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The Effects of Tai Chi on Cardiovascular Risk in Women

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Abstract

Purpose—This study examined the effects of tai chi (TC) on biobehavioral factors associated with cardiovascular disease (CVD) risk in women.

Design—A randomized trial used a wait-list control group, pretest-posttest design. Data were collected immediately before, immediately after, and 2 months following the intervention.

Setting—The study was community based in central Virginia.

Subjects—Women aged 35 to 50 years at increased risk for CVD.

Intervention—The 8-week intervention built on prior work and was designed to impact biobehavioral factors associated with CVD risk in women.

Measures—Biological measures included fasting glucose, insulin, and lipids as well as C-reactive protein and cytokines. Behavioral measures included fatigue, perceived stress, depressive symptoms, social support, mindfulness, self-compassion, and spiritual thoughts and behaviors.

Analysis—A mixed effects linear model was used to test for differences between groups across time.

Results—In 63 women, TC was shown to decrease fatigue (∂ [difference in group means] =9.38, p = .001) and granulocyte colony stimulating factor (∂ = 12.61, p = .052). Consistent with the study model and intervention design, significant changes observed 2 months post intervention indicated that TC may help down-regulate proinflammatory cytokines associated with underlying CVD risk, including interferon gamma (∂=149.90, p =.002), tumor necrosis factor (∂=16.78, p =.002), interleukin (IL) 8 (∂=6.47, p =.026), and IL-4 (∂=2.13, p =.001), and may increase mindfulness (∂ = .54, p = .021), spiritual thoughts and behaviors (∂ = 8.30, p = .009), and self-compassion (∂ = .44, p = .045).

Conclusion—This study contributes important insights into the potential benefits and mechanisms of TC and, with further research, may ultimately lead to effective strategies for reducing CVD risk in women earlier in the CVD trajectory.

Keywords

Psychoneuroimmunology; Biobehavioral; Cardiovascular Disease; Tai chi; Moving meditation; Spirituality; Self-compassion; Mindfulness; Prevention Research; Manuscript format: research;

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Research purpose: intervention testing; Study design: randomized trial; Outcome measure: biobehavioral; Setting: local community; Health focus: physical activity/stress management; Strategy: skill building/behavior change; Target population age: adult women; Target population circumstances: geographic location

INTRODUCTION

A unique psychoneuroimmunology (PNI)–based biobehavioral model of cardiovascular disease (CVD) has been developed wherein modifiable risk factors related to stress and inflammatory processes are hypothesized to influence the eventual development of CVD in women. Building on our prior research,[1,2] the current study examined the effects of a specific tai chi (TC) intervention designed to reduce stress-related psychosocial and inflammatory risk factors in women with early indicators of CVD risk. In a sample of 63 premenopausal women with abdominal adiposity who had a family history of CVD, the intervention was tested by using a wait-list, pretest-posttest design with repeated measures of potential indicators of intervention effectiveness derived from the research model. Ultimately, the goal of this line of research is to expand knowledge about potential mechanisms underlying evolving CVD risk in women that may lead to effective strategies for reducing risk at earlier points in the CVD trajectory.

BACKGROUND

Cardiovascular Disease

Despite increasing awareness, as well as better diagnostics and treatment, women remain more likely than men to present with advanced disease and experience higher CVD-related morbidity and mortality. Higher morbidity and mortality for CVD in women is related to its complex and unique development and presentation. Adding to this complexity is the fact that atherosclerosis, the hallmark feature of CVD, only partly explains CVD in women, with at least half of women with ischemic heart disease presenting with no coronary artery obstruction.[3] CVD risk factors include genetic predisposition as well as obesity, hypertension, dyslipidemia, smoking, and physical inactivity.[4] Additionally, psychosocial variables including stress, depression, and lack of social support significantly contribute to the manifestation of CVD, particularly in women. CVD evolves over decades; thus primary prevention, including stress management and psychosocial support strategies, may attenuate risk, particularly when implemented early in the trajectory of disease evolution.

Research Model

Integration of CVD risk factors within the PNI framework provided the research model upon which this study was based (Figure 1).

The research model depicts selected PNI indicators of psychosocial, neuroendocrine, immune (inflammation), and cardiovascular mechanisms associated with increased risk of CVD. The primary risk factors of adiposity and family history of CVD evolve through interactions of biological (age, body mass index) and psychosocial factors (stress, depressive symptoms, and social support).

By design, the TC intervention is thought to positively affect PNI mechanisms through effects on perceived stress and perhaps on self-compassion, mindfulness, and spirituality. Such effects might then influence PNI interactive mechanisms to reduce fatigue and ultimately, CVD risk. In our prior testing of the research model, increased body mass index (BMI) and waist circumference (WC) were associated with elevated blood pressure, insulin resistance, dyslipidemia, and increased proinflammatory cytokines, along with lower adiponectin levels and perceived social support.[5] We are continuing to test and refine this PNI-based research model. Background research for each indicator in the model is briefly outlined below.

PNI involves investigation of the mechanisms of multidimensional psychobehavioral-neuroendocrine-immune system interactions, including the influence of psychosocial, spiritual, and behavioral factors on immunologically moderated and mediated diseases. It is well known that chronic stress and the associated psychological distress activates the hypothalamic-pituitary-adrenocortical (HPA) and sympathetic-adrenomedullary (SAM) systems, generally inducing immunosuppression (comprehensively reviewed in Ader[6]). An acute-phase SAM response and activation of the HPA axis occur simultaneously with perceived stress. Chronic stress creates chronic HPA activation and is associated with fatigue, sleep disturbance, depressed mood, cognitive impairment, immune dysfunction, and ultimately, type 2 diabetes mellitus (DM) and CVD.[7,8]

Abdominal obesity, reflecting the presence of visceral adipose tissue and evidenced by increased WC, has been shown to be a significant predictor of CVD.[9] Adipose tissue acts as a proinflammatory endocrine organ, attracting monocytes and secreting adipocytokines such as adiponectin, resistin, and leptin, as well as the cytokines tumor necrosis factor (TNF) and interleukin (IL) 6, leading to chronic systemic inflammation. This inflammatory response may be the hallmark of CVD risk.[10] Additionally, chronic stress dysregulates the metabolic balance between cortisol and insulin, causing insulin resistance in the liver and skeletal muscle through multiple mechanisms, including inhibition of pancreatic insulin secretion and dysregulation of glucose transporter mechanisms.[11] Insulin resistance is associated with a state of chronic low-grade inflammation, involving increased proinflammatory cytokines and decreased adiponectin levels.[10] Circulating adiponectin levels have been found to be decreased in hypertension, obesity-related insulin resistance, hyperlipidemia, type 2 DM, and CVD.[12]

Elevations of the proinflammatory cytokines IL-6 and TNF stimulate hepatocyte production of C-reactive protein (CRP), an acute phase response protein. IL-1β, TNF, and IL-6 affect the levels of adipocytokines and have been implicated in the development of CVD.[13] CRP plays a direct role in promoting the inflammatory aspect of CVD, with elevated CRP shown to be associated with hyperglycemia, hyper-triglyceridemia, and lowered high density lipoprotein-cholesterol (HDL-C) in individuals with stable coronary disease.[14]

Adiponectin is a protein produced by adipocytes and macrophages[15]; it is antidiabetic, antiatherosclerotic, and anti-inflammatory.[16] Hence, low levels of adiponectin are associated with atherosclerotic CVD, type 2 DM, hypertension, and hyperlipidemia.[12] Adiponectin

circulates in at least three isoforms, with the high-molecular-weight (HMW) isoform thought to be the active form in terms of protecting against inflammation.[18]

Hypertension (HTN) is a known contributor to CVD risk and even minimal abnormalities in blood pressure (BP) increase CVD risk when clustered with other CVD risk factors.[19] HTN is frequently associated with atherogenic dyslipidemia, which encompasses elevated triglycerides, increased low density lipoprotein-cholesterol (LDL-C), and decreased HDL-C.[20] Lipid abnormalities, particularly when combined with other CVD risk factors, have been correlated with both incident and recurrent CVD events regardless of gender.[18]

The combination of acute and chronic perceived stress over time increases the development and progression of CVD, although the mechanisms are complex and incompletely understood. Recent research indicates that stress creates hemodynamic, endocrine, and immune changes that increase susceptibility to CVD.[20] Increased perceived stress has been implicated as an important predictor of cardiovascular dysfunction in women.[21]

A highly prevalent and distressing symptom across populations, fatigue may disproportionately affect women, particularly at mid-life.[22] Given cumulative stress effects, fatigue gives rise to dysregulation of metabolic processes and potentiates a trajectory of CVD risk related to decreased physical activity and inadequate self-care. Thus, both immune changes and chronic stress may account for the fatigue often associated with CVD risk factors.[23] Fatigue is postulated to be an outcome of interactive PNI mechanisms in our research model.

Depression is an independent risk factor for the development and progression of CVD.[24] Depressive symptoms have been shown to be sufficient to increase CVD risk in the absence of major depressive disorder,[25] carrying a 1.5- to 2.5-fold increase in CVD risk.[26] Potential mechanisms include depression-related fatigue and lack of interest, predisposing individuals to more sedentary lifestyles, decreasing adherence to healthy lifestyle behaviors and medical regimens, as well as decreasing heart rate variability, all of which increase CVD risk.[27]

Social support (SS) has been associated with positive health outcomes after controlling for CVD risk factors and health risk behaviors, and lack of SS has been shown to negatively affect cardiovascular health.[28] SS is thought to mediate stress by facilitating coping through cognitive reappraisal, thus attenuating neuroendocrine activation.[29]

Mindfulness is a unique aspect of consciousness and self-awareness thought to enhance well-being and also a presumed requisite for the development of self-compassion. This quality of awareness and attention can be cultivated with various mind-body techniques such as meditation, yoga, and tai chi, among others.[30] Mindfulness has been associated with amygdala deactivation suggesting down-regulation of negative emotions including decreased rumination, altered perception of stressors, and improved emotional regulation.[31,32]

Self-compassion encompasses being kind and understanding with one’s self, appreciating experiences as part of a larger human experience, and mindful awareness of painful experiences.[33] The presence of self-compassion has been negatively correlated with depression and anxiety and positively correlated with well-being and life satisfaction, likely

serving as a useful emotional regulation strategy to balance stress perception.[34] We propose that self-compassion can be developed through interventions such as tai chi.

Religiosity has been associated with decreased obesity and CVD risk.[35] While they are different concepts, religiosity and spirituality encompass similar constructs and likely share similar mechanisms of action.[36] Similar to self-compassion, spirituality may serve emotion regulation and hence, stress reactivity. Thus, we propose that spirituality also may contribute to stress management approaches such as tai chi.

Tai Chi

Tai chi is an ancient practice that originated as a martial art emphasizing harmony with nature and within oneself by accessing and manipulating one's *chi,* or *life force*. It has most recently been referred to as a "moving meditation" focused on the connection of body, mind, and spirit to facilitate health through focused breathing, relaxation, and movement.[37] While measuring the accessing and manipulation of chi is not yet possible, the effect of the practice has been studied in several disease states. Tai chi has been found to improve quality of life, psychological well-being, sleep, balance, strength, activity tolerance, immune function, and cardiovascular and pulmonary function.[38] Pertinent CVD-related outcomes of tai chi include favorable changes in lipid profiles and improved glycemic control.[39,40] A recent review did not support the effects of tai chi on glycemic control but did support its efficacy in lowering lipids and blood pressure.[40]

Although there is accumulating scientific evidence that tai chi improves a variety of clinical outcomes, there are few studies that focus on prevention as did the current study. While the complexities of CVD risk in women are far from defined, a number of factors have been implicated. Comprehensive biobehavioral models need to be tested to further specify risk factors, particularly those that may be early markers of risk.

METHODS

Design

The study used a wait-list control group, pretest-posttest design. After baseline data collection (time 1 [T1]), participants were randomized into either the wait-list control group or the intervention group. Data were then collected immediately post intervention at 8 weeks (T2) and at 2 months post intervention or the equivalent wait period (T3). At each data collection point, participants were fasting and had blood drawn, then completed psychosocial study instruments.

Sample

After Institutional Review Board approval, a sample of 96 women was recruited for this study in the metropolitan area of Richmond, Virginia. Participants were recruited by using local electronic newsletters as well as distribution of study flyers and brochures throughout the city of Richmond and surrounding counties. Eligible participants were nondiabetic women, aged 35 to 50 years with increased WC (≥35 inches), a family history of CVD in first- or second-degree relatives, and able to speak and read English. Participants were

excluded for history of CVD, morbid obesity (BMI > 40 kg/m^2) and uncontrolled HTN (BP ≥ 160/100 mm Hg). Once consented and enrolled, participants were randomized into either the TC intervention or the wait-list control group and baseline data were collected. Participants were administratively withdrawn if baseline measures indicated fasting glucose > 126 mg/dL (indicating DM) or fasting LDL-C > 160 mg/dL (consistent with existing CVD). Such results were shared with participants who were then referred for medical consultation. Wait-list control participants were instructed to return in 8 weeks then 16 weeks for data collection, at which time they received the 8-week TC class. At each data collection, participants were asked about changes in medications, health status, and lifestyle factors.

A total of 281 individuals were assessed for study eligibility; 185 were excluded (most of whom [n =147] did not meet inclusion criteria), resulting in 96 participants being randomized into the intervention (n = 47) or wait-list control group (n =49). Of these, 11 were lost to follow-up, 19 were administratively withdrawn because of abnormal laboratory values or missed TC classes, and 3 self-withdrew for personal reasons, resulting in a total of 63 women who completed the study (see diagram in Figure 2). Participants who did not complete the study had higher WC and were more likely to be African-American in comparison to participants who completed the study. This was largely related to high lipid levels (LDL > 160 mg/dL) that rendered them ineligible for the study.

Measures

Demographic data included self-reported age, race, income, and education.

Biological Measures—Samples of 3 mL of blood for plasma and 8 mL of blood for serum were drawn by using standard venipuncture procedures. Specimens were distributed to the Center for Biobehavioral Clinical Research (CBCR) laboratory and the Virginia Commonwealth University Medical Center (VCUMC) Department of Pathology laboratory.

Neuroendocrine measures included assessments of insulin resistance and impaired glucose metabolism. Clinical laboratory measures of fasting glucose and insulin levels were obtained, from which Homeostasis Model Assessment of Insulin resistance scores were calculated.

Indicators of inflammation included levels of type 1 cytokines (interferon gamma, TNF, IL-1β, IL-2, IL-8, and IL-12), type 2 cytokines (IL-4, IL-6, and IL-10), granulocyte colony stimulating factor (GCSF), and adiponectin (total and HMW), which were measured in the CBCR by experienced research laboratory personnel using plasma samples. Cryopreserved samples were batch-processed by using commercially prepared multiplex kits with a Bio-Plex Pro (BioRad, Inc., Hercules, California) magnetic bead system (intra-assay variability < 5%, interassay variability < 9 %, cross-reactivity < 1.0%). Additionally, serum levels of CRP (high-sensitivity assay [hsCRP]) were measured in the clinically certified pathology laboratory by using established assay procedures.

Adiposity was measured by using BMI and WC. BMI is an internationally used measure of obesity based on height and weight (BMI =weight in pounds × 703/[height in inches]^2). WC

was also measured because it is reflective of visceral adipose tissue, which is associated with increased CVD risk.[8]

BP was measured twice (and averaged) at each data collection point by using a calibrated sphygmomanometer.

Fasting lipid profiles (serum total cholesterol, HDL-C, LDL-C, and triglyceride levels) were assayed in the VCUMC Department of Pathology laboratory by using well-established measurement procedures.

Behavioral/Psychosocial Instruments—Fatigue was measured with the *Multidimensional Fatigue Symptom Inventory–Short Form* (MFSI-SF).[41] The MFSI-SF has a stable multidimensional factorial structure supporting five subscales of general, physical, emotional, and mental fatigue, and vigor. The MFSI-SF has been validated in medically ill and healthy populations and demonstrates excellent psychometric properties, including high subscale α coefficients (.74–.95) and high concurrent and convergent validity. Using a five-point Likert-type format, respondents provide information on overall level of fatigue and the extent to which fatigue has been experienced across the five domains during the past 7 days, with higher scores indicating a higher level of fatigue.

The *Perceived Stress Scale* (PSS)[42] measures the degree to which situations in an individual's life are appraised as stressful. The 10 questions are general in nature, with respondents indicating how often each statement applied to them during the past month. The PSS is a widely used general measurement of perceived stress and it has accrued considerable reliability and validity data since its inception, with internal consistency α's at . 85 and test-retest reliability of 0.87. Higher scores reflect higher levels of perceived stress.

The *Center for Epidemiological Studies–Depression* (CES-D) is a widely used, psychometrically sound instrument designed to detect depressive symptoms in the general population.[43] The CES-D is composed of 20 items reflecting the domains of depressive affect, somatic symptoms, positive affect, and interpersonal relations. Participants report the extent to which they experienced each symptom within the previous week. Higher scores reflect a higher level of depressive symptoms; while not diagnostic, a score ≥ 16 suggests the presence of clinical depression.

The revised *Social Provisions Scale*, a widely used, reliable, and valid measure of social support, is a 24-item Likert-type measure of six components of social support (attachment, social integration, reassurance of worth, reliable alliance, guidance, and opportunity for nurturance), allowing evaluation of various dimensions as well as overall perception of social support.[44] Higher scores indicate greater social support.

Mindfulness was measured by using the *Mindful Attention Awareness Scale* (MAAS), a 15-item Likert scale indicating *attention to* and *awareness of* what is presently occurring, with higher scores reflecting higher levels of mindfulness.[45] Although the concept of mindfulness arises from Eastern thought and philosophy, the MAAS was developed as an instrument for a general Western population by avoiding the need for highly sensitive awareness or knowledge of a special vocabulary. It has been shown to be a reliable and valid instrument

with good internal consistency (Cronbach α = .73–.95) as well as strong construct, criterion, convergent, and discriminant validity[45] in adolescent and adult populations, but its previous use in middle-aged women is limited. Higher scores reflect a higher level of dispositional mindfulness.

The revised *Self-Compassion Scale* (SCS-R) has demonstrated good internal consistency, discriminant and convergent validity, as well as test-retest reliability.[33] The SCS-R is a 26-item Likert scale reflecting three attributes of self-compassion: self-kindness versus self-judgment, common humanity versus isolation, and mindfulness versus overidentification. Its use is limited in middle-aged women but in one study of nurses, the subscales yielded relatively high internal consistency (.69–.90), as did the total instrument (.90).[46]

Spirituality as a potential modifier of stress was assessed by using the revised *Spiritual Involvement and Beliefs Scale* (SIBS-R).[47] The 26-item SIBS-R has demonstrated high reliability (Cronbach α = .92), test-retest reliability (r = .93), and strong construct validity in samples that included adult and elderly women. The SIBS-R captures spiritual thoughts and beliefs as well as actions in daily life that arise from these thoughts and beliefs, with higher scores reflecting greater spiritual beliefs and practices.

Intervention

The 8-week TC intervention was led by the principal investigator (J.L.R.), who began designing and implementing tai chi interventions in 2000. Group classes were conducted at the VCU School of Nursing or a community-based VCUMC-affiliated facility, depending on which was geographically more convenient for participants. Participants in the intervention group had not participated in prior studies of TC and only one participant had previously practiced TC.

From prior studies and a review of the literature, an 8-week, focused, short form of tai chi was developed for this study.[2] Each of eight weekly, 60-minute instructor-led group classes began with a focus on breathing and balance, both key elements in tai chi. A sequence of movements was taught focused on developing each individual's skills in balancing, focused breathing, gentle physical posturing and movement, and the active use of consciousness for relaxation. Movements were taught in a sequence that allowed repetitive instruction as well as a progressive building of skills. Training was designed to promote increased control of attention and an integrated mind-body relaxation experience. Participants in the intervention group were asked to practice daily at home by using professionally produced DVDs designed by and featuring the principal investigator. Because "dose" is a critical and largely unexplored issue in mind-body intervention efficacy, participants were encouraged to practice 15 minutes most days of the week and were asked to report their practice times each week. Participants were required to attend at least six of the eight classes. If a participant missed a class, she was instructed to review the DVD chapter for that week. Classes were structured such that all prior movements were reviewed before the week's new movement was introduced in order to reinforce learning.

Wait-list control group participants were instructed to refrain from engaging in new mind-body practices during the study period and were asked to report any change in diagnoses, medications, or lifestyle practices at each data collection visit.

Analysis

Power for the mixed linear model was calculated by using Helms method with data from our previous study.[48] Given a sample size of 30 per group and α =.05, power was calculated to be .90 when based on blood pressure changes and .99 when using changes in PSS scores.

Descriptive statistics were computed for demographic data to characterize the sample. Demographic variables were compared by using two-sample *t*-tests for continuous variables and χ^2 tests for categorical variables. As is common with most biological data, specifically cytokine data, the data are bounded below by zero and are asymmetric-skewed positively. Because the cytokine data from this study were skewed positively, the assumptions of normality and homoscedasticity of the mixed effects linear model residuals were not satisfied. Thus, log transformation was used on the cytokine data, which served the dual role of normalizing and stabilizing the variance of the residuals. Tests of significance were based on α = .05.

A mixed effects linear model was used to test for differences between the intervention and wait-list groups across time for all biological and behavioral/psychosocial variables. Following the intent-to-treat principle, all available data were used in the mixed effects linear model. Fixed effects included time (visit number: T1, T2, T3), intervention group, and time-by-group interaction. Covariates included age and WC. Subject was modeled as a random effect. Because this was an exploratory study, no adjustment for multiplicity was used; the results from this trial should be regarded as preliminary and should be confirmed in a larger trial.

RESULTS

The 8-week TC intervention was delivered to six groups. Group size ranged from 8 to 12 participants. Each week, participants were asked to turn in a practice log documenting daily home practice of TC. Approximately two-thirds (n = 44) of participants completed practice logs, which revealed an average of 12 minutes of daily practice.

Demographics

Demographic data are presented in Table 1. The mean age was 43.89 (SE, 0.0551) years; mean BMI was 32.28 kg/m^2 (SE, 0.515); mean WC was 39.94 inches (SE, 1.008); 24% were African-American and 76% were Caucasian. In terms of comorbidities that increase CVD risk, 18% of participants had been diagnosed with hypertension, 14% with hyperlipidemia, and 10% with hypothyroidism, all of which had been treated and were stable at the time of the study. Socioeconomic status variables also can be seen in Table 1. Analysis of demographic variables at baseline indicated significant differences between the treatment and control groups on two variables: age and WC. Thus, these variables were used as covariates in the mixed effects linear model analyses; however, *p* values were consistently <. 05, so that adjustments for these covariates had little effect on the group comparisons.

Biological Factors

CVD risk factor variable means are presented in Table 2. While there were no significant changes in measures associated with insulin resistance and dyslipidemia immediately post intervention (T2), there was a significant decrease in log-transformed GCSF, which persisted at T3, 2 months following the intervention. At T3 statistically significant decreases were found in four proinflammatory cytokines: IL-4, IL-8, TNF, and interferon gamma as well as a marginally significant decrease in IL-6 ($p = .06$) (Table 4).

Behavioral/Psychosocial Factors

Consistent with the changes in cytokines, changes in other behavioral/psychosocial variables were not observed at T2 (immediately post intervention), but were seen at T3, 2 months following the intervention. The only exception in this pattern was level of fatigue, which significantly decreased at T2 and persisted at T3. Additional changes seen at T3 were significant decreases in depressive symptoms, as well as increased mindfulness, self-compassion, and spirituality, and marginally significant decreases in perceived stress ($p = .06$) (Table 3).

DISCUSSION

While there were differences in age and WC at baseline, the means and standard deviations indicated these variables had no significant effects in the analytic model. Thus, we were able to validly analyze and interpret the biological data.

Fatigue was significantly improved immediately after the intervention. We hypothesize that the TC intervention created new perspectives on the role of stress in daily life and the ability to handle it, which in turn, influenced biological signaling as evidenced by changes in multiple cytokines that influence immune function and inflammation. Perhaps over a longer period these changes in cytokines would have resulted in changes in other biological outcomes.

GCSF was significantly decreased post intervention. Changes in other proinflammatory cytokines were observed at T3, 2 months post intervention, indicating a decrease in signaling associated with systemic inflammation. While clinical significance could be inferred from what is known about cytokine signaling, these measures remain experimental, which limits clinical interpretation.

In the context of the PNI framework, the absence of change in the biological measures at T2 was not expected. An important contributing factor was likely the limited intervention dose, which was chosen for this study in order to test for minimum effective dose and was based on feedback from participants in a prior feasibility and acceptability study. Additionally, participants' daily practice time was somewhat limited. The goal was for participants to practice on average 2 to 2.5 hours per week, which reflected class time plus 15 minutes of home practice most days of the week, which meets the lower threshold for practice represented in the literature. While tai chi is an ancient and powerful practice, it is well known that proficiency and the resultant impact on health and health outcomes requires routine practice time. Additionally, it was anticipated that changes in BP would be found, as

these changes would occur more quickly than changes in glucose metabolism and lipids; however, missing data prohibited analysis of participants' blood pressure.

While significant changes in the other behavioral measures were not observed until T3 and it is not clear whether these changes resulted from exposure to the intervention, they are consistent with the study model as well as the design and administration of the TC intervention. The intervention is novel in that it included well-established yet specifically chosen tai chi movements based on the meanings of the movements.[2] Teaching the meanings facilitates the opportunity to shift perceptions of stress and coping. For example, the Five Elements emphasizes the universal human experience of ongoing cycles of change in life, recognizing that change presents the opportunity for growth and transformation and that we have the ability to successfully navigate these changes. Consistent with our prior work[1,2] participants voiced that while finding time to practice during the week was challenging, they consistently remembered the meanings of the movements when dealing with situations in their daily lives, which helped them reframe thoughts in different, more successful ways. Additionally, based on the biological contributors to CVD, medical qigong (a specific type of tai chi and an integral component of traditional Chinese medicine) movements were chosen that purportedly address these biological contributors. For example, the endocrine and immune systems were targeted with the Thymus Tap and Kidney Rub movements, and there were changes in several cytokines associated with functional signaling within these systems. While we have no data related to causal relationships, these are interesting associations that should be further explored.

Significant changes in the other behavioral measures at T3 included decreased depressive symptoms as well as increases in mindfulness, self-compassion, and spiritual thoughts and behaviors. These changes are conceptually consistent with the research model. Mindfulness is often learned through various meditation-based practices, including tai chi. Theoretically, mindfulness is a prerequisite for the development of self-compassion and the current findings support this proposition. In addition to overall self-compassion, significant changes were observed in the identification and kindness subscales. Identification is associated with overidentification with painful experiences, and kindness is related to the ability to think kind thoughts about one's self. These changes are consistent with other behavioral changes including the decrease in fatigue observed at T2 and T3 as well as the meanings of the tai chi movements taught in this intervention.

Significant changes were observed in spirituality-related thoughts, beliefs, and behaviors at T3. These changes are consistent with the intervention used in this study. While tai chi is conceptualized and often taught as a moving meditation, a way of combining focused movement and breath for relaxation and present moment awareness, it is associated with Eastern thought and spiritual traditions. Additionally, intervention movements such as the Tai Chi Bow and Heaven and Earth Energy entail a belief in and relationship with a higher power.

Limitations in this study included its relatively small sample size. Additionally, while recruitment strategies targeted ethnically and racially diverse women with lower socioeconomic status, in part because they experience a higher incidence of CVD,[45] we

were not able to achieve adequate diversity in our sample. This may be in some measure related to the intervention; for example, tai chi currently is not well known or widely desired by underserved African-American women. Finally, missing data prohibited analysis of intervention impact on blood pressure, BMI, and WC.

Tai chi is a feasible and acceptable moving meditation practice that appears to decrease fatigue and may decrease inflammatory signaling. Additionally, it can increase mindfulness, spirituality, and self-compassion. Further testing of this research model may ultimately enhance disease prevention by creating a more holistic, comprehensive approach targeting both known indicators of risk and newer, potentially more powerful preclinical indicators and biomarkers.

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SO WHAT? Implications for Health Promotion Practitioners and Researchers

What is already known on this topic?

Tai chi, a form of moving meditation, is supported by an evidence base comprising several hundred studies in various populations and disease states.

What does this article add?

While recent study quality is improving, historically, methodologic rigor in tai chi investigations has been lacking. This study contributes to a growing body of increasingly well-designed studies of tai chi. Additionally, it builds on a decade of work focused on designing tai chi interventions to engage both physical activity and mindfulness for stress management by incorporating the meanings of the tai chi movements, which is a unique feature of this approach compared to other tai chi interventions.

What are the implications for health promotion practice or research?

This study provides insights into the potential anti-inflammatory mechanisms and effects of tai chi in the prevention of CVD disease in women. Given emerging evidence confirming the inextricable mind-body connection, moving meditations such as tai chi may reduce disease risk by targeting underlying inflammatory mechanisms in CVD and other diseases.

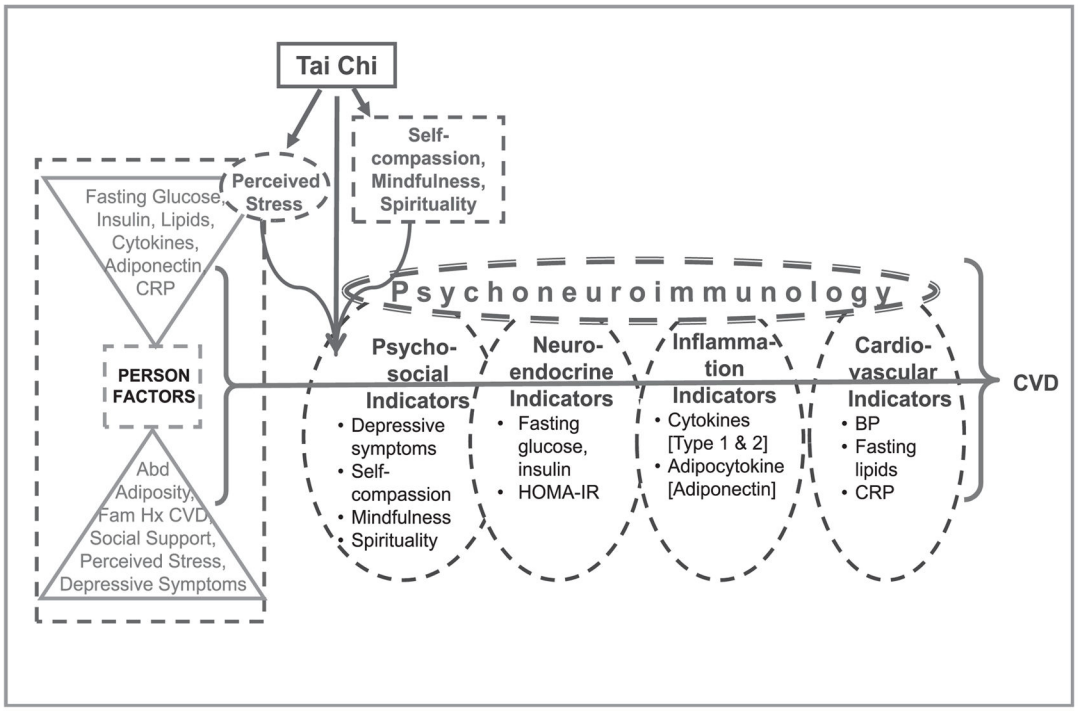

Figure 1. Cardiovascular Disease (CVD) Risk in Women*

*CRP indicates C-reactive protein; Abd, term to come; Fam Hx, term to come; HOMA-IR, Homeostasis Model Assessment of Insulin resistance; and BP, blood pressure.

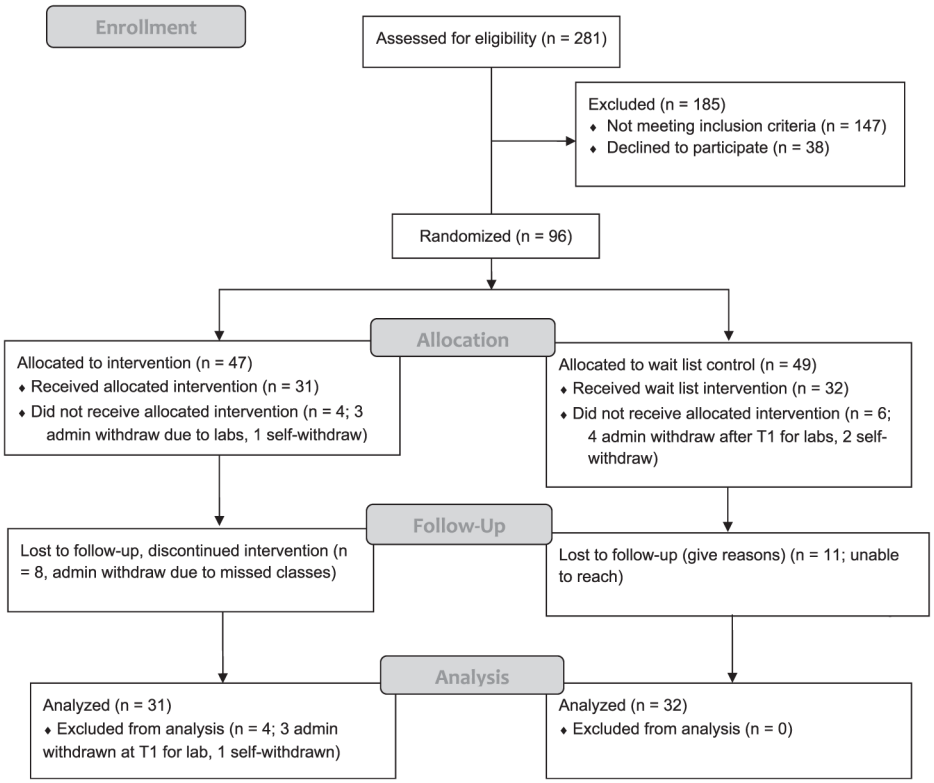

Figure 2.
Consolidated Standards of Reporting Trials 2010 Flow Diagram

Table 1

Demographic Data and Sample Characteristics

	Control (n = 32)		Intervention (n = 31)		
Variable	**Mean ± SE**	**No. (%)**	**Mean ± SE**	**No. (%)**	***p***
Age, y	45.22 ± 0.708		42.52 ± 0.784		0.0129 *
Body mass index, kg/m^2	33.38 ± 0.693		31.10 ± 0.714		0.0256 *
Waist circumference, inch	41.26 ± 1.850		38.53 ± 0.603		0.1781 *
Race					
African-American/more than one		7 (21.88)		8 (26.67)	0.6597 **
White		25 (78.13)		22 (73.33)	
Income					
<$45,000		9 (28.13)		3 (10.34)	0.1053 **
$45,000–$89,999		12 (37.50)		9 (31.03)	
≥$90,000		11 (34.38)		17 (58.62)	
Education					
High school/technical training		10 (31.25)		3 (10.00)	0.0607 ***
Baccalaureate degree or higher		22 (68.75)		27 (90.00)	

* *t*-test.

** Chi-square test.

*** Fisher test.

Table 2

Behavioral Risk Factors

		Control			Intervention			*p*	
Measure*	**Cronbach α**	**Visit 1 Mean ± SE n = 32**	**Visit 2 Mean ± SE n = 32**	**Visit 3 Mean ± SE n = 31**	**Visit 1 Mean ± SE n = 31**	**Visit 2 Mean ± SE n = 31**	**Visit 3 Mean ± SE n = 28**	**Visit 2 n = 63**	**Visit 3 n = 59**
MFSI-SF Total	0.85	12.08 ± 3.17	10.57 ± 3.52	10.50 ± 3.51	17.67 ± 3.90	5.94 ± 3.79	1.12 ± 2.90	0.021	0.001
PSS	0.88	16.63 ± 1.13	14.37 ± 1.33	14.57 ± 1.29	17.13 ± 1.26	14.68 ± 1.26	12.12 ± 1.10	0.90	0.061
CES-D	0.91	10.82 ± 1.65	10.84 ± 2.02	11.69 ± 1.83	12.23 ± 1.79	9.55 ± 1.89	5.64 ± 0.98	0.21	0.001
SPS	0.91	82.44 ± 1.93	81.63 ± 2.05	83.50 ± 2.07	85.65 ± 1.35	86.52 ± 1.44	85.04 ± 1.77	0.32	0.73
MAAS	0.90	3.79 ± 0.16	3.78 ± 0.16	3.70 ± 0.18	3.95 ± 0.17	4.03 ± 0.15	4.24 ± 0.15	0.53	0.021
SCS-R Total	0.94	3.18±0.13	3.20 ± 0.13	3.27 ± 0.14	3.32 ± 0.14	3.52 ± 0.14	3.71 ± 0.15	0.21	0.045
SIBS-R	0.92	122.30 ± 4.40	124.41 ± 3.90	121.23 ± 5.03	119.26 ± 4.75	126.68 ± 4.18	129.53 ± 4.37	0.094	0.009

*MFSI-SF indicates Multidimensional Fatigue Symptom Inventory–Short Form; PSS, Perceived Stress Scale; CES-D, Center for Epidemiological Studies–Depression; SPS, Social Provisions Scale; MAAS, Mindful Attention Awareness Scale; SCS-R, Self-Compassion Scale; and SIBS-R, Spiritual Involvement and Beliefs Scale.

Table 3

Biological Risk Factors

Measure*	Control			Intervention			*p*	
	Visit 1 Mean ± SE n = 32	Visit 2 Mean ± SE n = 32	Visit 3 Mean ± SE n = 31	Visit 1 Mean ± SE n = 31	Visit 2 Mean ± SE n = 31	Visit 3 Mean ± SE n = 28	Visit 2 n = 63	Visit 3 n = 59
Fasting blood glucose	91.63 ± 1.00	91.22 ± 1.16	92.00 ± 0.94	91.32 ± 1.62	92.97 ± 1.85	92.39 ± 1.52	0.30	0.68
Fasting insulin	5.73 ± 0.91	5.94 ± 0.81	7.31 ± 1.45	6.08 ± 0.65	7.08 ± 1.07	7.76 ± 1.81	0.42	0.54
HOMA-IR	1.32 ± 0.22	1.35 ± 0.19	1.68 ± 0.34	1.40 ± 0.16	1.69 ± 0.27	1.86 ± 0.48	0.45	0.71
Total cholesterol	179.63 ± 5.28	184.94 ± 4.17	184.77 ± 5.18	186.39 ± 3.62	190.87 ± 4.54	188.29 ± 4.98	0.86	0.75
HDL-C	51.16 ± 1.74	51.34 ± 1.56	50.77 ± 1.62	52.42 ± 2.09	52.52 ± 1.94	53.36 ± 2.09	0.95	0.42
LDL-C	109.84 ± 4.28	113.63 ± 3.76	115.20 ± 4.35	114.45 ± 3.32	117.52 ± 3.45	115.39 ± 3.58	0.84	0.50
Triglycerides	94.34 ± 10.08	100.09 ± 9.06	94.60 ± 7.90	97.39 ± 6.13	102.00 610.57	97.75 ± 7.20	0.89	0.98
Adiponectin total	5158.19 ± 382.17	5465.17 ± 444.63	5433.10 ± 429.70	4743.75 ± 373.03	5060.75 ± 409.91	5173.74 ± 448.35	0.63	0.88
Adiponectin HMW	2471.51 ± 294.43	2585.33 ± 313.09	2608.11 ± 281.29	2319.06 ± 252.32	2357.33 ± 244.36	2457.63 ± 275.31	0.96	0.81
hsCRP	0.59 ± 0.11	0.54 ± 0.06	0.62 ± 0.09	0.52 ± 0.09	0.58 ± 0.12	0.49 ± 0.08	0.83	0.41

*HOMA-IR indicates Homeostasis Model Assessment of Insulin resistance; HDL-C, high density lipoprotein-cholesterol; LDL-C, low density lipoprotein-cholesterol; HMW, high molecular weight; and hsCRP, high-sensitivity C-reactive protein.

Table 4

Statistically Significant Cytokines/Growth Factors (Log-Transformed Data) *

Cytokine/Growth Factor	Visit	Control Median (Min, Max)	Intervention Median (Min, Max)	*p*
Interferon gamma	1	67.64 (0.10, 695.74)	32.08 (0.10, 630.61)	
	2	109.91 (0.10, 954.57)	35.82 (0.10, 322.17)	0.311
	3	172.40 (0.10, 963.86)	22.50 (0.10, 380.07)	0.002
TNF-α	1	15.71 (1.30, 72.84)	14.47 (1.99, 103.30)	
	2	18.63 (0.49, 111.35)	15.39 (0.92, 56.49)	0.559
	3	32.42 (7.46, 114.56)	15.64 (0.01, 56.81)	0.002
Interleukin-8	1	12.01 (0.10, 33.98)	11.18 (0.10, 58.32)	
	2	14.01 (3.29, 57.68)	9.92 (4.21, 32.63)	0.303
	3	17.48 (6.04, 61.02)	11.01 (1.35, 37.99)	0.026
Interleukin-4	1	1.32 (0.01, 7.78)	1.08 (0.01, 13.96)	
	2	1.79 (0.01, 11.93)	1.08 (0.01, 9.55)	0.062
	3	3.11 (0.01, 10.96)	0.98 (0.01, 8.43)	0.001
GCSF	1	29.62 (10.76, 109.63)	27.74 (4.76, 191.54)	
	2	37.76 (18.76, 201.76)	25.15 (0.67, 100.59)	0.052
	3	56.94 (0.86, 157.92)	25.80 (6.02, 124.71)	0.087

* TNF indicates tumor necrosis factor; and GCSF, granulocyte colony stimulating factor.