Metabolic and Muscle Damage Profiles of Concentric versus Repeated Eccentric Cycling

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ABSTRACT

PEÑAILILLO, L., A. BLAZEVICH, H. NUMAZAWA, and K. NOSAKA. Metabolic and Muscle Damage Profiles of Concentric versus Repeated Eccentric Cycling. Med. Sci. Sports Exerc., Vol. 45, No. 9, pp. 1773–1781, 2013. Purpose: Eccentric cycling is an exercise modality that could elicit multiple health benefits with low metabolic cost, but unaccustomed performance results in significant muscle damage. It is not known whether muscle damage is attenuated when eccentric cycling is repeated; thus, this study compared metabolic and muscle damage responses to concentric (CONC) and two consecutive eccentric (ECC1 and ECC2) cycling bouts. Methods: Ten men (28 ± 8 yr) performed each cycling bout for 30 min at 60% of the maximal concentric power output at 60 rpm, with 2 wk between bouts. HR, oxygen consumption (VO2), blood lactate (BLa), RPE, and muscle activity (EMG) data were collected during cycling. Maximal voluntary isometric knee extensor (MVC) strength, squat (SJ), countermovement jump (CMJ) height, muscle soreness indicators, and plasma creatine kinase (CK) activity were measured before, immediately after, and 1–4 d after exercise. Results: Average HR, VO2, BLa, and RPE were lower (P < 0.05) during ECC1 than CONC, and EMG amplitude was also lower during ECC1 than CONC. Decreases in MVC, CMJ, and SJ and the increase in muscle soreness were greater (P < 0.05) after ECC1 than CONC. Increases in creatine kinase were minimal after all bouts. When comparing ECC1 and ECC2, HR and BLa were lower (P < 0.05) during ECC2 than ECC1, and decreases in MVC, CMJ, and SJ and the increase in muscle soreness were greater (P < 0.05) after ECC1 than ECC2. After ECC2, MVC, CMJ, and SJ did not change and no muscle soreness was developed. Conclusions: Eccentric cycling was less metabolically demanding than concentric cycling, and HR and BLa were further reduced during ECC2. Muscle damage is minimal after ECC2 and should not influence the choice to undertake eccentric cycling training. Key Words: LENGTHENING CONTRACTIONS, DELAYED ONSET MUSCLE SORENESS, RECUMBENT BICYCLE, OXYGEN CONSUMPTION, REPEATED BOUT EFFECT

Eccentric contractions are often performed during activities of daily living such as walking downstairs or sitting down on a chair, as well as in exercises such as downhill running or walking (11), stepping exercise (36), and a variety of resistance exercises (10,19). Eccentric cycling is also an exercise modality in which eccentric contractions predominate, as the knee extensor muscles perform eccentric contractions when resisting against the backward rotational movements of the cranks. Eccentric cycling was first introduced by Abbott et al. (2) in 1952, where two interlinked bicycles were used with one person pedaling forward (i.e., concentric) and the other resisting the backward movements (i.e., eccentric) imposed on their bicycle. In the classic study, Abbott et al. (2) reported that oxygen consumption (VO2) was 41%, 49%, and 66% lower during eccentric cycling performed at 25, 35, and 52 rpm, respectively, when compared with concentric cycling at intensities ranging between 24 and 245 W. These findings were later confirmed by Asmussen (4), Knutten et al. (24), and Bigland-Ritchie and Woods (6). Bigland-Ritchie and Woods (6) also showed that muscle activation was lower during eccentric than concentric cycling. More recently, electric motors have been used to drive the backward rotations of the cranks, against which the person works. Researchers have shown that eccentric cycling requires only 25–30% of the oxygen required for concentric cycling at the same workload (23,40) and that a four to seven times greater workload can be produced in eccentric cycling compared with concentric cycling at an intensity of 65%HRpeak (27) or at the same intensity (i.e., 1 L·min−1) VO2 (28). In addition, several studies have shown that eccentric cycling training produces greater increases in muscle strength and size compared with concentric cycling training (26–28). Therefore, it has been advocated that eccentric cycling might be an ideal exercise to induce muscle mass and strength gains in the elderly and for use by patients with pulmonary or coronary disease, where cardiorespiratory fitness is reduced but that increases in muscle mass and strength are required (41).

One possible negative aspect of eccentric-dominant exercise is the risk of muscle damage, which is characterized by muscle weakness and delayed-onset muscle soreness (DOMS) after
exercise. This is especially prevalent when it is performed for the first time or after a long interval from the previous exercise bout (32). Muscle damage after eccentric exercise is directly evidenced by histological changes such as disruption of contractile and/or noncontractile proteins and plasma membrane (17,29). However, more common markers of muscle damage are increases in muscle proteins in the blood (e.g., creatine kinase [CK]), prolonged loss of muscle function, swelling, and DOMS (39). When eccentric exercise is repeated within several weeks of the initial bout, changes in muscle damage markers are attenuated and recovery is enhanced, and this adaptation is called the repeated bout effect (38).

Several studies have reported muscle damage induced by eccentric cycling exercise. Friden et al. (17) reported that 30 min of eccentric cycling performed at 80%–100% of VO$_{2\text{max}}$ resulted in a 13%–24% decrease in maximal isometric knee extensor strength (MVC) for 3 d after exercise, accompanied by myofibrillar Z band disruption predominantly seen in type II muscle fibers. Klossner et al. (22) reported a 6% decrease in countermovement jump (CMJ) performance 1 d after 15 min of eccentric cycling at 50% of the concentric maximal power output (PO$_{\text{max}}$). In addition, several studies have shown increases in muscle proteins in the blood (5,9,15,18,22,43) and muscle soreness (5,22) after eccentric cycling exercise (15–60 min) at different intensities ranging from 50% to 150% of concentric PO$_{\text{max}}$. To safely apply eccentric cycling training in elderly and/or clinical populations, it is necessary to understand the characteristics of eccentric cycling, including metabolic and muscle damage responses to the initial and secondary eccentric cycling bouts. Friden et al. (16) showed a decreased magnitude of muscle architectural disruption after 8 wk of eccentric cycling training evidenced by well-preserved muscle fibers and nonaffected Z bandwidths after the last session of eccentric cycling training when compared with that after the first eccentric cycling session. These data indicated that prolonged exposures to eccentric cycling might allow for a protective effect to accumulate. However, no previous studies have systematically examined muscle damage profile of eccentric cycling when it is repeated after an initial bout.

The purposes of this study, therefore, were to compare the metabolic costs of concentric cycling to both initial and secondary eccentric cycling bouts and to subsequently compare changes in muscle damage markers after the three cycling bouts. We hypothesized that eccentric cycling would be less metabolically demanding than concentric cycling. Regarding muscle damage, we hypothesized that concentric cycling would not induce muscle damage and also that severe muscle damage would be present after the first eccentric cycling bout, but only minimal muscle damage would be present after the second bout.

**METHODS**

**Participants.** Ten healthy men who had not performed lower limb resistance training regularly in the past 6 months and who reported no history of neurological disorders or orthopedic lower limb injuries completed a written informed consent form and a medical questionnaire before participating in the study. Ethical approval from the Institutional Human Research Ethics Committee was sought before the study. The participants’ mean ± SD age, height, body mass, body mass index, and peak oxygen consumption were 28.4 ± 8.3 yr, 179.0 ± 4.6 cm, 81.6 ± 13.1 kg, 25.5 ± 3.8 kg m$^{-2}$, and 3.1 ± 0.5 L min$^{-1}$, respectively. The sample size was estimated using the data from a previous study (7) in which changes in MVC strength of the knee extensors after isokinetic eccentric exercise (50 maximal isokinetic eccentric contractions of the knee extensors) were compared between the first and the second bouts. On the basis of an α level of 0.05 and a power (1 – β) of 0.8, with a potential 8% difference in the isometric strength between bouts at 1 d postexercise, it was found that 10 subjects would be sufficient.

**Study design.** Participants reported to the laboratory on three occasions each separated by 2 wk, in which they performed one bout of 30 min of concentric cycling (CONC; visit 1) followed by two 30-min eccentric cycling bouts (ECC1 and ECC2; visits 2 and 3). To minimize possible effects of eccentric cycling on concentric cycling (we considered the effects of concentric cycling on eccentric cycling to be minimal), all subjects performed concentric cycling first. Metabolic variables, including HR, oxygen consumption (VO$_2$), blood lactate (BLa), tympanic temperature, and RPE data, were obtained during the 30-min cycling bouts. Also, surface EMG data were recorded from the vastus lateralis (VL) during cycling. In addition, maximal voluntary isometric knee extensor (MVC) strength and squat (SJ) and CMJ height were measured before, immediately after, and 1–4 d after each eccentric cycling bout and 1–2 d after concentric cycling. Plasma CK activity and muscle soreness ratings were measured before and 1–4 d after each eccentric cycling and 1–2 d after concentric cycling. The shorter follow-up after concentric cycling (i.e., 2 d) compared with eccentric cycling (i.e., 4 d) was due to the lack of significant changes in MVC, SJ, and CMJ after concentric cycling found in a pilot study.

**Cycling exercise.** Both the concentric and the eccentric cycling bouts were performed at 60 rpm for 30 min at 60% of maximal concentric power output (PO$_{\text{max}}$) based on the VO$_{2\text{peak}}$ test. Our pilot studies showed that 60% of the concentric PO$_{\text{max}}$ was close to the highest concentric power output that could be maintained for 30 min by our subjects. The VO$_{2\text{peak}}$ test was performed at least 96 h before the concentric cycling and consisted of an incremental test using an electro-magnetically braked recumbent ergometer (Tunturi F30R, Australia). The test started at 50 W for 4 min followed by 25-W increments every minute until volitional exhaustion. Cadence was kept at 60 rpm, and participants received verbal encouragement during the test. Concentric cycling was performed on the same ergometer as that used for the VO$_{2\text{peak}}$ test, and eccentric cycling was performed on a recumbent ergometer with a motor that moved the cranks of the ergometer backward at a selected cadence (Eccentric Trainer, Metitur, Finland). Participants were instructed to resist the
backward movements of the cranks and to maintain a steady level of power output displayed on a screen, in which a line was drawn at the target power output. This required eccentric contractions of mainly the knee extensor muscles. A familiarization period was performed immediately before the first eccentric cycling bout, which consisted of 5 min of cycling at ∼50 W.

**Metabolic parameters.** Metabolic parameters included VO$_2$ measured using a metabolic cart (TrueOne 2400; ParvoMedics, Sandy, UT), HR recorded by a Polar HR monitor (Polar RS800sd; Polar Electro Oy, Kempele, Finland), BLA obtained from finger prick and measured by a Lactate Pro analyzer (Arkray KDK, Kyoto, Japan), and RPE measured using Borg’s 6–20 scale. VO$_2$ and HR were recorded throughout the 30-min cycling bouts, and BLA and RPE measurements were taken at 10, 20, and 29 min of exercise. These time points were chosen because a pilot study showed that steady state was reached within 5 min, and no further changes in the metabolic parameters were found during cycling. Tympanic temperature was measured before and immediately after cycling by a digital thermometer (First Temp, Genius), and the magnitude of change in the temperature from pre- to postexercise was used for further analysis.

**Surface electromyography.** The surface EMG was recorded from VL during cycling using a Bagnoli-8 desktop EMG system (Delsys Inc., Boston, MA) with a bipolar electrode configuration (DE-2.1 SEMG sensor; Delsys Inc.) with a 10-mm interelectrode distance. Skin was shaved and cleansed with alcohol, and the electrodes were placed at two-thirds of the distance from the anterior superior iliac spine to the patella according to SENIAM guidelines. The sampling frequency was set at 2000 Hz, and an offline digital filter was applied with a band-pass filter of 10–450 Hz. EMG was recorded throughout the 30-min cycling bouts, but analysis was made for selected time points because a pilot study data did not show significant changes in root mean square (RMS) sEMG amplitude after 1 min of cycling. Thus, EMG analyses were made for the data at 1–2, 15–16, and 29–30 min of each cycling. The 1-min time point was included because it would provide data in a nonfatigued state. RMS analysis was performed for 10 complete revolutions, and the average of the 10 revolutions was calculated and used as representation of each time point. EMG epochs were determined when EMG amplitudes increased more than 2 SD above the baseline amplitude. The median frequency (MDF) of the power spectrum was obtained by a fast Fourier transform of 1024 points using a Hanning window with 50% overlap using the same epochs as for the RMS analyses (31).

**MVC strength.** MVC strength of the knee extensors of the dominant leg (i.e., kicking leg) was measured at 70° of knee flexion (1) in a custom-made rigid chair with a load cell (Xtran S1W, Applied Measurement, Melbourne, Australia). After the participants performed a warm-up of 5-min cycling on an ergometer (Monark 828E, Vansbro, Sweden) at 1 kp and 60 rpm, they were seated in the chair and performed three submaximal contractions (i.e., 50%, 50%, and 80% of perceived maximum for 3 s each and 30 s of rest between contractions). The participants performed three maximal isometric contractions with a 1-min rest between contractions, and the maximum value was used for further analysis. The participants were instructed to contract as fast and hard as possible, and visual feedback was provided in real time on a computer screen.

**Vertical jump.** After performing the MVC strength test, the participants were assessed for maximal squat jump (SJ) and CMJ height, in this order. For SJ height measurement, participants positioned themselves in a squat position (90° of knee flexion) and were instructed to jump from the position without any countermovement. CMJ height was measured in a jump where they started from a stand position and used countermovement before the upward (concentric) phase to jump as high as they could. Jump height was measured by a jump mat (Jump–MD, TKK 5106, Takei Scientific Instruments, Tokyo, Japan). The highest of three jumps was used for further analysis.

**Muscle soreness.** Thigh muscle soreness was quantified using a 100-mm visual analog scale (VAS), in which 0 indicates no pain and 100 represents the worst pain imaginable (38). The participants were asked to mark the level of perceived pain of the quadriceps femoris muscle on the VAS while sitting and standing from a 42-cm chair three times (3). Pressure pain threshold (PPT) was also assessed at three sites using a digital algometer (Somedic AB, Sweden), including vastus medialis (VM) at 80% of the distance between anterior superior iliac spine (ASIS) and the patella, VL at 50% of the distance between ASIS and the patella, and rectus femoris at 50% of the distance between the ASIS and the patella. The probe of the PPT algometer (1 cm$^2$ stimulation area) was placed perpendicular to the site, and the investigator gradually applied force at a rate of 50 kPa·s$^{-1}$ until the participants reported a pain from each muscle. The average of three measurements was used for further analysis (3).

**Plasma CK activity.** A 35-µL blood sample was taken by a finger prick, and plasma CK activity was measured by a Reflotron (Roche Diagnosis, Germany) using standard procedures.

**Statistical analysis.** A one-way ANOVA was used to compare the average power output performed during each cycling bout. Average HR, VO$_2$, RPE, and BLA during cycling were compared between the concentric cycling (CONC) and the first eccentric cycling (ECC1), CONC, and the second eccentric cycling (ECC2), and ECC1, and ECC2 by a paired t-test. A two-way repeated-measures ANOVA (bout × time) was used to compare changes in MVC, CMJ, and SJ, VAS, and PPT over time between CONC and ECC1 and CONC and ECC2, and between ECC1 and ECC2. A two-way repeated-measures ANOVA was also used to compare changes in RMS and MDF during the 30-min of cycling in the same way to the muscle damage parameters. If a significant bout, time or interaction effect was found, a
Bonferroni post hoc test was used for pairwise comparisons. The significance level was set at $P < 0.05$. All statistical analyses were performed with PASW Statistics 19 software for Mac (SPSS Inc., IBM company, USA). Data are presented as mean ± SEM.

RESULTS

Metabolic parameters. The average cycling power output was 158.5 ± 9.2, 169.9 ± 26.7, and 179.3 ± 6.1 W for CONC, ECC1, and ECC2, respectively, with no significant difference being found between bouts ($P = 0.79$). As shown in Figure 1, HR was 19% lower (126.7 ± 8.5 vs 156.1 ± 5.9 beats min$^{-1}$, $P = 0.002$), BLa was 65% lower (2.7 ± 0.5 vs 7.6 ± 0.8 mmol L$^{-1}$, $P = 0.000$), VO$_2$ was 50% lower (1.2 ± 0.1 vs 2.3 ± 0.1 L min$^{-1}$, $P = 0.000$), and RPE was 22% lower (10.6 ± 2.9 vs 13.6 ± 2.4, $P = 0.0003$) in ECC1 when compared with CONC. HR, BLa, VO$_2$, and RPE during ECC2 were also lower than those during CONC (29%, 77%, 51%, and 23%, respectively, $P < 0.05$). In addition, a 12% lower HR (111.4 ± 8.5 vs 126.7 ± 8.5 beats min$^{-1}$, $P = 0.003$) and 35% lower BLa (1.7 ± 0.4 vs 2.7 ± 0.5 mmol L$^{-1}$, $P = 0.002$) were seen in response to ECC2 when compared with ECC1. A significant increase in tympanic temperature from pre- to postexercise (0.45 ± 0.16°C) was found only after CONC.

Surface electromyography. A significant main effect for bout was found for RMS EMG amplitude between CONC and ECC1 ($P = 0.01$), CONC and ECC2 ($P = 0.00$), and ECC1 and ECC2 ($P = 0.016$). Although there was no interaction effect, pairwise comparisons revealed that RMS EMG amplitude was greater ($P = 0.01$) during CONC than ECC1 and ECC2 at all time points (1, 15, and 29 min), and EMG amplitude was lower ($P = 0.02$) during ECC2 than ECC1 at 15 min (Fig. 2A). Significant interaction effects were found when MDF was compared between CONC and ECC1 ($P = 0.002$) and between CONC and ECC2 ($P = 0.012$) without a main effect for bout. Pairwise comparisons revealed that MDF was greater ($P = 0.04$) during ECC1 compared with CONC only at 1 min (Fig. 2B), and significant increases in MDF over time were observed only for CONC.

MVC strength. No significant difference in the baseline MVC strength was evident between CONC (282.3 ± 14.7 N m), ECC1 (268.4 ± 15.5 N m), and ECC2 (282.1 ± 13.1 N m). Nonetheless, significant bout-by-time interaction effects were found between CONC and ECC1 ($P = 0.04$) and between ECC1 and ECC2 ($P = 0.044$). Also, significant main effects for bout were found between CONC and ECC1 ($P = 0.045$) and between ECC1 and ECC2 ($P = 0.001$) for the changes in MVC strength (Fig. 3A). MVC strength was lower ($P = 0.005$) immediately after and 1–2 d after ECC1 compared with CONC and ECC2 and was lower ($P = 0.001$) at 1–4 d after ECC1 than ECC2. MVC strength returned to baseline by 1 d after CONC and ECC2 but remained below the baseline until 4 d after ECC1. An increase in MVC strength (6.5% ± 2.7%; $P = 0.04$) was seen at 4 d post-ECC2.

Vertical jump. No significant differences in SJ (mean ± SD; 41.3 ± 5.2 cm) or CMJ height (45.4 ± 4.4 cm) were evident before exercise between CONC, ECC1, and ECC2. However, significant bout effects were found between CONC and ECC1 ($P = 0.045$) and ECC1 and ECC2 ($P = 0.037$), with an interaction effect between ECC1 and ECC2 ($P = 0.015$). Significant decreases in CMJ height (7%–12%) from baseline...
were seen at 1–3 d after ECC1 only (Fig. 3B). CMJ height was lower \((P = 0.045)\) 1–2 d post-ECC1 compared with CONC, and it was lower \((P = 0.037)\) than ECC2 at 2–3 d. Similarly, significant bout effects were found between CONC and ECC1 \((P = 0.046)\), between ECC1 and ECC2 \((P = 0.045)\), with significant interaction effects between CONC and ECC1 \((P = 0.025)\), and between ECC1 and ECC2 \((P = 0.0016)\) for SJ. Pairwise comparison revealed that SJ height decreased significantly only after ECC1 and was 17%–22% lower \((P = 0.046)\) immediately and 1 d postexercise for ECC1 than CONC (Fig. 3C). When comparing between ECC1 and ECC2, SJ height was 12%–14% lower \((P = 0.045)\) at 1–2 d after ECC1.

**Muscle soreness.** Significant bout and interaction effects were evident between CONC and ECC1 \((P = 0.001\) and \(P = 0.001\), respectively) and between ECC1 and ECC2 \((P = 0.003\) and \(P < 0.001\), respectively) for muscle soreness assessed by VAS. There was significant muscle soreness 1–2 d after ECC1 compared with CONC, and 1–4 d when compared with ECC2, but no increase in muscle soreness was found after CONC (Fig. 4A). PPT results were similar for VM, VL, and RF, and Figure 4B shows data for VM. The baseline PPT was 839.7 ± 82.5 kPa, and no significant difference was seen between CONC, ECC1, and ECC2. Significant main bout and interaction effects \((P = 0.006\) and \(P = 0.005\), respectively) were found when CONC and ECC1 were compared, and an interaction effect was found between CONC and ECC2 \((P = 0.05)\).

Significant decreases in PPT were observed 1–3 d after ECC1, 1–2 d after ECC2 and 1 d after CON.

**Plasma CK activity.** Small but significant increases in plasma CK activity were evident 1 d after CONC \((219.5 ± 40.3 \text{ IU·L}^{-1})\) and ECC1 \((246.5 ± 33.0 \text{ IU·L}^{-1})\), but not after ECC2 \((173.1 ± 16.0 \text{ IU·L}^{-1})\) from the baseline \((147.4 ± 21.1 \text{ IU·L}^{-1})\). No significant difference in the change was seen between the bouts.

**DISCUSSION**

Oxygen consumption during eccentric cycling was approximately 50% of that during concentric cycling for the same workload \((~165 \text{ W})\), confirming the findings of previous studies and highlighting the fact that eccentric cycling is less metabolically demanding than concentric.
centric cycling (23,40). A new finding of the present study, how-

ever, was that HR and blood lactate responses were further
reduced by 10%–12%, when eccentric cycling was re-
peated. This suggests a further reduction in metabolic stress
during the repeat exercise bout. Regarding muscle damage,
eccentric cycling resulted in loss of muscle function (i.e.,
MVC strength and vertical jump) and noticeable DOMS.
However, almost no symptoms of muscle damage were
observed when it was performed 2 wk later. These results
support our hypothesis that muscle damage would be
minimal after the second eccentric cycling bout.

Metabolic profile. Previous researchers have reported
that eccentric cycling requires only 25%–30% of the oxygen
(VO₂) required for concentric cycling at the same workload
(6,23,40), that HR during eccentric cycling is approximately
two thirds of that during concentric cycling at maximal in-
tensity (13), and that eccentric cycling does not promote an
noticeable increase in BLa (23). However, as shown in
Figure 1, we found that VO₂ during eccentric cycling was
50% of that measured during concentric cycling, HR during
eccentric cycling was 81% of that in concentric cycling, and
a small increase in BLa (2.7 mmol·L⁻¹) was found after the
first eccentric cycling bout. Thus, the metabolic demand of
eccentric cycling found in the present study was greater than
that reported in previous studies. A possible explanation is
that the magnitude of the difference in the metabolic cost
between eccentric and concentric cycling becomes greater at
higher workloads and that eccentric cycling is relatively
more metabolically efficient at higher loads. The workload
was set at 60% of the concentric POmax (~165 W) in the
present study, and it was close to the limit that could be
maintained by participants for the 30-min concentric cycling
bout. However, this is much lower than that (330–600 W
maintained only for 30-s to 6 min) used in previous studies
(23,40). It is also important to note that the participants in
the present study were unaccustomed to eccentric cycling
and were deliberately given no familiarization, whereas
participants in previous studies were accustomed to it. When
comparing the first and second eccentric cycling bouts, a
12% lower HR was elicited in the second bout compared
with the first, and no significant increase in BLa was
detected in the second bout. Although no significant differ-
ences between the first and the second eccentric cycling
bouts were found for VO₂ and RPE (Fig. 1), it may be that
VO₂ and RPE during eccentric cycling decrease with re-
peated exposures, and the metabolic differences between
eccentric cycling and concentric cycling become greater.

It was found that eccentric cycling did not stimulate
an increase in tympanic temperature during either the first or
second bouts, whereas a significant increase in temperature
was evident after concentric cycling. Nadel et al. (34) found
that internal (i.e., esophageal) temperature was consistently
lower (~0.7°C) during eccentric cycling (~37.6°C) com-
pared with concentric cycling (~38.3°C), but quadriceps
intra muscular and skin temperatures were higher during ec-
centric cycling (40°C and 34°C, respectively) compared

![Figure 4](http://www.acsm-msse.org)

**FIGURE 4**—Changes in muscle soreness responses assessed by a VAS (A) and normalized changes in pressure pain threshold of vastus
medialis (B) from baseline (100%) before (Pre) and 1–4 d after con-
centric (CONC) and the first (ECC1) and second eccentric (ECC2)
cycling bouts. *Significant (P < 0.05) bout-by-time interaction effect.
**Significantly (P < 0.05) different from CONC. **Significantly
(P < 0.05) different from ECC1.

with concentric cycling (38.8°C and 31°C, respectively) at
same intensity (30–40 min cycling). It is interesting that the
substantial muscle work performed during eccentric cycling
did not increase internal temperature in the present study.
Importantly, the lack of response could be beneficial for
cardiac and respiratory patients or elderly people with im-
paired thermoregulatory capacity. Eccentric cycling thus
seems to be a low-risk exercise from a temperature regu-
lation perspective, when compared with concentric cycling.

The lower metabolic cost of eccentric cycling is likely at-
tributable to the lesser muscle activity when compared with
concentric cycling. As shown in Figure 2A, EMG amplitude
was consistently lower for eccentric cycling compared with
concentric cycling. Kellis and Baltzopoulos (21) reported
that VL EMG amplitudes during maximal eccentric iso-
kinetic knee extensor contractions at different velocities
were 11%–52% lower than during concentric contractions,
which may be attributed to a smaller portion of the moto-
near pool being recruited during eccentric contractions
(14). It is possible, therefore, that the 38% (ECC1) and the
53% (ECC2) lower EMG amplitudes obtained during ec-
centric cycling compared with concentric cycling are also
reflective of a reduced motoneuron activation. The lesser
muscle activity during ECC2 compared with ECC1 could
represent an enhanced cycling efficiency (i.e., maintenance
of power output with a lesser muscle activity), which is evidenced
by the decreased HR and BLa in ECC2. LaStayo et al. (25) observed a decreased EMG amplitude during eccentric cycling in subjects after 8 wk of eccentric cycling training compared with an group of subjects unaccustomed to eccentric cycling and speculated that the decrease was related to a lower level of motoneuron activation or to an activation of only a subset of the entire motor unit population within the muscle, or both (25). Interestingly, the EMG MDF was 13% greater during ECC1 than CONC at the beginning of exercise as shown in Figure 2B (i.e., 1 min), which could be taken to indicate that a greater proportion of fast-twitch motor units were activated or that motor units were fired at a higher rate during eccentric cycling (30). McHugh et al. (33) found a greater MDF and lesser VL EMG amplitude during maximal eccentric compared with concentric isokinetic (60°·s⁻¹) contractions of the knee extensors and speculated that a greater proportion of fast-twitch motor units were active during the eccentric contractions (33,35). However, controversy exists as to whether the greater MDF is indicative of a preferential recruitment of fast-twitch motor units during eccentric contractions, and a faster motor unit firing rate has been proposed to explain the difference in mean or MDF between eccentric and concentric contractions (30). Indeed, it has been recently proposed that changes in EMG amplitude and frequency are also affected by adaptations in the muscle fibers themselves (12). Dimitrov et al. (12) suggested that the shift in the EMG frequency content toward lower frequencies and the decreases in EMG amplitude during the second bout of eccentric contractions could be attributed also to changes in the shape of the intracellular action potential (i.e., increase in intracellular action potential duration and in the negative after potentials). They speculated that these were due to the long-lasting elevated resting cytoplasmic Ca²⁺ caused by an increase in membrane permeability after eccentric exercise and the motor unit synchronization after muscle damage induced by eccentric contractions. Therefore, it could be that peripheral (i.e., intracellular action potential shape) in addition to central (i.e., motor unit recruitment) adaptations affect the amplitude and frequency changes observed when eccentric exercise was performed repeated.

Muscle damage profile. Several researchers have reported a significant muscle damage being induced by eccentric cycling (5,9,15,17,22,43). However, we have compared, for the first time, the magnitude of muscle damage between two consecutive eccentric cycling bouts and a concentric cycling bout. The results revealed that 10%–25% decreases in muscle function after the first eccentric cycling bout, which lasted for 2–3 d (Fig. 3), and that moderate DOMS was developed after the cycling (Fig. 4). However, the increase in plasma CK activity in the present study was small after both the first and the second eccentric cycling bouts and was not different from concentric cycling. If a large increase in plasma CK activity indicates muscle fiber necrosis (37), then the small increases in CK activity detected in the present study suggest that myocellular disruption was minimal. We speculate that the decreased muscle function and DOMS after eccentric cycling resulted from damage and inflammation to the muscle extracellular matrix occurring without significant myocellular disruption. Unfortunately, markers of connective tissue damage were not measured in the present study, and this should be examined in the future.

Muscle damage markers after the first eccentric cycling bout were similar in magnitude to those reported after other eccentric exercise modalities. For instance, the magnitude of loss in MVC strength was similar to that seen 1–3 d after 45 min of downhill (−10% slope) running (42) and 1–2 d after 72 submaximal (75%) isokinetic eccentric contractions of the knee extensor muscles (20). However, MVC strength loss after eccentric cycling was smaller when compared with that (30%–40% decrease) measured 1–3 d after 50 maximal isokinetic eccentric contractions (8), and MVC strength in the present study recovered faster than after 30 min of downhill (−15% slope) running (11). Increases in plasma CK activity after eccentric cycling were smaller compared with those found after 30 min of downhill running on a −15% slope (peak CK activity: 462 IU·L⁻¹), possibly due to the greater muscle mass involved in the exercise (11) and the smaller than after 50 knee extensor maximal isokinetic eccentric contractions (2815 IU·L⁻¹) (8), most likely due to the maximal instead of submaximal eccentric contractions. However, muscle soreness levels seemed to be similar to those observed after other eccentric exercise modalities for the knee extensors (8,11,20). Therefore, the magnitude of knee extensor muscle damage induced by eccentric cycling at 60% of concentric PO_max seems similar to other submaximal eccentric exercises.

After the second eccentric cycling bout, however, changes in MVC strength, jump height, and muscle soreness were minimal and were not different from those after concentric cycling. This represents a typical repeated bout effect that is characterized by smaller changes in, and faster recovery of, muscle damage markers after the second eccentric bout compared with the first, which has been reported previously for numerous other eccentric exercises (10,32,38). The repeated bout effect has been speculated to be associated with neural, mechanical, and cellular adaptations (32). Typically, neural adaptations include a change in the motor unit activation pattern for a given muscle force, an increasing activation of slow-twitch fibers, and a decreasing stress on fast-twitch fibers; mechanical adaptations may include changes in tendon and/or muscle connective tissue stiffness or an improved efficiency of muscle–tendon force transmission during the second bout; and cellular adaptations might include a reduced inflammatory response and requirement for remodeling of muscle fibers and the extracellular matrix (32). From the present data, it is not possible to determine the mechanisms underpinning the repeated bout effect; however, an important finding is that muscles seem to adapt to eccentric cycling rapidly to minimize muscle damage in the subsequent bout of eccentric cycling. Thus, the potential for muscle damage after

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eccentric cycling training should not be a major factor influencing the decision to use eccentric cycling in the longer term. Because only young, healthy men were used in the present study, further studies are required to examine the muscle damage profile in response to eccentric cycling in older individuals and clinical populations.

In conclusion, eccentric cycling was less metabolically demanding than concentric cycling, and the metabolic stress of eccentric cycling was further reduced in a second bout performed 2 wk later. In addition, less metabolic stress was induced during eccentric cycling shown by a smaller increase in HR and BLa, a lower RPE, and a lack of change in EMG MDF during cycling compared with concentric cycling. Eccentric cycling resulted in moderate muscle damage when it was performed for the first time, but the second bout of eccentric cycling resulted in little or no sign of muscle damage. Thus, the potential for muscle damage and the subsequent muscle soreness should not be a factor influencing the decision to use eccentric cycling, and eccentric cycling could be an effective and well-tolerated exercise modality for elderly individuals and clinical populations.

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REFERENCES


