Changes in joint range of motion and muscle-tendon unit stiffness after varying amounts of dynamic stretching

Running title: Dose–response effects after dynamic stretching

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Abstract

The purpose of this study was to examine the effects of varying amounts of dynamic stretching (DS) on joint range of motion (ROM) and stiffness of the muscle–tendon unit (MTU). Fifteen healthy participants participated in four randomly ordered experimental trials, which involved one (DS1), four (DS4), and seven (DS7) sets of DS, or control conditions/seated at rest (CON). Each DS set consisted of 15 repetitions of an ankle dorsiflexion–plantarflexion movement. The displacement of the muscle–tendon junction (MTJ) was measured using ultrasonography while the ankle was passively dorsiflexed at 1°/s to its maximal dorsiflexion angle. Passive torque was also measured using an isokinetic dynamometer. Ankle ROM was significantly increased after DS4 and DS7 compared with the pre-intervention values ($P < 0.05$), but there were no significant differences in ankle ROM between DS4 and DS7. No differences were observed in ankle ROM after DS1 and CON. In addition, the stiffness of the MTU, passive torque, and displacement of the MTJ at submaximal dorsiflexion angles did not change in any of the experimental conditions. These results indicate that DS4 increased ankle ROM without changing the mechanical properties of the MTU, and that this increase in ankle ROM plateaued after DS4.

Introduction

Stretching is generally performed during warm-up to improve exercise performance and reduce the risk of injury. Traditionally, static stretching (SS) has been practiced during the warm-up before exercise rather than dynamic stretching (DS). However, previous studies have shown that SS impairs muscle strength (Cramer et al., 2005; Evetovich, Nauman, Conley, & Todd, 2003; Fowles, Sale, & MacDougall, 2000; Mizuno, Matsumoto, & Umemura, 2014; Ryan et al., 2008b) and explosive performance (Yamaguchi & Ishii, 2005), although it improves joint range of motion (ROM) (Boyce & Brosky Jr, 2008; Mizuno, Matsumoto, & Umemura, 2013b; Morse, Degens, Seynnes, Maganaris, & Jones, 2008). There is also limited evidence that SS reduces injury risk (McHugh & Cosgrave, 2010). In contrast, DS improves exercise performance parameters such as muscle strength (Sekir, Arabaci, Akova, & Kadagan, 2010), muscle power (Yamaguchi, Ishii, Yamanaka, & Yasuda, 2007), and jump height (Hough, Ross, & Howatson, 2009). Hence, it was recently recommended that DS should be performed during warm-up prior to competition or exercise (Behm & Chaouachi, 2011); however, whether DS improves joint ROM and reduces the risk of injury has not been fully investigated.

Only a limited number of studies have examined the effects of DS on joint

ROM (Bandy, Irion, & Briggler, 1998; Herda et al., 2013; O'Sullivan, Murray, & Sainsbury, 2009; Samukawa, Hattori, Sugama, & Takeda, 2011). Joint ROM depends on both mechanical and neural factors (Magnusson, Simonsen, Aagaard, & Kjaer, 1996b; Mizuno, Matsumoto, & Umemura, 2013a). Joint ROM is mechanically affected by the stiffness of the muscle–tendon unit (MTU), which is related to exercise performance and the risk of MTU strain injuries (Cross & Worrell, 1999; Mizuno et al., 2014). Neural factors influence joint ROM through stretch tolerance, or the pain tolerance threshold (Magnusson et al., 1996a). However, although DS has been reported to increase joint ROM, there is a dissociation between joint ROM and the mechanical or neural factors affecting it; thus, changes in these factors are not directly measured by changes in joint ROM (Herda et al., 2013; Samukawa et al., 2011). Thus, it is not clear whether DS affects either mechanical or neural factors, or both; however, it has been demonstrated that SS affects both factors (Magnusson, 1998; Mizuno et al., 2013b; Morse et al., 2008). Additionally, as the joint ROM response to varying numbers of repetitions of DS has not been demonstrated, the dose–response relationship between DS and joint ROM is not yet understood.

The purpose of the present study was to examine the effects of varying

repetitions of DS on joint ROM and stiffness of the MTU. This work was conducted to investigate two hypotheses. First, I hypothesised that there would be a positive dose–response relationship between DS and joint ROM, such that more stretching repetitions would elicit greater increases in joint ROM until a plateau was reached. This phenomenon has been previously demonstrated with SS (Boyce & Brosky Jr, 2008). Second, I hypothesised that DS would not affect the stiffness of the MTU, regardless of the number of repetitions of DS performed. Decreased MTU stiffness after SS is related to a “stretching-induced force deficit” (Ce et al., 2015; Cramer et al., 2005; Mizuno et al., 2014; Ryan et al., 2008b); however, the majority of previous studies have reported that DS elicits an increase in exercise performance rather than a force deficit (Hough et al., 2009; Sekir et al., 2010; Yamaguchi et al., 2007). In addition, the stiffness of the MTU has not been shown to be one of the mechanisms of the positive performance effects of DS (Behm & Chaouachi, 2011).

Methods

Participants

Fifteen healthy participants (eight males and seven females) volunteered for the study (mean ± SD, age 23 ± 2 years, height 168.6 ± 7.6 cm, weight 62.7 ± 7.6 kg). No participants reported any history of recent musculoskeletal injuries or neuromuscular diseases specific to the lower limb. All participants were fully informed of the purposes, procedures, and possible risks of the study. Each participant gave their written informed consent for their participation in the experiments, which were conducted according to the principles in the Declaration of Helsinki and approved by the Human Subjects Committee at Chukyo University Graduate School of Health and Sports Sciences (approval number: 2013-18).

Experimental design

The participants visited the laboratory on five occasions, and the visits were separated by more than 24 h. The first visit involved a familiarisation trial, and the subsequent four visits included the following experimental conditions in random order: a) control condition/resting in a seated position (CON); b) one set of 15 repetitions of DS (DS1); c) four sets of 15 repetitions of DS (DS4); and d) seven

sets of 15 repetitions of DS (DS7) of the plantar flexors. During the familiarisation trial, each participant practiced the passive-dorsiflexion test to minimise any potential learning effects and to adjust to the procedures. During the experimental sessions, the participants underwent two pre-intervention passive-dorsiflexion tests, an intervention (DS or resting in a seated position) and a post-intervention passive-dorsiflexion test. During the passive-dorsiflexion test, I measured passive torque (i.e., involuntary resistive torque against passive dorsiflexion) and displacement of the muscle–tendon junction (MTJ) at different joint angles, the ROM of the ankle joint, and the electromyographic (EMG) activities of the medial head of the gastrocnemius (MG) and tibialis anterior (TA) muscles. A post-intervention passive-dorsiflexion test was performed as soon as possible after DS.

Dynamic stretching

Repeated DS was performed using the isokinetic dynamometer (Biodex System3, Biodex, NY, USA) in the same posture as for the passive-dorsiflexion test. DS was administered to the right lower leg of each participant. The participant's leg was secured to the isokinetic machine with the knee in full extension. The footplate of the isokinetic machine was fixed securely to the right foot of each participant and

the participants were then instructed to perform active dorsiflexion and passive plantarflexion of their right ankle at a rhythm of 60 beats per minute, set by a metronome. Participants were instructed to perform dorsiflexion in as wide a range as possible. Participants performed isokinetic voluntary dorsiflexion contractions at a speed of 300°/s controlled by a dynamometer; when they performed plantarflexion of their ankle joint, they were asked to completely relax and not offer any voluntary contraction. In addition, to ensure the voluntary contraction during dorsiflexion and passive movement during plantarflexion, an investigator monitored EMG tracings during DS. This dorsiflexion-plantarflexion movement was continued for 30 seconds (15 repetitions) per set. Sets were repeated during each stretching session as previous described for each experimental condition, with a 20-s rest between sets.

Passive-dorsiflexion test

To determine passive torque, displacement of the MTJ, ankle ROM, and EMG activity, each participant underwent two passive-dorsiflexion tests before the treatment intervention and one passive-dorsiflexion test at the post-intervention assessment. The passive-dorsiflexion test was performed using an approach similar

to that described in previous studies (Mizuno et al., 2013a; Morse et al., 2008). Participants were secured to an isokinetic machine with the right knee in full extension and the footplate fixed to their right foot. The angle of the back of the seat was 110° (90° was defined as perpendicular to the floor). The lateral malleolus was aligned with the axis of the dynamometer. In this study, all reported ankle angles are the angle of the footplate, and the ankle angle was defined as 0° when the footplate was perpendicular to the floor. Values were defined as positive for dorsiflexion. Passive ankle ROM was assessed by passively and isokinetically dorsiflexing the participant's foot at a speed of 1°/s from −30° to the angle at which the participant felt discomfort and stopped the dynamometer by activating a safety trigger. The maximal angle of the footplate was defined as the ankle ROM. During this test, the passive torque generated on the footplate was determined both when the ankle was submaximally dorsiflexed and at the maximal dorsiflexion angle. Throughout the passive-dorsiflexion test, the participants were asked to completely relax, not offer any voluntary resistance, and to wear an eye mask to eliminate any visual input from the immediate environment that might otherwise have provided the participant with a reference point for their joint ROM (Magnusson, Aagard, Simonsen, & Bojsen-Moller, 1998). The value from the trial in which the participant

reached the greatest ankle joint ROM value during the two pre-intervention passive-dorsiflexion tests was used in all subsequent analyses. Passive torque and ankle angle were converted from analogue to digital at a sampling rate of 1.5 kHz (LX-10, TEAC, Tokyo, Japan).

B-mode ultrasonography (LOGIQ P5, GE Healthcare, CT, USA) was used to determine displacement of the MTJ of the MG during the passive-dorsiflexion test. The MTJ was visualised as a longitudinal ultrasonic image using a 4.5-cm, 12.0-MHz linear-array probe (12L probe, GE Healthcare). The probe was secured to the skin using a specially made styrene frame. Displacement of the MTJ was measured as its position relative to a reflective marker placed between the skin and ultrasonic probe as a landmark. Ultrasonic images were recorded on videotape at 30 Hz (SR-VSI30, Victor, Kanagawa, Japan) via digital timer (VTG-33, FOR-A, Tokyo, Japan). Ultrasonic images were synchronised to the passive torque and joint angle output using a trigger switch that simultaneously activated the digital timer and analogue-to-digital converter (Mizuno et al., 2013b). Displacement of the MTJ was manually traced with software developed in-house using Visual C# (Microsoft, WA, USA) and DirectShow (Microsoft).

The submaximal passive torque and submaximal displacement of the MTJ

were determined at every fourth degree during the final 13° (at 1°, 5°, 9° and 13°) that were common to both assessment periods (pre- and post-intervention) (Ryan et al., 2008a). Using these values (i.e., passive torque and ankle ROM at 1°, 5°, 9° and 13° during the common final 13°of ROM), the stiffness of the MTU was calculated as the slope of the second-order polynomial passive torque-ankle angle regression curve (Mizuno et al., 2013b) at each of the four measurement points (1°, 5°, 9°, and 13°) during the final 13° of ROM. Stiffness values of the MTU were also determined at 1°, 5°, 9°, and 13° during the final 13° of ROM. The same absolute degree values that were common to each assessment period (within each experimental condition) were used to calculate the submaximal passive torque, submaximal displacement of the MTJ, and stiffness of the MTU for each participant, although the absolute values of these degrees differed between the experimental and CON conditions for each subject.

Electromyographic evaluation

To ensure that the passive-dorsiflexion test was truly passive, I measured EMG activity using bipolar, 13-mm Ag/AgCl surface electrodes (S&ME; Biolog, Tokyo, Japan) placed on the most prominent bulge of the MG and the TA with a 25-mm

interelectrode distance. EMG activity was recorded at a bandwidth of 5–500 Hz. EMG signals were transmitted to a digital data recorder at a sampling rate of 1.5 kHz. To remove any potential contribution of MG or TA contraction during dorsiflexion, the investigator monitored EMG tracings < 50 μV above baseline during the passive stretch cycles of the passive-dorsiflexion tests (Gajdosik, Vander Linden, & Williams, 1999). In this study, the EMG amplitudes during the passive-dorsiflexion tests were calculated with a root mean square function for MG and TA throughout the initial 10° of dorsiflexion and the final 5° of dorsiflexion, respectively.

Statistical analysis

The MTJ displacement data were available from only 14 of the 15 participants, as the ultrasonographic image of one participant was not recorded; all other analyses were conducted using data from all 15 participants. A 3-way analysis of variance (ANOVA; time [pre or post] × condition [DS1, DS4, DS7, or CON] × angle [1°, 5°, 9°, or 13° during final the 13°]) was used to analyse the submaximal passive torque, submaximal displacement of the MTJ, and stiffness of the MTU. A 3-way ANOVA (time [pre or post] × condition [DS1, DS4, DS7, or CON] × portion [initial 10°, or

final 5°]) was used to analyse the MG and TA EMG amplitudes. Two-way ANOVA (time [pre or post] × condition [DS1, DS4, DS7, or CON]) was used to analyse the ankle ROM, passive torque at the maximal dorsiflexion angle, and displacement of the MTJ at the maximal dorsiflexion angle. When appropriate, follow-up analyses were performed using *t*-tests with Bonferroni corrections. Differences were considered statistically significant if $P \leq 0.05$. All data are reported as means ± SD; however, means ± SEM values are used in the figures. Based on the results of Samukawa et al. (2011) and the following parameters (power = 0.80, alpha = 0.05, ES = 0.76), a minimum of eight participants were needed.

Results

Ankle ROM

A significant two-way interaction between time and condition was detected for ankle ROM ($F_{(3, 42)} = 4.191$, $P = 0.011$, $\eta_p^2 = 0.230$). Post hoc testing revealed a significant post-intervention increase in ankle ROM after DS4 ($P = 0.007$, 95% confidence interval [CI]: 0.765 to 4.106) and DS7 ($P = 0.002$, 95% CI: 1.386 to 4.886). However, no significant differences in ankle ROM were seen after either DS1 ($P = 0.442$, 95% CI: −1.032 to 2.237) or CON ($P = 0.581$, 95% CI: −0.843 to 1.447; Fig. 1).

Passive torque at maximal dorsiflexion angle

No significant two-way interaction between time and condition ($F_{(3, 42)} = 1.807$, $P = 0.161$, $\eta_p^2 = 0.114$) and no significant main effects for time ($F_{(1, 14)} = 1.637$, $P = 0.222$, $\eta_p^2 = 0.105$) or condition ($F_{(3, 42)} = 0.803$, $P = 0.499$, $\eta_p^2 = 0.054$) were detected for passive torque at the maximal dorsiflexion angle (Fig. 2).

Displacement of the muscle–tendon junction at the maximal dorsiflexion angle

A significant two-way interaction between time and condition was detected for MTJ displacement at the maximal dorsiflexion angle ($F_{(3, 39)} = 3.626$, $P = 0.021$, $\eta_p^2 = 0.218$). Post hoc testing revealed that DS increased the displacement of the MTJ at the maximal dorsiflexion angle after DS4 ($P = 0.019$, 95% CI: 0.202 to 1.850) and DS7 ($P = 0.009$, 95% CI: 0.229 to 1.317). However, there were no significant differences in the displacement of the MTJ at the maximal dorsiflexion angle after DS1 ($P = 0.853$, 95% CI: −1.018 to 1.213) and CON ($P = 0.223$, 95% CI: −1.552 to 0.397; Fig. 3).

Passive torque during the final 13° of range of motion

No significant three-way interaction between time, condition, and joint angle ($F_{(2.264, 31.698)} = 0.467$, $P = 0.655$, $\eta_p^2 = 0.032$) and no significant two-way interactions between time and condition ($F_{(1.424, 19.931)} = 0.464$, $P = 0.571$, $\eta_p^2 = 0.032$) or condition and joint angle ($F_{(3.174, 44.430)} = 1.261$, $P = 0.300$, $\eta_p^2 = 0.083$) were detected for passive torque, but a significant two-way interaction between time and joint angle ($F_{(1.798, 25.178)} = 4.743$, $P = 0.021$, $\eta_p^2 = 0.253$) was identified. Post hoc testing revealed that passive torque during the final 13° of ROM increased with increases in the ankle angle both pre- and post-intervention (both $P < 0.05$; Table

1).

Displacement of the muscle–tendon junction during the final 13° of range of motion

No significant three-way interaction between time, condition, and joint angle ($F_{(9, 117)} = 1.125$, $P = 0.351$, $\eta_p^2 = 0.080$) and no significant two-way interactions between time and condition ($F_{(3, 39)} = 0.296$, $P = 0.828$, $\eta_p^2 = 0.022$), condition and joint angle ($F_{(3.270, 42.504)} = 0.525$, $P = 0.682$, $\eta_p^2 = 0.039$), or time and joint angle ($F_{(3, 39)} = 0.156$, $P = 0.925$, $\eta_p^2 = 0.012$) were detected for MTJ displacement. In addition, no significant main effects were detected for time ($F_{(1, 13)} = 0.006$, $P = 0.939$, $\eta_p^2 < 0.001$) or condition ($F_{(3, 39)} = 0.655$, $P = 0.585$, $\eta_p^2 = 0.048$), but there was a significant main effect of joint angle ($F_{(3, 39)} = 175.186$, $P < 0.001$, $\eta_p^2 = 0.931$; Table 2).

Stiffness of the muscle–tendon unit

No significant three-way interaction between time, condition, and joint angle ($F_{(2.429, 34.003)} = 2.284$, $P = 0.108$, $\eta_p^2 = 0.140$) and no significant two-way interactions between time and condition ($F_{(1.572, 22.005)} = 0.140$, $P = 0.821$, $\eta_p^2 = 0.010$) or time

and angle ($F_{(1, 14)} = 1.534$, $P = 0.236$, $\eta_p^2 = 0.099$) were detected for MTU stiffness. However, a significant interaction was seen between condition and joint angle ($F_{(2.625, 36.611)} = 4.115$, $P = 0.016$, $\eta_p^2 = 0.227$). Post hoc testing revealed that the stiffness of the MTU increased with increases in the ankle angle in each experimental condition (all $P < 0.05$; Table 3).

Electromyography of the medial head of the gastrocnemius and the tibialis anterior

No significant three-way interactions between time, condition, and portion were seen for the EMG values from the MG and TA (MG: $F_{(1.518, 19.731)} = 0.950$, $P = 0.380$, $\eta_p^2 = 0.068$, TA: $F_{(1.498, 19.480)} = 1.060$, $P = 0.346$, $\eta_p^2 = 0.075$) and no two-way interactions for time and condition (MG: $F_{(3, 39)} = 0.180$, $P = 0.909$, $\eta_p^2 = 0.014$, TA: $F_{(3, 39)} = 0.644$, $P = 0.591$, $\eta_p^2 = 0.047$), time and portion (MG: $F_{(1, 13)} = 0.743$, $P = 0.404$, $\eta_p^2 = 0.054$, TA: $F_{(1, 13)} = 0.403$, $P = 0.537$, $\eta_p^2 = 0.030$) or condition and portion (MG: $F_{(2.054, 26.706)} = 2.155$, $P = 0.135$, $\eta_p^2 = 0.142$, TA: $F_{(1.643, 21.353)} = 0.258$, $P = 0.732$, $\eta_p^2 = 0.019$) were detected. In addition, no significant main effects were detected for time (MG: $F_{(1, 13)} = 1.106$, $P = 0.312$ $\eta_p^2 = 0.078$, TA: $F_{(1, 13)} = 2.733$, $P = 0.122$, $\eta_p^2 = 0.174$), condition (MG: $F_{(3, 39)} =$

0.814, $P = 0.460$, $\eta_p^2 = 0.059$, TA: $F_{(3, 39)} = 0.738$, $P = 0.536$, $\eta_p^2 = 0.054$), or portion (MG: $F_{(1, 13)} = 1.197$, $P = 0.294$, $\eta_p^2 = 0.084$, TA: $F_{(1, 13)} = 0.141$, $P = 0.713$, $\eta_p^2 = 0.011$) for both the MG and TA.

Discussion

The present study investigated the effects of varying repetitions of DS on joint ROM and MTU stiffness. DS4 and DS7 increased ankle ROM relative to pre-intervention values, whereas ankle ROM did not change after DS1. Additionally, the stiffness of the MTU, passive torque during the final 13° of ROM, and displacement of the MTJ during the final 13° of ROM were unaffected by DS.

This study is the first to describe the dose–response relationship between DS repetitions and joint ROM, although several previous studies have estimated the effect of DS on joint ROM. Samukawa et al. (2011) demonstrated that five sets of 15 repetitions of DS increased ankle ROM. Similarly, Herda et al. (2013) demonstrated that four 30-s sets of 12–15 repetitions of DS increased the ROM of the knee. However, no previous study has demonstrated the dose–response relationship between DS repetitions and joint ROM. The results of the current study revealed that ankle ROM significantly increased after DS4 (from 19.5 ± 7.4° to 22.2 ± 6.8°) and DS7 (from 18.8 ± 6.4° to 21.9 ± 6.3°), although ankle ROM was not changed after DS1 (21.6 ± 7.6° versus 22.2 ± 5.5°). In addition, there was no significant difference between the effects of DS4 and DS7. In summary, the increase in ankle ROM after DS plateaued after four sets. This is similar to findings for SS;

joint ROM increased with an increase in the number of 15-s SS sets until it plateaued at five sets (Boyce & Brosky Jr, 2008). The results of the current study suggest that performing at most four sets of 15 DS repetitions will increase joint ROM.

Regardless of the number of repetitions of DS, mechanical factors such as stiffness of the MTU, passive torque during the final 13° of ROM, and displacement of the MTJ during the final 13° of ROM were not changed by DS in the current study. This contrasts with a previous study that reported a decrease in MTU stiffness after DS (Herda et al., 2013). This contradiction might be due to differences in DS techniques. Various DS techniques have been used in previous studies (Herda et al., 2013; Samukawa et al., 2011; Yamaguchi et al., 2007). One technique involves contracting the muscle group “antagonist” to the target muscle group, as was performed in this study. Another method involves contracting the muscle group “agonist” to the target muscle group. Taylor et al. (Taylor, Brooks, & Ryan, 1997) found that 10 repeated isometric contractions of the rabbit anterior tibialis muscle resulted in decreased passive tension of that muscle. In addition, Kubo et al. (Kubo, Kanehisa, & Fukunaga, 2002) reported that 50 repetitions of 3-s isometric maximum voluntary contractions of the MG decreased its tendon stiffness. Direct

stimulation is considered necessary to change the mechanical properties of the MTU (Taylor et al., 1997; Kubo et al., 2002). Thus, previous studies in which DS involved agonist muscle group contractions demonstrated decreased MTU stiffness (Herda et al., 2013), while in the present study in which DS involved antagonist muscle group contractions the MTU stiffness did not change.

The results of the current study suggest that the increase observed in ankle ROM after DS was due to changes in neural factors, especially increased stretch tolerance. There were no significant mechanical changes after DS, although the passive torque at the maximal dorsiflexion angle tended to increase after DS4 and DS7. This tendency was supported by the results of additional statistical testing; one-way ANOVA revealed a significant percent change in passive torque from pre- to post-intervention at the maximal dorsiflexion angle ($F_{(3, 42)} = 3.391$, $P = 0.027$, $\eta_p^2 = 0.195$). The percent change in passive torque at the maximal dorsiflexion angle was significantly greater after DS4 (13.1 ± 20.2 %) than after DS1 (3.5 ± 18.7 %, $P = 0.047$, 95% CI: 0.146 to 19.056) and CON (−1.2 ± 11.8 %, $P = 0.045$, 95% CI: 0.352 to 28.298), and was greater after DS7 (17.4 ± 28.7 %) than after CON ($P = 0.022$, 95% CI: 3.047 to 34.164). This result indicates that the participants had a greater pain threshold (i.e., increased stretch tolerance) after DS. Several previous

studies have also demonstrated an increase in joint ROM due to increased stretch tolerance without mechanical changes to the MTU (Konrad & Tilp, 2014; Magnusson, 1998; Magnusson, Simonsen, Aagaard, Sorensen, & Kjaer, 1996c). Therefore, the present findings indicate that DS by antagonist muscle group contraction increased ankle ROM as a result of increased stretch tolerance. In addition, as mechanical factors such as stiffness of the MTU, passive torque during the final 13° of ROM, and displacement of the MTJ during the final 13° of ROM were not changed by DS, the increase in displacement of the MTJ at maximal dorsiflexion angle would be due to increased ROM, as a result of increased stretch tolerance. The mechanism underlying the altered stretch tolerance is not fully understood; however, it is possible that nociceptive nerve endings in the joint and muscles, as well as the primary somatosensory cortex, play a role (Antal et al., 2008; Kenshalo & Isensee, 1983; Marchettini, 1993; Peyron, Laurent, & Garcia-Larrea, 2000).

This study had some limitations. The present study did not control for the potential effect of the menstrual cycle stage of the female participants on their muscular function. Furthermore, a previous study reported a sex difference in the passive muscle stiffness during passive dorsiflexion movement (Morse, 2011).

However, the effects of SS on stiffness of the MTU is reportedly not significantly different between male and female participants (Hoge et al., 2010). Therefore, it might be considered that sex differences would not have affected the current results, even though passive muscle stiffness may potentially differ between male and female participants.

In summary, this study found significant increases in ankle ROM after DS4 and DS7 relative to pre-intervention values, but ankle ROM did not significantly increase after DS1 or CON. There were no significant differences between DS4 and DS7 post-intervention values, indicating that the initial increase in ankle ROM after DS plateaued after four sets. In addition, there were no stretching-induced changes in MTU stiffness, passive torque during the final 13° of ROM, and displacement of the MTJ during the final 13° of ROM. Several previous review articles have reported that DS augments subsequent exercise performance (Behm & Chaouachi, 2011; Yamaguchi & Ishii, 2014). Therefore, if the purpose of the warm-up is to improve exercise performance and joint ROM, based on the results of the current study and those of previous studies, I recommend that DS4 should be performed during the warm-up prior to competition or exercise (Hough et al., 2009; Sekir et al., 2010; Yamaguchi et al., 2007). However, if the purpose of the warm-up is to

reduce the risk of injury by decreasing MTU stiffness, then SS should be performed during the warm-up (Cross & Worrell, 1999). Further studies are needed to determine whether the chronic effects of DS on joint ROM and mechanical properties are similar to its acute effects, and whether the dose–response relationship between DS and joint ROM is different for each muscle. An optimal DS protocol to improve joint ROM is also unclear. Future studies that clarify these points will expand the clinical applications of DS.

Conflict of Interest: The author declares no conflicts of interest.

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Figure legends

Figure 1: Stretching-induced changes in ankle range of motion. CON and DS1, DS4, and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively. *Significantly different from pre-intervention ($P < 0.05$). Data are expressed as mean ± SEM.

Figure 2: Stretching-induced changes in passive torque at the maximal dorsiflexion angle. CON and DS1, DS4, and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively. Data are expressed as mean ± SEM.

Figure 3: Stretching-induced changes in the displacement of the muscle–tendon junction at the maximal dorsiflexion angle. CON and DS1, DS4, and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively. *Significantly different from pre-intervention ($P < 0.05$). Data are expressed as mean ± SEM.

Table 1. Stretching-induced changes in passive torque (Nm) during the final 13° of ROM.

	1°	**5°†**	**9°††**	**13°†††**
CON				
Pre	10.7 ± 5.5	13.5 ± 6.4	16.7 ± 8.0	20.8 ± 9.5
Post	10.7 ± 5.2	13.4 ± 6.2	16.5 ± 7.9	20.3 ± 9.4
DS1				
Pre	9.7 ± 3.8	12.1 ± 5.0	15.3 ± 6.1	19.6 ± 8.2
Post	9.5 ± 3.6	11.9 ± 4.7	15.1 ± 6.0	18.9 ± 7.7
DS4				
Pre	9.2 ± 4.0	11.6 ± 5.1	14.5 ± 6.4	18.6 ± 8.3
Post	8.9 ± 3.8	11.2 ± 5.0	14.1 ± 6.4	18.0 ± 8.4
DS7				
Pre	9.3 ± 4.8	11.7 ± 6.2	14.6 ± 7.8	18.1 ± 9.6
Post	8.6 ± 4.7	10.9 ± 5.9	13.7 ± 7.8	17.2 ± 9.5

Values are given as the mean ± SD. There was a significant two-way interaction between time and joint angle. Post hoc testing revealed that passive torque during the final 13° of range of motion increased with increases in ankle angle ($^{\dagger}P < 0.05$ compared with 1°, $^{\dagger\dagger}P < 0.05$ compared with 1° and 5° and $^{\dagger\dagger\dagger}P < 0.05$ compared with 1°, 5° and 9° both pre- and post-intervention). CON and DS1, DS4, and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively.

Table 2. Stretching-induced changes in displacement of the muscle-tendon junction (mm) during the final 13° of range of motion.

	1°	5°†	9°††	13°†††
CON				
Pre	0.0 ± 0.0	1.7 ± 0.8	3.2 ± 1.4	4.9 ± 2.0
Post	0.0 ± 0.0	1.7 ± 1.0	3.3 ± 1.4	4.5 ± 2.0
DS1				
Pre	0.0 ± 0.0	1.9 ± 0.7	3.3 ± 1.0	4.8 ± 1.3
Post	0.0 ± 0.0	1.8 ± 0.6	3.4 ± 0.8	4.7 ± 1.1
DS4				
Pre	0.0 ± 0.0	2.0 ± 0.8	3.8 ± 1.2	5.0 ± 1.7
Post	0.0 ± 0.0	2.0 ± 0.9	3.6 ± 1.5	5.3 ± 2.4
DS7				
Pre	0.0 ± 0.0	1.5 ± 0.8	3.4 ± 1.2	4.6 ± 1.4
Post	0.0 ± 0.0	1.8 ± 0.8	3.4 ± 1.2	4.7 ± 1.6

Values are given as the mean ± SD. A significant main effect was seen for joint angle ($^{\dagger}P < 0.05$ compared with 1°, $^{\dagger\dagger}P<0.05$ compared with 1° and 5°, $^{\dagger\dagger\dagger}P < 0.05$ compared with 1°, 5° and 9°). CON and DS1, DS4, and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively.

Table 3. Stretching-induced changes in the stiffness of the muscle–tendon unit (Nm/ °)

	1°	5°†	9°††	13°†††
CON				
Pre	0.6 ± 0.3	0.8 ± 0.4	0.9 ± 0.5	1.1 ± 0.6
Post	0.6 ± 0.3	0.7 ± 0.4	0.9 ± 0.4	1.0 ± 0.5
DS1				
Pre	0.5 ± 0.2	0.7 ± 0.3	0.9 ± 0.4	1.2 ± 0.5
Post	0.5 ± 0.2	0.7 ± 0.3	0.9 ± 0.4	1.1 ± 0.5
DS4				
Pre	0.5 ± 0.3	0.7 ± 0.3	0.9 ± 0.4	1.1 ± 0.6
Post	0.5 ± 0.3	0.7 ± 0.3	0.8 ± 0.5	1.0 ± 0.6
DS7				
Pre	0.5 ± 0.3	0.7 ± 0.4	0.8 ± 0.5	0.9 ± 0.5
Post	0.5 ± 0.4	0.6 ± 0.4	0.8 ± 0.5	0.9 ± 0.6

Values are given as the mean ± SD. There was a significant interaction between condition and angle. Post hoc testing revealed that the stiffness of the muscle–tendon unit increased with increases in ankle angle ($^{\dagger}P < 0.05$ compared with 1°, $^{\dagger\dagger}P < 0.05$ compared with 1° and 5° and $^{\dagger\dagger\dagger}P < 0.05$ compared with 1°, 5° and 9° both pre- and post-intervention). CON and DS1, DS4 and DS7 represent control and 1, 4, and 7 sets of 15 repetitions of dynamic stretching, respectively.