

The effect of passive stretching on delayed onset muscle soreness, and other detrimental effects following eccentric exercise

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The aim of this study was to measure if passive stretching would influence delayed onset muscle soreness (DOMS), dynamic muscle strength, plasma creatine kinase concentration (CK) and the ratio of phosphocreatine to inorganic phosphate (PCr/Pi) following eccentric exercise. Seven healthy untrained women, 28–46 years old, performed eccentric exercise with the right m. quadriceps in an isokinetic dynamometer (Biodex, angle velocity: $60^\circ \cdot s^{-1}$) until exhaustion, in two different experiments, with an interval of 13–23 months. In both experiments the PCr/Pi ratio, dynamic muscle strength, CK and muscle pain were measured before the eccentric exercise (day 0) and the following 7 d. In the second experiment daily passive stretching (3 times of 30 s duration, with a pause of 30 s in between) of m. quadriceps was included in the protocol. The stretching was performed before and immediately after the eccentric exercise at day 0, and before measurements of the dependent variables daily for the following 7 d. The eccentric exercise alone led to significant decreases in PCr/Pi ratio ($P < 0.001$) and muscle strength ($P < 0.001$), and an increase in CK concentration ($P < 0.01$). All subjects reported pain in the right m. quadriceps with a peak 48 h after exercise. There was no difference in the reported variables between experiments one and two. It is concluded that passive stretching did not have any significant influence on increased plasma-CK, muscle pain, muscle strength and the PCr/Pi ratio, indicating that passive stretching after eccentric exercise cannot prevent secondary pathological alterations.

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Athletes and other physically active subjects are recommended to stretch their muscles in relation to strenuous and unaccustomed exercise in order to prevent delayed onset muscle soreness (DOMS) (1, 2). Scientific documentation for this is, however, poorly elucidated (3). Several studies have investigated the effects of stretching in relation to DOMS. De Vries (4–6) demonstrated a decrease in pain following static stretching, but later studies have been unable to reproduce the pain reduction following stretching. Abraham (7) found a small pain reduction lasting only 1–2 min following stretching, but McGlynn et al. (8) found no pain reduction. Two other studies (9, 10), specifically looking at stretching as a prevention for DOMS, could not find any difference in perceived pain between the intervention group and the control

group. Recently, Rodenburg et al. (11) tested a combination of warm-up before eccentric exercise and stretching and massage after but found only scant and inconsistent results, and no clear conclusion could be drawn.

DOMS is assumed to be initiated by a mechanical disruption of the muscle fibre at the cellular level (12, 13), and a reduction in maximal muscle strength, an increasing concentration of plasma-CK and a decreasing PCr/Pi ratio (measured with ³¹P-magnetic resonance spectroscopy) seem to be indirect signs of mechanical disruptions in relation to DOMS (14–17). Studies to investigate any influence of stretching on these parameters together are completely lacking. Thus, the aim of this study was to measure if passive stretching of the quadriceps muscle would signifi-

cantly influence DOMS, maximal muscle strength, concentration of plasma-CK and the PCr/Pi ratio.

Experimental design

Two identical experiments (exp. I and II), except for passive stretching of m. quadriceps in the second experiment (exp. II), were performed with an interval of 18 months (range: 13–23 months) to avoid possible longterm effects of exhausting eccentric exercise (17). Measurements were obtained before (control) and daily for 7 d after the subjects performed the eccentric exercise. All measurements were performed bilaterally.

Subjects

Seven healthy female volunteers, aged 28–46 years, participated. The subjects also participated in the method-study (17). The time interval between the experiments in the method study and this study was from 6 to 7 months. They all had a medium level of fitness with $\text{VO}_{2\text{max}}$ between 33.6 and 46.4 ml $(\text{kg} \cdot \text{min})^{-1}$ and a mean weekly physical activity level of 4 h (2.1–7.6 h). None of the subjects had performed any specific exercise regime for m. quadriceps for at least 3 months before the test. They were asked to maintain the same level of activity during the study period and not to begin any new recreational or training programme.

All subjects registered their physical activity weekly as long as they participated in the study. Informed consent was obtained in accordance with the scientific ethics committee of the county of Copenhagen.

Eccentric exercise

The muscle damage was induced (day zero) by a bout of lengthening contractions until exhaustion, with the

right m. quadriceps (except subject no. 1) in a isokinetic dynamometer (angle velocity $60^\circ \cdot \text{s}^{-1}$, Biodex, system 2). The exercise was performed with 60% of maximum eccentric peak torque in each contraction (measured in each experiment) and a pause between contractions of 1.4 s (18). The subject was defined exhausted when she was no longer able to perform 60% of her maximum eccentric strength in five consecutive contractions. The range of motion was from 20° to 90° flexion in the knee. This range of motion might result in a 25% stretch of the muscle fibre length (L_f : muscle fibre length) (12), and a contraction velocity of $1.04 L_f \cdot \text{s}^{-1}$ (19) assuming fibre length of the vastus lateralis to be 65.7 mm (20) and the radius for the knee flexion to be 65 mm. Eccentric peak torque force, total work, exercise duration and number of repetitions were registered.

Stretching procedure

The subject was placed in the prone position on a bench. The right knee was then passively (by the same experimenter) flexed until the subject felt it uncomfortable and/or tight or the experimenter perceived resistance to stretch. This position was kept and/or flexed further as much as the subject allowed for 30 s (21). The stretching was repeated two more times with a rest interval of 30–50 s between each stretch (2). M. quadriceps was thus stretched 90 s in each session. The time schedule is presented in Table 1; stretching was performed each day before measurements took place, as well as immediately before and after the eccentric exercise on day 0.

Plasma CK

Blood samples were drawn by venipuncture from the cubital region into a vacutainer. The blood was

Table 1. Time schedule for passive stretching and measurements in exp. II

Time	0 min	about 20 min	about 30 min	about 50 min	about 125 min	about 130 min	about 150 min
Interventions day 0	Blood sample	Muscle pain registration	Muscle strength measurement	MR scanning	Stretching	Eccentric exercise	Stretching
Time average (range)	0 min	14 (1–50) min	28 (15–57) min		43 (24–67) min		123 (95–154) min
Interventions day 1–7	Stretching	Blood sample	Muscle pain registration		Muscle strength measurement		MR scanning

Table 2. The eccentric exercise leading to DOMS, mean (range)

Number of contractions		Total work (N · m)		Time (s)		Peak torque (N · m)	
Exp. I	Exp. II	Exp. I	Exp. II	Exp. I	Exp. II	Exp. I	Exp. II
438 (361–641)	401 (282–641)	36 641 (24 305–47 164)	38 557 (26 199–49 655)	1211 (701–1821)	1173 (749–1853)	201 (180–225)	274 (212–364)

allowed to clot for 30 min and centrifugated. The total plasma creatine kinase (CK) was measured spectrophotometrically at 340 nm and 37°C (IFCC method).

Muscle pain

The pain experienced by the subjects at rest, during walking, and during downstairs walking, was registered on a horizontal Visual Analogue Scale, 10 cm in length, and with no marking on the line. The subjects were only allowed to see where they had put the marks the days before in the same experiment. All tests were performed bilaterally.

Muscle strength

Quadriceps muscle strength was tested as a modified maximum peak torque test on a isokinetic dynamometer (Biodex, system 2). Each test consisted of both an eccentric and a concentric test, for both legs separately (involved=the leg performing eccentric exercise and being stretched, noninvolved=the control leg). The highest of three repetitions at $60^\circ \cdot s^{-1}$ was chosen as the maximum level.

PCr/Pi ratio

All measurements of the ratio of phosphocreatine to inorganic phosphate (PCr/Pi ratio) were performed with ^{31}P magnetic resonance spectroscopy in a 1.5 T whole-body magnet (Siemens Medical Systems Inc., Erlangen, Germany). An elliptic 40 · 80 mm one-turn surface coil and a 90° radio frequency pulse were used for excitation, giving a half ellipsoid-shaped sensitive volume (with major/minor/depth axis=80/40/20 mm) of approximately 33 ml (22).

Positioning of the surface coil was governed by a 40 · 80 mm elliptical mark on the thigh. The centre of the mark was placed at an equal distance from the proximal part of the patella to the spina iliaca anterior superior, on the vastus lateralis. Optimization of the static magnetic field was done using the proton signal.

All NMR spectra were obtained using an average of 64 acquisitions with 7 s repetition time giving a high signal-to-noise ratio spectrum, and were processed using the VARPRO fitting routine (23). All tests were performed bilaterally.

Data analysis

To test for a systematic effect over time, a two-way analysis of variance (ANOVA) was applied. The difference between experiments was examined by applying a two-way ANOVA on the difference between the two experiments, testing for time homogeneity. Significance level was $\alpha=0.05$.

Results

The subjects performed on average 340 eccentric contractions (range 244–641), and continued on average 15.7 min (range 11.7–30.4) in exp I (Table 2). No difference in the performed eccentric exercise between experiment I and II was observed, considering number of contractions, total work performed, power and

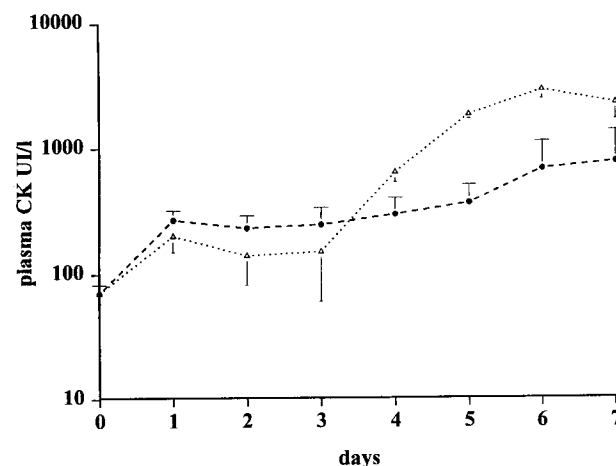


Fig. 1. Changes in plasma creatine kinase following eccentric exercise alone (experiment I ●) and eccentric exercise and stretching combined (experiment II). Values are mean ($\pm$ SEM). All subjects in exp. I and II showed an increasing plasma-CK activity from day 0 to day 1 ($P<0.05$), but there was no significant difference between exp. I and II ($P=0.48$). Due to the great interindividual variation, the creatin kinase values are presented on a logarithmic scale.

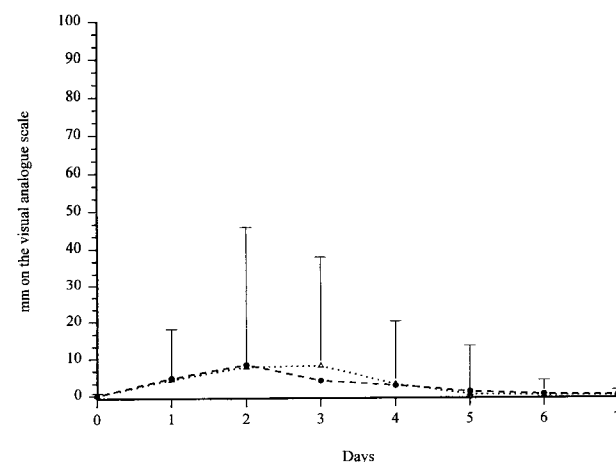


Fig. 2. Subjective muscle pain reported on a visual analogue scale (presented as mm) before and for seven days after eccentric exercise alone (experiment I ●) and eccentric exercise and stretching combined (experiment II Δ). The values are median (range) of the experienced pain during rest, walking and walking downstairs. The maximum pain score was 48 h after exercise, and all subjects reported pain on day 3 in both experiments, thus no difference between eccentric exercise alone (exp. I) and eccentric exercise and stretching combined (exp. II).

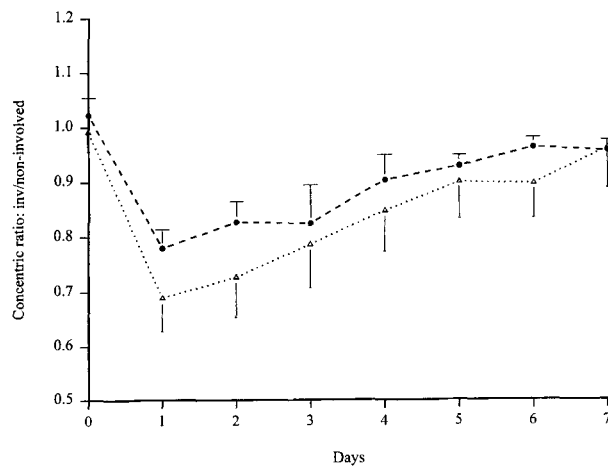


Fig. 3. Changes in concentric maximum muscle strength following eccentric exercise alone (experiment I ●) and eccentric exercise and stretching combined (experiment II Δ). Values are given as the ratio between the concentric muscle strength in the involved and noninvolved thigh presented as mean ($\pm$ SEM). All subjects in exp. I and II showed a decreasing concentric muscle strength with a systematic effect over time ($P<0.001$), but still there was a significant difference between exp. I and II ($P<0.01$), with a greater decrease in concentric muscle strength following exp. II (the experiment which included stretching).

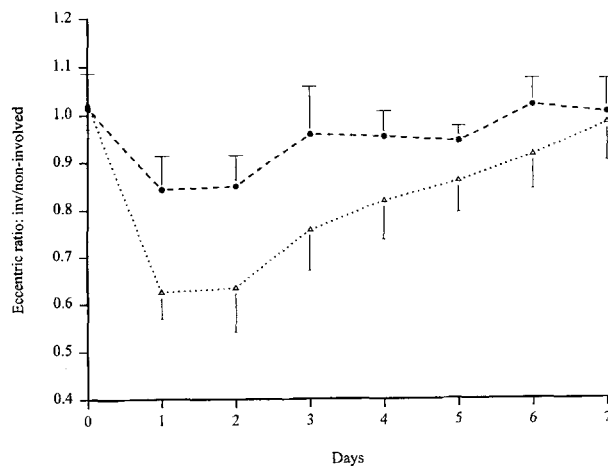


Fig. 4. Changes in eccentric maximum muscle strength following eccentric exercise alone (experiment I ●) and eccentric exercise and stretching combined (experiment II Δ). Values are given as the ratio between the eccentric muscle strength in the involved and noninvolved thigh presented as mean ($\pm$ SEM). All subjects in exp. I and II showed a decreasing eccentric muscle strength with a systematic effect over time ($P<0.001$), but still there was a significant difference between exp. I and II ($P<0.01$), with a greater decrease in eccentric muscle strength following exp. II (the experiment which included stretching).

time. However, a significant increase in eccentric peak torque from experiment I to II from 201 ± 17.5 Nm to 274 ± 49.3 Nm (mean $\pm$ SD) was observed ($P=0.02$).

An increasing concentration of CK following the eccentric exercise was observed in both exp. I and II

($P<0.05$), with no significant difference ($P=0.48$) (Fig. 1).

In both experiments the subjects reported maximal pain on day 2 (48 h after eccentric exercise), and all subjects had pain in the thigh at day 3 (three subjects in exp. I and one in exp. II still experienced pain when registration ceased) (Fig. 2). However, four subjects reported spontaneously less pain immediately following stretching, but this lasted only a few minutes. When muscle pain was registered on average 28 min after the stretching no difference between exp. I and II was observed.

Both concentric and eccentric maximal muscle strength decreased from day 0 to day 1 and showed a significant systematic effect over time ($P<0.001$) and a significant difference between experiment I and II ($P<0.01$) (Fig. 3, 4). The decrease in muscle strength was greater in experiment II (the experiment which included stretching).

The PCr/P_i ratio showed a decrease from day 0 to day 1 and a systematic effect over time for both experiments ($P<0.001$), with no significant difference between experiment I and II (Fig. 5).

Discussion

This study showed no difference in the plasma-CK, DOMS, muscle strength, or PCr/P_i ratio response following either eccentric exercise alone or eccentric exercise combined with stretching. Recently it has been shown (17) that two bouts of eccentric exercise leading to DOMS, and separated by 7–16 months, lead

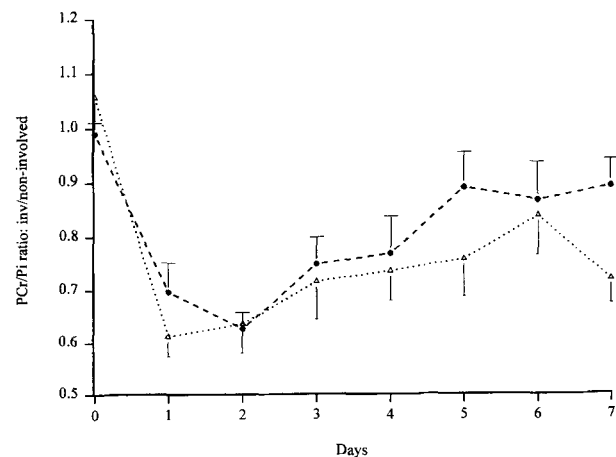


Fig. 5. Changes in the ratio of creatine phosphate to inorganic phosphate (PCr/P_i) measured on the ^3P -NMR spectroscopy, following eccentric exercise alone (experiment I ●) and eccentric exercise and stretching combined (experiment II Δ). Values are given as the ratio between the PCr/P_i ratio in the involved and noninvolved thigh presented as mean ($\pm$ SEM). All subjects in exp. I and II showed a decreasing PCr/P_i ratio with a systematic effect over time ($P<0.001$), but no difference between exp. I and II.

to identical responses for DOMS, muscle strength, plasma-CK concentration and PCr/Pi ratio. If stretching would have any effect on the four parameters, it would thus be expected that we would detect this difference in this experiment.

Eccentric exercise leading to DOMS is often characterized with streaming or smearing of the Z-band immediately following exercise and an increasing number of Z-bands affected in the following two or three days (24, 25). Recently it was proposed that the eccentric exercise leading to DOMS might be initiated by a disruption of some of the intermediate filaments (e.g. desmin, titin) (13, 26). Titin is likely involved as a "protein ruler" to regulate the assembly of myosin and actin precisely, and as the elastic component when a passive muscle is stretched (27). It is located between the M-line and the Z-band. If a resting muscle is stretched passively it would thus affect the Z-bands, by analogy to that which is seen during eccentric exercise. It could thus be argued that both stretching and eccentric exercise leading to DOMS affect the muscle tissue in almost the same way, and it seems therefore irrelevant to stretch the muscle in order to avoid DOMS. Eccentric exercise is part of normal daily activity, and it could be questioned whether it should be looked upon as harmful to the muscle. It has been proposed that eccentric exercise and the ultrastructural alterations in relation to DOMS might be an important initiator of the myofibrillogenesis (28). If stretching is affecting the same part of the muscle structure as eccentric exercise, it could be suggested that stretching might have the same effect as eccentric exercise on the muscle structure. This is supported by the fact that the subjects showed a greater decrease in muscle strength following both eccentric exercise and stretching (experiment II).

Some of the subjects in this study spontaneously reported that immediately following the stretch-procedure they felt a pain relief, which however lasted only a few minutes. This might explain the anecdotal claim that stretching reduces DOMS. During the period with DOMS it could be proposed that the oedema evolved in the sore muscle (29, 30) could be squeezed away by stretching and thus decrease the pain experience. In support of this hypothesis, some studies have indicated a close relation between inflammation, DOMS and oedema (31–34). However, if DOMS aggravates in relation to pressure on the muscle or in relation to muscle contraction, it seems unlikely that stretching, which means decreasing the space in the muscle, should lead to less pain. On the other hand, all subjects in this study experienced severe pain during the stretching, and in one-third of the stretch sessions there was more than 10° increase in range of motion (ROM) from first stretch to second or third, maybe indicating squeezing of water out

of the muscle. The fact that the pain returned within a few minutes and Abraham's (7) findings of pain relief only 1–2 min after stretch, seem also to support the "oedema model".

The eccentric exercise performed in order to induce DOMS showed a significant higher peak torque in experiment II compared to experiment I. This is of minor importance, however, because an earlier study (17) showed that a similar difference between two bouts of eccentric exercise nevertheless showed an equal response. In addition, the force is of minor importance compared to the muscle strain in order to induce DOMS (12). In this study the strain was the same in both experiments (knee flexion from 20° to 90°).

Some of the subjects' CK responses showed a shift of plasma CK peak from day 3, 4 or later to day 1. This might indicate that stretching reduces some of the pathological alterations following unaccustomed and strenuous eccentric exercise. But, as mentioned, we could not detect any effect of stretching on muscle strength, PCr/Pi ratio and muscle pain. An increased concentration of CK is assumed to indicate sarcolemma disruption (15), but because CK is a very large molecule (80 000 Da) CK should not be able to enter the blood directly due to the supposed small pore radius in the capillaries (40–70 Å (35)), but is thought to be released into the blood stream via the lymph system. This explains the delayed CK concentration peak following eccentric exercise. However, Paaske & Sejrsen (36) found a larger pore radius in the capillaries being 145–160 Å rather than 40–70 Å, which would allow CK to enter the blood stream directly from the interstitial space. Since it could be argued that the CK release is a one-time event (during and right after the eccentric exercise), stretching might help in removing CK from the interstitial space close to the damaged muscle fibres.

In this study we have chosen passive stretching, partly because we intended it to be similar to general practice, and partly because we wanted to be sure that the muscle really had been to the limits of its ROM. It could be argued that 30 s repeated stretch three times for 8 d, (two times on day 0, i.e. 810 s in total) was too short a stretching time to achieve any difference. But Bandy & Irion (21) found no difference between 900 s and 1800 s total stretching time in the increase in ROM, and Gajdosik (37) used 3150 s and achieved the same increase in ROM as that of Bandy & Irion.

The results may indicate that stretching is not that beneficial after strenuous eccentric exercise as the general recommendations seem to indicate. More studies of the physiological background for stretching are needed, especially with respect to the "oedema model", and clinical controlled studies are lacking too.

In conclusion: passive stretching had no influence

on increased plasma-CK, muscle pain, decreased muscle strength and decreased PCr/P_i ratio following eccentric exercise, indicating that passive stretching as it was performed in our experiment could not prevent secondary pathological alterations.

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