: timing of blood draws, and method of assessment. First, it was our intention to adopt a conservative approach by performing the blood tests 72 h after the last exercise bout. This ensured that observed effects of TC on insulin resistance were not overstated by measuring acute effects on glucose and insulin, which would have mostly receded by 72 h after the last exercise bout [7]. It is possible that we missed an effect of TC on insulin resistance that was only present acutely. Future studies could include earlier time-points to assess this possibility. Nevertheless, improved insulin sensitivity has been measured at least 72 h after the last bout of exercise in the majority of recent literature [18, 19], thus, our lack of efficacy stands in contrast to other proven exercise modalities. Second, we estimated insulin resistance with the HOMA2 model rather than the 'gold standard' euglycemic clamp technique [20], and it is possible that subtle changes were missed. However, HOMA2 is closely correlated to the euglycemic clamp method [13]. Additionally, the lack of improvement in insulin resistance demonstrated by the previous TC study [3] using a more sensitive index (intravenous insulin tolerance test, at an unspecified time after the last bout of exercise) further supports the absence of metabolic benefits associated with TC. It is possible that a type II error may explain the lack of statistical significance observed for HbA1c. However, the clinical relevance of changes in HbA_{1c} as small as those associated with TC in this study would be minimal. On the other hand, there was no evidence of improvement in insulin sensitivity at all after TC, making it unlikely that a type II error underlies this finding.

Our findings confirm the importance of body composition with respect to metabolic fitness [7]. Long-term glucose control was associated with reduced %BF, and insulin resistance with lowered %BF, waist circumference and increased FFM. Although both groups lost a small amount of weight and body fat, neither group significantly improved central fat mass or FFM estimates. Change in body mass was not related to either primary outcome, which is consistent with a recent meta-analysis of exercise and diabetes [10], in that weight loss was not associated with reduced HbA₁₆. Visceral adiposity is more closely related to metabolic fitness than body mass, and our results confirm that exercise studies should include estimates of this compartment to define mechanisms of intervention efficacy. The change in body mass in our study was similar to the findings of Boule et al. [10] who reported a 0.9 kg decrease in body mass following moderate-high intensity aerobic or resistance

T. Tsang et al.

training in people with diabetes. Of note, a more frequent (3 days/week with practice in between sessions), and longer (12 months) duration of TC [3] was, similarly, unable to induce favourable body composition changes, or better glucose homeostasis/insulin sensitivity. Increases in FFM are generally only seen with anabolic exercise such as resistance training [3, 8, 21], and our study confirms the relationship between changes in FFM and improved insulin sensitivity reported by others [22]. Four months of TC does not appear to be sufficiently anabolic to improve insulin resistance via this mechanism.

In conclusion, twice-weekly, supervised participation in a 16-week TC program utilising the TCD form did not improve blood glucose control or insulin resistance, measured 72 h after the last exercise bout in older adults with long-standing and pharmacologically treated T2DM. Shifts in body composition (decreased fat, increased muscle) appear to be necessary for metabolic benefits to accrue in this diabetic cohort.

Funding declaration and acknowledgements

This study was funded by the School of Exercise and Sport Science, The University of Sydney, Australia. Blood tests and collection tubes were supplied by our sponsor, Douglass Hanly Moir Pathology. The authors would like to express their gratitude to Douglass Hanly Moir Pathology for all their assistance and generosity; and to the subjects for their dedication and commitment to the study.

Conflicts of interest declaration

There were no conflicts of interests, and the results of the present study do not constitute endorsement of the product by four of the five authors. One of the authors, however, was the creator of the "Tai Chi for Diabetes' form and producer of its video, and is also the founder of a company which distributes these videos and similar products and services.

Key points

- The 'Tai Chi for Diabetes' form was not effective in improving insulin resistance or glucose homeostasis in older adults with long-standing, pharmacologically treated type 2 diabetes.
- The importance of body composition changes relative to improvements in metabolic factors was re-confirmed.

Supplementary data

Supplementary data for this article is available online at http://ageing.oxfordjournals.org.

References

- 1. Bjork S. The cost of diabetes and diabetes care. Diabetes Res Clin Pract 2001; 54(Suppl. 1): S13-8.
- Mokdad A, Ford E, Bowman B et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. JAMA 2003; 289: 76–9.
- **3.** Thomas GN, Hong AWL, Tomlinson B *et al.* Effects of Tai Chi and resistance training on cardiovascular risk factors in elderly Chinese subjects: a 12-month longitudinal, randomized, controlled intervention study. Clin Endocrinol 2005; 63: 663–9.
- Lam P. Tai Chi for diabetes: designed to help prevent and control diabetes. On East Action Videos. Narwee Tai Chi Productions 2001.
- **5.** Fiatarone MA, O'Neill EF, Ryan ND *et al.* Exercise training and nutritional supplementation for physical frailty in very elderly people. N Engl J Med 1994; 330: 1769–75.
- Butterworth DE, Nieman DC, Perkins R et al. Exercise training and nutrient intake in elderly women. J Am Diet Assoc 1993; 93: 653-7.
- **7.** Albright A, Franz M, Hornsby G *et al.* American college of sports medicine position stand. Exercise and type 2 diabetes. Med Sci Sports Exerc 2000; 32: 1345–60.
- **8.** Castaneda C, Layne JE, Munoz-Orians L *et al.* A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. Diabetes Care 2002; 25: 2335–41.
- **9.** Boule NG, Kenny GP, Haddad E *et al.* Meta-analysis of the effect of structured exercise training on cardiorespiratory fitness in Type 2 diabetes mellitus. Diabetologia 2003; 46: 1071–81.
- 10. Boule NG, Haddad E, Kenny GP et al. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA 2001; 286: 1218–27.
- **11.** Lukaski HC, Bolonchuk WW, Hall CB *et al.* Validation of tetrapolar bioelectrical impedance method to assess human body composition. J Appl Physiol 1986; 60: 1327–32.
- **12.** Washburn RA, Smith KW, Jette AM *et al.* The physical activity scale for the elderly (PASE): development and evaluation. J Clin Epidemiol 1993; 46: 153–62.
- **13.** Wallace TM, Levy JC, Matthews DR. Use and abuse of HOMA modeling. Diabetes Care 2004; 27: 1487–95.
- **14.** Willey K, Fiatarone Singh M. Battling insulin resistance in elderly obese people with type 2 diabetes—bring on the heavy weights. Diabetes Care 2003; 26: 1580–8.
- **15.** Tessier D, Menard J, Tamas F *et al.* Effects of aerobic physical exercise in the elderly with type 2 diabetes mellitus. Arch Gerontol Geriatr 2000; 31: 121–32.
- **16.** Yeh SH, Chuang H, Lin LW *et al.* Tai chi chuan exercise decreases A1C levels along with increase of regulatory T-cells and decrease of cytotoxic T-cell population in type 2 diabetic patients. Diabetes Care 2007; 30: 716–8.
- 17. Carroll S, Dudfield M. What is the relationship between exercise and metabolic abnormalities? A review of the metabolic syndrome. Sports Med 2004; 34: 371–418.
- Hughes VA, Fiatarone MA, Fielding RA et al. Exercise increases muscle GLUT-4 levels and insulin action in subjects with impaired glucose tolerance. Am J Physiol 1993; 264: E855–62.

- **19.** Short K, Vittone J, Bigelow M *et al.* Impact of aerobic exercise training on age-related changes in insulin sensitivity and muscle oxidative capacity. Diabetes 2003; 52: 1888–96.
- **20.** Bonora E, Targher G, Alberiche M *et al.* Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity—studies in subjects with various degrees of glucose tolerance and insulin sensitivity. Diabetes Care 2000; 23: 57–63.
- **21.** Nichols JF, Omizo DK, Peterson KK *et al.* Efficacy of heavy-resistance training for active women over sixty: muscular strength, body composition, and program adherence. J Am Geriatr Soc 1993; 41: 205–10.
- **22.** Stewart KJ, Bacher AC, Turner K *et al.* Exercise and risk factors associated with metabolic syndrome in older adults. Am J Prev Med 2005; 28: 9–18.
- Dallal GE. (2003); Randomization.com. Retrieved 15/3/2004, from www.randomization.com
- **24.** Hermans MP, Pepersack TM, Godeaux LH *et al.* Prevalence and determinants of impaired glucose metabolism in frail elderly patients: the Belgian Elderly Diabetes Survey (BEDS). J Gerontol A Biol Sci Med Sci 2005; 60: 241–7.

- **25.** Hutchinson F. The Fred Hutchinson Cancer Research Center Food Frequency Questionnaire, Version 01.6.88. Seattle, WA: Cancer Research Center, 1988.
- **26.** Nutrition Coordinating Center. Minnesota Nutrition Data System software (Version 10A). Minneapolis, MN: University of Minnesota.
- 27. Epidemiology and Dietary Assessment Research Program. Nutrient Database (Version 25). Boston, MA: USDA Human Nutrition Research Center on Aging, Tufts University, 1995.
- **28.** Ware JE Jr, Snow KK, Kosinski M *et al.* SF-36 Health Survey: Manual and Interpretation Guide. Lincoln, RI: QualityMetric Incorporated, 1993, 2000.
- 29. National Cholesterol Education Program. Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): Final Report. Bethesda, MD: National Heart, Lung, and Blood Institute, 2002.

Received 19 July 2006; accepted in revised form 27 June 2007