Twenty minutes of passive stretching lowers glucose levels in an at-risk population: an experimental study

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Question: Can passive static stretching lower blood glucose in an at-risk population? Design: Randomised, within-participant experimental study. Participants: 22 adults (17 males) either at increased risk of Type 2 diabetes or with Type 2 diabetes. Intervention: The participants reported to the laboratory 2 hr after eating a meal, and drank 355 ml of fruit juice (~43 g carbohydrate). Thirty minutes later, they underwent either a 40-min passive static stretching regimen or a mock passive stretching regimen. Stretching consisted of six lower body and four upper body static passive stretches. For the mock stretches, the same positions were adopted, but no tension was applied to the musculature. Outcome measures: Blood glucose levels for both the stretching and mock stretching were analysed from a finger prick sample using a hand-held glucometer. Values were obtained at baseline (0 min), during the regimen (20 min), and after the regimen (40 min) on both study days. Results: Compared to mock stretch, stretching resulted in a significantly greater drop in blood glucose at 20 min (mean difference 28 mg/dL, 95% CI 13 to 43; or 1.57 mmol/L, 95% CI 0.72 to 2.39). This effect was also statistically significant at 40 min (mean difference 24 mg/dL, 95% CI 9 to 39; or 1.35 mmol/L, 95% CI 0.50 to 2.17). Conclusion: These results suggest that passive static stretching of the skeletal muscles may be an alternative to exercise to help lower blood glucose levels. [Nelson AG, Kokkonen J, Arnall DA (2011) Twenty minutes of passive stretching lowers glucose levels in an at-risk population: an experimental study. Journal of Physiotherapy 57: 173–178]

Key words: Type 2 diabetes, Pre-diabetes, Blood glucose, Pacific Islanders, Caucasians

Introduction

It is possible to prevent or delay the onset of Type 2 diabetes by reducing lifestyle risk factors through moderate weight loss and increased physical activity. Several studies have shown that lifestyle changes that include exercise can significantly delay and possibly prevent diabetes (Tudor-Locke et al 2000, Wei et al 2000). Moreover, in people with Type 2 diabetes using insulin, a single bout of light exercise significantly reduces the prevalence of hyperglycemia during the subsequent day by about 40% (Manders et al 2010). Also, considerable amounts of data have accumulated showing that muscle contraction triggers glucose uptake (for reviews see Dohm 2002, Henriksen 2002). In contrast, if good glucose control is not achieved over time, prolonged hyperglycemia can lead to negative and severe outcomes such as retinopathy, nephropathy, neuropathy, cardiovascular disease, stroke, pressure ulcers, neuropathic wounds, loss of peripheral protective sensation, gangrene, limb amputation, and death.

Notwithstanding the benefits derived from regular exercise, there are many people with Type 2 diabetes who do not exercise. For some individuals, the secondary complications arising from diabetes (eg, lower limb neuropathies, lower limb amputations, hypertension, kidney disease, and retinopathies) can either contraindicate exercise or make it more difficult. Also, many elderly people with Type 2 diabetes residing in extended care facilities are either extremely frail, wheelchair bound, or bed bound, and do not have sufficient physical work capacity to exercise aerobically and thus have problems maintaining euglycemia (Zarowitz et al 2006). Hence, for most of these patients, the physician is constrained to use a sliding-scale insulin plan in an attempt to control hour-to-hour glucose levels.

Passive static stretching of the skeletal muscles may be a modality that could accrue the benefits of exercise without its accompanying physical stress. Passive static stretching occurs when sustained tension develops within a person’s muscle through actions performed by an outside source. Several studies, using either cell culture or isolated animal muscles, suggest that passive stretching of a person’s muscles could result in increased cellular glucose uptake. Hatfullady and colleagues (1989) exposed cultured avian pectoralis muscle cells to repetitive cycles of stretch and relaxation. After four hours, uptake of a marker of tissue glucose use (3H) deoxy-D-glucose increased 34%. Similarly, Mitsumoto and colleagues (1992) subjected L6 muscle cells to 24 hours of intermittent stretch and relaxation (25% maximum elongation at 30 cycles per minute), and saw as much as a 2-fold increase in glucose marker (2-deoxy-D-glucose) uptake. Also, Iwata and colleagues (2007) reported increased glucose marker (2-deoxy-D-glucose) uptake in mechanically stretched cultured C2C12 myotubes, which they attributed to a Ca²⁺-dependent mechanism. Correspondingly, using isolated muscle, Thiemann and colleagues (1999) stretched rat soleus passively for five minutes, and found a 50% increase in uptake of the same glucose marker (2-deoxy-D-glucose). Lastly, in an in situ study, Nie and colleagues (2000) reported an increase in glucose transporters (GLUT 1) in denervated hemidiaphragm. They postulated that the increase in the glucose transporters could have resulted by the passive stretched imposed on the denervated hemidiaphragm by the activity of the contralateral side.

It is therefore possible that an individual could experience a noticeable decrease in blood glucose following a program of successive sustained muscle stretches. Passive stretching requires minimum effort by the person experiencing the
To be eligible to participate, the volunteer had to have been diagnosed either as having Type 2 diabetes, or as being ‘at risk’ for Type 2 diabetes by having at least three of the following four risk factors: sedentary, aged at least 45 yr, BMI at least 25 kg/m², and a family history of Type 2 diabetes.

Can a regimen of passive stretching lower blood glucose levels following a glucose challenge in people who have Type 2 diabetes? Can people who are reluctant or unable to exercise may be willing to submit to a stretching protocol. The research question was:

### Method

#### Design

Participants were tested twice with three days between tests. For each test the participants reported to the laboratory two hours after eating a meal, and immediately drank a 355 ml (12 fl. oz.) can of fruit juice (~43 g carbohydrate). Thirty minutes after drinking the fruit juice, the participants went through either a 40-min passive static stretching regimen or a mock passive stretching regimen (ie, participants assumed the stretch positions, but no tension was placed upon the musculature). The order of the interventions (ie, stretching or mock stretching) was assigned in a random, balanced order.

#### Participants

Adults were recruited from the population of Laie, Hawaii (population approximately 5000) to participate in the study. To be eligible to participate, the volunteer had to have been diagnosed either as having Type 2 diabetes, or as being...

### Description of Stretches Used

<table>
<thead>
<tr>
<th>Stretch Description</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seated knee flexor (bilateral)</td>
<td>Each person sat on the floor with the legs extended and arms above the head. From this position, each person lowered their head toward the knees, while the experimenter pushed down on their back.</td>
</tr>
<tr>
<td>Seated knee flexor – hip adductor (bilateral)</td>
<td>The participants sat on the floor in the lotus position. From this position, each person lowered their head toward the floor, while the experimenter pushed down on their back.</td>
</tr>
<tr>
<td>Seated shoulder lateral flexor (bilateral)</td>
<td>The person sat in a chair with fingers interlocked and placed behind the head. Keeping the arms in this position, the experimenter stood behind the person and pulled the elbows back toward the body’s midline.</td>
</tr>
<tr>
<td>Supine hip flexor-knee extensor (unilateral)</td>
<td>The participants lay on their backs with their leg hanging over the edge of the table with the knee flexed at approximately 90°. The hip was then hyperextended by the experimenter pushing down on the thigh.</td>
</tr>
<tr>
<td>Seated hip external rotators, extensors (unilateral)</td>
<td>Each person sat on the floor with one leg extended. The opposite leg was flexed at the knee, and the foot placed flat against the extended leg’s inner thigh. The person then lowered their head toward the extended knee, while the experimenter pushed down on their back.</td>
</tr>
<tr>
<td>Supine knee flexor-plantar flexor (unilateral)</td>
<td>Each person lay on their back with the legs extended. The experimenter then raised one leg, and simultaneously flexed the hip and dorsiflexed the ankle.</td>
</tr>
<tr>
<td>Prone hip flexor (unilateral)</td>
<td>Each person lay on their stomach and flexed one knee at approximately 60°. Keeping the knee at the flexed position, the experimenter lifted the thigh to hyperextend the hip.</td>
</tr>
<tr>
<td>Seated shoulder flexors, depressors (bilateral)</td>
<td>Each subject sat on the floor with the legs extended. The experimenter then grabbed the wrists and, while keeping the back and elbows straight, hyperextended the shoulder by raising the arms behind the back and up towards the head.</td>
</tr>
<tr>
<td>Seated shoulder and elbow flexors (unilateral)</td>
<td>Each subject sat on the floor with the legs extended, with one elbow flexed and brought up near the ear. From this position the shoulder was hyperflexed by the experimenter pushing the upper arm down towards the floor.</td>
</tr>
</tbody>
</table>
flexor; seated shoulder flexor and depressor; and seated shoulder flexor and elbow extensor. A description covering how each of these stretches was performed is presented in Box 1. Twenty minutes after the initiation of either the stretching or mock stretching, the regimen was interrupted for a blood glucose measure. After this, the participants continued the treatment regimen, but only up to 40 minutes at which point the treatments were ended.

**Outcome measures**

Blood glucose levels were analysed from a finger prick drop of blood, using a hand-held glucometer whose accuracy was checked against a company supplied standard before each participant’s use. Values were obtained at baseline (0 min), during the regimen (20 min), and after the regimen (40 min) on both study days.

**Data analysis**

A two-way (treatment × time) repeated measures ANOVA was used for data analysis. Significance was set at \( p < 0.05 \). To ascertain whether any treatment differences were due to a day 1-to-day 2 variation in glucometer readings, an additional two-way (day × time) repeated measures ANOVA was used to determine whether there was a difference between the two different days (ie, the results were collapsed across days). Effect size (\( \eta_p^2 \)) was calculated using the formulas recommended by Bakeman (2005). Post-hoc ANOVA analysis involved, where appropriate, the use of Bonferroni t-tests.

A total of 22 patients entered this crossover study. The probability was 80 percent that the study would detect a treatment difference at a two-sided 0.05 significance level, if the true difference between treatments was 17 mg/dL or 0.94 mmol/L. This is based on the assumption that the standard deviation of the difference in the response variable is 27 mg/dL or 1.50 mmol/L.

**Results**

**Characteristics of participants**

Twenty-two adults (15 males, 7 females) participated in the study. The baseline characteristics of the participants are presented in Table 1. Seven of the participants (4 males, 3 females) had been previously diagnosed with Type 2 diabetes, and the rest (11 males, 4 females) were in the ‘at risk’ category. In addition, six participants (4 males, 2 females) were Caucasian, and the rest were of mixed race (Asian, Caucasian, and Pacific Islander).

**Table 1. Mean (SD) of baseline characteristics of participants.**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All (n = 22)</th>
<th>Males (n = 17)</th>
<th>Females (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>47 (10)</td>
<td>48 (11)</td>
<td>48 (4)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173 (10)</td>
<td>177 (6)</td>
<td>163 (7)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>103 (27)</td>
<td>133 (24)</td>
<td>81 (18)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>34.2 (7.2)</td>
<td>35.8 (7.6)</td>
<td>30.6 (5.2)</td>
</tr>
</tbody>
</table>

**BMI = body mass index**

**Stretching and blood glucose levels**

The effect of the stretching and mock stretching programs upon blood glucose values are presented in Table 2. The
individual patient data are presented in Appendix 1 on the eAddenda. The main effect for treatment ($F (1, 21) = 6.33, p = 0.02, \eta^2 = 0.23$), the main effect for time ($F (1, 21) = 35.26, p < 0.001, \eta^2 = 0.63$), and the interaction between treatment and time ($F (1, 42) = 10.45, p < 0.001, \eta^2 = 0.33$) were significant. The best estimate of the magnitude of the effect of 20 min of stretching on the change in blood glucose was a reduction of 28 mg/dL, with a 95% CI of 13 to 43. The best estimate of the magnitude of the effect of 40 min of stretching on the change in blood glucose was a reduction of 24 mg/dL, with a 95% CI of 9 to 39. Post hoc analysis of the interaction between treatment and time showed that for the mock stretch the 40 min value was significantly less than either 0 min or 20 min, while for stretching both the 20 min and 40 min values were significantly less than 0 min. In addition, the stretching 20 min and 40 min values were significantly less than their mock stretching counterparts.

**Variation from Day 1 to Day 2**

The analysis of day-to-day variation (ie, the stretching and mock stretching results collapsed across days) showed that both the main effect for days and the interaction between days and measurement times were not significant. The main effect for time, however, was significant. The blood glucose levels at 0 min were significantly greater than those at 40 min.

**Discussion**

The purpose of this study was to determine if a program of passive static stretching could significantly lower blood glucose in people with Type 2 diabetes or ‘at risk’ for developing Type 2 diabetes. The results suggest that engaging in 20 minutes or more of passive static stretching will lower blood glucose values to a greater extent than doing nothing. This finding is noteworthy especially considering that the study design placed stretching in a ‘worst case’ scenario for demonstrating a treatment effect. First, instead of having the participants lie motionless for the control portion, the subjects engaged in mock stretching. Since even light activity can start to lower blood glucose, having the people move around into different positions increased the likelihood of having both of the study conditions lower blood glucose. Thus, having the stretching treatment lower blood glucose significantly more than the mock stretching strengthens the argument that the stretching by itself influences blood glucose. Second, stretching may possibly cause discomfort and pain during the stretch. Emotional and physical stress can cause the release of cortisol and catecholamines, both of which can raise blood glucose via activation of liver glycogenolysis. However, the stretching used in the experimental condition was not ‘eased off’ to the point of no discomfort. Nevertheless, the stretching regimen still produced significantly lower blood glucose levels at 20 and 40 minutes than the control condition.

There are several possible mechanisms that could explain why passively stretching muscles yields a lower blood glucose concentration. First, numerous studies have shown that despite a lack of sarcolemma depolarisation or cross-bridge cycling, a stretched muscle cell can not be considered metabolically dormant. In 1932, Feng (1932) showed that a passively stretched in vitro muscle was metabolically active. He found that passively stretched muscles exhibited increased heat production and oxygen consumption. Later research corroborated these findings; Clinch (1968) reported increased heat production, while Whalen and colleagues (1962) and Barnes (1987) added reports of increased oxygen consumption. In other related studies, passive stretch increased carbon dioxide production (Eddy and Downs 1921), increased glycogen breakdown (Barnes and Worrell 1985), increased lactic acid production (Barnes 1987), and decreased phosphocreatine concentrations (Barnes 1987). Since increased metabolic activity is related to increased activation of the adenosine monophosphate kinase (AMPK) facilitated glucose transporter (GLUT 4) activation pathway (Dohm 2002), it is possible that the increased metabolic activity accompanying passive muscle stretching could have activated the incorporation of GLUT 4 into the stretched muscles.

Other research also points to the possibility of stretching increasing GLUT4 incorporation. For instance, protein kinase B activity partially controls GLUT 4 incorporation and activation, and Sakamoto and colleagues (2003) found that protein kinase B was stimulated by passively stretching isolated muscles for ten minutes. Second, mitogen-activated protein kinase activity stimulates muscle cell glucose uptake (Ho et al 2004), and the activity of mitogen-activated protein kinases directly reflects the magnitude of the mechanical stress (ie, actively or passively generated tension) applied to the muscle (Martineau and Gardiner 2001). Third, exercise-induced increases in nitric oxide result in increased glucose transport (Roberts et al 1997), and nitric oxide released from excised soleus muscles can be increased 20% by a single two-minute passive stretch (Tidball et al 1998). Finally, ischaemia can increase GLUT 4 translocation to the sarcolemma as well as increasing glucose uptake (Sun et al 1994, Young et al 1997), and passive stretching has the potential to cause ischaemia (Poole et al 1997, Wines and Kirkebo 1976). Wisnes and Kirkebo (1976) found an increased resistance to blood flow during passive stretching. In addition, Poole and colleagues (1997) reported that muscle stretching reduces bulk blood delivery, alters capillary flow dynamics, and impairs blood tissue oxygen exchange.

Regardless of the responsible mechanisms, it is clear that passive static stretching had a significant positive effect on blood glucose levels. This finding has important clinical applications because numerous patient populations are sedentary due to chronic illness, no availability of equipment, or a lack of motivation to exercise. Despite encouragement from the medical professions, most people fail to meet the most minimal level of daily exercise that would prevent the deleterious effects of hypomobility (American Diabetes Association 2008, Tudor Locke et al 2000, Wei et al 2000). Thus, the finding that static stretching has the potential to be a viable treatment for hyperglycemia provides an alternative treatment modality in the absence of the patient’s desire to exercise. In addition, stretching skeletal muscles similarly to that demonstrated in this study is a hopeful alternative to exercise for those patients with Type 2 diabetes who are too disabled to exercise. Some patient groups that could benefit from a stretching program for improved glucose control might be patients who have sustained a spinal cord injury, patients who have New York Class III/IV rheumatoid arthritis, stroke patients, and those individuals who are constrained to long term bed rest. As physical therapists and nurses interact with these hypomobile patients, 20–40 minutes of passive static stretching could be incorporated into the patient plan of care.
Also, many nursing homes do not have a policy to evaluate the effectiveness of a treatment algorithm in their resident population with diabetes to determine if the staff is able to control the glucose peaks and nadirs in these patients (Feldman et al 2009). Few nursing homes, for example, have a policy to evaluate the patient’s HbA1c values routinely (Feldman et al 2009), a fundamental recommendation by the American Diabetes Association (2008). Failure to control blood glucose levels adequately in the diabetic population represents nearly 50% of all deaths in nursing homes (Russell et al 2005). If a stretching program (either passive or active) under the supervision of a physical therapist or other trained personnel was established, these patients could realise better blood glucose control and health at a substantial financial saving.

We acknowledge that this study looked only at the immediate effect of stretching and did not ascertain if this effect could be carried over successive days of stretching. Nevertheless, Kokkonen and colleagues (2007) have shown that a program of 40 minutes static stretching done three times a week can increase muscle strength and endurance. In addition, Nelson and colleagues (2005) have presented data showing that static stretching raises the metabolic rate similar to the rate estimated for walking 40 m/min. These findings, coupled with the results of this study, suggest that stretching daily for 20–40 min may help a person to control or lower blood glucose levels.

In conclusion, this study shows that static stretching is an additional viable activity that can help regulate blood glucose acutely. Since it requires little effort by the individual, it appears to be an advantageous treatment for those with reduced physical capabilities. Also, it can be done without any additional equipment, facilities, or other expenses. Thus it should easily fit into the repertoire of treatment modalities of people with Type 2 diabetes.

**eAddenda:** Table 3 and Appendix 1 available at jop.physiotherapy.asn.au

**Footnotes:** aAscensia Breeze, Bayer Diabetics.

**Ethics approval:** The Brigham Young University-Hawaii and Louisiana State University Ethics Committees approved this study. All participants gave written informed consent before data collection began.

**Competing interests:** None declared.

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**References**


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