The Effect Of Yoga Practice On Glycemic Control Of Type 2 Diabetes Mellitus Patients

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THE EFFECT OF YOGA PRACTICE ON GLYCEMIC CONTROL OF TYPE 2 DIABETES PATIENTS

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By

Maricarmen Vizcaíno

2011
For my husband, whose immense love, support, and faith in me made the completion of this thesis possible. For my parents, for their constant encouragement in the realization of my goals and for their guidance in the world of reason since my childhood.
THE EFFECT OF YOGA PRACTICE ON GLYCEMIC CONTROL OF TYPE 2 DIABETES MELLITUS PATIENTS

by

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THESIS

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ABSTRACT

The primary purpose of this study was to investigate the effect of yoga practice on glycemic control of Type 2 diabetes mellitus patients by evaluating changes in physiological and psychological stress measures. Participants (n=10) were non-insulin-dependent diabetes mellitus patients free of complications derived from diabetes. The 6-week intervention consisted of structured yoga classes with a registered yoga teacher three times a week. Assessments were completed at baseline and post-intervention. Glycemic control parameters under investigation included fasting blood glucose (FBG), glycated hemoglobin (HbA1c), and insulin sensitivity (QUICKI). Psychological stress was assessed with the perceived stress scale (PSS) and the State-Trait Anxiety Inventory (STAI); while physiological stress was assessed by cortisol content at midnight. Additional measures included diabetes regimen adherence, quality of life, active range of motion, and balance. No significant changes were observed in any of the glycemic control parameters following the yoga intervention. Nonetheless, significant decreases in all stress measures were found following yoga practice. Similarly, significant improvements in regimen adherence, flexibility, and balance were found. The findings indicate that a longer and more frequent yoga practice may be necessary to induce significant improvements in glycemic control, and that this improvement may be mediated by decreases in anxiety and cortisol of diabetes patients.
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INTRODUCTION

The benefits that practitioners and teachers attribute to the practice of hatha yoga are numerous, including positive effects on chronic diseases such as diabetes mellitus. The majority of studies investigating the effect of yoga practice alone (Amita, Prabhakar, Manoj, Harminder, & Pavan, 2009; Gokal, Shillito, & Maharaj, 2007; Gordon et al., 2008; Malhotra, Singh, Tandon, & Sharma, 2005; Singh, Malhotra, Singh, Madhu, & Tandon, 2004; Singh et al., 2001) or combined with another non-medical intervention (Agrawal et al., 2003; Agte & Tarwadi, 2004; Jain, Uppal, Bhtanagar, & Talukdar, 1993) have shown potential benefits of this ancient practice on glycemic control of non-insulin-dependent diabetes patients by reporting reductions in fasting blood glucose (FBG) (Amita et al., 2009; Agrawal et al., 2003; Agte & Tarwadi, 2004; Gokal et al., 2007; Gordon et al., 2008; Jain et al., 1993; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001), post-prandial blood glucose (Amita et al., 2009; Agte & Tarwadi, 2004; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001), glucose tolerance (Jain et al., 1993), and even glycated hemoglobin (HbA$_1c$) (Agrawal et al., 2003; Singh et al., 2004; Singh et al., 2001). Nonetheless, data are still inconclusive because of the lack of control for confounding variables such as additional physical activity (Agrawal et al., 2003; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001), failure to monitor diet or medication (Malhotra et al., 2005), poor description of the intervention (Gordon et al., 2008), or inadequate description of the sample population (Agrawal et al., 2003). Furthermore, some studies have reported no change in HbA$_1c$ following yoga practice.
(Agte & Tarwadi, 2004; Mercuri, Olivera, Souto, & Guidi, 2003) and only two studies proposed potential mechanisms to explain the beneficial impact of yoga for Type 2 diabetes. Manjunatha, Vempati, Ghosh, & Bijlani (2005) proposed that yogic postures increase the rate of glucose utilization after observing a decrease in insulin levels immediately after practice, and Malhotra et al. (2005) suggested that yoga may increase glucose utilization by redistributing fat in diabetes patients. Finally, none of the studies reported control of factors that could affect the measurement of FBG, such as female hormones or ingestion of certain drugs, or factors that could affect the assessment of HbA1c like red blood cell abnormalities or excessive doses of vitamin C and E (Unger, 2007).

Many authors have suggested that additional research is needed to clarify the potential benefit of yoga on glycemic control (Agte & Tarwadi, 2004; Gokal et al., 2007; Manjunatha et al., 2005; Singh et al., 2004). And most importantly, there is a need to explain the possible mechanism underlying the improvement in glycemic control that yoga may confer to Type 2 diabetes mellitus patients. A plausible explanation may stem from the effect that yoga generates by alleviating psychological stress of the diabetes patient. Psychological stress can act indirectly by influencing regimen adherence to diabetes self-care behaviors (Nakahara et al., 2006; Cohen & Kanter, 2004), or directly by the effect of stress hormones on glucose metabolism (Surwit & Feinglos, 1983, 1984).
Stress and self-care behaviors in diabetes

Nakahara et al. (2006) found that self-efficacy directly influenced adherence to self-care behaviors, which itself directly influenced HbA$_{1c}$ levels of Type 2 diabetes patients. Self-efficacy was, in turn, positively influenced by social support and inversely influenced by daily hassles and diabetes-related distress. Similarly, Cohen and Kanter (2004) found that the less ability to cope with stress the greater the deterioration of diabetic regimen; that is, adherence to self-care behaviors such as regular clinic attendance, blood tests, medication, diet, and physical activity, was positively affected by the belief of self-efficacy and negatively affected by psychological distress. Hence, by the previous scientific evidence we can infer that psychological stress, lack of social support, and poor belief of having control over life’s challenges or lack of self-efficacy indirectly impact the stability of glucose control in Type 2 diabetes by decreasing adherence to a proper diet regimen, exercise program, medication, or even consistent glucose monitoring. Accordingly, a way of maintaining constant and appropriate self-care behaviors from diabetes patients is to find venues to deal with everyday stress and major life events, along with a method to increase self-efficacy.

Anxiety and diabetes

Previous research has provided evidence of the relationship between psychological distress, in the form of anxiety, and glucose control of Type 2 diabetes patients. Okada, Hamada, Ishii, Ichiki, Tanokuchi, and Ota (1995) found significant reductions in anxiety levels, but most importantly, significant reductions in HbA$_{1c}$ from
8.4% to 7.3% for Type 2 diabetes patients following a 12-week treatment with an anti-anxiety drug. Diet, exercise, and medical therapy remained constant for all participants throughout the study. Similarly, Lane, McCaskill, Ross, Feinglos, and Surwit (1993) found that subjects who demonstrated improvements in glucose tolerance after a pharmacological intervention to decrease anxiety, also improved in glucose tolerance after a progressive muscle relaxation (PMR) treatment. Furthermore, those subjects that showed a greater deterioration in blood glucose levels with an epinephrine infusion that resembled secretion during episodes of stress had a trend toward greater improvements in glucose tolerance after relaxation treatment. They concluded that relaxation therapy may produce significant improvements in glucose tolerance for those individuals who suffer from anxiety and are more susceptible to adrenergic stimulation by catecholamines (Lane et al., 1993). The influence of yoga practice on reductions in anxiety may then facilitate improved glucose tolerance. Yoga has been found previously to significantly reduce both, state anxiety and trait anxiety, in patients with a variety of medical problems including hypertension, obesity, and diabetes (Gupta, Khera, Vempati, Sharma, & Bijlani, 2006).

The role of cortisol on glycemic control in diabetes

According to the General Adaptation Syndrome (GAS) theory by Hans Seyle, there are three phases of the body’s adaptation to stress, which are: alarm reaction, stage of resistance, and stage of exhaustion. Alarm reaction is the “fight or flight” response originally described by Canon, characterized by sudden elevations of
epinephrine and norepinephrine. During the stage of resistance there is a reduction of catecholamines; however, there is a marked increase in cortisol secretion in order to sustain heightened levels of functioning in the body (Asterita, 1985). It is this stage of the body response and adaptation to stress that may pose significant challenges to diabetes patients because of the effect of cortisol on glucose metabolism.

The work by Eigler, Sacca, and Sherwin (1979) demonstrated that cortisol maintains the elevated glucose production initially generated by epinephrine and glucagon, and that changes in glucose metabolism during simulated episodes of stress are a result of the synergistic interaction between these hormones and not just one hormone alone. Therefore, continuous elevations of cortisol produced from chronic stress are expected to be more detrimental to diabetes patients because they already have difficulty metabolizing plasma glucose due to insulin resistance or irregular insulin secretion, and consequently glycemic control will be compromised. Additionally, research has already provided some evidence of altered hypothalamic-pituitary-adrenal (HPA) axis and subsequent cortisol elevations in diabetes patients (Chiodini et al., 2007; Hudson et al., 1984; Roy, Collier, & Roy, 1990). Previous research has also reported a significant correlation between elevated cortisol levels at midnight and poor glycemic control and diabetes complications (Chiodini et al., 2007).

In light of the previous scientific evidence, we can hypothesize that individuals with glucose intolerance and elevated cortisol levels, such as diabetes patients, may be even more susceptible to the negative effects of stress. Therefore, it can be postulated that a reduction in psychological stress, similar to that incurred through yoga practice,
may be beneficial for diabetes patients by decreasing adrenal-cortical activity and consequently cortisol levels.

Surwit and Feinglos (1983) analyzed the effect of PMR training and biofeedback sessions on glucose tolerance among non-insulin dependent diabetes patients. All participants were hospitalized and provided with weight-maintenance diets for 5 days; however, only the treatment group practiced PMR twice a day during that week. At the end of the study significant decreases in the area under the curve were observed during an oral glucose tolerance test (OGTT) for the relaxation group; however, there was no change in the control group. Insulin sensitivity, as measured by an insulin tolerance test (ITT), also showed a trend towards improvement but the observed changes failed to achieve statistical significance. Furthermore, significant decreases in plasma cortisol were reported for the PMR treated patients during post-testing whereas cortisol was significantly increased for the control patients; catecholamines remained unchanged for both groups (Surwit & Feinglos, 1984). The authors suggested that relaxation training may be related to decreases in adrenal cortical activity; specifically, that variations in plasma cortisol levels could alter adrenergic receptor sensitivity to circulating catecholamines thereby changing glucose tolerance (Surwit & Feinglos, 1984).

Nonetheless, another study examining the effect of PMR on metabolic control of diabetes patients found no significant improvements in glucose tolerance (Jablonski, Naliboff, Gilmore, & Rosenthal, 1997). One of the main differences between this study and that of Surwit and Feinglos (1983) was patient compliance; while Surwit and Feinglos (1983) closely monitored the twice daily practice of PMR in a hospital setting,
Jablon et al. (1997) used an outpatient protocol in which subjects were instructed to practice twice a day on their own. A lack of PMR practice compliance could affect the ultimate results of the study. Moreover, Jablon et al. (1997) did not find significant differences in anxiety scores, as measured by the State-Anxiety Inventory (STAI), between the PMR group and the control group at the end of the study. In addition, subjects responded to a subjective relaxation ability scale where 0 represented “not at all able to relax” and 10 “able to relax extremely well.” Despite significant improvement in the scale response (pre: 3.4 vs. post: 5.7), the average values may indicate that the PMR group did not achieve proficiency in their relaxation ability.

In the study by Surwit and Feinglos (1983), the lack of psychological stress assessment of participants was the major weakness; however, under highly controlled conditions it was demonstrated that the treatment itself, and not extraneous variables such as modifications in diet or additional physical activity, produced the significant improvements in glucose tolerance and decreases in physiological markers of stress like cortisol. Nevertheless, subsequent studies are challenged to establish a more clear relationship between psychological stress and glucose control in diabetes.
Yoga has been previously investigated as an alternative therapy for glycemic control in non-insulin dependent diabetes patients (Amita et al., 2009; Agrawal et al., 2003; Agte & Tarwadi, 2004; Gokall, et al., 2007; Gordon et al., 2008; Jain et al., 1993; Malhotra et al., 2005; Mercuri et al., 2003; Singh et al., 2004; Singh et al., 2001). Research has provided evidence of the potential benefit of yoga on glycemic control parameters (Amita et al., 2009; Agrawal et al., 2003; Agte & Tarwadi, 2004; Gokall et al., 2007; Gordon et al., 2008; Jain et al., 1993; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001); nonetheless, results are inconclusive since the majority of research had no control over extraneous variables, especially those factors that may affect fasting glucose and glycated hemoglobin measurement. Furthermore, there is no consensus within the literature to explain the improvement in glycemic control after yoga practice. Therefore, the primary purpose of this study was to investigate the effect of yoga practice on glycemic control of Type 2 diabetes mellitus patients under controlled conditions. The second purpose of the study was to investigate the physiological and psychological impact of yoga practice on glycemic status for these patients by evaluating changes in the stress hormone cortisol and psychological stress measures. It is hypothesized that yoga may act indirectly by increasing measures of regimen adherence, and directly by decreasing measures of stress and anxiety and consequently decreasing measures of physiological stress such as cortisol. Hence, it is expected that following the yoga intervention, measures of both short-term and long-term glycemic control will improve in patients.
The hypotheses are as follows:

H₁: FBG will decrease following 6 weeks of yoga practice
H₂: HbA₁c will decrease following 6 weeks of yoga practice
H₃: Cortisol will decrease following 6 weeks of yoga practice
H₄: Perceived stress and anxiety will decrease following 6 weeks of yoga practice
H₅: Measures of quality of life and regimen adherence will improve following 6 weeks of yoga practice
H₆: Additional benefits such as increased flexibility and balance will be observed following 6 weeks of yoga practice
METHODS

Participants

Participants (n=10) were recruited from the university and surrounding community through campus announcements, brochures at medical clinics, presentations at El Paso Diabetes Association, and a public service announcement in a local newspaper. Interested individuals were pre-screened with a health questionnaire to determine qualification to participate in the study. Inclusion criteria were the following: (1) women had to be post-menopausal and not taking any kind of hormone replacement therapy; (2) no complications derived from diabetes such as neuropathy, retinopathy, nephropathy, etc; (3) no cardiovascular disease such as coronary heart disease, cerebrovascular disease, peripheral arterial disease, deep vein thrombosis, etc; (4) no conditions that alter the 120-day life-span of red blood cells such as hemolytic anemia, kidney disease, liver disease, sickle-cell disease, recent blood loss, or folate deficiency as these conditions can affect the accuracy of the HbA\textsubscript{1c} measurement; (5) no major musculoskeletal disorders or injuries, especially of the spine; (6) being able to stand unassisted for at least 30 minutes; (7) sedentary, defined as no more than once a week of moderate exercise and no vigorous exercise during the previous three months; and (8) no previous experience with yoga or meditation practice. All participants were previously diagnosed by their personal physician with Type 2 diabetes mellitus; none of them were undergoing insulin therapy and therefore were non-insulin dependent. Characteristics of participants are shown in Table 1. A written statement from their personal physician providing clearance to participate in an exercise program was provided by all participants before the beginning of the intervention and all participants
provided written informed consent to participate. This entire study was approved by the Institutional Review Board for research involving human subjects of the University of Texas at El Paso.

<table>
<thead>
<tr>
<th>Table 1. Mean (± SD) characteristics of Type 2 diabetes patients at baseline and following the 6-week yoga intervention.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre-intervention</strong></td>
</tr>
<tr>
<td>Age (yrs)</td>
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<tr>
<td>Length of disease (yrs)</td>
</tr>
<tr>
<td>Height (cm)</td>
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<tr>
<td>Body Mass (kg)</td>
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<tr>
<td>BMI (kg/m²)</td>
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<td>Fat mass (kg)</td>
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<tr>
<td>Lean mass (kg)</td>
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<td>Bone mineral content (kg)</td>
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No significant differences between pre- and post-intervention ($p>0.05$).
Yoga Intervention

The 6-week intervention consisted of hatha yoga classes composed of asanas (physical poses), pranayama (breathing exercises), relaxation, and meditation exercises. Classes were taught by a qualified registered yoga teacher (RYT) and were held in the Biomechanics Laboratory on the University of Texas at El Paso campus. The duration and frequency of classes were approximately 50-60 minutes, three days per week. Because all participants were previously sedentary, overweight, and above the age of 50 years, all poses were modified to accommodate limited levels of flexibility, balance, and strength. Props such as chairs, belts, or blankets were used for such modifications to allow for appropriate alignment, technique, and balance, and especially to provide participants the opportunity to obtain full benefit from the asanas within their particular limitations (see Appendix). During each class, participants were instructed to center their attention on their breathing throughout the practice session and to increase the awareness of their body position in each pose. Some of the classes had the focus of a particular topic such as “Yoga for the neck and shoulders,” “Yoga for the back,” “Yoga for insomnia,” and “Yoga as a meditative practice.” For instance, during the session of insomnia participants were taught restorative yoga poses which consisted of modified supine poses. Participants placed blankets underneath the lower back and rolled blankets underneath the neck so they could have their spine supported and they could relax completely into the poses while at the same time stretching certain areas of the body. The closure of each session consisted of a relaxation exercise in which participants were asked to scan their entire body to locate any tension still present in
their muscles and to relax those specific muscles with every exhalation emphasizing focus on the natural pattern of breathing and its sound.

Yoga mats were provided to all participants throughout the intervention, as well as chairs for modifications. However, other props such as belts, towels or blankets were brought from home by the participants. Handouts with yoga poses and meditation exercises were provided approximately every 2 weeks so that participants could continue practice at home and after the end of the intervention.

Finally, although hypoglycemia is uncommon during exercise in individuals who are not treated with insulin (ADA, 2010), such as the participants of this study, caution was implemented at all times. All participants were encouraged to check their glucose levels before each yoga class and were instructed to ingest a carbohydrate snack if their glucose levels were below 100 mg/dL. In addition, glucose tablets (Reli On®, CanAm Care, LLC., Alpharetta, GA) were available during yoga classes as a preventive measure for the unlikely occurrence of a hypoglycemic event. Four of these glucose tablets provide 15 grams of carbohydrate as suggested by the ADA to offset a sudden drop in circulating plasma glucose (ADA, 2010). No hypoglycemic events occurred during this intervention.

Each participant completed a battery of assessments at baseline and following the 6-week yoga intervention. To assess the overall effectiveness of the yoga program, anthropometric, body composition, blood pressure, flexibility, range of motion, and balance were measured. The effect of the intervention on markers of glucose control was assessed from fasting blood samples, while alterations in perceived stress, anxiety,
quality of life and self-care were assessed by questionnaires.

**Anthropometric measurements**

Height was measured to the nearest 0.1 cm without shoes on a mobile Seca 225 stadiometer (Seca, Ontario, CA, USA). Body mass was measured to the nearest 0.1 kg with a Tanita WB-110A load cell scale (Tanita, Illinois, USA). Body mass index (BMI) was calculated by dividing mass in kilograms by height in meters squared. Waist circumference was measured at the end of a normal exhalation to the nearest 0.1 cm using a Gulick II measuring tape with tension spring (Country Technology Inc., Gays Mills, WI, USA) with the participant standing upright and abdomen relaxed. The waist circumference was measured at the narrowest part of the torso, above the umbilicus and below the xiphoid process, by placing the tape on the skin surface without compressing the subcutaneous adipose tissue as described by the manufacturers’ instructions. Duplicate measurements were recorded and the procedure was repeated if measures were not within 5 mm (ACSM, 2010).

**Blood pressure**

Blood pressure was measure with an automated Bp TRU™ vital sign monitor (BPM-300, VSM Med Tech Ltd., Canada). Participants were asked to sit quietly on a chair for at least five minutes prior to measurement. The upper arm was positioned at heart level and supported by the arm of the chair. The appropriately sized cuff was wrapped firmly around the upper arm and aligned with the brachial artery. The cuff was
automatically inflated to approximately 20 mmHg above the first Korotkoff sound. Pressure was slowly released at a rate of approximately 5 mmHg per second. Both, systolic and diastolic pressure were displayed on the LCD screen of the instrument and recorded in mmHg. Blood pressure was measured in duplicate with three minutes between each measurement and the average of the two measurements for each participant was recorded (ACSM, 2010). Each participant was provided with the blood pressure results immediately after measurement.

Body composition

Body composition was assessed using dual energy x-ray absorptiometry (DEXA) (Lunar DPX-NT, GE Lunar Corp., Madison, WI). Participants lie supine on a padded table while a whole body x-ray scan was performed. The entire procedure for this particular population lasted approximately 30 minutes. Each subject was exposed to approximately 0.03 mrad of radiation during a single test and had a total radiation exposure for this project of approximately 0.06 mrad. This low exposure is less than 2% of the radiation received from a chest x-ray, and less than half the radiation exposure from six hours outdoors. Since only post-menopausal women participated in this study, there was no concern for pregnant women exposed to radiation. A quality assurance test (QA), which calibrates and verifies the correct operation of the densitometer, was performed at the start of each testing day to examine the functionality, accuracy, and precision of the system. Each participant was appropriately positioned on the padded table and was asked not to move during the measurement. One strap was placed
around the knees and another one around the ankles to allow subject to relax while maintaining a correct position. Any metals such as watches or rings were removed before testing started. After the scan was completed, a brief explanation of the results was provided to each participant and any questions regarding the test were answered. Fat mass, lean mass, and bone mineral content were recorded to the nearest 0.1 kg. Percentage body fat was also recorded.

**Flexibility**

*Sit-n-reach test.* Flexibility of the lower back and hamstrings was measured with a sit-n-reach test using a sit-n-reach box (Acuflex I Trunk Flex Tester, Country Technology Inc., Gays Mills, WI). Participants sat on the floor without shoes, toes pointing upward, knees extended, and soles of the feet against the box. They were instructed to slowly reach forward with both hands as far as possible moving the slide bar across the long bar of the box with their fingers and held the maximum reach momentarily while the flexion measurement was recorded to the nearest 0.1 cm from the calibrated bar. Participants were allowed a warm-up trial, then, the best of three trials was recorded as the sit-n-reach score.

*Range of motion at the hip.* Active range of motion (AROM) at the hip was assessed with a double-armed universal goniometer (Baseline™, Medelco, Inc., Ontario, Canada) for the following movements on both sides of the body: (1) flexion, (2) adduction and abduction, and (3) internal and external rotation. All measurements were recorded in degrees. Three consecutive trials of each movement were performed on
each side with the participant in a supine position and the average of the three was recorded as the participant’s AROM.

**Hip flexion.** Participants were positioned supine over a testing table. The axis of the goniometer was positioned over the greater trochanter of the femur with the stationary arm parallel to the midaxillary line of the trunk. The moveable arm was aligned with the longitudinal axis of the femur, using the lateral epicondyle as a reference. Participants were asked to hold the leg of the non-tested side in a flexed position with foot flat on the table to allow a neutral position of the lumbar spine. Participants were then instructed to flex the knee and the hip of the tested side, bringing the knee as close to the chest as possible to perform maximum hip flexion. The procedure was repeated on the opposite side of the body (Greene & Heckman, 1994).

**Hip abduction and adduction.** Participants were positioned supine over a testing table. For adduction, the leg of the non-tested side was abducted to allow full ROM during adduction on the tested side. The axis of the goniometer was positioned over the anterior superior iliac spine (ASIS) of the extremity being measured. The stationary arm was placed along the line that joins the two ASISs; while the moveable arm was aligned with the longitudinal axis of the femur, using the midline of the patella for reference. Participants were instructed to perform adduction or abduction in the frontal plane without any degree of hip flexion or rotation. The procedure was repeated on the opposite side of the body (Greene & Heckman, 1994).

**Hip internal and external rotation.** Participants sat straight at the edge of the testing table, with the back of the knees against the table, and legs slightly further apart
than hip-width. The axis of the goniometer was positioned at the patella, while both the stationary and the moveable arm were aligned with the longitudinal axis of the tibia. Participants were then instructed to perform either an internal or external rotation by moving the foot laterally or medially, respectively, avoiding lifting the thigh or the hips off the table. The moveable arm stayed aligned with the tibia through the performance of the movement, while the stationary arm remained perpendicular to the floor (Greene & Heckman, 1994).

Range of motion of the trunk. AROM of the trunk was assessed with two inclinometers (Performance Attainment Associates, MedNet Technologies Inc., St. Paul, MN) and a double-armed universal goniometer. Movements included (1) lumbar extension and lateral flexion, and (2) thoracolumbar rotation. All AROM assessments were measured in degrees, performed on both sides of the body, and the average of three trials was recorded as the participant’s AROM. The cervical, thoracic, and lumbar spine, were at 0 degrees at the start of all movements.

Lumbar extension and lateral flexion. Participants stood upright with feet shoulder-width apart, arms by the sides for lateral flexion and hands over the hips for lumbar extension. One inclinometer was placed at the base of the spine over the sacrum, and another inclinometer was positioned in the middle of the back, approximately at the T12 vertebra. Both inclinometers were zeroed before the beginning of the measurements. For extension, subjects were instructed to bend backwards as far as possible. For lateral flexion, subjects were instructed to bend laterally as far as possible in the frontal plane avoiding flexion and extension in the sagittal plane. The
inclinometers were kept firmly against the spine during the performance of the movements. The degrees from both inclinometers were recorded at the end of motion. The degrees from the sacral inclinometer were subtracted from the degrees on the T12 inclinometer to obtain true lumbar AROM. For lateral flexion, the procedure was repeated on both sides of the body. (Greene & Heckman, 1994).

Thoracolumbar rotation. Participants sat on a chair with back straight, arms crossed in front of chest, and feet flat on the ground. The center of the goniometer was placed over the center of the cranial aspect of the participant's head. Both arms of the goniometer were aligned parallel along the imaginary line between the top of the head and the acromial process of the tested side. Participants were instructed to rotate the trunk to one side as far as possible, maintaining a straight back and feet on the floor. Participants were also instructed to avoid flexion, extension, and lateral flexion of the spine during the performance of rotation (Norkin & White, 2003).

Balance

Unipedal stance test. Balance was assessed by the unipedal stance test. The unipedal stance test is a validated measure of standing balance and risk for falls in adults aged 50 years and older (Hurvitz, Richardson, Werner, Ruhl, & Dixon, 2000). In addition, it has been used previously in studies examining the factors associated with multiple and injurious falls (Richardson, 2002); and as an outcome measure of exercise regimen for diabetes patients suffering from peripheral neuropathy (Richardson, Sandman, & Vela, 2001). Participants were asked to stand barefoot on one foot, with
the other foot raised near the ankle of their stance foot. Participants were instructed to focus on a point on the wall at eye level in front of him/her. Arms were crossed against the chest. A stopwatch was used to measure the amount of time the subject was able to stand on one limb. Time started as soon as the participant’s foot rose off the floor. Recorded time ended when the subject performed one of the following: (1) uncrossed the arms to maintain balance; (2) moved the raised foot forward or backwards away from the standing limb; (3) moved the weight-bearing foot to maintain balance (i.e., rotate foot on the ground); (4) touched the floor with the raised foot; or (5) a maximum of 45 seconds elapsed. Three trials were performed on each foot and the best trial for each foot was recorded as the balance time.

**Blood collection, processing, and handling**

Blood collection was performed by venipuncture from an antecubital vein following an overnight fast. The procedure was performed by a trained technician and universal safety precautions were observed at all times. Venous blood was collected with a syringe using a blood collection set (BD Vacutainer® Safety-Lok™ Blood Collection Set, BD Vacutainer Systems, Franklin Lakes, NJ). From the syringe 1 µL of blood was obtained for HbA\(_{1c}\) analysis, 200 µL of blood was used for FBG analysis, and the rest was transferred into a blank serum tube (BD Vacutainer® Plastic Tubes, BD Vacutainer Systems, Franklin Lakes, NJ) and allowed to clot. The clotted blood was then centrifuged for approximately 15 minutes and the obtained serum was immediately transferred to cryrule vials in 500 µL aliquots and stored at -80°C for later analysis of
insulin. Blood samples were collected at baseline and the end of the 6-week yoga intervention.

**Physiological parameters**

*Glycated hemoglobin (HbA\textsubscript{1c}).* Long-term glycemic control was assessed by percentage of HbA\textsubscript{1c}. The American Diabetes Association (ADA) states that HbA\textsubscript{1c} should be the primary target for glycemic control with target values of less than 7%, equivalent to pre-prandial capillary plasma glucose of 70-130 mg/dL (ADA, 2010). According to the ADA, lowering HbA\textsubscript{1c} levels to ~7% has been observed to decrease microvascular complications of diabetes. The U.S. Department of Health and Human Services has stated that research indicates a strong, graded relation between glycated hemoglobin exposure and risk for retinopathy and nephropathy in Type 2 diabetes. Also, cohort studies have indicated a positive association between glycated hemoglobin and cardiovascular complications (U.S. Department of Health and Human Services, 2003). In addition, levels of glycated hemoglobin have been shown to predict dyslipidemia in Type 2 diabetes patients regardless of gender and age. The percentage of HbA\textsubscript{1c} correlates positively and significantly with total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglycerides (Khan, 2007). High blood cholesterol raises the risk for cardiovascular disease (WHO, 2011). Finally, a 1% decrease in HbA\textsubscript{1c} levels has been associated with a 37% reduction in relative risk for microvascular complications and with a 21% decrease in relative risk of any endpoint or death associated to diabetes (Stratton et al., 2000). Glycated hemoglobin was measured with
a DCA 2000 + Analyzer (Bayer HealthCare LLC., Elkhart, IN). The machine was calibrated according to operating instructions. Whole blood obtained from venipuncture was collected into the glass capillary holder, which was immediately inserted into the reagent cartridge. The cartridge was scanned and inserted into the machine for HbA$_{1c}$ analysis. The measurement was recorded as a percentage to the nearest 0.1 percent.

*Fasting blood glucose (FBG).* Short-term glycemic control was assessed through FBG. The ADA recommends a fasting plasma glucose level between 70-130 mg/dL for adults with diabetes in order to avoid complications derived from the disease (ADA, 2010). Fasting blood glucose was measured with a glucose analyzer (YSI Model 2300 STAT, YSI Incorporated, Yellow Springs, OH). Blood previously obtained by venipuncture was collected into an YSI preservative tube (YSI Incorporated, Yellow Springs, OH). Blood was sampled twice and the average of the two results was recorded as FBG (mg/dL) for each participant.

*Plasma insulin.* Plasma insulin was analyzed with an insulin kit (Insulin microplate ELISA, MP Biomedicals, Inc., Solon, OH). The assay produces a sandwich between two monoclonal antibodies and human insulin, MAb1-human insulin-MAb2, after an incubation period. The amount of bound labeled enzyme was measured through a chromogenic reaction after the appropriate solution is added to the well. The microplate was read at the indicated wavelength and absorbance was measured, which was proportional to insulin concentration. Each sample was analyzed in duplicate and the average of the two results was recorded as the insulin value (pmol/L).
Insulin sensitivity. After analysis of FBG and plasma insulin, insulin resistance was estimated by the Quantitative Insulin Sensitivity Check Index (QUICKI) method with the following formula:

\[
\frac{1}{\log I_0} + \log G_0
\]

where, \(I_0\) is the fasting plasma insulin value (in µU/mL) and \(G_0\) is the fasting plasma glucose value (in mg/dL). Previous studies have demonstrated the validity of QUICKI to estimate insulin sensitivity of Type 2 diabetes patients (Katz et al., 2000).

Salivary cortisol. Research has indicated that nighttime salivary cortisol is a valid measure of hypercortisolism compared to 24-hr urinary free cortisol excretion (Viardot et al., 2005). Each participant collected a saliva sample at home at 0000 hrs using investigator provided collection materials. Within one hour of collecting the saliva sample, participants were instructed not to consume food or drink, except water, and not to perform any oral or dental care to prevent contamination of the sample by blood. Subjects were instructed to place an oral cotton swab (Salimetrics Oral Swab, Salimetrics LLC., USA) under the tongue for approximately 5 minutes or until the swab was saturated with saliva. Then, the saturated swab was placed into the appropriate container (Salimetrics LLC., USA), which was placed in the participants' home freezer until delivery to the lab the following day. After delivery to the lab, all samples were stored at -80°C until cortisol analysis. On the day of the analysis, all samples were brought to room temperature (20-25°C), centrifuged for approximately 15 minutes to separate saliva from the swab, and analyzed for cortisol content with an immunoassay kit (High Sensitivity Salivary Cortisol Immunoassay Kit, Salimetrics LLC., USA). For the
assay, samples were placed in a microtitre plate coated with monoclonal anti-cortisol antibodies, and a cortisol enzyme conjugate was added. After an incubation period, the unbound components were washed away and a substrate solution was added to the plate. The microplate was read at the indicated wavelength and absorbance was measured. The quantity of cortisol peroxidase detected was inversely proportional to the quantity of cortisol present. Each sample was analyzed in duplicate and the average of the two results was recorded as the cortisol value (nmol/L).

**Psychological parameters**

*Perceived stress scale (PSS).* Stress was measured by the PSS 14-item version developed by Cohen and Williamson (1988). The scale assesses the extent to which the respondent’s situations in life are perceived as stressful, unpredictable, and uncontrollable. The PSS contains 14 items, 7 positive and 7 negative, appraising how often the subject felt or thought in a particular way during the previous month. Items are answered using a Likert scale from 0=never to 4=very often. The total score was obtained by reversing the scores on the positive items and then summing across all items. The higher the score, the greater the individual’s perceived stress. The validity and reliability of the PSS has been assessed previously (Cohen & Williamson, 1988). It has been used in stress and diabetes research (Lazcano & Salazar, 2007), in research investigating the effect of PMR training on glycemic control (Surwit et al., 2002), and in several studies investigating the effect of yoga and meditation practice on stress relief (Carmody & Baer, 2008; Cowen & Adams, 2005; Granath, Ingvarsson, von Thiele, & Lundberg, 2006; West, Otte, Geher, Johnson, & Mohr, 2004).
State-Trait Anxiety Inventory (STAI). Anxiety was measured by the STAI developed by Spielberger, Gorsuch, and Lushene (1970). The STAI consists of two subscales, the State scale (S-scale) and the Trait scale (T-scale); each contains 20 items rated on a 4-point Likert scale. The higher the score obtained, the greater the anxiety. State anxiety is defined as a temporary emotional state, while trait anxiety is defined as a relatively stable personal characteristic in anxiety proneness (Spielberger et al., 1970). The STAI scale has been used in research investigating the effect of PMR on diabetic populations (Jablon et al., 1997; Lammers, Naliboff, & Straatmeyer, 1984; Surwit et al., 2002), in studies about the effect of yoga on psychological states (Agte & Chiplonkar, 2008; Gupta et al., 2006; Javnbakht, Kenari, & Ghasemi, 2009), for research evaluating psychological states of Type 2 diabetes patients in which levels of anxiety where correlated with fasting blood glucose (Mosaku, Kolawole, Mume, & Ikem, 2008), and for research where the improvement of anxiety was associated with decreased HbA$_{1c}$ levels of Type 2 diabetes patients (Okada et al., 1995).

Diabetes-39 questionnaire (D-39). Diabetes-related quality of life was assessed with the D-39 questionnaire developed by Boyer and Earp (1997). The D-39 consists of 39 items covering several dimensions of health targeted specifically to diabetes patients. Subscales utilized included: energy and mobility, diabetes control, anxiety and worry, and social burden. The validity and reliability of this psychological instrument has been confirmed previously (Boyer & Earp, 1997).

Summary of Diabetes Self-Care Activities Questionnaire. Regimen adherence to diabetes self-care was assessed through the Summary of Diabetes Self-Care Activities
Questionnaire (SDSCA) (Toobert & Glasgow, 1994). The questionnaire measures four different dimensions including blood glucose testing, medications, diet, and exercise. In general, questions record the number of days during the previous week (0-7) the individual engages in a specific health behavior. Total number of days across dimensions was the total score minus the smoking status and number of cigarettes smoked; hence, the higher the score, the greater the adherence to self-care behaviors. The validity and reliability of the questionnaire have been previously confirmed for Type 2 diabetes patients (Toobert, Hampson, & Glasgow, 2000).

**Extraneous variables**

To decrease the possibility that external factors, not of interest in this study, confound the results, participants were instructed not to change their levels of physical activity or their usual diet during the intervention. Physical activity was documented pre- and post-intervention through the International Physical Activity Questionnaire (IPAQ) targeted to adults 18-65 years of age (Craig et al., 2003). The validity and reliability of the IPAQ has been previously demonstrated in several studies around the world (Craig et al., 2003). Additionally, the IPAQ questionnaire was used in a research study investigating the intensity of physical activity and its relationship with gender and age of Type 2 diabetes mellitus patients aged 41-85 years (Kuduzovic, Nuhbegovic, Ljuca, & Imamovic, 2009).

Diet was also documented before and after the intervention through diet recalls assessed for caloric intake and macronutrient composition using dietary software (The Food Processor 2004, ESHA Research, Salem, OR). In addition, a medication log
documenting all prescribed medication, such as diabetes-specific medication, and over-the-counter medication was provided by all participants before the beginning of the study and at the end of the intervention.

*Statistical Analysis*

Data were analyzed using the Statistical Package for the Social Sciences (SPSS, version 16.0, Chicago, IL). Standard descriptive statistics was used to describe all variables under investigation. Descriptive variables included age, years since diabetes diagnosis, height, body mass, BMI, waist circumference, systolic and diastolic blood pressure, percentage body fat, fat mass, lean mass, and bone mineral content. Glycemic control was assessed from HbA$_{1c}$, FBG, and insulin analysis, and from estimation of insulin sensitivity by QUICKI. Physiological stress was assessed from cortisol content, whereas psychological stress was assessed from the perceived stress and anxiety questionnaires. Other psychological variables included measures of quality of life and self-care behaviors. An initial analysis revealed that the data violated the assumption of normality. Several transformation methods failed to normalize the data. Because the data were not normally distributed coupled with the limited sample size, nonparametric tests were applied for statistical analysis. The Wilcoxon Signed Rank Test was performed to assess changes between pre-intervention and post-intervention values for all variables under investigation. In addition, Spearman’s rho correlation was conducted to analyze the relationship between measures of glycemic control and measures of both physiological and psychological stress. Statistical significance was set at $\alpha=0.05$. 
RESULTS

A total of 17 individuals were enrolled in the study and completed initial baseline testing. Four participants withdrew prior to starting the intervention because of schedule conflicts, or lack of medical access that could provide clearance to participate in the program. Two more participants withdrew after the first week of the intervention because of schedule conflicts or unrelated health complications. In addition, the data from one participant was excluded because of reported insulin therapy. Ten participants were included in the final analysis. The average attendance rate of the yoga classes was 81.6%. Type of medication used by participants included metformin (n=9), sulfonylureas (n=1), alpha-glucosidase inhibitors (n=1), meglitinide (n=1), incretin (n=1), meglitinide (n=1), and glitazone (n=1). Participants reported no change in medication use during the duration of the study.

Physical characteristics of participants (n=10) were similar between baseline and following the yoga intervention (Table 1). Although not statistically significant, a modest decrease from pre- to post-intervention was observed for body mass ($p=0.06$), waist circumference ($p=0.07$), BMI ($p=0.07$), and lean mass ($p=0.13$), with a concurrent increase in bone mineral content ($p=0.13$), percent body fat ($p=0.12$), and fat mass ($p=0.44$). There was no significant change ($p=0.72$) in physical activity patterns as demonstrated by pre- and post- values for the physical activity questionnaires. In contrast, the dietary analysis revealed a significant decrease ($p=0.017$) in total calories and protein consumption after the intervention; although, there was no change in calories from fat ($p=0.26$), saturated fat ($p=0.32$), or fat intake ($p=0.26$).
Baseline and post-intervention parameters of glycemic control are presented in Table 2. Compared to baseline, modest but non-significant differences were observed following the yoga intervention for HbA$_{1c}$ ($p=0.18$), FBG ($p=0.08$), and fasting insulin ($p=0.10$). Insulin sensitivity, as measured by QUICKI, also remained unchanged ($p=0.38$) from pre- to post-intervention.

Table 2. Mean (± SD) glycemic control measures of Type 2 diabetes patients at baseline and following the 6-week yoga intervention.

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HbA$_{1c}$ (%)</strong></td>
<td>8.35 ± 2.79</td>
<td>8.28 ± 2.44</td>
</tr>
<tr>
<td>Fasting glucose (mg/dL)</td>
<td>130.59 ± 81.83</td>
<td>119.81 ± 65.57</td>
</tr>
<tr>
<td>Fasting insulin (pmol/l)</td>
<td>97.85 ± 73.55</td>
<td>112.86 ± 80.63</td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.33 ± 0.03</td>
<td>0.32 ± 0.04</td>
</tr>
</tbody>
</table>

No significant differences between pre- and post-intervention ($p>0.05$).

Measures of physiological and psychological stress improved following the yoga intervention (Table 3). Compared to baseline, salivary cortisol significantly decreased after the 6-week intervention ($p=0.04$). There were also decreases in measures of perceived stress from the PSS ($p=0.01$) and state anxiety from the STAI ($p=0.006$). Trait anxiety from the STAI remained unchanged ($p=0.07$). In addition, the SDSCA indicated a significant improvement for measures of diabetes self-care after the intervention ($p=0.004$), while the D-39 measures of quality of life also improved but the change was not statistically significant ($p=0.16$) (Table 4).
Table 3. Mean (± SD) physiological and psychological measures of stress for Type 2 diabetes patients at baseline and following the 6-week yoga intervention.

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol (nmol/l)</td>
<td>6.89 ± 7.72</td>
<td>2.76 ± 1.65*</td>
</tr>
<tr>
<td>PSS</td>
<td>22.80 ± 7.96</td>
<td>17.50 ± 7.12*</td>
</tr>
<tr>
<td>State anxiety</td>
<td>39.80 ± 13.27</td>
<td>29.20 ± 8.56†</td>
</tr>
<tr>
<td>Trait anxiety</td>
<td>36.80 ± 11.22</td>
<td>31.10 ± 8.13</td>
</tr>
</tbody>
</table>

Perceived Stress Scale (PSS). State and Trait anxiety determined from State and Trait Anxiety Inventory (STAI). Significantly different from baseline * (p<0.05), † (p<0.01).

Table 4. Mean (± SD) psychological measures of self-care and quality of life for Type 2 diabetes patients at baseline and following the 6-week yoga intervention.

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>SDSCA</td>
<td>42.96 ± 12.88</td>
<td>56.04 ± 15.26†</td>
</tr>
<tr>
<td>D-39</td>
<td>34.61 ± 18.60</td>
<td>30.63 ± 20.87</td>
</tr>
</tbody>
</table>

Data for both questionnaires are presented as a total score across all measured dimensions. Summary of Diabetes Self-Care Activities Questionnaire (SDSCA); Diabetes-39 Questionnaire (D-39). An increase in score on the SDSCA indicates improvement, while a decrease in score on the D-39 indicates improvement. Significantly different from baseline † (p<0.01).
Bivariate analysis revealed that post-intervention cortisol values had no significant correlation with post-intervention FBG or HbA$_{1c}$ values. However, partial correlation analysis demonstrated a significant correlation between post-intervention cortisol values and post-intervention FBG ($r^2=0.77$, $p=0.02$) when controlling for baseline FBG values. The correlation between post-intervention cortisol and HbA$_{1c}$ values only approached significance ($r^2=0.62$, $p=0.08$). That is, at the end of the intervention cortisol values had a significant relationship with FBG relative to initial levels of FBG.

Spearman’s rho demonstrated a negative correlation between baseline PSS and baseline insulin sensitivity scores ($r_s=-0.58$, $p=0.08$), and between baseline state anxiety scores and baseline insulin sensitivity scores ($r_s=-0.60$, $p=0.07$). That is, the greater the initial scores for stress and anxiety, the poorer the insulin sensitivity of these diabetes patients. Unfortunately, these correlations approached but failed to reach statistical significance. Moreover, there was no significant correlation between post-intervention PSS and insulin sensitivity scores ($r_s=0.27$, $p=0.46$), or between post-intervention anxiety and insulin sensitivity scores ($r_s=0.12$, $p=0.75$) (Table 5).
Table 5. Spearman rho correlations between baseline and post-intervention values of psychological measures and insulin sensitivity of Type 2 diabetes patients.

<table>
<thead>
<tr>
<th></th>
<th>QUICKI pre</th>
<th>QUICKI post</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PSS pre</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Correlation</em></td>
<td>-0.58</td>
<td>-0.20</td>
</tr>
<tr>
<td><em>Sig. (2-tailed)</em></td>
<td>0.08</td>
<td>0.60</td>
</tr>
<tr>
<td><strong>S-anxiety pre</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Correlation</em></td>
<td>-0.60</td>
<td>-0.40</td>
</tr>
<tr>
<td><em>Sig. (2-tailed)</em></td>
<td>0.07</td>
<td>0.26</td>
</tr>
<tr>
<td><strong>PSS post</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Correlation</em></td>
<td>-0.24</td>
<td>0.27</td>
</tr>
<tr>
<td><em>Sig. (2-tailed)</em></td>
<td>0.51</td>
<td>0.46</td>
</tr>
<tr>
<td><strong>S-anxiety post</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Correlation</em></td>
<td>-0.38</td>
<td>0.12</td>
</tr>
<tr>
<td><em>Sig. (2-tailed)</em></td>
<td>0.28</td>
<td>0.75</td>
</tr>
</tbody>
</table>

Perceived stress scale (PSS); State anxiety (S-anxiety); Quantitative Insulin Sensitivity Check Index (QUICKI). No significant correlations ($p>0.05$).

A similar pattern was observed for cortisol with several correlations approaching significance (Table 6). Percent change in cortisol appeared to have a positive correlation with percent change in insulin sensitivity ($r_s=0.58$, $p=0.08$) following the yoga intervention, which means that those who had positive changes in cortisol values also had positive changes in insulin sensitivity. Also, there was a positive correlation between percent change in state anxiety and percent change in FBG ($r_s=0.62$, $p=0.06$), and between percent change in state anxiety and percent change in HbA$_1c$ ($r_s=0.53$, $p=0.11$). That is, those who had positive changes in anxiety also had positive changes
in glycemic control. Nonetheless, these relationships failed to reach statistical significance.

Table 6. Spearman rho correlations between changes in cortisol and state anxiety with glycemic control after the 6-week yoga intervention.

<table>
<thead>
<tr>
<th>%change cortisol</th>
<th>%change HbA1c</th>
<th>%change QUICKI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation</td>
<td>0.16</td>
<td>0.48</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0.65</td>
<td>0.17</td>
</tr>
</tbody>
</table>

%change anxiety

<table>
<thead>
<tr>
<th>Correlation</th>
<th>%change HbA1c</th>
<th>%change QUICKI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.62</td>
<td>0.53</td>
<td>-0.15</td>
</tr>
<tr>
<td>0.06</td>
<td>0.11</td>
<td>0.67</td>
</tr>
</tbody>
</table>

No significant correlations (p>0.05)

Finally, all measures of AROM and balance improved following the yoga intervention, except for trunk left lateral flexion and lumbar extension (Table 7). Hip flexion (p≤0.007), abduction (p≤0.02), adduction (p=0.02), internal rotation (p≤0.04), and external rotation (p≤0.006) for both sides of the body significantly improved following the 6-week intervention. There were also increases in AROM of the trunk including right lateral flexion (p=0.02), right rotation (p=0.002), and left rotation (p=0.004). There were no significant changes for trunk left lateral flexion (p=0.06) or lumbar extension (p=0.20). Flexibility of the hamstrings and lower back, as measured by the sit-n-reach
test, also improved ($p=0.005$). Likewise, unipedal balance scores significantly improved for both the right and the left foot following the intervention ($p\leq0.04$).

Table 7. *Mean (± SD) active range of motion and balance of Type 2 diabetes patients at baseline and following the 6-week yoga intervention.*

<table>
<thead>
<tr>
<th></th>
<th>Pre-intervention</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hip flexion (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>116.5 ± 5.7</td>
<td>122.8 ± 6.6†</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>113.5 ± 6.7</td>
<td>124.3 ± 6.9†</td>
</tr>
<tr>
<td><strong>Hip abduction (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>36.3 ± 8.7</td>
<td>43.7 ± 9.5*</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>37.3 ± 9.8</td>
<td>46.8 ± 9.3*</td>
</tr>
<tr>
<td><strong>Hip adduction (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>28.9 ± 8.9</td>
<td>35.5 ± 6.8*</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>34.0 ± 8.5</td>
<td>39.8 ± 6.7*</td>
</tr>
<tr>
<td><strong>Hip internal rotation (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>24.2 ± 7.3</td>
<td>29.5 ± 7.0*</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>25.4 ± 5.3</td>
<td>29.0 ± 7.4*</td>
</tr>
<tr>
<td><strong>Hip external rotation (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>22.8 ± 7.7</td>
<td>33.8 ± 8.2†</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>23.9 ± 7.0</td>
<td>32.0 ± 7.7†</td>
</tr>
<tr>
<td><strong>Trunk lateral flexion (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>18.7 ± 10.0</td>
<td>26.7 ± 9.4*</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>19.7 ± 11.6</td>
<td>26.8 ± 9.5</td>
</tr>
<tr>
<td><strong>Trunk rotation (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>25.6 ± 9.3</td>
<td>39.4 ± 4.6†</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>27.0 ± 10.4</td>
<td>38.1 ± 4.3†</td>
</tr>
<tr>
<td><strong>Lumbar extension (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>11.0 ± 5.7</td>
<td>12.8 ± 7.9</td>
</tr>
<tr>
<td><strong>Sit-n-reach (cm)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Right</em></td>
<td>20.6 ± 7.7</td>
<td>25.8 ± 6.9†</td>
</tr>
<tr>
<td><em>Left</em></td>
<td>16.4 ± 17.0</td>
<td>24.0 ± 17.6†</td>
</tr>
</tbody>
</table>

Significantly different from baseline * ($p<0.05$), † ($p<0.01$).
DISCUSSION

The primary purpose of this study was to investigate changes in glycemic control of Type 2 diabetes patients after a 6-week yoga practice intervention. Glycemic control parameters, specifically FBG and HbA1c, showed a trend toward improvement, but change was not statistically significant. Nonetheless, the mean post-intervention FBG value (119.81 ± 65.57 mg/dL) achieved the ADA suggested fasting glucose target goal of less than 130 mg/dL for adults with diabetes (ADA, 2010).

The results from this study contrast the findings from previous research conducted in India reporting significant decreases in glycemic control parameters such as FBG, HbA1c, and post-prandial glucose (Amita et al., 2009; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001). The length of the intervention in these studies was 40 days, similar to the 42-day intervention of the present investigation, except for the Amita et al. (2009) study that conducted a 3-month yoga meditation intervention. However, the subjects described by Malhotra et al. (2005) had a substantially lower body mass compared to the participants of this study (62.94 ± 2.4 kg vs. 95.25 ± 24.48 kg, respectively). Singh et al. (2004) and Singh et al. (2001) did not report anthropometric characteristics. In fact, based on BMI classifications (ACSM, 2010), subjects in the Amita et al. (2009) study were normal-weight diabetics (22.64 kg/m²) and subjects in the Malhotra et al. (2005) study were considered only overweight (26.81 ± 0.9 kg/m²), while 20% of the participants in this investigation were in the obesity class II category (37.67 ± 2.82 kg/m²) and 40% were in the obesity class III category (43.73 ± 1.18 kg/m²). Insulin resistance has been shown to be more prevalent among individuals with obesity and abdominal adiposity (Vanhala, Pitkajarvi, Kumpusalo, & Takala, 1998).
Fasting insulin and insulin responses to an oral glucose challenge have also been found to be significantly higher for obese diabetics compare to normal weight diabetics (Turkoglu et al., 2003). It is postulated that the diabetes patients of this study may have achieved greater changes in glucose control if they had experienced a concomitant reduction of body mass. Singh, Kyizom, Singh, Tandon, & Madhu (2008) found significant decreases ($p<0.01$) in FBG and post-prandial glucose, with a simultaneous decrease ($p<0.05$) in body mass, for Type 2 diabetes patients following a 45-day yoga intervention. They also found significant decreases ($p<0.05$) in serum insulin, possible indicating an improved insulin sensitivity following yogic exercises as suggested by the authors.

Accordingly, a longer duration intervention may be necessary to achieve both weight reduction and glucose control. Gordon et al. (2008) implemented a 6-month yoga intervention to a sample of Type 2 diabetes patients and reported significant decreases ($p<0.05$) for FBG from baseline (211.53 ± 6.13 mg/dL) at 3 months (156.94 ± 4.32 mg/dL) and 6 months (153. 52 ± 4.32 mg/dL) in the yoga group, while no changes were observed in the control group. However, Gordon et al. (2008) did not report specific anthropometric characteristics of their subjects; hence, a concurrent decrease in body mass in this study is not known. Additionally, other studies used considerably larger samples than the present study allowing for greater statistical power. Gokal et al. (2007) implemented a 7-day yoga intervention to 258 participants with a variety of medical conditions, including Type 2 diabetes, and reported significant decreases in FBG (from 126.13 ± 46.85 to 120.72 ± 45.05 mg/dL, $p<0.001$) following the intervention. This modest change in FBG ($\Delta \approx 5.4$ mg/dL) (Gokal et al., 2007) was approximately one half
that observed for the present study ($\Delta \approx 10.8$ mg/dL) (Table 2) suggesting that greater statistical power would have elicited a significant FBG improvement.

Finally, perhaps frequency of yoga practice is of vital importance to achieve glycemic control improvements. Studies that reported a significant decrease for FBG, post-prandial glucose, or HbA$_{1c}$ involved daily yoga practice (Amita et al., 2009; Malhotra et al., 2005; Singh et al., 2004; Singh et al., 2001; Singh et al., 2008). Participants of this study were encouraged to practice daily but the actual intervention only involved structured yoga sessions three days per week. In addition, home practice was not recorded during this study, therefore, the extent of participants’ independent practice is not known. Rosenzweig et al. (2007) implemented an 8-week mindfulness-based stress reduction program for a similar sample of diabetes patients (mean ± SD age: 59.2 ± 2.57 years; body mass: 107.2 ± 21.81 kg) that involved 27 practice hours per week. At the end of the intervention there was only a trend toward improved glycemic control; however, at 1-month follow-up assessments significant decreases were found for HbA$_{1c}$, indicating detectable improvements after 12 weeks of continuous practice (Rosenzweig et al., 2007). In contrast, Skoro-Kondza, Tai, Gadelrab, Drincevic, and Greenhalgh (2009) implemented a twice a week, 3-month yoga intervention for a Type 2 diabetes population and found no significant changes in HbA$_{1c}$. However, the authors reported a 50% yoga class attendance, and participants declared not practicing at home on a regular basis. Lack of continuous attendance and low frequency of practice may explain the absence of glycemic control improvement in this particular study.
From these results and previous reports we can infer that for a yoga intervention to have significant effects on measures of glycemic control among obese diabetes patients, an intervention longer than 6 weeks and with more frequent practice is required. Most importantly, yoga practice still appears promising as an alternative method of glucose control for Type 2 diabetes mellitus.

_Diabetes regimen adherence and glycemic control_

The second purpose of this study was to investigate the psychological and physiological impact of yoga practice on glycemic status of Type 2 diabetes patients. The first hypothesis stated that yoga may act indirectly by improving measures of regimen adherence, and consequently improving glycemic control. In this study, SDSCA scores were significantly higher post-intervention compared to baseline indicating improved adherence to the diabetic regimen of participants following 6 weeks of yoga practice. Specifically, the SDSCA enquired about the frequency of behaviors that improve glycemic control such as following an adequate diet, increasing physical activity, taking all diabetes medication, and regularly monitoring blood glucose. Even when dietary analysis and the physical activity questionnaire revealed no change in fat intake or physical activity patterns beyond yoga practice, it is possible that through an improved adherence to prescribed medication and glucose monitoring participants were able to achieve the modest improvement observed in fasting glucose levels following the yoga intervention. Such adherence may be the result of decreased levels of stress and anxiety as indicated by post-intervention scores for psychological questionnaires and cortisol. Amador, Marquez, and Sabido (2007) found that absence of depression
and anxiety is a favorable factor in self-care behaviors, such as following proper pharmacological treatment and foot care, of Type 2 diabetes patients. These authors also found significantly higher FBG levels among patients that did not follow self-care behaviors compared to patients that practice self-care (Amador et al., 2007). Similarly, Hall, Rodin, Vallis, and Perkins (2009) found that higher levels of anxiety were significantly associated with decreased physical activity as a measure of self-care behavior in Type 2 diabetes, while Goetsch, Abel, and Pope (1994) found that during “high stress days” diabetes patients also reported significantly lower physical activity. Consequently, we can postulate that reducing symptoms of anxiety among diabetes patients improves adherence to self-care behaviors, resulting in an improved glycemic status.

**Anxiety, stress, and glycemic control**

In the present study, significant decreases in state anxiety were observed following the 6-week yoga intervention. Okada et al. (1995) reported that decreasing anxiety by an anxiolytic drug improved glycemic control of Type 2 diabetes patients and that decreased anxiety was significantly correlated with decreased HbA$_{1C}$. The anxiolytic drug was fludiazepam, which produces a relaxing effect by inhibition of the central nervous system, consequently inducing skeletal muscle relaxation and sedation, among other effects (Katzung, 2007). Perhaps, the effect of yoga practice was similar by relaxing skeletal muscle with yoga poses and calming the mind with meditation. The added benefits are that yoga does not pose any drug interaction or side effects produced by most pharmacological agents. In addition, there is no risk for physiological
dependence with long-term use that is common for sedative-hypnotic drugs, which produces increases in anxiety, insomnia, and central nervous system excitability (Katzung, 2007).

The improvement in anxiety scores following the yoga intervention is of special significance for Type 2 diabetes patients because reports indicate a higher prevalence of anxiety among diabetes patients compared to healthy individuals (Barker, Ford, Zhang, Strine, & Mokdad, 2008; Collins, Corcoran, & Perry, 2008; Okada et al., 1995). In addition, symptoms of depression and anxiety correlate with poor glycemic control (Mosaku, Kolawole, Mume, & Ikem, 2008) and decreased quality of life (Chyun et al., 2006). Therefore, it would be expected that decreasing anxiety symptoms of diabetes patients will improve glycemic control. In this study, a borderline significant relationship ($p=0.06$) was found between percent change in state anxiety and percent change in fasting blood glucose, indicating that as state anxiety decreased fasting glucose decreased as well. The exact mechanism of this relationship is still to be elucidated; however, as mentioned previously, decreases in anxiety may act as an indirect mechanism by improving feelings of well-being and hence improving diabetes regimen adherence. Zihl, Schaaf, & Zillmer (2010) pointed out that there is a need to clarify whether changes in mood associated with diabetes are a consequence of altered brain function or a result of the burden of coping with everyday demands.

In addition, although not significant, a moderate correlation was found between baseline scores of stress and insulin sensitivity ($r_s=-0.58$, $p=0.08$), and between baseline scores of anxiety and insulin sensitivity ($r_s=-0.60$, $p=0.07$), indicating a possible relationship between the psychological state of these diabetes patients and
glucose metabolism before the intervention. On the contrary, a poor correlation was observed between post-intervention scores of PSS and insulin sensitivity ($r_s = 0.27$, $p=0.46$), or between post-intervention scores of anxiety and insulin sensitivity ($r_s = 0.12$, $p=0.75$). Interestingly, even when no significance was found, the direction of the relationship changed following the intervention; the small positive correlation indicated that those with greater stress and anxiety scores had the greater insulin sensitivity scores. This may be due to the considerable variance for post-intervention insulin sensitivity scores between participants; while some participants experienced small improvements others experienced small decreases in QUICKI scores or no change at all. In contrast, the majority of participants experienced significant reductions in both stress and anxiety scores. The divergent pattern of psychological measures and insulin sensitivity scores following the yoga intervention may partially explain why the relationship between these variables was opposite to that anticipated. Moreover, a poor correlation may indicate that the modest glucose reduction achieved by the participants of this study following the intervention was a better reflection of decreased gluconeogenesis as a result of decreased cortisol produced by lower stress and anxiety.

As mentioned before, perhaps a body mass reduction in patients with high degrees of obesity is necessary to observe substantial changes in insulin sensitivity.

**Stress hormones and glycemic control**

The second hypothesis stated that yoga practice may act directly by decreasing measures of both psychological and physiological stress, and consequently glucose metabolism will be improved. In the present study, simultaneous significant decreases
in cortisol, perceived stress, and anxiety were observed following the 6-week yoga intervention.

As mentioned previously, cortisol secretion increases during periods of stress (Asterita, 1985). Furthermore, cortisol amplifies the glucose response to stress by maintaining the elevation in circulating glucose produced by catecholamines (Eigler et al., 1979). Past research also has demonstrated that the availability of glucose amplifies the cortisol response to psychological stress (Gonzalez-Bono, Rohleder, Hellhammer, Salvador, & Kirschbaum, 2002). A significantly greater cortisol response following a stress test was observed for subjects that ingested glucose compared to control groups ingesting fat, protein, or water, indicating that the specific availability of glucose produces elevations of cortisol during periods of stress (Gonzalez-Bono et al., 2002). This may be of special significance to diabetes patients, who may have an even greater cortisol response to psychological stress because of chronic hyperglycemia.

Research has shown that acute anxiety produces elevations in cortisol, along with significant elevations in plasma glucose (Armario, Marti, Molina, de Pablo, & Valdes, 1996). In addition, it has been observed that individuals with higher trait anxiety scores have sustained elevations of cortisol throughout the day compared to low-anxiety individuals that show a normal cortisol pattern that peaked in the morning and decreased throughout the day (Taylor et al., 2008). It is possible that the significant decreases in stress and anxiety experienced by the participants of this study lead to the significant reduction in cortisol. Decreased anxiety has been associated with reduced cortisol secretion following a pharmacological intervention (Lenze et al., 2011). Additionally, a yoga intervention implemented for cancer patients with similar frequency
and length of the present study (3 days per week for 6 weeks) found significant decreases in perceived stress, anxiety, and cortisol (Vadiraja et al., 2009). Such findings are similar to the responses experienced by the diabetes patients of this investigation.

In this study, no relationship was found between post-intervention values of cortisol and anxiety ($p=0.55$). A possible explanation is the variable under investigation. This study examined salivary cortisol content at midnight, which has been shown previously to be a marker of excess circulating cortisol during the day (Viardot et al., 2005). Nonetheless, cortisol at midnight may not be the ideal method to investigate anxiety effects on the HPA axis regulation. Vreeburg et al. (2010) found a significant ($p=0.002$) correlation between anxiety and higher awakening cortisol responses, while this association was not observed for individuals without anxiety disorders; in contrast, they found no correlation between anxiety status and evening cortisol levels. The difference between midnight cortisol and the awakening cortisol response may lie on the secretion pattern rather than 24-hour cortisol content. In the study by Taylor et al. (2008) differences were found in the cortisol diurnal pattern between high-anxiety and low-anxiety subjects; however, 24-hour cortisol was similar between the two groups. This may suggest that it is not total cortisol content that may be affected by anxiety, but the cortisol secretion pattern followed throughout the day.

Therefore, it is suggested that future investigations explore the association of anxiety and cortisol of diabetes patients utilizing different collection methods of this hormone.
The significant cortisol reduction following the 6-week yoga intervention may have directly improved glucose metabolism of diabetes patients, which may explain the modest improvement in FBG. Partial correlation analysis showed a significant relationship between post-intervention cortisol and post-intervention FBG values ($r^2=0.77$, $p=0.02$) when controlling for baseline FBG values. In addition, there was a correlation between post-intervention cortisol and HbA$_{1c}$ values that approached significance ($r^2=0.62$, $p=0.08$). These results may demonstrate the association between positive changes in cortisol and glycemic control improvement. Surwit & Feinglos (1983, 1984) reported significant improvements in glucose tolerance in conjunction with significant decreases in cortisol among non-insulin dependent diabetes patients.

Cortisol is known to counteract the actions of insulin by increasing insulin resistance in peripheral tissues, and increasing gluconeogenesis from the liver (Goodman, 2009). Hence, decreases in cortisol are expected to improve insulin sensitivity and decrease gluconeogenesis. Although not statistically significant ($r_s=0.58$, $p=0.08$), this study showed a possible relationship between percent change in cortisol and percent change in QUICKI, indicating that those who had greater decreases in cortisol also show improvements in insulin sensitivity. In addition, the factor of decreased glucose output from the liver may have played a role in the reduction of circulating glucose concentration; however, it was not assessed during this study.

Another possible explanation for the improvement of glycemic status of diabetes patients is the decrease of circulating catecholamines as a result of decrease stress and anxiety. In this study, measures of perceived stress and state anxiety significantly decreased following the yoga intervention. Binding of epinephrine and norepinephrine to
α-adrenergic receptors of the pancreatic beta cells will inhibit insulin secretion (Powders & Howley, 2007). Research has shown that non-insulin-dependent diabetes patients are more susceptible to the effects of circulating catecholamines due to α-adrenergic receptor hypersensitivity (Robertson, Halter, & Porter, 1976) and consequently experience significantly higher plasma glucose responses to catecholamines compared to matched healthy controls (Bruce, Chisholm, Storlien, Kraegen, & Smythe, 1992). Furthermore, several investigations have demonstrated that the insulin response to glucose is increased for diabetes patients following an α-adrenergic blocker drug administration (Broadstone, Pfeifer, Bajaj, Stagner, & Samols, 1987; Kashiwagi et al., 1986; Robertson et al., 1976). Therefore, if a lower α-adrenergic stimulation improves glucose-mediated insulin secretion, then a higher α-adrenergic stimulation will worsen hyperglycemia by blunting the insulin response to glucose. Consequently, it can be postulated that reduced stress attenuates adrenergic stimulation by catecholamines thereby improving glucose-induced insulin secretion of diabetes patients. In this study, a trend towards increase insulin secretion was observed following the yoga intervention (97.85 ± 73.55 pmol/L vs. 112.86 ± 80.63 pmol/L). Nonetheless, a direct relationship cannot be established since catecholamines were not assessed. Future studies should focus on establishing a direct relationship between catecholamines changes and insulin secretion regulation, and hence, clarify the impact of the psychological state of the diabetes patient on glycemic control. As Surwit & Feinglos (1988) suggested, regardless of whether changes in sympathetic nervous system activity are related to the pathophysiology of Type 2 diabetes or are secondary to hyperglycemia, interventions
that decrease sympathetic nervous activity should be helpful in the regulation of hyperglycemia.

Additional benefits from yoga practice

In addition, significant increases in most AROM movements of the hip and trunk, and increases in balance were observed following the yoga intervention, which corroborate findings from previous studies (Chen & Tseng, 2008; Cowen & Adams, 2005; Schure, Christopher, & Christopher, 2008; Tekur, Singphow, Nagendra, & Raghuram, 2008). Increases in ROM seem to be of particular importance for these patients since some authors suggest that adults with diabetes have limited joint mobility, even without neuropathy (Shinabarger, 1987; Schulte, Roberts, Zimmerman, Ketler, & Simon, 1993; Turner, Helliwell, Burton, & Woodburn, 2007). Improved flexibility and balance of diabetes patients may contribute to the ability to perform everyday tasks independently, and hence maintain an active lifestyle.

Finally, extraneous variables did not appear to influence the results of this study. No change was reported in physical activity patterns (p=0.72) or dietary fat consumption (p=0.26). There was a statistically significant decrease in protein intake following the intervention (p=0.017), which may explain the decrease in total calories consumed (p=0.017). The change in dietary pattern may be due to the period of the year in which this study was conducted. During lent, individuals from certain religious backgrounds tend to decrease meat consumption and compensate with legumes intake. The reduction in protein intake may also explain the trend towards decreased lean mass.
(\(p=0.13\)) of participants following the intervention, especially when increased protein turnover is known to exist with diabetes. Nonetheless, it is very unlikely that reductions in protein consumption may have resulted in improved glycemic control. On the contrary, some authors have suggested that adequate protein intake is needed for glucose control and to correct whole-body protein metabolism (Franz, 2000).

The major limitation of this study was the lack of adequate statistical power that most likely prevented significant intervention effects to be observed for the glycemic control variables under analysis. Comparing the results of this investigation with previous studies, it is probable that significant changes would have been observed in the primary variables, such as FBG and HbA\(_1c\), if a larger sample size would have been available. In addition, although improbable, a lack of a control group may prevent to establish whether changes observed following yoga practice were only caused by the intervention or coincidental. Another important limitation was the internal validity of the physiological marker of chronic stress. Salivary cortisol at midnight may not be the most accurate reflection of prolonged stress, especially because cortisol content is expected to reflect episodes of stress experienced during the day of collection and not the overall stress load accumulated during a week or a month. Research in the future may elucidate the ideal method of assessing physiological stress in the human body. Also, the subjective nature of survey responses to psychological questionnaires may pose a limitation, making it difficult to relate psychological stress with physiological stress responses. Lastly, unreported changes in diet, physical activity, or medication may have a strong influence on all the glycemic control variables under investigation.
CONCLUSIONS

The findings from this study indicate that a yoga practice intervention greater than 6 weeks in duration and more frequent than 3 sessions per week may be necessary to induce significant improvements in glycemic control parameters, especially in individuals with obesity and abdominal adiposity. The potential benefits of yoga practice on glycemic control of diabetes patients may be the result of two different processes. First, yoga may improve glycemic control by decreasing stress and anxiety, and consequently enhancing adherence to a diabetes-care regimen. Second, yoga may improve glycemic status by decreasing stress and anxiety, and subsequently decreasing stress hormones such as cortisol. Decreases in cortisol are thought to decrease hyperglycemia by reducing glucose output from the liver and improving insulin sensitivity. Future investigations should focus on exploring other measures of physiological stress like catecholamine excretion and diurnal variations in cortisol. Even when only modest improvements were observed in the present investigation, yoga practice still seems promising as an alternative therapy for glycemic control of the diabetes patient. Yoga practice can also help to increase and maintain proper mobility and balance, positively impacting quality of life.
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LITERATURE REVIEW

YOGA AND TYPE 2 DIABETES
Yoga is an ancient spiritual practice that most likely originated in India approximately 4000 years ago. It was originally developed as the first step in a spiritual path that is to be practiced throughout a lifespan, and it is thought to help the practitioner to ultimately achieve a higher degree of consciousness, but most of all, to attain the union with the “universal spirit.” The practice of yoga has evolved throughout the centuries. Regardless of its spiritual significance, in the present day there is a growing interest in the physical and physiological benefits that can be derived from this ancient practice. Although there are numerous styles and schools of yoga, Hatha Yoga is the form most commonly practiced throughout the world. It is generally comprised of a series of poses called asanas, breathing exercises called pranayama, and meditation.

The benefits that practitioners and teachers attribute to the practice of hatha yoga are numerous, including positive effects on chronic diseases such as diabetes mellitus. Early investigations of the effects of yoga on physical, physiological, and psychological parameters were conducted on different populations in India; the main objective was to scientifically evaluate the hypothesis of old writings which state that yoga improves physical and mental health (Joseph et al., 1981; Udupa & Singh, 1972; Udupa, Singh, & Yadav, 1973; Udupa, Singh, & Settiwar, 1975a, 1975b, 1975c; Udupa, Singh, Dwivedi, Pandey, & Rai, 1975). Later investigations focused primarily on changes in blood chemistry, including blood glucose, induced by the practice of yoga in healthy subjects (Bagga, Gandhi, & Bagga, 1981; Naruka, Mathur, & Mathur, 1986). Recent studies have focused specifically on non-insulin dependent diabetes patients practicing either yoga alone (Amita, Prabhakar, Manoj, Harminder, & Pavan, 2009;
Gokall, Shillito, & Maharaj, 2007; Gordon et al., 2008; Malhotra, Singh, Tandon, & Sharma, 2005; Mercuri, Olivera, Souto, & Guidi, 2003; Singh et al., 2001; Singh, Malhotra, Singh, Madhu, & Tandon, 2004) or combined with another non-medical intervention (Agrawal et al., 2003; Agte & Tarwadi, 2004; Jain, Uppal, Bhtanagar, & Talukdar, 1993). The majority of research reported beneficial effects in glycemic control including reductions in fasting blood glucose (FBG) (Amita et al., 2009; Agrawal et al., 2003; Agte & Tarwadi, 2004; Gokal et al., 2007; Gordon et al., 2008; Jain et al., 1993; Malhotra et al., 2005; Singh et al., 2001; Singh, et al., 2004), post-prandial blood glucose (PPG) (Amita et al., 2009; Agte & Tarwadi, 2004; Malhotra et al., 2005; Singh et al., 2001; Singh et al., 2004), and even glycated hemoglobin (HbA$_1c$) (Agrawal et al., 2003; Singh et al., 2001; Singh et al., 2004). In contrast, other authors reported no change in HbA$_1c$ after yoga practice (Agte & Tarwadi, 2004; Mercuri et al., 2003).

**Research on healthy populations**

Early research investigating the effects of yoga on the human body was conducted on young, healthy male subjects and included a variety of physiological parameters. The first studies on the impact of asanas and pranayama found a decrease in FBG and an increase in serum proteins, among other benefits, after 6 months of practice; however, none of them reported statistical significance (Udupa & Singh, 1972; Udupa et al., 1975a, 1975b, 1975c).

Additionally, some studies focused on endocrine changes after yoga practice; nonetheless, the results were contradictory. Plasma epinephrine and norepinephrine decreased in the first study by Udupa et al. (1975a). In the second study,
catecholamines only decreased in two of the three groups of subjects practicing different sets of asanas (Udupa et al., 1975b). Similarly, Udupa, Singh, Dwivedi, et al. (1975) found significant decreases in catecholamines in Indian practitioners but not on Westerners. Moreover, only Udupa, Singh, Dwivedi, et al. (1975) attempted to explain observed changes in epinephrine and norepinephrine by stating they were the result of a heightened neurophysiological state after meditation; nevertheless, only one group in their study showed significant increases in these hormones and no explanation was provided for the significant decrease in catecholamines observed for the other group. In addition, an increase in catecholamines is seen in the literature as part of the usual stress response, and not as a positive outcome of improved cognitive function.

Similarly, cortisol exhibited either a decrease (Udupa et al., 1975a; Udupa, Singh, Dwivedi, et al., 1975) or an increase (Udupa et al., 1975a, 1975b, 1975c) depending on the aspect of yoga practiced or even the type of asanas utilized. For instance, Udupa et al. (1975a) examined three different sets of asanas and after 6 months of practice found different effects in plasma cortisol levels between subjects practicing the different sets. Udupa, Singh, Dwivedi, et al. (1975) studied only the effect of meditation, which is an important aspect of traditional yoga, and observed that after only 10 days of intense practice there was a decline of cortisol levels in participants; nonetheless, the change did not reach statistical significance. Authors concluded that asana and pranayama practice improved adrenocortical function in subjects (Udupa et al., 1975b, 1975c), which can provide stress competence in practitioners (Udupa et al., 1975c); while meditation produced a less stressful state by decreasing metabolic activities (Udupa, Singh, Dwivedi, et al., 1975). However, while it is certain that optimal
secretion of cortisol is critical for the survival of mammalian species, subjects in the previous studies were not reported to have deficiencies in cortisol secretion. Consequently, it is inaccurate to state that yoga practice improved adrenocortical function or stress competence if the cortical function was not reported to be compromised. In summary, explanations of findings appear contradictory. Except for the study by Udupa, Singh, Dwevedi, et al. (1975), none of the studies demonstrated statistical significance. Moreover, no study specified the time of the day for blood collection, which can greatly influence measured cortisol levels because of the inherent diurnal rhythm of this hormone (Cohen, Kessler, & Gordon, 1995). Similarly, there was no reported control for extraneous variables that can highly influence catecholamine secretion in just a matter of seconds, such as psychological stress induced by venipuncture (Cohen, Kessler, & Gordon, 1995).

Subsequent studies in the 1980’s also focused on healthy, young subjects; however, larger populations were observed, female participants were included, duration of the intervention shorten, and the experimental design of the studies varied. Joseph et al. (1981) performed a detailed study on male soldiers who practiced asanas, pranayama, and meditation, and found that FBG significantly decreased and total proteins significantly increased after 4 months of practice. Likewise, Naruka et al. (1986) found significant decreases in FBG for two groups of male subjects practicing a different set of pranayama everyday for 12 weeks. In contrast, a study comparing female yogic practitioners with non-practitioners found no differences in blood glucose levels between the two groups; no statistical significance was reported, nor duration of practice in yogis
(Bagga et al., 1981). No explanation on glycemic changes was provided for these studies (Bagga et al., 1981; Joseph et al., 1981; Naruka et al., 1986).

Early research in India initiated the first scientific endeavor to evaluate the effect of yoga on blood glucose and other physiological parameters. Nonetheless, some of these first scientific studies did not report statistical significance (Bagga et al., 1981; Udupa & Singh, 1972; Udupa et al., 1975a, 1975b, 1975c), did not report control for extraneous variables such as diet or other types of physical activity (Bagga et al., 1981; Naruka et al., 1986; Udupa & Singh, 1972; Udupa et al., 1973; Udupa et al., 1975a, 1975b, 1975c), or did not describe frequency of the intervention (Udupa & Singh, 1972; Udupa et al., 1975a, 1975b). In addition, only two possessed more than 20 subjects (Bagga et al., 1981; Naruka et al., 1986), and only one possessed a control group (Naruka et al., 1986). Furthermore, none of the authors explained a possible mechanism for the observed changes in glycemic status of these healthy subjects. Udupa et al. (1975a) only mentioned that yoga may produce rehabilitative effects on different vital organs by means of improved circulation, thereby improving their function; however, such speculation had no scientific base.

Research on non-insulin dependent diabetes mellitus (NIDDM) patients

Following initial yoga research suggesting improved glycemic status among healthy subjects, subsequent studies focused on the effect of yoga in non-insulin dependent diabetes mellitus (NIDDM) patients. These new studies used mainly middle-age populations, both genders, and greater number of participants. Also, novel parameters were under investigation and a variety of practice modalities were used
including additional components to traditional yoga. The result was new research with very diverse interventions and investigation approaches.

The first study on NIDDM patients was a 40-day intervention that combined asana and pranayama practice with a vegetarian diet and shatkriyas (Jain et al., 1993). Oral glucose tolerance tests (OGTT’s) and FBG were assessed pre- and post-intervention. The response to yoga therapy in 149 participants was categorized based on responses to the second OGTT. The poor response group (n=45) had peak values exceeding 300 mg/dL of glucose response and no reduction in hypoglycemic drug requirements following the intervention. The fair response group (n=28) had peak values not exceeding 210 mg/dL and hypoglycemic drugs requirements decreased after the intervention. The good response group (n=76) had peak values not exceeding 210 mg/dL and hypoglycemic drugs no longer required for maintenance of normoglycemia. Before the beginning of the study 60.4% of participants were taking oral hypoglycemic drugs; after yoga therapy this number was reduced to 38.3%. In addition, significant reductions in FBG and glucose intolerance were observed for all three categories following the intervention, even for those subjects classified in the poor response group (Jain et al., 1993). Although it is difficult to determine the element of this particular yoga therapy that caused the change in glycemic status of these subjects, this is the only study that has shown a yoga effect on all categories of diabetes, from severe to well-controlled. No explanation of the findings was provided by the authors.

Studies including supplementary components to yoga practice appeared decades later with interventions equal to or longer than 3 months, and started employing relaxation techniques. Agrawal et al. (2003) incorporated a diabetic diet,
“health rejuvenating exercises”, abdominal exercises, and relaxation exercises; while Agte & Tarwadi (2004) added nutrition counseling and stress-management techniques to yoga practice. Agrawal et al. (2003) analyzed the influence of yogic lifestyle on glycemic control and quality of life in diabetes patients. Hence, in addition to physiological parameters they included psychological measures such as satisfaction score, impact score, and worry score. In contrast, Agte & Tarwadi (2004) focused on lipid profile and oxidative stress in addition to measures of glycemic control. For both studies, participants were Type 2 diabetes patients treated with conventional prescribed medication and were further divided into experimental and control groups. At the end of both interventions, decreases in FBG and HbA₁c were observed in the experimental groups; however, the change in HbA₁c was not statistically significant in the Agte & Tarwadi (2004) study. Additionally, Agrawal et al. (2003) reported a significant decrease in oral hypoglycemic drugs and insulin use following the intervention, and Agte & Tarwadi (2004) reported a significant decrease in PPG. No changes were reported for the control groups. Agrawal et al. (2003) also reported improvements in all psychological scores measured, and although not mentioned by the authors, this may be the first study that indicates some relationship between glycemic status improvement and stress-release with yoga practice. Finally, neither study provided an explanation of their findings.

Studies focusing only on asana and pranayama practice in NIDDM patients include those by Malhotra et al. (2005), Singh et al. (2001), and Singh et al. (2004). Participants of these three studies were between the ages of 30-60 years, Type 2 diabetes patients, free of diabetes complications, and on recommended diet and oral
hypoglycemic drugs. For these studies, yoga practice was implemented for 40 days, 30-40 minutes per day, with a qualified instructor. However, variables investigated between these studies varied. At the completion of the interventions, all measured glycemic control parameters significantly decreased: FBG and PPG (Malhotra et al., 2005; Singh et al., 2001; Singh et al., 2004), and HbA1c (Singh et al., 2001; Singh et al., 2004). In addition, Malhotra et al. (2005) observed that serum insulin levels tended to decrease in subjects with a body mass index (BMI) greater than 25 kg/m², but tended to increase in subjects with a BMI lower than 25 kg/m²; nevertheless, changes in insulin were not significant. Singh et al. (2001) and Singh et al. (2004) provided no explanation for their findings, or specification of control for extraneous variables such as additional physical activity. Conversely, Malhotra et al. (2005), by observing significant decreases in waist-to-hip ratio in 70% of their subjects, concluded that yoga asanas aid glucose utilization and fat redistribution of NIDDM patients by shifting central obesity to peripheral obesity. They also suggested that yoga asanas may reduce insulin resistance, particularly for patients with high initial insulin levels such as those observed in overweight individuals (Malhotra et al., 2005).

The only known study comparing the effects of yoga practice to traditional exercise in Type 2 diabetes patients was that of Gordon et al. (2008). In their 24-week study, one group followed traditional physical therapy exercise, another group practiced yoga, while a control group did not perform any kind of exercise. A significant reduction in FBG was found in the traditional exercise and yoga groups at 3 and 6 months; although, there was no significant difference in FBG concentration between these groups. No changes were reported in the control group. Additionally, the percentage of
insulin binding receptor significantly increased in both experimental groups compared to the control group after 6 months. No other significant change was observed for the rest of the parameters measured, which included cortisol. No specification about the type of yoga intervention implemented was provided (asana, pranayama, frequency, duration of practice, etc.).

Moreover, Mercuri et al. (2003) is the only known study reporting no significant changes in any of the glycemic parameters measured. Participants were all females (mean ± SD age: 61 ± 11 years) with previous yoga experience. After three months of yoga practice, no significant changes in HbA$_{1c}$ or any of the parameters measured were observed. This was the only study that has reported previous yoga experience in subjects, which may explain why no significant changes were observed; patients may have been accustomed to yoga practice to the extent that no further results could be obtained. And most importantly, the baseline levels for HbA$_{1c}$ (7.4% ± 2.0) were already around the target value recommended by the American Diabetes Association (ADA) of <7%.

Finally, only one known study (Manjunatha et al., 2005) attempted to clarify the mechanism by which yogic postures may aid in the treatment of diabetes mellitus by analyzing serum insulin levels immediately after yoga practice. The authors investigated the hypothesis that asanas involving twisting or folding of the abdomen release insulin by compressing the pancreas, and hence help in diabetes. Nevertheless, the study only included healthy subjects and no diabetes patients. Male and female volunteers randomly practiced four different sets of asanas throughout a 4-week period. On days 4 and 5 of each week, glucose and insulin levels were measured before the asanas and
within 10 minutes after finishing the asanas. In addition, a standard 75 g OGTT was conducted on all subjects before the beginning of the study, each week right after the corresponding set of asanas, and at the end of the study. No significant change in FBG levels was found immediately after any asana set compared to pre-practice values. Furthermore, contrary to the hypothesis, serum insulin levels significantly decreased following yogic postures compared to before practice for all asana sets. No significant changes were observed between the pre- and post- values of the OGTT, except for the 30 minutes after glucose ingestion value that decreased significantly following the intervention. The authors suggested that the decrease in serum insulin levels immediately after asanas may be the result of increased sensitivity of the pancreas to glucose changes, which in turn produces a decrease in insulin release when there is accelerated glucose utilization because of yoga practice (Manjunatha et al., 2005). This study may have demonstrated that the practice of yoga asanas has the same effect on insulin dynamics as other types of exercise where insulin levels decrease (Powers & Howley, 2007); nevertheless, it may highlight the fact that insulin starts to significantly decrease with very low exercise intensities such as yoga, an activity previously catalogued as eliciting only 14.5% of maximal oxygen consumption reserve (VO$_2$R) (Clay, Lloyd, Walker, Sharp, & Pankey, 2005).

*Research on populations with other medical conditions*

Gokal et al. (2007) conducted a pilot study evaluating the impact of yoga, pranayama, and meditation on subjects suffering from diabetes, obesity, hypertension, and hyperlipidemia. Individuals attended a yoga camp for a period of 7 days and 428 of
the 8000 people attending were randomly selected as a representative cohort group. Surprisingly, at the end of the week significant reductions in weight, blood glucose, cholesterol, and blood pressure were reported for the selected group. Authors acknowledge that such findings may be questionable since confounding factors were not controlled; hence, they recommended a larger randomized controlled trial during a longer intervention to confirm the positive changes observed during their pilot study. In addition, there was no specification on the protocol used to test blood glucose. Finally, significant changes could be due to the high power allowing subtle variability in the parameters measured to achieve statistical significance.

Research during the last decades has shown potential benefits of yoga practice on glycemic control in Type 2 diabetes patients. However, similar to earlier reports, data are still inconclusive because of the lack of control for confounding variables such as additional physical activity (Agrawal et al., 2003; Malhotra et al., 2005; Singh et al., 2004), failure to monitor diet or medication (Malhotra et al., 2005), poor description of the intervention (Gordon et al., 2008), or inadequate description of the sample population (Agrawal et al., 2003). Only two studies proposed potential mechanisms to explain the beneficial impact of yoga in Type 2 diabetes. Manjunatha et al. (2005) proposed that yogic postures increase the rate of glucose utilization after observing a decrease in insulin levels immediately after practice. Malhotra et al. (2005) suggested that yoga may increase glucose utilization by redistributing fat in diabetes patients. Furthermore, none of the studies reported control of factors that could affect the measurement of FBG, such as female hormones or ingestion of certain drugs, or factors
that could affect the assessment of Hb_{A1c} like red blood cell abnormalities or excessive doses of vitamin C and E (Unger, 2007).

There have been several scientific studies on the physiological effects of yoga practice, and specifically, the impact of yoga on diabetes mellitus. Several have reported improvements in both, short-term and long-term glycemic control in patients after yoga practice (Agrawal et al., 2003; Singh et al., 2001; Singh et al., 2004). However, many authors suggested that additional research is needed to clarify the potential benefit of yoga on glycemic control (Agte & Tarwadi, 2004; Gokal et al., 2007; Manjunatha et al., 2005; Singh et al., 2004). And most importantly, there is a need to explain the possible mechanism underlying the improvement that yoga can confer to Type 2 diabetes mellitus patients.

A POSSIBLE MECHANISM OF ACTION FOR YOGA PRACTICE TO IMPROVE GLYCEMIC STATUS OF TYPE 2 DIABETES PATIENTS

A substantial volume of research has demonstrated that exercise benefits diabetes by increasing insulin sensitivity (Borghouts & Keizer, 2000; Braun, Zimmermann, & Kretchmer, 1995; Devlin, 1992; Devlin, Hirshman, Horton, & Horton, 1987; Ibanez et al., 2005; Lampman & Schteingart, 1991) and decreasing several risks for cardiovascular disease that are inherently high in patients with this condition (ADA, 2011; Schneider & Ruderman, 1990; Steppel & Horton, 2004). Nonetheless, these benefits are only derived from exercising at a certain mode, intensity, or frequency. Brown & Thompson (1988) suggested that the major muscles of the body should be exercised in a continuous, repetitive motion such as in walking, running, or biking to condition the cardiovascular system and decrease the need for insulin. Nagi (2005)
pointed out, the greater the exercising muscles mass, the greater the glucose uptake and subsequent enhanced insulin sensitivity. Furthermore, it appears that benefits, like improved lipid profile, are only derived when a person exercises at moderate to high intensities for most days of the week (Steppel & Horton, 2004). So far, there is no consensus about the frequency or intensity of the exercise needed to improve insulin sensitivity of Type 2 diabetes patients (Nagi, 2005). However, the general recommendation for patients with type 2 diabetes mellitus is 20-60 minutes of cardiorespiratory exercise at an intensity of 50-80% of $\text{VO}_2\text{R}$, 3-4 days a week (ACSM, 2010). The rationale are the previous benefits mentioned, in addition to weight control, increased physical work capacity for everyday activities, improved posture, and an increased sense of well-being.

As already reviewed, numerous research studies in the last three decades have demonstrated the benefits of yoga practice on Type 2 diabetes. Nevertheless, such studies have failed to demonstrate a mechanism by which yoga aids in glycemic control. Traditional yoga, such as hatha, consists primarily of isometric muscle contractions and passive stretching during poses of 30 seconds or longer, in addition to the mental element of focusing the mind on the movements of the body and breathing control. Therefore, by the relaxing nature of yoga practice the cardiovascular system is not stressed sufficiently to derive the cardiovascular benefits obtained from regular aerobic exercise. Hence, yoga does not provide improvements in maximal aerobic capacity, or weight control by means of caloric expenditure (Clay et al., 2005; DiCarlo, Sparling, Hinson, Snow, Rosskopf, 1995). The question then remains, how can yoga possibly
improve glycemic status in Type 2 diabetes? The answer may lie on the effect that yoga generates by alleviating psychological stress of the diabetes patient.

*Psychological stress and Type 2 diabetes*

Acute psychological stress is known to affect the body by activating the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, consequently increasing heart rate, blood pressure, and concentrations of the catecholamines (Asterita, 1985). There are theories that state that chronic activation of the HPA axis may lead to negative health consequences, including insulin resistance (Bjorntorp, Holm, & Rosmond, 1999). Furthermore, some scientists have proposed that chronic psychological stress, through the major stress hormones such as norepinephrine and cortisol, produces a chronic inflammatory response in visceral fat and blood vessels. This process in turn, may generate insulin resistance, which if prolonged, may increase the likelihood of developing cardiovascular disease and Type 2 diabetes (Black, 2006).

Early research demonstrated glucose fluctuations due to stressful events in diabetes patients. In particular, Hinkle & Wolf (1952) observed that blood glucose levels tended to increase when patients reported feeling “great fear or overwhelming anger,” represented as a common response, while remembering real past stressful situations during interviews. However, they did not report any statistical significance. Similarly, Grant, Kyle, Teichman, & Mendels (1974) found a significant positive correlation between negative events and worsening of physical symptoms, suggesting a possible relationship between negative life events and an aggravated diabetic state in a group of
patients answering questionnaires about specific positive and negative life situations during medical examinations.

Acute stress and glycemic status

Several recent studies have demonstrated the adverse effect of acute psychological stress on glycemic status of Type 2 diabetes patients. In such studies, significant elevations of blood glucose were observed in patients after the implementation of acute stressors such as mental arithmetic tasks (Goetsch, Wiebe, Veltum, & VanDorsten, 1990), threat of shock (Goetsch, VanDorsten, Pbert, Ullrich, & Yeater, 1993), and a video showing adverse symptoms of severe diabetes (Wang, Xue, & Yao, 2000). One study in particular demonstrated significant glucose elevations after psychological stress in NIDDM patients, but no change in healthy subjects of similar age, weight, and gender, even when both groups showed usual physiological stress responses such as elevated heart rate and systolic blood pressure, in addition to increases in subjective stress ratings (Goetsch et al., 1993). Nevertheless, others have reported no significant increase in blood glucose levels following an arithmetic task in diabetes patients (Naliboff, Cohen, & Sowers, 1985). The differences between studies may be due to lack of statistical power to demonstrate a significant change; Naliboff et al. (1985) only used 8 subjects in their intervention group, while Goestch et al. (1993) and Wang et al. (2000) used more than 20 participants in their experimental groups. Also, variation in the glucose response may be attributed to the different methods for inducing psychological stress among studies; Naliboff et al. (1985) used a mental arithmetic test, while Goestch et al. (1993) utilized threat of shock and Wang et al.
(2000) a video demonstrating adverse symptoms of severe diabetes. It is possible that the mental arithmetic test did not produce sufficient psychological stress to induce observable elevations in blood glucose levels of diabetes patients.

**Daily stress and glycemic status**

The effect of daily life stress on glycemic status of Type 2 diabetes patients has also been investigated. In the study by Goetsch et al. (1990), patients completed a subjective stress scale prior to each glucose measurement at four different times per day for 12 days. A significant positive correlation was found between subjective stress ratings and blood glucose values; that is, as stress increased, glucose levels tended to increase as well (Goetsch et al., 1990). A follow-up of the previous study examined the effects of daily life stress and coping on blood glucose in NIDDM patients while controlling the variables of diet and physical activity (Goetsch, Abel, & Pope, 1994). Again, blood glucose levels were found to be significantly higher for “high stress days” compared to “low stress days.” However, levels of physical activity were found to be significantly lower for “high stress days” suggesting that the lack of activity may have contributed to the increased glucose levels (Goestch et al., 1994). Although, these studies did not possess non-diabetic control groups to compare the differences in stress responses, the findings suggest that daily psychological stress may have an indirect effect on glycemic control that is mediated by decreases in physical activity levels.
Chronic stress, coping, and glycemic control

Diverse observational studies have established a possible relationship between chronic stress, coping, and glycemic control (Lazcano & Salazar, 2007; Peyrot, McMurry, & Kruger, 1999; Toshihiro et al., 2008). Peyrot et al. (1999) investigated the relationship of psychological stress, coping styles, socio-demographic and biologic factors, and regimen adherence with metabolic control in Type 1 and Type 2 diabetes mellitus treated with daily insulin injections. Metabolic control was determined by HbA$_{1c}$ testing. Through statistical analysis, it was found that for Type 1 diabetes patients, negative measures of the “self-controlled” coping style and positive measures of the “emotional arousability” coping style, along with stress were significantly related to poor glycemic control; however, the significance was diminished when regimen adherence was controlled. Conversely, in Type 2 diabetes patients it was found that BMI and negative measures of the “self-controlled” coping style were significantly related to poor glycemic control even when controlled by treatment adherence. Moreover, for Type 1 diabetes marital status, education, duration of disease, a less negative “self-control” coping style, a less positive “emotional arousability” coping style, and regimen adherence were associated with stable glycemic control. In contrast, for Type 2 diabetes, only a less negative “self-control” coping style was associated with stable glycemic control. Authors stated that their findings may reveal a possible psycho-physiologic mechanism with the observed relationship between a coping style and glycemic control in Type 2 diabetes independent of regimen adherence (Peyrot et al., 1999). Nonetheless, the study did not analyze levels of physical activity in participants,
which may impact the relationship between glycemic control and stress (Goestch et al., 1994).

Similarly, Lazcano & Salazar (2007) evaluated the influences of perceived stress, coping strategies, and physiological and psychosocial adaptation in patients with Type 2 diabetes. They found, by means of Spearman correlation, an inverse relationship between perceived stress and coping strategies and psychosocial adaptation; that is, the more perceived stress, the less coping strategies and psychosocial adaptation. However, a linear regression model indicated no significant relationship between perceived stress and physiological measurements including HbA$_{1c}$, cholesterol, and triglycerides (Lazcano & Salazar, 2007).

Toshihiro et al (2008) examined possible risk factors for diabetes, including psychosocial and psychological factors, in Japanese workers with impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT). In addition to FBG and an OGTT, several anthropometric measurements, biochemical analyses, and questionnaires were completed at the beginning of the study, and then once a year for the next five years to all subjects. Impaired fasting glucose was defined as a FBG level of 6.1-6.9 mmol/l and a 2-hr plasma glucose level < 7.8 mmol/l during an OGTT. Impaired glucose tolerance was defined as a FBG level < 7.0 mmol/l and a 2-hr plasma glucose level of 7.8-11.1 mmol/l. Type 2 diabetes was defined as a FBG ≥ 7.0 mmol/l and a 2-hr plasma glucose level > 11.1 mmol/l. At the end of the study, statistical analyses were conducted to determine independent risk factors for progression to diabetes mellitus (DM) from IFG and/or IGT. Authors found that night duty, high FBG levels, stress in daily life, and having an administrative position were significant
independent risk factors for the development of DM from IFG and/or IGT. Conversely, lower FBG levels, having a white-collar job, and being a non-smoker were significant independent factors for the recovery of normal glucose tolerance from IFG and/or IGT. It was concluded that in addition to abnormal glucose levels, social and psychological factors also impact the progression to Type 2 DM or recovery to normal glucose levels in Japanese workers (Toshihiro et al., 2008).

Despite differences in methodologies, the majority of research indicates a possible relation between psychological stress and sudden increases in blood glucose levels (Hinkle & Wolf, 1952; Goetsch et al., 1990; Goetsch et al., 1994; Grant et al., 1974; Wang et al., 2000). In particular, the work by Goestch et al. (1993) demonstrated an exacerbated glucose response to laboratory stress in diabetes patients compared to healthy subjects. Research of daily-life stress also indicated a likely relationship between high perceptions of subjective stress and elevations in blood glucose levels (Goetsch et al., 1990; Goetsch et al., 1994).

Furthermore, longitudinal studies have shown that the less coping strategies, the poorer long-term glycemic control as measured by HbA1c, in individuals suffering from diabetes (Lazcano & Salazar 2007, Peyrot et al., 1999). Finally, Toshihiro et al. (2008) suggested that, in addition to physiological markers, psychological factors may be implicated in the progression of glucose intolerance to Type 2 diabetes mellitus. Therefore, despite the limitations of previous research, such as control for additional factors that may affect blood glucose levels, it is apparent that constant stressful situations along with social issues directly or indirectly affect glycemic control in these patients.
Indirect influence of stress on glycemic control: self-efficacy and regimen adherence

The work by Nakahara et al. (2006) attempted to clarify the relationship between stress and blood glucose changes in diabetes mellitus by studying the direct and indirect impact of psychosocial factors on glycemic control of Japanese Type 2 diabetes patients during 12 months. They analyzed the relationship between HbA1c levels and several psychological, social, and self-care behaviors measures of 256 patients. They found a positive correlation between HbA1c and (1) psychosocial scores (Problem Areas in Diabetes Scale) and (2) daily hassles; in addition, there was a negative correlation between HbA1c and (1) adherence for self-care and (2) diabetes related self-efficacy. They also created a causal model and observed self-efficacy directly influencing adherence, which itself directly positively influenced HbA1c. Self-efficacy was positively influenced by social support and inversely influenced by daily hassles and diabetes-related distress. The model was confirmed utilizing the 12-month HbA1c levels during a follow-up (Nakahara et al., 2006).

Similarly, an observational study by Cohen & Kanter (2004) examined the relationship between sense of coherence and glycemic control determined by HbA1c, in addition to the role of psychological distress on adherence to self-care behaviors. Sense of coherence was defined as the individual’s resources and ability to cope successfully with challenges presented throughout a lifetime that protect the individual from negative effects of stressors at the psychological and physical level. After statistical analysis, it was shown that measures of psychological distress were significantly higher in diabetes patients compared to healthy controls. Scores of coherence had a significant negative relationship with adherence, that is, the less ability to cope with stress the greater the
deterioration of diabetic regimen. Also, their model indicated a direct effect of adherence, type of diabetes, and psychological distress on HbA1c levels. Consequently, it was concluded that the better the adherence to self-care behaviors such as regular clinic attendance, blood tests, medication, diet, and physical activity, the better the glycemic control would be in diabetes patients. Such adherence was then influenced by the belief of self-efficacy and psychological distress, therefore indicating an indirect effect of stress on glycemic control in type 2 diabetes patients (Cohen & Kanter, 2004).

Hence, by the previous scientific evidence we can conclude that psychological stress, psychosocial factors, and poor belief of having control over life challenges or lack of self-efficacy indirectly impact the stability of glucose control of individuals suffering from Type 2 diabetes. Such indirect mechanisms may be mediated by the effect of the previous negative factors on adherence to a proper diet regimen, exercise program, medication, or even consistent glucose monitoring. Accordingly, a way of maintaining constant and appropriate self-care behaviors in diabetes patients is to find venues to deal with everyday stress and major life events, along with a method to increase self-efficacy.

**Direct influence of stress on glycemic control: the role of stress hormones**

Surwit & Feinglos (1983) analyzed the effect of progressive muscle relaxation (PMR) training on glucose tolerance in NIDDM patients. All participants were hospitalized and provided with weight-maintenance diets for 5 days; however, only the treatment group practiced PMR twice a day during that week. Pre- and post-study measurements included an OGTT with measurements of plasma glucose and insulin at
0, 30, 60, 90, 120, 150, and 180 min; and an insulin tolerance test (ITT) with measurements of plasma glucose at 5-min intervals for 30 minutes following an insulin infusion relative to body weight. In addition, three blood samples taken at 5-minutes intervals before the pre and post OGTT’s were analyzed for catecholamines and plasma cortisol. At the end of the study significant decreases in the area under the curve were observed during the OGTT of the relaxation group; however, there was no change in the control group. Insulin sensitivity, as measured by the ITT, also showed a trend towards improvement but changes were not significant. Furthermore, on a following publication of the same study, significant decreases in plasma cortisol were reported in treated patients during post-testing while control patients showed a significant increase in this hormone; catecholamines remained unchanged for both groups (Surwit & Feinglos, 1984). The authors suggested that relaxation training may be related to decreases in adrenal cortical activity; specifically, that variations in plasma cortisol levels could alter adrenergic receptor sensitivity to circulating catecholamines thereby changing glucose tolerance (Surwit & Feinglos, 1984).

Nonetheless, another study examining the effect of PMR on metabolic control of diabetes patients found no significant reductions in glucose tolerance (Jablon, Naliboff, Gilmore, & Rosenthal, 1997). One of the main differences between this study and that of Surwit and Feinglos was patient compliance; while Surwit and Feinglos (1983) closely monitored the twice daily practice of PMR in a hospital setting, Jablon et al. (1997) used an outpatient protocol in which subjects were instructed to practice twice a day on their own. A lack of PMR compliance could affect the ultimate results of the study. Moreover, Jablon et al. (1997) did not find significant differences in anxiety scores, as measured
by the State-Anxiety Inventory (STAI), between the PMR group and the control group at the end of the study. In addition, subjects responded to a subjective relaxation ability scale where 0 represented “not at all able to relax” and 10 “able to relax extremely well.” Despite significant improvement in the scale response (pre: 3.4 vs. post: 5.7), the average values may indicate that the PMR group did not achieve proficiency in their relaxation ability.

In the study by Surwit & Feinglos (1983) the lack of psychological stress assessment of participants was the major weakness; however, under highly controlled conditions it was demonstrated that the treatment itself, and not extraneous variables such as modifications in diet or additional physical activity, produced the significant improvements in glucose tolerance and decreases in physiological markers of stress like cortisol. Nevertheless, subsequent studies are challenged to establish a more clear relationship between psychological stress and glucose control in diabetes.

Later research provides evidence of the interaction between psychological stress, in the form of anxiety, and glucose control in Type 2 diabetes patients (Lane, McCaskill, Ross, Feinglos, & Surwit, 1993; Okada et al., 1995). Lane et al. (1993) also conducted OGTT tests in diabetes patients before and after the implementation of 48 weeks of PMR practice and conventional diabetes therapy. However, they compared the results with a control group that underwent diabetes therapy alone. In addition to the regular OGTT protocol, the researchers conducted another OGTT one hour following the ingestion of alprazolam in all patients to observe glucose responses to this drug. Alprazolam is used for the treatment of anxiety disorders, it produces a decrease in neuronal excitability of the nervous system and therefore has a relaxing effect (Katzung,
2007); its peak blood level is approximately 1-2 hrs, which corresponds to the implementation of the OGTT. Additionally, an epinephrine infusion resembling secretion during stress was conducted between OGTT test days; blood glucose samples were taken at baseline, and after infusion at 30-min intervals for 120 min. Finally, the STAI inventory was administered before the beginning of the study to assess anxiety levels in participants before the intervention. After 48 weeks, significant improvements in glucose response were observed in both the experimental and the control group. Nonetheless, Lane et al. (1993) found that relative improvements in glucose tolerance, as measured by reductions in the incremental glucose area, were significantly correlated to improvements associated with alprazolam therapy and high anxiety scores in the relaxation group; no such correlation was found in the control group. That is, subjects who demonstrated improvements in glucose tolerance after alprazolam treatment also improved in glucose tolerance after relaxation treatment. Similarly, subjects with initial elevated anxiety scores responded better to relaxation treatment. Furthermore, those subjects that showed a greater deterioration in blood glucose levels with epinephrine infusion had a trend toward greater improvements in glucose tolerance after relaxation treatment. Therefore, authors concluded that relaxation therapy may produce significant improvements in glucose tolerance for those individuals who suffer from anxiety and are more susceptible to adrenergic stimulation by catecholamines (Lane et al., 1993). Likewise, Okada et al. (1995) utilized a pharmacological agent to study the relationship between anxiety and glucose control; however, the focus of the intervention was different. The authors administered another anti-anxiety drug, fludiazepam, to diabetes patients for 12 weeks; pre and post assessments of HbA1c were conducted as a
measure of glycemic control. Diet, exercise, and medical therapy remained constant in all participants throughout the study. At the end of the intervention, diabetes patients showed significant reductions in anxiety levels, but most importantly, patients showed significant reductions in HbA$_1c$ from 8.4% to 7.3%. Multiple regression analysis further indicated a significant correlation between decreased anxiety and improved glucose control. It was concluded that anxiety may be associated with metabolic control in patients with diabetes (Okada et al., 1995).

Surwit & Feinglos (1983) found improvements in glucose tolerance after a relaxation intervention, while Lane et al. (1993) and Okada et al. (1995) found improvements in glucose tolerance and long-term glycemic control, respectively, after pharmacological interventions that relieved anxiety. Therefore, a detailed examination of adrenergic stimulation by catecholamines and cortisol during stress and anxiety is necessary to understand the impact of negative psychological states on the deterioration of glycemic control in diabetes.

*Adrenergic stimulation by catecholamines*

Cell in all tissues of the body possess receptors that will bind to the catecholamines epinephrine and norepinephrine; such receptors are further divided into $\alpha_1$, $\alpha_2$, $\beta_1$, $\beta_2$, and $\beta_3$ subtypes (Goodman, 2009). Depending to which adrenoreceptor they bind, catecholamines can exert an entirely different effect. For instance, binding to $\alpha_1$ will produce smooth muscle contraction, while binding to $\beta_2$ will promote smooth muscle relaxation (Katzung, 2007). Specifically to the subject of interest, epinephrine and norepinephrine stimulate the $\alpha$-adrenergic receptors on the beta cells of the
pancreas which will ultimately decrease insulin secretion (Powers & Howley, 2007). As a result, we can hypothesize that just as in exercise, when elevations in circulating catecholamines decrease insulin secretion via stimulation of α-adrenergic receptors (Powers & Howley, 2007), episodes of stress that cause elevations in epinephrine and norepinephrine will also produce a decrease in insulin secretion. The main difference between exercise and psychological stress in terms of the positive and negative consequences of each in glycemic control may reside in the fact that exercise induces changes in glucose transporters, consequently increasing muscles' sensitivity to insulin and decreasing whole-body insulin resistance (Powers & Howley, 2007). In contrast, psychological stress may simply blunt insulin secretion without simultaneously increasing insulin sensitivity. Nonetheless, the question remains on whether the stress response is augmented in diabetes mellitus compared to healthy individuals in order to state that stress will further deteriorate glycemic control in these patients.

Robertson, Halter, & Porte (1976) reported an absence of acute insulin response to a glucose infusion in NIDDM patients compared to weight-matched healthy controls, even when both groups did not differ in fasting serum insulin levels. However, insulin response to glucose was re-established in diabetes patients after an α-adrenergic blocker drug. Increases in insulin secretion were also observed in the control group after drug administration, but the increment was significantly lower compared to diabetics (patients, 14 ± 9; controls, 3 ± 2; p<0.01). In addition, simultaneous infusions of an α-adrenergic blocker drug and a β-adrenergic blocker drug were administered to some diabetes patients. Interestingly, an improved insulin response to glucose was still observed in diabetics in spite of beta blockade. Finally, circulating total plasma
catecholamine levels were shown to be significantly greater in NIDDM patients compared to controls, both at basal state and after glucose infusion. The authors concluded that α-adrenergic inhibition of insulin secretion is exaggerated in diabetes mellitus; they suggested that such α-adrenergic hyperactivity may be caused by elevated circulating catecholamines levels in these patients (Robertson, Halter, & Porte, 1976). Later research by Bruce, Chisholm, Storlien, Kraegen, & Smythe (1992) showed that diabetes patients also have significantly higher plasma glucose responses to a norepinephrine infusion compared to healthy controls matched for age and blood pressure. In this study, diabetics also showed significantly greater blood pressure responses after norepinephrine infusion compared to controls.

Subsequent research continued demonstrating the impact of adrenoreceptor dysregulation on glucose homeostasis (Davani et al., 2004; Largis, Burns, Muenkel, Dolan, & Claus, 2004). In the work by Davani et al. (2004), transgenic (TG) mice with elevated glucocorticoid sensitivity were found to have higher FBG concentrations compared to control mice. Also, TG mice showed significantly lower insulin response during an intraperitoneal glucose tolerance test (IGTT) compared to controls, producing significantly higher elevations of glucose during the test. Nevertheless, glucose clearance during an intraperitoneal insulin tolerance test (IPITT) was similar between both groups of mice when insulin was administrated relative to body weight to all mice, indicating that it was not insulin sensitivity, but dysregulations in insulin secretion causing hyperglycemia in TG mice. Furthermore, analysis of islets in vitro indicated that TG islets showed significantly less insulin release at 16.7 mmol/l glucose while control islets had normal insulin responses. Interestingly, insulin response to glucose was
normalized in TG islets after they were treated with an $\alpha_2$-adrenergic receptor antagonist; control islets had no such response after treatment. The authors concluded that chronic augmentation of glucocorticoid signaling to $\alpha_2$-adrenoreceptors in pancreatic $\beta$-cells produce hyperglycemia and impaired glucose tolerance (Davani et al., 2004).

Similarly, Largis et al. (2004) observed significant decreases in hyperglycemia, with subsequent restoration to euglycemia, in diabetic mice after treatment with a $\beta_3$-adrenoceptor agonist compared to lean, healthy mice with no previous $\beta$-receptor desensitization; in addition, glucose tolerance improved along with reductions in plasma insulin in treated mice. Finally, Asensio et al. (2005) demonstrated that mice lacking $\beta$-adrenoreceptors ($\beta$-less mice) had an altered metabolism compared to healthy mice, by showing higher percentages of body fat and glucose intolerance. $\beta$-less mice also showed significantly lower insulin responses to a glucose challenge compared to controls, despite comparable basal insulin levels in both groups. Similar to previous research, Asensio et al. (2005) found increases in glucose uptake after insulin administration in both $\beta$-less mice and controls, suggesting that it was glucose-induced insulin secretion, and not insulin sensitivity, the mechanism affected in mice lacking $\beta$-adrenoreceptors.

Actually, long before experimentation in mice, pharmacological interventions aimed to block $\alpha_2$–adrenergic receptors have successfully improved glucose-stimulated insulin secretion and glucose disposal in NIDDM patients (Broadstone, Pfeifer, Bajaj, Stagner, & Samols, 1987; Kashiwagi et al., 1986). Furthermore, another possible mechanism of action of sulfonylureas, widely used in the treatment of Type 2 diabetes,
is desensitization of $\alpha_2$–adrenergic receptors to catecholamines (Katzung, 2007), which will further augment insulin secretion.

Therefore, evidence suggests that individuals suffering from diabetes mellitus may show an increased sensitivity to circulating epinephrine and norepinephrine compared to healthy subjects. Such sensitization is specifically present in $\alpha_2$–adrenergic receptors, which in turn will augment the inhibition of insulin release from the pancreas. Consequently, it will be expected that for every psychological stress episode, in which elevations of circulating catecholamines intensify the adrenergic activity of the sympathetic nervous system (Asterita, 1985), the glucose response experienced by diabetes patients will be exaggerated due to their heightened adrenergic sensitivity.

*The possible role of cortisol on further deterioration of glycemic control in diabetes patients*

According to the General Adaptation Syndrome (GAS) theory by Hans Seyle, there are three phases of the body’s adaptation to stress, which are: alarm reaction, stage of resistance, and stage of exhaustion. Alarm reaction is the “fight or flight” response originally developed by Canon, characterized by sudden elevations of epinephrine and norepinephrine. During the stage of resistance there is a reduction of catecholamines; however, there is a marked increase in cortisol secretion in order to sustain heightened levels of functioning in the body (Asterita, 1985). Hence, it is this stage of the body response and adaptation to stress that may pose significant challenges to diabetes patients because of the influence of cortisol on glucose metabolism.
Previous research has indicated the effect of cortisol on the enhancement of the glucose response during simulated episodes of stress. Eigler, Sacca, & Sherwin (1979) investigated the effect of counterregulatory hormones on glucose metabolism by simultaneously infusing epinephrine, cortisol, and glucagon to healthy dogs in doses that resembled hormonal changes during severe stress; single hormone infusions also took place. They found that the combination of epinephrine + cortisol produced a significantly higher augmentation in plasma glucose compared to epinephrine infusion alone. Similarly, the combination of glucagon + cortisol also produced a significant increase in glucose production compared to glucagon infusion alone. The authors concluded that cortisol maintains elevations in glucose production initially generated by epinephrine and glucagon, and that changes in glucose metabolism during stress are a result of the synergistic interaction between these hormones and not just one hormone alone (Eigler et al., 1979). Therefore, continuous elevations of cortisol produced by chronic stress are expected to be more detrimental to diabetes patients because they already have difficulty metabolizing plasma glucose due to insulin resistance or irregular insulin secretion, and consequently glycemic control will be compromised.

Additionally, research has already provided some evidence of altered hypothalamic-pituitary-adrenal (HPA) axis and subsequent cortisol elevations in diabetes patients (Chiodini et al., 2007; Hudson, et al., 1984; Roy, Collier, & Roy, 1990). Significantly higher plasma cortisol levels at 0900 hrs and 1600 hrs have been observed in diabetes patients compared to healthy controls (Roy et al., 1990). In addition, previous investigations have found that plasma cortisol levels remained significantly elevated after a dexamethasone test in diabetes patients compared to controls (Hudson
et al., 1984; Roy et al., 1990). A dexamethasone test is used to detect hypercortisolism; cortisol levels are expected to drop after an overnight dose of dexamethasone (Katzung, 2007). Finally, Chiodini et al. (2007) investigated cortisol secretion in Type 2 diabetes patients and its possible relationship with metabolic control and diabetes complications; they found significant elevations in 24-hr urinary free cortisol and serum cortisol levels at 12am in diabetes patients with present complications and a mean HbA$_1c$ of 10.2% compared to patients without complications and a mean HbA$_1c$ of 9.3% and matched controls. Moreover, significant associations were found between presence of complications and duration of diabetes with serum cortisol levels at midnight (Chiodini et al., 2007).

In summary, it appears that chronic cortisol elevations in diabetes patients are present, especially among those with poorer levels of glycemic control and longer duration of diabetes (Chiodini et al., 2007; Hudson et al., 1984; Roy et al., 1990). Research indicates that cortisol will likely augment glucose production during episodes of stress (Eigler et al., 1979), making individuals with glucose intolerance and elevated cortisol levels, such as diabetics, even more susceptible to the negative effects of stress. Therefore, it can be proposed that a reduction in psychological stress may be beneficial for these patients by decreasing adrenal-cortical activity and consequently cortisol levels.
Prevalence of depression and anxiety in diabetes patients: implications in quality of life of patients

Several studies have reported problems of depression and anxiety and its subsequent effects on quality of life among patients suffering from diabetes mellitus (Chyun et al., 2006; Mosaku, Kolawole, Mume, & Ikem, 2008; Okada et al., 1995; Van der Does et al., 1996). Okada et al. (1995) found significantly higher anxiety levels, both state and trait, in diabetes patients compared to healthy controls matched by age, sex, and body mass index. Mosaku et al. (2008) observed that symptoms of depression were most prevalent among diabetics compared to asthmatic patients and healthy controls. In their study, poor glycemic control assessed by FBG was significantly correlated with symptoms of depression and anxiety; additionally, FBG, depression, and anxiety were negatively correlated with measures of general well-being and positive well-being subscales of the diabetic well-being questionnaire (Mosaku et al., 2008). Similarly, Chyun et al. (2006) reported that depressive symptoms and anxiety among diabetes patients negatively correlated with almost all measures of quality of life, as measured by the Medical Outcomes Study Short Form-36 (SF-36), and with the domains of the Diabetes Quality of Life (D-QOL) measure. Lastly, Van der Does et al. (1996) examined outcome measures of the Type II Diabetes Symptom Checklist (DSC-Type 2) and Profile of Mood States (POMS) questionnaire and their relationship with HbA$_{1c}$ values in type 2 diabetes patients; they found a significant correlation between areas such as depression, anger, and cognitive distress and high levels of HbA$_{1c}$. Hence, they suggested that poor glycemic control is related to low measures of well-being in these patients (Van der Does et al., 1996). After reviewing the research
evidence, it can be determined that depression, anxiety, and stress produce a deterioration in well-being, as measured by a large variety of psychological instruments. Consequently, it can be concluded that alleviation of psychological symptoms is critical to sustain good levels of quality of life in type 2 diabetes patients.

Psychological benefits induced by yoga practice

Psychological stress and anxiety

Extensive research has demonstrated the benefits of yoga practice for psychological stress relief, although methodology to measure stress has varied between studies (Agte & Chiplonkar, 2008; Carmody & Baer, 2008; Cowen & Adams, 2005; Granath, Ingvarsson, von Thiele, & Lundberg, 2006; Gupta, Khera, Vempati, Sharma, & Bijlani, 2006; Javnbakht, Kenari, & Ghasemi, 2009; Schell, Alloio, & Schonecke, 1993; Schure, Christopher, & Christopher, 2008; Smith, Hancock, Blake-Mortimer, & Eckert, 2007; West, Otte, Geher, Johnson, & Mohr, 2004; Wheeler & Wilkin, 2007). Studies focusing on the acute effects of this ancient discipline have found that a single hatha yoga session produces significant decreases in perceived stress (West et al., 2004), and decreases in excitability and aggressiveness as measured by a personality inventory (Schell et al., 1993). Likewise, significant decreases in perceived stress have been observed in studies examining the long-term effects of yoga on the psychological state of healthy adults (Cowen & Adams, 2005; Granath, 2006; Wheeler & Wilkin, 2007). Moreover, in a qualitative investigation examining the impact of mind-body medicine in graduate students undergoing counseling treatment, a greater ability to deal with stress and strong emotions was reported after yoga practice for 15 weeks; in
addition, participants claimed to experience new ways to respond to fears, anxieties, and doubts (Schure et al., 2008).

Implementing yoga as part of an intervention has also demonstrated significant decreases in anxiety as measured by the STAI. Gupta et al. (2006) found significant reductions in both, state anxiety and trait anxiety, after 8 days of rigorous yoga practice in patients with a variety of medical problems including hypertension, obesity, diabetes, and coronary artery disease; meanwhile, the control group showed no change in psychological state. Similarly, Smith et al. (2007) found significant decreases in state anxiety after a 10-week yoga intervention in physically healthy adults, while Agte & Chiplonkar (2008) and Javnbakht et al. (2009) also found overall decreases in anxiety after two months of yoga practice in subjects reporting no physical symptoms.

Depression

Authors have previously observed significant decreases in symptoms of depression and anxiety after asana, pranayama, relaxation, and meditation practice for 6 weeks (Campbell & Moore, 2004). In addition, a study examining the impact of yoga and meditation in cancer patients have found significant decreases in anxiety and depression, through the Hospital Anxiety and Depression Scale (HADS) (Ando et al., 2009). Moreover, psychological research has found significant score reductions in the Beck Depression Inventory and the STAI in moderately depressed subjects after five weeks of yoga practice compared to a similar control group that showed no difference pre- and post- testing (Woolery, Myers, Sternlieb, & Zeltzer, 2004). Even more interesting, this same study found no correlation between changes in depression and anxiety of participants, indicating that decreases in depression were independent of
other psychological changes; consequently the authors suggested that yoga has therapeutic effects in individuals experiencing moderate levels of depression (Woolery et al., 2004).

Quality of life

Smith et al. (2007) and Robertshawe (2007) implemented a yoga intervention and found significant improvements in quality of life of participants, as measured by the SF-36, a health status questionnaire measuring eight different dimensions including mental health, physical role function, and emotional role function. Similarly, Carlson, Speca, Patel, & Goodey (2004) implemented 8 weeks of meditation and gentle yoga practice to cancer patients and observed significant enhancements in quality of life and significant reductions in reported stress symptoms, through the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ C-30) and the Symptoms of Stress Inventory (SOSI), respectively.

Multiple dimensions of well-being

In the study by Carmody & Baer (2008) medical students with a variety of problems including chronic pain, anxiety, and illness-related stress, underwent a mindfulness-based stress reduction program that included formal instruction in meditation practices such as sitting meditation and mindful yoga. After 7 weeks, participants reported significant reductions in perceived stress and increases in psychological well-being. Most importantly, subjects reported significant reductions of symptoms, as measured by the Medical Symptom Checklist, and significant reductions in all dimensions of the Brief Symptom Inventory that included a global severity index,
anxiety, and depression (Carmody & Baer, 2008). Finally, improvements in a general well-being inventory were shown in healthy males following hatha yoga practice for 3 months (Harinath, et al., 2004).

Additional benefits of yoga

Research has shown significant improvements in flexibility, and trunk muscle strength and endurance after 6 weeks of formal yoga practice in healthy adults aged 20-58 years (Cowen & Adams, 2005). Similarly, improvements in body fat percentage, balance, sleep disturbances, and range of motion at the shoulder and hip were observed following a 4 week yoga program in women aged 60 to 86 years (Chen & Tseng, 2008). Moreover, yoga practice has been shown to be more effective than physical therapy alone in the treatment of chronic low back pain, decreasing pain and producing increases in spinal flexion and extension, and lateral flexion (Tekur, Singphow, Nagendra, & Raghuram, 2008). In the qualitative investigation by Schure et al. (2008) hatha yoga practice also produced increases in flexibility, strength, balance, energy levels, and body awareness in students. Finally, following yoga practice reductions in systolic blood pressure (Chen & Tseng, 2008), diastolic blood pressure (Cowen & Adams, 2005), and mean arterial pressure (Harinath, et al., 2004) have been observed in individuals with normal blood pressure; while normal blood pressure has been reestablished in individuals suffering from hypertension in a study with adults aged 35-65 years (Murugesan, Govindarajulu, & Bera, 2000).
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APPENDIX

MODIFICATIONS OF YOGA POSES
Figure 1. Triangle pose

Figure 2. Modified triangle pose

Figure 3. Extended side stretch pose

Figure 4. Modified extended side stretch pose

Figure 5. Revolved triangle pose

Figure 6. Modified revolved triangle pose
Figure 7. Modified bound angle pose

Figure 8. Modified legs-up-the-wall pose
CURRICULUM VITA

Maricarmen Vizcaino was born in Chihuahua, Chih, Mexico. Daughter of Jesus Vizcaino and Maria del Carmen Campos, and wife of Pedro Lopez-Gomez. She entered the graduate program at the University of Texas at El Paso (UTEP) in Fall 2006. While pursuing her masters’ degree with concentration in exercise science, she worked with UTEP’s Track and Field Team as a graduate assistant and program coordinator, as a yoga instructor with the Recreational Department, and as a Teacher Assistant in the Biomechanics Laboratory with the Kinesiology Department. A registered yoga teacher (RYT) by Yoga Alliance and a certified personal trainer by the National Strength and Conditioning Association (NSCA), she plans to continue a research career in the area of alternative and complementary therapies, specifically on yoga and meditation, and its impact of chronic diseases such as Type 2 diabetes mellitus. She currently lives in El Paso, TX with her husband and her dog Mozart.