Clinical Medicine Insights: Women’s Health

ORIGINAL RESEARCH

Effects of Martial Arts Exercise on Body Composition, Serum Biomarkers and Quality of Life in Overweight/Obese Premenopausal Women: A Pilot Study

Ming-Chien Chyu¹, Yan Zhang², Jean-Michel Brismée³, Raul Y. Dagda⁴, Eugene Chaung⁵, Vera Von Bergen⁵, Susan Doctolero⁶ and Chwan-Li Shen⁴

¹Graduate Healthcare Engineering, Whitacre College of Engineering, Texas Tech University, Lubbock, TX. ²Department of Family and Community Medicine, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX. ³Department of Rehabilitation Science, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX. ⁴Department of Pathology, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX. ⁵School of Medicine, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX. ⁶Clinical Research Institute, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX.
Corresponding author email: leslie.shen@ttuhsc.edu

Abstract: Various exercise interventions have been shown to benefit weight control and general health in different populations. However, very few studies have been conducted on martial arts exercise (MAE). The objective of this pilot study is to evaluate the efficacy of 12 weeks of MAE intervention on body composition, serum biomarkers and quality of life (QOL) in overweight/obese premenopausal women. We found that subjects in the MAE group did not lose body weight, while they significantly decreased fat-free mass and muscle mass as compared to those in the control group, who demonstrated an increase in these parameters. The MAE group demonstrated an increase in serum IGF-I concentration, but no change in others. MAE may be a feasible and effective approach to improve body composition and QOL in overweight/obese premenopausal women. Our study underscores the need for further studies using larger samples to establish possible benefits of MAE in various populations.

Keywords: exercise, obesity, body composition, bone biomarker, quality of life, women

Clinical Medicine Insights: Women’s Health 2013:6 55–65

doi: 10.4137/CMWH.S11997

This article is available from http://www.la-press.com.

© the author(s), publisher and licensee Libertas Academica Ltd.

This is an open access article published under the Creative Commons CC-BY-NC 3.0 license.
Introduction

American women (mainly premenopausal women) typically gain 0.5–1 kg yearly.\(^1,2\) Such weight gain, mainly in terms of fat gain,\(^3,5\) leads to an increased prevalence of unhealthy weight and obesity with age.\(^5\) Excess adiposity, particularly excess abdominal fat, has been shown to increase the risk of morbidity related to a number of health conditions, including cardiovascular diseases and type 2 diabetes.\(^6\) To achieve weight control, both the prevention of further weight gain as well as the stimulation of weight loss are important for overweight/obese women.

There is an increasing body of evidence suggesting a connection between chronic inflammation and the development of obesity.\(^7,8\) A study shows that significant decrease of body weight lowers C-reactive protein (CRP), the prototypic biomarker for inflammation.\(^9\) However, substantial weight loss may cause bone loss and resulting in bone fragility in middle aged and older obese women,\(^10,11\) irrespective of current weight or intention to lose weight.\(^12\) Weight loss of 5–10\% is associated with a decrease in bone mass and an increase in bone resorption in obese women.\(^13\) Therefore, it is important to sustain optimal bone health during weight loss.

Although the etiology of the rising prevalence of obesity is still not well understood, lower physical activity level has been linked to greater weight gain,\(^14\) and higher physical activity level to greater weight loss and better weight-loss maintenance.\(^15\) Various exercise programs have been shown to benefit weight control, promote weight loss,\(^16,17\) prevent percentage body fat and intra-abdominal fat increase,\(^17,19\) and improve lipid profile,\(^17,18\) physical function,\(^20\) and quality of life (QOL).\(^21\)

Among various types of exercises, martial arts exercise (MAE), a form of aerobic exercise, has been gaining popularity. Unlike traditional martial arts involving full contact sparring as an important part of the training, MAE can be implemented as a non-competitive, safe, individual exercise without any body contact between participants. The advantages of MAE over other types of exercises include the following: (i) it is one of the highest calorie-consumption exercises\(^22\); (ii) it combines trainings in endurance/aerobic, strength, stretch/flexibility, and balance/coordination\(^23\); (iii) its intensity is easily adjustable for different levels of physical capability; (iv) it requires no equipment; (v) it can be practiced in a limited space. A recent review established a relation between karate and low body fat in male athletes, but studies on females are scarce.\(^24\) One of the most studied martial arts for health related outcomes is Tai Chi, which has been shown to improve quality of life,\(^25\) reduce stress\(^26\) and be cardio protective.\(^27\) However, Tai Chi is an “internal” (or “soft”) style of martial art that features slow, gentle, and flowing movements and are different than the commonly known martial arts (“external” or “hard” styles) characterized by fast and explosive movements with a focus on physical strength and agility\(^28\) that the current MAE is based on. No study has been conducted on the impact of MAE in overweight/obese premenopausal women.

This pilot study was designed to evaluate the effects of 12 weeks of MAE intervention on body composition, serum bone biomarkers and QOL in overweight/obese premenopausal women. In addition, obesity-related parameters including lipid profiles, insulin-like growth factor-I (IGF-I), leptin, and CRP were also assessed. The population of overweight/obese premenopausal women was selected because they are at risk of continued unhealthy weight levels/obesity beyond menopause, and because of expected better adherence to the MAE intervention due to possible higher interest and physical capability than postmenopausal women. We hypothesized that MAE exercise would improve body composition, favor bone turnover biomarkers (increasing bone formation and decreasing bone resorption), reduce inflammation, and benefit lipid profiles and QOL in the overweight/obese premenopausal women.

Methods

Experimental design

This was a 12-week assessors-blinded pilot randomized controlled trial to investigate the efficacy of MAE on obesity- and bone remodeling-related parameters in overweight and obese premenopausal women. The outcome measures were body composition, bone biomarkers including osteocalcin (bone formation biomarker) and C-terminal telopeptide (CTX, bone resorption biomarker), serum cytokines including IGF-I, leptin, and high-sensitivity CRP (hs-CRP), serum lipid profiles, and QOL. The assessors were blinded to the participant’s treatment group.
assignment. Outcome data were collected at the baseline, 6 and 12 weeks. Dietary intake and physical activity data were collected at baseline using a 3-day food intake survey (Food Processor SOL edition, version 10.0.0, ESHA Research, Salem, OR) and the Godin Leisure-time Exercise Questionnaire, respectively.

Since this is the first study to explore MAE’s impact on overweight and obese premenopausal women and no existing data is available, we used Cohen’s effect size index and estimated the required sample size to 50 subjects. With an anticipated attrition rate of 20% over 12 weeks of intervention, the final sample size would be 40 subjects (n = 20 per group). This sample size may detect a moderate to large effect size at an α level of 0.05 and 0.80 power.

Participants
Participants were recruited through local television, newspaper advertisements and flyers. Inclusion criteria were premenopausal women (≥30 years old) with regular menstrual cycle, body mass index (BMI) ≥25 kg/m² according to the WHO’s definition of overweight, stable weight, non-smoking, no previously diagnosed cardiovascular, kidney or liver disorders, or diabetes mellitus, and sedentary daily behaviors (≤20 min of aerobic exercise, 2 times/week).

Exclusion criteria were the following: (i) history of metabolic bone diseases or treatments that might have influenced bone turnover; (ii) use of any weight loss medication, supplement, or program within the last 3 months, (iii) a history of any weight-reducing surgery, (iv) cancers, except for skin cancer, (v) taking any lipid-lowering drug or medication, (vi) uncontrolled intercurrent or terminal illness, or physical condition that would be a contraindication to exercise, (vii) unwillingness to accept randomization, and (viii) pregnancy based on self-report.

Qualified participants gave their written, informed consent and were then randomly assigned to the control and the MAE groups at 1:1 ratio. All participating subjects were asked to maintain their regular diet, medication if any, normal daily activities and lifestyle throughout the study period. At 6 and 12 weeks, subjects were asked to document any deviation of dietary intake and physical activity from the baseline. This study was approved by the local Institutional Review Board.

Treatments
Participants in the MAE group met for 60 minutes per session, for 3 sessions per week on non-consecutive days, for 12 weeks. Each 60-minute exercise session consisted of 10 minutes of warm-up exercise, 40 minutes of MAE, and 10 minutes of cool-down exercise. The MAE program, developed by Chyu, was structured, systematic, comprehensive, and suitable for a general population of different age groups and physical conditions. The curriculum featured a non-competitive, non-contact, safe and fun personal/group exercise based on traditional martial arts training, covering a wide spectrum of techniques of hand strikes, kicks, elbow strikes, knee strikes, blocks, their combinations, take downs/throws, and self-defense, in addition to warm-up and cool-down. There are different styles of martial arts, but many of them share common techniques. The current MAE covers these common techniques taught in major styles of martial arts such as Taekwondo, Karate, Kung Fu, boxing, Kickboxing, Muay Thai, Judo, Aikido, and Jujitsu. MAE class attendance for each participant was recorded to calculate the attendance rate as the number of sessions completed divided by the total number of sessions prescribed.

Participants wore commercial heart rate monitors to quantify exercise intensity and to ensure a moderate-intensity exercise workload. Recent reviews of clinical evidence indicated that moderate-intensity activity effectively prevents obesity. For moderate-intensity physical activity, the target heart rate should be 50 to 70% of the maximum heart rate which was estimated by subtracting the age from 220.

The heart rate data during exercise always indicated a moderate-intensity exercise workload, and the heart rate monitors were set to beep if the heart rate reading falls outside the target range. Participants who were in the control group were asked to continue their regular activities throughout the study period.

Adverse events
Participants were questioned about the occurrence of any adverse event, including pain, malaise, and more serious conditions that may be related to the MAE intervention, before and after each MAE session. The MAE instructor also monitored participants during the exercise sessions for any sign of adverse event. All observed or volunteered adverse events in both groups were recorded.
Measurement of outcomes

Body composition including body weight (BW), fat mass (FM), fat-free mass (FFM, the combined non-fat body mass including muscle, bone, skin, organs, etc.), total body water (TBW), musculoskeletal muscle mass (MM), and bone mass (BM) were determined through bioimpedance measurement (SC-331S Body Composition Analyzer, Tanita Corporation of America, Inc., Arlington Height, IL, USA). Body height was measured with a stadiometer installed on the bioimpedance machine, and body mass index (BMI) was calculated based on body height and mass.

Serum osteocalcin, CTX, and IGF-I (Immuno-diagnostic Systems, Inc., Scottsdale, AZ), leptin (GenWay Biotech, Inc., San Diego, CA), and hsCRP (ALPCO Diagnostics, Salem, NH) were measured using respective kits. Serum lipid profile including triglyceride (TG), total cholesterol, and high-density lipoprotein (HDL) were analyzed by Quest Diagnostics (Irving, TX). Low-density lipoprotein (LDL) was calculated using a formula (LDL = total cholesterol – HDL – TG/5.0 (mg/dL)).

The SF-36 survey (version 2) was employed to assess general health status and QOL. It consisted of 8 domains: physical functioning (PF), role limitation due to physical health (RP), bodily pain (BP), general health (GH), vitality (VT), social function (SF), role of limitation due to emotional health (RE), and mental health (MH) in the conduct of daily activities.

Statistical analysis

Statistical analysis was performed using SPSS Statistics (version 20, IBM Corp). Descriptive statistics (e.g., normality) for each variable at the baseline were determined to justify parametric or non-parametric methods. All continuous variables were expressed as means ± standard deviation (SD) unless otherwise specified. Therefore, only the baseline and the end-point data were analyzed and presented. Preliminary findings showed no statistically significant differences of demographic characteristics between the subjects who withdrew from the study and those who completed the study; hence, we only presented and assessed the results of those who completed the study based on a per-protocol analysis. Independent t-tests were conducted to compare the differences in outcomes between the MAE and the control groups at the baseline and 12 weeks (end) for preliminary bi-variate assessment. The change of each outcome between the baseline and the end-point was calculated and also analyzed by independent t-test. All outcome changes were analyzed using MANOVA procedure to determine differences between groups while controlling for age and BMI. Results were considered significant if p < 0.05.

Results

Figure 1 shows the study flow chart of participant recruitment and retention. 24 subjects participated in the control group and 23 in the MAE group at the baseline. 14 subjects dropped out (7 in control, 7 in MAE) before completing the intervention, due to family, job, personal, and schedule issues. Baseline characteristics were similar between the control and the MAE participants who completed the study (Table 1). The adherence rate for MAE classes was 83.4% (ranging from 50% to 100%). The adverse events due to study treatment reported by the participants included minor muscle soreness, and shoulder and knee pain only during the first two weeks of intervention.

There was no significant difference in BMI and body composition parameters including BW, FM, FFM, TBW, MM, and BM between the control and the MAE groups at baseline and 12 weeks (Table 2). The pre-to-post changes in FFM (p = 0.007) and MM (p = 0.022) were significantly different between the 2 groups, with a decrease in the MAE group as compared to an increase in the control group (Table 2).

Table 3 shows no statistical difference in the serum biomarkers and lipid profiles between the 2 groups at baseline or 12 weeks. Over the 12-week intervention, there was no significant difference in the pre-to-post change of any serum biomarker and lipid profiles between the 2 groups, except the pre-to-post change in serum IGF-I (p = 0.047), with a decrease in the control group whereas an increase in the MAE group.

Data in Table 4 show no statistically significant differences in any SF-36 subscale between the 2 groups at baseline. At the end of the intervention, there was no significant difference in PF, BP, or GH subscales between the 2 groups, while the RP (p = 0.03), VT (p = 0.03), RE (p = 0.03), and MH (p = 0.02) scores were higher in the MAE group than control. The pre-to-post changes in GH (p = 0.001),
VT ($p = 0.009$), and MH ($p = 0.006$) were significantly different between the 2 groups, with a decrease in the control group as compared to an increase in the MAE group.

**Discussion**

This pilot study was the first to explore the MAE intervention in overweight and obese premenopausal women. Results of this 12-week pilot study suggest that MAE tends to modify body composition, elevates serum IGF-I levels, and improves QOL.

According to the 3-day dietary intake data collected at the baseline, there was no difference in caloric intakes ($1058 \pm 524$ Kcal for the control group versus (vs.) $1062 \pm 645$ Kcal for the MAE group) and major macronutrients between the control and MAE groups ($p > 0.05$) (data not shown). At the end of study, all subjects self-reported that they have maintained their

**Table 1. Baseline characteristics of study population.**

| Variables | Control | MAE | *p value |
|-----------|---------|-----|----------|
|           | Enrolled | Completed | Enrolled | Completed |          |          |
| Age (years) | 41.0 ± 6.7 | 41.7 ± 6.8 | NS | 40.2 ± 5.7 | 41.4 ± 5.5 | NS | NS |
| Weight (kg) | 97.6 ± 22.3 | 99.3 ± 25.0 | NS | 96.1 ± 15.2 | 96.1 ± 17.1 | NS | NS |
| Height (m) | 1.65 ± 0.08 | 1.64 ± 0.07 | NS | 1.63 ± 0.06 | 1.64 ± 0.06 | NS | NS |
| BMI (kg/m²) | 35.60 ± 7.13 | 36.24 ± 7.72 | NS | 36.14 ± 5.42 | 35.74 ± 5.97 | NS | NS |

Data were presented as mean ± standard deviation. *p value was for a comparison between the enrolled participants and the completed participants in the same intervention arm by independent $t$-test. *p value was for a comparison between the completed control and completed MAE groups by independent $t$-test. NS, no statistical difference ($p > 0.05$).
regular diet, medication, normal daily activities and lifestyle (except for MAE intervention in the MAE group) throughout the study period. Therefore, the differences in the outcome parameters should be due to the intervention.

In the present study, there was no difference in any body composition parameter between the control and the MAE groups at either the baseline or after 12 weeks. On the other hand, MAE intervention did not result in weight loss, as shown by a pre-to-post (0–12 week) decrease in BW (Table 2), possibly due to decreases in FFM, TBW, MM and BM, although change in TBW and BM did not reach statistical significance. The discrepancy between the data collected at each time point and the data derived at different times (12 week–0 week) could be due to the small sample size with a wide range of caloric intakes (435–2126 Kcal in the control group vs. 322–2711 Kcal in the MAE group) among subjects, reducing the sensitivity to detect the statistical differences at each collection time. Our data also show that the control group gained BW along with increased FFM, TBW, MM, and BM, while the MAE group lost BW without a significant change in FFM. Riesco et al.\(^{21}\) also reported that a 16-week walking program did not affect lean mass in both premenopausal and early postmenopausal obese women.

Low-grade chronic inflammation is one of the key metabolic alterations linked to excessive caloric intake and adiposity,\(^{35}\) and systemic low-grade inflammation is related to both circulating and adipose tissue tumor necrosis factor-\(\alpha\) (TNF-\(\alpha\)), leptin, and

### Table 2. Data of body composition.

| Outcome                  | Control (n = 17) | MAE (n = 16) | \(p\) value |
|--------------------------|------------------|--------------|-------------|
| **BMI (kg/m\(^2\))**     |                  |              |             |
| Baseline                 | 36.34 ± 7.66     | 36.12 ± 5.90 | 0.92        |
| 12 weeks                 | 36.24 ± 7.72     | 35.74 ± 5.97 | 0.83        |
| Change (0–12 week)       | −0.18 ± 3.9      | −1.01 ± 4.32 | 0.56        |
| **Body weight (BW), kg** |                  |              |             |
| Baseline                 | 97.6 ± 22.3      | 96.1 ± 15.2  | 0.85        |
| 12 weeks                 | 99.3 ± 25.0      | 96.1 ± 17.1  | 0.67        |
| Change (0–12 week)       | 0.46 ± 1.89      | −0.96 ± 3.84 | 0.093       |
| **Fat mass (FM), kg**    |                  |              |             |
| Baseline                 | 47.25 ± 17.82    | 45.06 ± 10.68| 0.67        |
| 12 weeks                 | 46.71 ± 18.43    | 44.05 ± 11.69| 0.62        |
| Change (0–12 week)       | −0.67 ± 2.11     | −1.19 ± 2.88 | 0.55        |
| **Fat-free mass (FFM), kg** |            |              |             |
| Baseline                 | 51.13 ± 6.77     | 51.98 ± 6.85 | 0.72        |
| 12 weeks                 | 52.58 ± 6.85     | 51.90 ± 6.31 | 0.77        |
| Change (0–12 week)       | 1.33 ± 1.35      | −0.004 ± 1.32| 0.007       |
| **Total body water (TBW), kg** |          |              |             |
| Baseline                 | 36.07 ± 3.91     | 37.70 ± 5.52 | 0.34        |
| 12 weeks                 | 37.06 ± 3.76     | 36.51 ± 7.21 | 0.78        |
| Change (0–12 week)       | 1.30 ± 1.41      | −1.42 ± 5.98^ | 0.086     |
| **Muscle mass (MM), kg** |                  |              |             |
| Baseline                 | 48.55 ± 6.43     | 49.36 ± 6.51 | 0.72        |
| 12 weeks                 | 49.94 ± 6.51     | 48.70 ± 6.79 | 0.60        |
| Change (0–12 week)       | 1.34 ± 1.34      | −0.55 ± 2.93 | 0.022       |
| **Bone mass (BM), kg**   |                  |              |             |
| Baseline                 | 2.58 ± 0.34      | 2.62 ± 0.34  | 0.78        |
| 12 weeks                 | 2.64 ± 0.33      | 2.63 ± 0.31  | 0.95        |
| Change (0–12 week)       | 1.19 ± 1.64      | 0.26 ± 1.14^ | 0.069       |

Data are presented as mean ± standard deviation. Data were analyzed by independent t-test at each collection time.

^Indicated \(p < 0.05\), ^^indicated 0.05 < \(p\) < 0.1.
Table 3. Data of serum biomarkers and lipid profiles.

| Outcome                        | Control (n = 17) | MAE (n = 16) | p value |
|--------------------------------|-----------------|-------------|---------|
| Osteocalcin, ng/mL             |                 |             |         |
| Baseline                       | 13.25 ± 5.24    | 10.32 ± 3.21^^ | 0.06    |
| 12 weeks                       | 12.71 ± 4.62    | 11.11 ± 5.23 | 0.72    |
| Change (0–12 week)             | 1.99 ± 32.00    | 15.95 ± 28.29 | 0.19    |
| CTX, pg/mL                     |                 |             |         |
| Baseline                       | 253.32 ± 117.85 | 230.68 ± 109.47 | 0.57    |
| 12 weeks                       | 251.67 ± 112.50 | 248.06 ± 104.33 | 0.92    |
| Change (0–12 week)             | −4.89 ± 35.27   | 5.82 ± 21.93 | 0.30    |
| IGF-I, µg/L                    |                 |             |         |
| Baseline                       | 89.41 ± 28.99   | 81.16 ± 26.01 | 0.39    |
| 12 weeks                       | 82.70 ± 25.34   | 86.82 ± 25.64 | 0.64    |
| Change (0–12 week)             | −5.65 ± 18.38   | 9.37 ± 22.78^ | 0.047   |
| Leptin, ng/mL                  |                 |             |         |
| Baseline                       | 49.08 ± 14.79   | 45.34 ± 15.15 | 0.47    |
| 12 weeks                       | 50.37 ± 20.93   | 44.15 ± 13.24 | 0.31    |
| Change (0–12 week)             | 1.91 ± 28.91    | −0.20 ± 21.58 | 0.81    |
| hs-CRP, pg/mL                  |                 |             |         |
| Baseline                       | 10460.8 ± 7293.8 | 10031.1 ± 6519.7 | 0.86    |
| 12 weeks                       | 11340.6 ± 8001.9 | 8604.4 ± 4939.0 | 0.25    |
| Change (0–12 week)             | 26.71 ± 85.26   | −7.32 ± 28.91 | 0.14    |
| Triglyceride, mg/dL            |                 |             |         |
| Baseline                       | 142.1 ± 65.6    | 156.0 ± 64.10 | 0.54    |
| 12 weeks                       | 139.5 ± 63.9    | 147.4 ± 65.6 | 0.72    |
| Change (0–12 week)             | 2.7 ± 33.6      | −4.9 ± 24.3 | 0.46    |
| Total cholesterol, mg/dL       |                 |             |         |
| Baseline                       | 198.1 ± 25.9    | 202.9 ± 45.8 | 0.17    |
| 12 weeks                       | 193.0 ± 28.9    | 200.1 ± 36.8^^ | 0.07    |
| Change (0–12 week)             | −2.4 ± 9.1      | −0.3 ± 10.3 | 0.11    |
| LDL, mg/dL                     |                 |             |         |
| Baseline                       | 119.4 ± 24.6    | 123.4 ± 44.5 | 0.74    |
| 12 weeks                       | 116.8 ± 28.4    | 123.9 ± 37.0 | 0.54    |
| Change (0–12 week)             | −2.0 ± 14.1     | 2.9 ± 14.9 | 0.33    |
| HDL, mg/dL                     |                 |             |         |
| Baseline                       | 50.2 ± 12.8     | 48.3 ± 16.4 | 0.71    |
| 12 weeks                       | 48.2 ± 10.9     | 46.8 ± 13.0 | 0.73    |
| Change (0–12 week)             | −1.8 ± 16.8     | −1.1 ± 14.6 | 0.93    |

Data were presented as mean ± standard deviation. Data were analyzed by independent t-test at each collection time. ^Indicated p < 0.05, ^^indicated 0.05 < p < 0.1.

Abbreviations: IGF-I, insulin-like growth factor-I; CTX, C-terminal telopeptide; hs-CRP, high-sensitivity C-reactive protein; LDL, low density lipoprotein; HDL, high density lipoprotein.

interleukine (IL-6) levels in obese women. Visceral fat is a source of several molecules such as leptin, adiponectin, TNF-α, and IL-6, that are collectively called adipokines. Recent evidence shows that visceral fat in particular was associated with chronic inflammation, whereas subcutaneous fat may be a “metabolic sink” that prevents accumulation of visceral fat and thereby limits inflammation. Ziccardi et al reported that after 1 year of a multidisciplinary program of weight reduction (diet, exercise, behavioral counseling), all obese premenopausal women lost at least 10% of original body weight and such sustained weight loss was associated with reduction of inflammatory cytokines (i.e., TNF-α and IL-6). Fisher et al further demonstrated that the effect of weight loss was mediated by changes in total fat mass or intra-abdominal adipose tissue, and the decrease in total fat mass was strongly associated with decrease in markers of inflammation.
### Table 4. Data of quality of life.*

| Outcome               | Control (n = 17) | MAE (n = 16) | p value |
|-----------------------|-----------------|--------------|---------|
| **Physical function (PF)** |                 |              |         |
| Baseline              | 87.33 ± 18.88   | 88.13 ± 11.38 | 0.88    |
| 12 weeks              | 89.06 ± 15.62   | 92.00 ± 5.91  | 0.50    |
| Change (0–12 week)    | 10.93 ± 54.44   | 6.78 ± 13.67  | 0.77    |
| **Role physical (RP)** |                 |              |         |
| Baseline              | 90.83 ± 15.99   | 92.57 ± 10.99 | 0.72    |
| 12 weeks              | 78.12 ± 28.50   | 95.41 ± 7.26a | 0.03    |
| Change (0–12 week)    | −12.50 ± 34.33  | 3.98 ± 13.45 ^^ | 0.09    |
| **Bodily pain (BP)**  |                 |              |         |
| Baseline              | 82.60 ± 17.49   | 79.68 ± 17.59 | 0.64    |
| 12 weeks              | 71.25 ± 26.55   | 82.20 ± 12.68 | 0.15    |
| Change (0–12 week)    | −5.34 ± 25.60   | 6.13 ± 27.89  | 0.25    |
| **General health (GH)** |                 |              |         |
| Baseline              | 71.53 ± 15.06   | 63.18 ± 17.25 | 0.16    |
| 12 weeks              | 66.12 ± 19.95   | 74.06 ± 17.52 | 0.24    |
| Change (0–12 week)    | −10.32 ± 18.00  | 19.67 ± 23.48 ^ | 0.001   |
| **Vitality (VT)**     |                 |              |         |
| Baseline              | 59.58 ± 17.17   | 48.43 ± 17.60 ^^ | 0.08    |
| 12 weeks              | 52.73 ± 24.14   | 69.58 ± 17.81 ^ | 0.03    |
| Change (0–12 week)    | −12.04 ± 30.99  | 69.42 ± 107.45 ^ | 0.009   |
| **Social function (SF)** |                 |              |         |
| Baseline              | 85.83 ± 18.21   | 73.43 ± 29.53 | 0.17    |
| 12 weeks              | 77.34 ± 20.00   | 90.00 ± 17.80 ^^ | 0.07    |
| Change (0–12 week)    | −7.93 ± 22.80   | 73.00 ± 182.85 | 0.11    |
| **Role emotional (RE)** |                 |              |         |
| Baseline              | 83.33 ± 18.36   | 81.77 ± 20.23 | 0.82    |
| 12 weeks              | 81.77 ± 20.46   | 95.00 ± 10.81 ^ | 0.03    |
| Change (0–12 week)    | −1.09 ± 24.96   | 26.84 ± 51.81 ^ | 0.07    |
| **Mental health (MH)** |                 |              |         |
| Baseline              | 73.33 ± 12.77   | 67.50 ± 17.88 | 0.30    |
| 12 weeks              | 69.69 ± 21.56   | 84.00 ± 7.60 ^ | 0.02    |
| Change (0–12 week)    | −4.88 ± 27.19   | 31.89 ± 38.76 ^ | 0.006   |

Data were presented as mean ± standard deviation. Data were analyzed by independent t-test at each collection time.

*SF-36 scales were scored from 1 to 100, with a higher score representing a better functioning. A positive change in SF-36 scores from previous time point indicated improvement of symptoms, whereas a negative change indicated worsening of symptoms.

^Indicated p < 0.05, ^^indicated 0.05 < p < 0.1.

(i.e., TNF-α, CRP) in overweight premenopausal women, regardless of intervention assignments (diet, diet + aerobic, or diet + resistance training).

In the present study, after 12 weeks, the serum hs-CRP level in the MAE group decreased, accompanied with a decrease in FM, in agreement with Fisher et al.40 On the other hand, the hs-CRP level in the control group increased. However, the difference in the pre-to-post change of hs-CRP level between the 2 groups did not reach statistical significance (p = 0.14). The difference between the 2 groups in the pre-to-post change in FM was not statistically significant, which may explain why the pre-to-post change in hs-CRP was not significantly different between the 2 groups in the current study.

One of the purposes in this study was to investigate the impact of short-term MAE on bone turnover biomarkers. Lester et al41 reported that 8 weeks of physical training increased biomarkers of bone formation without substantial alterations in the markers of bone resorption in inactive young women. Villarreal et al42 found that a 1-year weight exercise program
increased bone turnover biomarkers without BMD changes in overweight women. However, the present study shows no change in bone turnover biomarkers in overweight and obese premenopausal women after 12 weeks of MAE intervention. Such difference in results may be due to the age of participants (41 years in our study vs. 20 years in the study by Lester et al41 and 57 years in the study by Villareal et al42) and length of study. Furthermore, the finding of no impact on bone turnover biomarkers in the present study suggests no deleterious effect of MAE on bone biomarkers. However, a long-term study with a larger sample size is needed to better understand the effects of MAE on bone turnover, bone mass, and their relation with weight loss in premenopausal overweight and obese women.

Evidence indicates that the growth hormone/IGF pathway plays an important role in muscle hypertrophy and strength gain resulting from exercise in adults.43 In the present study, we did not observe any differences in IGF-I concentration between MAE and control groups after the intervention. Such a result is consistent with reports from Arikawa44 who found that 16 weeks of exercise had no effect on circulating IGF-I, insulin, and glucose in young women (average age 25 years with BMI range 18.5–40 kg/m2). On the other hand, a significant difference in the pre-to-post change in serum IGF-I between the MAE and control groups was observed in the current study, with a decrease in the control group but an increase in the MAE group. MAE may increase hepatic production of IGF-I followed by elevated circulating IGF-I through the secretion of growth hormone directly or indirectly.45 However, circulating growth hormone was not measured in the present study. Nevertheless, such a greater increase in IGF-I in the MAE group agrees with findings from Borst et al16 that during the first 13 weeks of resistance training, circulating IGF-I increased in both men and women (mean age = 37 ± 7 years), and no further increase afterward.

Previous studies show that weight loss and improved physical condition by diet or exercise can bring forth significant health and psychological benefits including QOL in obese premenopausal women.45,46 The present results suggest that 12 weeks of MAE benefit overweight and obese premenopausal women in terms of improved QOL. Such results appear to be consistent with exercise studies reported by Garcia-Martinez et al47 in women with fibromyalgia, and by Adamsen et al48 in cancer patients undergoing chemotherapy.

The present pilot study is limited in its small sample size and short study period. The mid-point data were not analyzed due to a very small sample size. Based on the 3-day food intake results, the wide variation range of calcium intake (91–1186 mg for control group vs. 121–429 mg for MAE) could have potentially interfered with bone turnover biomarkers. Body composition parameters including FM, FFM, MM, and BM were determined indirectly through bioimpedance analysis, which has been validated but may have measurement errors.49

**Conclusion**

The results of this pilot study suggest that MAE for 12 weeks (3 hours/week) is a feasible exercise intervention for overweight and obese premenopausal women, and may have positive effects on weight control and changes in circulating IGF-I. MAE also significantly improves QOL in terms of role physical, vitality, role emotional, and mental health in the study population.

**Author Contributions**

Conceived and designed the experiments: MCC, JMB, CLS. Analyzed the data: YZ. Wrote the first draft of the manuscript: MCC, CLS, YZ. Contributed to the writing of the manuscript: JMB. Agree with manuscript results and conclusions: MCC, YZ, JMB, RYD, EC, VVB, SD, CLS. Jointly developed the structure and arguments for the paper: JMB, YZ, MCC. Made critical revisions and approved final version: MCC, JMB, YZ, CLS. All authors reviewed and approved of the final manuscript.

**Funding**

This study was supported by Laura W. Bush Institute for Women’s Health and University Medical Center Women’s Innovation Fund, Texas Tech University Health Sciences Center.

**Competing Interests**

Authors disclose no potential conflicts of interest.
Disclosures and Ethics
As a requirement of publication the authors have provided signed confirmation of their compliance with ethical and legal obligations including but not limited to compliance with ICMJE authorship and competing interests guidelines, that the article is neither under consideration for publication nor published elsewhere, of their compliance with legal and ethical guidelines concerning human and animal research participants (if applicable), and that permission has been obtained for reproduction of any copyrighted material. This article was subject to blind, independent, expert peer review. The reviewers reported no competing interests.

References
1. Lewis CE, Jacobs DR Jr, McCreath H, et al. Weight gain continues in the 19990s: 10-year trends in weight and overweight from the CARDIA study. Coronary Artery Risk Development in Young Adults. Am J Epidemiol. 2000;151:1172–1181.

2. Williamson DF. Descriptive epidemiology of body weight and weight changes in U.S. adults. Ann Intern Med. 1993;119:646–649.

3. Going S, Williams D, Lohman T. Aging and body composition: biological changes and methodological issues. Exere Sport Sci Rev. 1995;23:411–458.

4. Forbes GB. Longitudinal changes in adult fat-free mass: influence of body weight. Am J Clin Nutr. 1999;70(6):1025–1031.

5. Flegal KM, Carrol MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA. 2002;288:1723–1727.

6. Smith SC Jr. Multiple risk factors for cardiovascular disease and diabetes mellitus. Am J Med. 2007;120(3 Suppl 1):S3–S11.

7. Patsch JM, Kiefer FW, Varga P, et al. Increased bone resorption and impaired bone microarchitecture in short-term and extended high-fat diet-induced obesity. Metabolism. 2011;60(2):2430–2439.

8. Cao JJ. Effects of obesity on bone metabolism. J Orthop Surg Res. 2011;15;6:30.

9. Dietrich M, Jialal I. The effect of weight loss on a stable biomarker of inflammation, C-reactive protein. Nutr Rev. 2005;63(1):22–28.

10. Meyer HE, Tverdal A, Selmer R. Weight variability, weight change and the risk in older women. Int J Obes Relat Metab Disord. 2001;25(10):1536–1540.

11. Langlois JA, Mussolino ME, Visser M, Looker AC, Harris T, Madans J. Intentional and unintentional weight loss increase bone loss and hip fracture risk in older women. J Bone Miner Res. 2002;17:1421–1428.

12. Ensrud KE, Ewing SK, Stone KL, Cauley JA, Bowman PJ, Cummings SR. Strength training and adiposity in premenopausal women: strong, healthy, and empowered study. Am J Clin Nutr. 2007;86:566–572.

13. Chyb SK, Turko, Bernard G, et al. Impact of Walking on eating behaviors and quality of life of premenopausal and early postmenopausal obese women. Menopause. 2010;17(3):529–538.

14. Ricci TA, Heymsfield SB, Pierson RNJr, Stahl T, Chowdhury HA, Warren Jr, Berg A. Effect of a weight loss intervention on anthropometrical measures and metabolic risk factors in pre- versus postmenopausal women (review article). BMC Nutr J. 2007;6:31.

15. Deibert P, König D, Vitolinis MZ, Landmann U, Frey I, Zahradnik HP, Berg A. Effect of a weight loss intervention on anthropometric measures and metabolic risk factors in pre- versus postmenopausal women (review article). BMC Nutr J. 2007;6:31.

16. Bond RJ, Barry HC, Pilkington PE, et al. Long-term effects of a weight loss intervention on adiposity, body composition and metabolic parameters in premenopausal women. Am J Clin Nutr. 2007;85:663–669.

17. Schmitz KH, Hansson PJ, Stovitz SD, Bryan CJ, Warren B, Jensen MD. Strength training and adiposity in premenopausal women: strong, healthy, and empowered study. Am J Clin Nutr. 2007;86:566–572.

18. Dechamps A, Gatta B, Bourdel-Marchasson I, Tabarin A, Roger P. Pilot study of a 10-week multidisciplinary Tai Chi intervention in sedentary obese women. Clin J Sport Med. 2009;19(1):49–53.

19. Risco ES, Tesser S, Pérusse F, et al. Impact of Walking on eating behaviors and quality of life in premenopausal and early postmenopausal obese women. Menopause. 2010;17(3):529–538.

20. Lee SB, Hong JJ, Lee TS. Analysis of physical activities in wu-shu training. Conf Proc IEEE Eng Med Biol Soc. 2007;2007:632–635.

21. Nguyen MH, Kruse A. A randomized controlled trial of Tai chi for balance, sleep quality and cognitive performance in older Vietnamese. Clin Interv Aging. 2012;7:185–190.

22. Chaubine H, Hachana Y, Franchini E, Mkaouer B, Chamari K. Physical and physiological profile of elite karate athletes. Sports Med. 2012;42(10):829–843.

23. Zhang F, Kong LL, Zhang YY, Li SC. Evaluation of impact on health-related quality of life and cost effectiveness of Traditional Chinese Medicine: a systematic review of randomized clinical trials. J Altern Complement Med. 2012;18(12):1180–1190.

24. Nedeljkovic M, Ausfeld-Hafer B, Streithberger K, Seiler R, Wirtz PH. Taiji practice attenuates psychological stress reactivity—a randomized controlled trial in healthy subjects. Psychoneuroendocrinology. 2012;37(8):1171–1180.

25. Ng SM, Wang CW, Ho RT, et al. Tai chi exercise for patients with heart disease: a systematic review of controlled clinical trials. Alther Ther Heart Dis. 2012;18(3):16–22.

26. Chyu MC, Shen CL. Martial arts: potential alternative exercise for weight control. ACGM’s Certified News. 2009;19(3):4–6.

27. Cohen J. A power primer. Psychol Bull. 1992;112(1):155–159.

28. Silverman NE, Nicklas BJ, Ryan AS. Addition of aerobic exercise on a weight loss program increases BMD, with an associated reduction in inflammation in overweight postmenopausal women. Calcif Tissue Int. 2009;84:257–265.

29. Thorogood A, Mottillo S, Shimony A, et al. Isolated aerobic exercise and weight loss: a systematic review and meta-analysis of randomized controlled trials. Am J Med. 2011;124(8):747–755.

30. Centers for Disease Control and Prevention. Target Heart Rate and Estimated Maximum Heart Rate. 2011; Available at http://www.cdc.gov/physicalactivity/everyone/measuring/heartrate.htm. Accessed May 13, 2013.

31. Ware JR, Kosinski M, Keller SA. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. Med Care. 1996;34(3):220–233.

32. Pickard AS, Jeffrey AJ, Penn A, Lau F, Noseworthy T. Replicability of SF-36 summary scores in the SF-12 in stroke patients. Stroke. 1990;30:1213–1217.

33. Fontana L, Eagon JC, Trujillo ME, Scherer PE, Klein S. Viscer al fat deposition is associated with systemic inflammation in obese humans. Diabetes. 2006;56:1010–1013.

34. Matich A, Pirozzi L, Bruckert E, et al. Systemic low-grade inflammation is related to both circulating and adipose tissue TNFalpha, leptin and IL-6 levels in obese women. Int J Obes Relat Metab Disord. 2004;28(8):993–997.

35. Ferroni P, Basili S, Falco A, Davì G. Inflammation, insulin resistance, and obesity. Curr Atheroscler Rep. 2004;6(4):424–431.

36. Mathieu P, Poirier P, Pibarot P, Lemieux I, Desprès JP, Parent E. Intentional and unintentional weight loss increase bone loss and hip fracture risk in older women. Int J Obes Relat Metab Disord. 2001;25:1740–1747.

37. Ricci TA, Heymsfield SB, Pierson RN Jr, Stahl T, Chowdhury HA, Shaples SA. Moderate energy restriction increases bone resorption in obese postmenopausal women. Am J Clin Nutr. 2001;73(2):347–352.

38. Poelman ET, Toth MJ, Bunyard LB, et al. Physiological predictors of walking exercise weight loss. How much is enough? Int J Obes. 2002;26:1484–1493.

39. Schmitz KH, Hansson PJ, Stovitz SD, Bryan CJ, Warren B, Jensen MD. Strength training and adiposity in premenopausal women: strong, healthy, and empowered study. Am J Clin Nutr. 2007;86:566–572.
40. Fisher G, Hyatt TC, Hunter GR, Oster RA, Desmond RA, Gower BA. Effect of diet with and without exercise training on markers of inflammation and fat distribution in overweight women. *Obesity (Silver Spring)*. 2011;19(6):1131–1136.

41. Lester ME, Urso ML, Evans RK, et al. Influence of exercise mode and osteogenic index on bone biomarker responses during short-term physical training. *Bone*. 2009;45(4):768–776.

42. Villareal DT, Fontana L, Weiss EP, et al. Bone mineral density response to caloric restriction-induced weight loss or exercise-induced weight loss: a randomized controlled trial. *Arch Intern Med*. 2006;166(22):2502–2510.

43. Borst SE, De Hoyos DV, Garzarella L, et al. Effects of resistance training on insulin-like growth factor-I and IGF binding proteins. *Med Sci Sports Exerc*. 2001;33(4):648–653.

44. Arikawa AY, Kurzer MS, Thomas W, Schmitz KH. No effect of exercise on insulin-like growth factor-I, insulin, and glucose in young women participating in a 16-week randomized controlled trial. *Cancer Epidemiol Biomarkers Prev*. 2010;19(11):2987–2990.

45. Lemoine S, Rossell N, Drapeau V, et al. Effect of weight reduction on quality of life and eating behaviors in obese women. *Menopause*. 2007;14(3 Pt 1):432–407.

46. Rippe JM, Price JM, Hess SA, Kline G, DeMers KA, Damitz S, Kreidieh I, Freedson O. Improved psychological well-being, quality of life, and health practices in moderately overweight women participating in a 12-week structured weight loss program. *Obes Res*. 1998;6(3):208–218.

47. García-Martínez AM, De Paz JA, Márquez S. Effects of an exercise programme on self-esteem, self-concept and quality of life in women with fibromyalgia: a randomized controlled trial. *Rheumatol Int*. 2012;32(7):1869–1876.

48. Adamsen L, Quist M, Andersen C, et al. Effect of a multimodal high intensity exercise intervention in cancer patients undergoing chemotherapy: randomised controlled trial. *BMJ*. 2009;339:b3410.

49. Janssen I, Heymsfield SB, Baumgartner RN, Ross R. Estimation of skeletal muscle mass by bioelectrical impedance analysis. *J Appl Physiol*. 2000;89(2):465–471.