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Original article

The impact of a long training run on muscle damage and running economy in runners training for a marathon

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Abstract

The purposes of this study were to evaluate whether there were changes in markers of cardiovascular and muscle damage and then to determine whether running economy (RE) was adversely affected due to those changes in a group of runners training for a marathon following a 26-km moderately paced outdoor long training run (LTR). Fifteen, healthy male participants (age: 35.2 ± 11.1 years; height = 171.2 ± 14.5 cm; body mass = 73.6 ± 11.9 kg; and VO_{2max} = 63.6 ± 12.7 mL/kg/minute) completed the protocol. The LTR consisted of a 26-km run on a marked outdoor course with water stops and heart rate (HR) and rating of perceived exertion checkpoints every 5 km. Muscle damage [serum creatine kinase (CK)], delayed onset muscle soreness (DOMS), cardiorespiratory measures (HR, pulmonary ventilation, respiratory exchange ratio), muscle power, step rate (SR), and RE (oxygen consumption during treadmill running at 3.1, 3.6, and 4.0 m/second) were assessed before and 24, 48, and 72 hours after exercise. A repeated-measures analysis of variance showed that serum CK levels were significantly elevated at 24, 48, and 72 hours (145%, 112%, and 72% increase above baseline, respectively; $p < 0.05$). However, these significant increases in serum CK were not associated with an increase in DOMS. In addition, none of the RE measures or other dependent variables showed changes during the study. It was concluded that the muscle damage caused by a 26-km LTR was not reflected by changes in RE at submaximal speeds. Therefore, an LTR of this duration and intensity can be well tolerated in participants training for a marathon and routine training can be followed in the days after this run with few adverse consequences.

Keywords: Creatine kinase; Delayed onset muscle soreness; Endurance running; Fatigue; Movement economy; Muscle damage

Introduction

It is common for runners focusing on distance events to endure extensive training programs in pursuit of success in distance running. The majority of these programs include regular long-duration training runs (LTRs; 20–45 km) that are often followed closely by higher intensity workout days. The aim of these LTRs is to develop and/or maintain maximum aerobic power. In addition, LTRs are intended to enhance running economy (RE) by training the athlete to run at a pace as efficiently as possible.[1] Essentially, RE is the "aerobic demand" of a pace and is defined as the steady-state oxygen uptake (VO_2) related to that velocity.[1–5] It is likely that after an LTR, athletes exhibit a considerable amount of muscle damage and soreness as well as changes in several determinants of RE.[3] The impact of an LTR on RE has not been previously studied but it may be likely that RE would be adversely affected due to changes in markers of muscle damage.

In recent years a number of studies have described the physiological consequences of a demanding competition or training regimen and the subsequent effect on RE. Kyröläinen et al found that after a marathon, oxygen uptake, ventilation (V_E), and heart rate (HR) increased while the respiratory exchange ratio (RER) decreased. In addition, serum creatine

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kinase (CK) and delayed onset muscle soreness (DOMS) were elevated up to 6 days after the run. Later, in 2003, Braun and Dutto found that RE was compromised during the course of DOMS following downhill running.[6] In 2005, Paschalis et al found that while several RE and muscle damage indicators were changed following eccentric exercise, RE itself was not altered up to 96 hour postexercise.[7] However, none of the aforementioned studies involved training routines that an endurance runner might incorporate leading up to the completion of a marathon.

None of the previously described studies have documented the impact of a distance commonly run during training in the lead up to a marathon and how that might impact muscle damage and RE. Knowing this may help runners training for a marathon or their coaches develop appropriate programs with adequate recovery in an effort to enhance performance. Therefore, it is upon this dearth of evidence that the following questions were derived: (1) are physiological and metabolic changes (i.e., muscle damage, cardiorespiratory) evident following a typical LTR? and (2) if changes do manifest, how long will they endure and will RE be affected in distance runners preparing for a marathon? Thus, the purposes of this investigation were to examine the effect of a typical LTR on physiological and muscle damage parameters and how those changes might impact RE in the days that follow. It was hypothesized that (1) an LTR would elicit muscle damage changes (i.e., increased CK and DOMS values), and (2) an LTR would negatively effect RE during the days immediately following the exercise bout.

Methods

Participants

Fifteen male runners, recruited from local running clubs in the New England area, agreed to participate in this study (Table 1). Participants were experienced endurance-trained runners and triathletes (≥16-year training experience) running on average 56.3 km in at least 4−5 days/week. All participants were training for a marathon and none of the participants had run more than 15 km in one session. Participants were instructed to abstain from strenuous exercise activities before and during data collection. In addition, participants were asked to run no more than 15% of their total weekly mileage during each day of data collection.

Table 1
Participant characteristics (n = 15).

Variable	Mean ± SD
Age (y)	35.2 ± 11.1
Height (cm)	171.2 ± 14.5
Body mass (kg)	73.6 ± 11.9
Body fat (%)	13.0 ± 5.1
VO_{2max} (mL/kg/min)	63.6 ± 12.7
Average (km/wk)	56.3 ± 15.4
Training (y)	16.2 ± 10.1
Long run time (min)	125.5 ± 12.2
Long run pace (min/km)	4.9 ± 0.40
Long run HR (bpm)	156.4 ± 12.2

HR = heart rate; SD = standard deviation.

Procedures

Participants reported to the laboratory a total of five times throughout the study. Trials were conducted on consecutive days at 24-hour intervals with the exception of baseline testing (visit 1). To ensure reliable and representative data, each participant was evaluated at the same time of day wearing the same clothing and footwear. Participants were allowed an initial period of treadmill accommodation before data collection if they were unfamiliar with the treadmill (n = 3).[8] This initial familiarization period was approximately 10 minutes of running at a self-selected speed, and a 0% grade. This was repeated three times with a 5-minute recovery between each 10-minute run to yield a total of 30 minutes of treadmill familiarization. In addition, before any treadmill running, the belt speed was checked for calibration by placing a piece of tape and timing the revolutions for speeds used in this study. The accuracy was above 99% for all trials.

Before beginning data collection, a refractometer was used to measure urine-specific gravity (USG) to ensure that all participants were being tested in a euhydrated state (USG < 1.019 g/mol).[9] It was important that participants be euhydrated to avoid potential changes in RE related to hypohydration brought about primarily by a decrease in body mass.

The first visit was reserved for baseline measures of RE and VO_{2max} testing. Visit 1 also included baseline measures of flexibility (FLEX), muscle power, CK, and anthropometric data. The second visit was used for the 26-km LTR while the remaining visits were reserved for follow-up measurements 24, 48, and 72 hours after the run.

Visit 1: baseline measures

Upon arriving at the Robert Kertzer Exercise Physiology Lab, all participants read and signed a written informed consent in accordance with guidelines of the Institutional Review Board at the University of New Hampshire. Participants' health was assessed via a health history questionnaire. A training questionnaire was also used to determine training state and for how many years each participant had been training. Visit 1 took place 1 week before visit 2. When the appropriate paperwork was completed, there was a short familiarization period of the laboratory equipment and protocol before performing data collection.

Data collection commenced with a 4 mL blood drawn from an antecubital vein into a BD Vacutainer SST (Franklin Lakes, NJ, USA). Blood was centrifuged at 3000g for 10 minutes. The serum layer was removed and stored in Fischer-Brand Cryovials at −4°C. CK levels were analyzed within 72 hours using a commercially available kit (Stanbio Laboratory, Boerne, TX, USA).

Anthropometric measures, including height (cm), body mass (kg), and body composition, were then obtained. Shoes were removed when measuring for height and body mass.

Skinfold measurements were taken at six sites using a Harpenden caliper (Body Care, Ann Arbor, MI, USA): triceps, subscapula, suprailiac, umbilicus, pectoral, and anterior midthigh. The Jackson and Pollock equation was used to determine body fat percentage (% Fat).[10] The same trained technician performed all of the body composition assessments.

Lower back and hamstring FLEX baseline measures were determined using the standard sit-and-reach method. Participants performed light stretching before the assessment and then sat on the floor with feet (nonshod) placed against the flexibility box. Participants then extended their arms and bent their torso toward their feet with knees flat in an effort to push the moveable slide on the box as far forward as possible. The best of three trials was recorded in centimeter.

Muscle power was assessed using a modification of the Sargent vertical jump (VJ) test.[11] Participants warmed up and then stood with their dominant arm against a wall and extended the arm upward along a wall-mounted scale and this height was recorded as the standing reach (cm). Participants chalked their hand and then performed three countermovement jumps and the highest jump height (cm) was recorded. Standing reach was subtracted and this value was used with body mass to calculate VJ power using the Lewis equation.[12]

At last, before the RE/VO_{2max} test, a subjective measure of muscle soreness was recorded using a previously documented scale from 1 to 10 with 1 being "no soreness" and 10 being "extreme soreness."[13] Muscle groups [quadriceps, hamstring, and lower, posterior leg (calf)] were manually palpated over the belly of the muscles for the soreness ratings.

Following a 5-minute warm-up of light running on the treadmill, RE was measured at three speeds: 3.1 m/second (approximately 187.8 m/minute; 7 mph), 3.6 m/second (approximately 214.6 m/minute; 8 mph), 4.0 m/second (approximately 241.4 m/minute; 9 mph). Each RE trial was 5 minutes long followed by a 5-minute recovery period. Step rate (SR) was counted between the 3rd and 4th minute. RE was calculated as the steady state VO_2 during the last minute of each trial.[14] In addition, % of VO_{2max}, minute V_E, RER, HR, and rating of perceived exertion (RPE) using Borg's 6–20 scale were determined.[15] Temperature conditions in the laboratory remained stable during all RE trials (25–27°C). Following the completion of the RE assessment, participants were allowed a 10-minute break preceding the VO_{2max} test.

Maximal oxygen uptake was evaluated using a modification of the Costill–Fox treadmill protocol 1 week before data collection.[16] A metabolic measurement cart (Sensormedics, Vmax, Yorba Linda, CA, USA) was calibrated using known standards of oxygen (O_2) and carbon dioxide (CO_2) according to manufacturer's instructions before each participant was tested. The protocol used a self-selected run velocity based on the participant's personal training pace. Velocity remained constant, while the grade incrementally increased by 2% every minute. The following criteria were used to indicate VO_{2max}: an RER $>$ 1.08, a plateau in VO_2, and an HR within one-standard deviation of the age-based predicted maximal value.[17] Maximum HR was determined from this test.

Visit 2: 26-km LTR

Each participant reported to the laboratory (1 week after visit 1) and a blood and urine sample were provided; he was fitted with a Polar HR monitor (Polar Electro Oy, Kempele, Finland), and was prepared to begin the 26-km LTR. The run followed a marked outdoor course that included water stations at 5-km intervals. Water was allowed *ad libitum* in an attempt to offset the effect of dehydration in addition to allowing for successful completion of the run. A support vehicle equipped with a cellular phone and first-aid kit followed the runners and noted HR and RPE at each water station, as well as providing motivation. Safety precautions regarding the heat–humidity index were established. Pace was submaximal between 60% and 75% of HR_{max}.

Once the run was completed, the participants were instructed to resume normal daily activities, but to abstain from strenuous exercise and the use of nonsteroidal anti-inflammatory drugs. Participants were then instructed to run no more than 15% of their weekly mileage during data collection. They were also asked to remain hydrated by following the recommended guidelines for hydration before returning to the lab 24 hours later for visits 3–5.

Visits 3–5: 24, 48, 72 hours postrun

Following the LTR, participants returned to the laboratory at 24-hour intervals in which all of the previously described tests (except VO_{2max}) were repeated.

Statistical analysis

A 1 $\times$ 4 repeated-measures analysis of variance (ANOVA) was performed to reveal the differences between all measured parameters (RE, cardiorespiratory measures, muscle power, FLEX, muscle soreness, and CK). A Newman–Keuls *post-hoc* analysis was used when significant main effects were noted and statistical significance was set at $p < 0.05$.

Results

The 26-km run was completed in 125.5 $\pm$ 13.7 minutes at an average HR of 153.1 $\pm$ 12.2 bpm. Runners completed the run at an intensity of 79.3% $\pm$ 7.1% of HR_{max}. Fluids were provided *ad libitum* at regular intervals; however the runners lost an average of 1.37 $\pm$ 0.59 kg over the course of the long run.

Muscle damage

The repeated-measures ANOVA revealed that CK was significantly elevated ($p < 0.05$) above the baseline measures 24, 48, and 72 hours post-LTR (Fig. 1). However, no significant changes were noted with regard to muscle soreness ratings at any time-point assessment in the three tested muscle groups (quadriceps, calf, and hamstring).

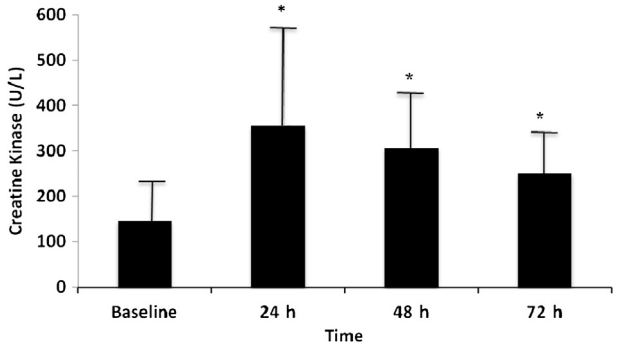

Fig. 1. Change in creatine kinase (CK) levels following a 16-mile training run. *Statistical significance compared with baseline ($p < 0.05$).

RE

No significant changes in RE at any speed or time-point assessment (24, 48, and 72 hours) (Table 2) were observed. In addition, no changes were noted regarding V_E or RER. Further, no significant differences were observed in RPE, SR, FLEX, or VJ.

Discussion

The purposes of this investigation were to examine the effect of a typical LTR on physiological and muscle damage parameters and how those changes might impact RE in the days following. Participants ran a submaximal, 26-km run on a marked road course before undergoing follow-up assessments 24, 48, and 72 hours postrun. Fluctuations in a training regimen, such as abrupt increases in running intensity or duration, have been associated with changes in RE.[1,18] The present study induced a fairly abrupt change in training by adding an LTR that was approximately 70% above the longest run of any previous effort in this group of runners training for their first marathon. Although this was not a typical increase in mileage leading up to a marathon, it was an increase that the authors felt would induce some elevations in muscle damage markers. Indeed, CK, a muscle damage indicator, was significantly elevated at all time points (24, 48, and 72 hours). However, statistical analyses revealed that neither RE (VO_2, % VO_2) nor the variables thought to be related to RE (V_E, RER, HR, SR, and VJ) showed significant changes after the run compared with baseline data at each post time point. The results suggest that a 26-km submaximal LTR did cause some muscle damage but this impairment did not negatively impact RE in the days following the run.

Table 2
Running economy and associated indices at 24, 48, and 72 hours post-long training run.

Indicators	Baseline value[a]	24 h	48 h	72 h
VO_2: 3.1 m/s	35.3 ± 3.1	33.4 ± 6.7	34.6 ± 3.1	33.7 ± 4.8
VO_2: 3.6 m/s	40.6 ± 4.0	39.6 ± 3.1	38.2 ± 3.5	38.3 ± 5.0
VO_2: 4.0 m/s	44.9 ± 3.0	44.8 ± 3.9	43.9 ± 4.8	42.8 ± 5.9
%VO_2: 3.1 m/s	61.8 ± 11.6	55.8 ± 15.2	56.7 ± 11.1	54.8 ± 11.8
%VO_2: 3.6 m/s	69.3 ± 14.5	66.4 ± 16.1	64.5 ± 12.5	63.7 ± 14.0
%VO_2: 4.0 m/s	77.8 ± 13.3	75.8 ± 19.4	76.3 ± 15.9	75.1 ± 16.8
V_E: 3.1 m/s	65.1 ± 13.6	61.3 ± 12.4	63.9 ± 11.2	62.1 ± 12.6
V_E: 3.6 m/s	75.2 ± 17.6	74.6 ± 15.3	74.9 ± 15.4	75.0 ± 16.6
V_E: 4.0 m/s	87.8 ± 20.6	90.0 ± 21.3	87.8 ± 20.0	87.9 ± 20.6
RER: 3.1 m/s	0.87 ± 0.05	0.85 ± 0.03	0.86 ± 0.01	0.86 ± 0.03
RER: 3.6 m/s	0.89 ± 0.06	0.88 ± 0.07	0.89 ± 0.03	0.89 ± 0.02
RER: 4.0 m/s	0.91 ± 0.07	0.91 ± 0.01	0.91 ± 0.03	0.91 ± 0.06
HR_{bpm}: 3.1 m/s	135.5 ± 8.4	132.8 ± 8.7	130 ± 9.1	128.8 ± 10.5
HR_{bpm}: 3.6 m/s	146.8 ± 10.4	143.9 ± 10.6	140.5 ± 10.0	141.1 ± 9.6
HR_{bpm}: 4.0 m/s	162.9 ± 12.4	154.2 ± 9.5	153.6 ± 9.2	152.8 ± 9.1
SR: 3.1 m/s	167.2 ± 5.8	168.4 ± 5.7	169.6 ± 4.8	169.8 ± 7.0
SR: 3.6 m/s	171.4 ± 4.1	171.2 ± 4.3	172.8 ± 4.7	170.1 ± 8.6
SR: 4.0 m/s	175.2 ± 3.3	173.6 ± 5.9	174.3 ± 5.9	175.1 ± 5.4
VJ (cm)	35.8 ± 12.9	34.8 ± 7.7	33.7 ± 6.9	33.9 ± 7.4

%VO_2 = % of VO_{2max}; HR = heart rate (beats/min); RER = respiratory exchange ratio; SR = step rate (steps/min); V_E = ventilation (L/min); VJ = vertical jump (cm); VO_2 = oxygen uptake (mL/kg/min).
[a] Values are expressed as mean ± SD.

Muscle damage and soreness

The first hypothesis regarding muscle damage was supported as the current investigation revealed significantly elevated levels of CK at 24, 48, and 72 hours post-LTR. CK, a marker frequently used to indicate muscle damage, is an enzyme that passes into the lymph system via interstitial fluid; peak values are typically seen anywhere from 2 to 4 days after intense exercise.[19] Kyröläinen et al found that after a marathon-distance run, CK levels peaked 2 days after the event but did not return to baseline levels until 6 days after the run. These findings coincide with our results. Most other documented studies have shown changes in CK levels similar to the changes seen in our study.[3,7,20]

Muscle soreness was assessed on a scale of 1 (no soreness) to 10 (extreme soreness) in an effort to indirectly indicate and assess the severity of muscle damage, and this scale has been used successfully by others.[21,22] Muscle soreness showed no significant changes throughout the period of data collection. Soreness assessment was a subjective measure and therefore it is possible that participants did not have a sufficient accommodation period with the scale. In addition, it is possible that the length and intensity of the run was not sufficient enough to elicit changes in muscle soreness. Several other research studies have examined the relationship between DOMS, the change in CK, and the ensuing effect on submaximal running performance.[2,3,7,20] The previously mentioned studies support the findings of the present study in that muscle damage indicators were elevated; however, the subjective evaluation of muscle soreness varied.

RE

The second hypothesis was that an LTR would negatively affect RE during the days immediately following the exercise bout and this hypothesis was rejected as no changes were noted with regard to RE up to 72 hours post-LTR. Our findings are in agreement with the work of Paschalis et al who induced muscle damage via eccentric exercise using an isokinetic dynamometer.[7] Paschalis et al showed that while muscle

damage indicators (CK levels) were significantly altered following the exercise, RE was unchanged. It was suggested that RE may have been less sensitive in a recreational runner whose gait pattern may not be as well refined as that of a trained runner.[6,7] In the same way, this may have accounted for the large degree of RE variability amongst the runners in the present study because some of the participants were triathletes who incorporated several days of cross-training into their training cycle. However, it is not an uncommon practice to use a combination of triathletes and runners in RE studies.[5,6]

Despite using a higher intensity and shorter duration, an earlier study by Morgan et al also supports the findings of the present study.[23] A 30-minute prolonged maximal run (PMR) at a velocity equivalent to 85–90% of VO_{2max} was used to identify the effect of a PMR on RE and running mechanics. A significant rise in HR and blood lactate was recorded 24, 48, and 72 hours after the PMR; however no changes in RE were reported throughout the testing period. The relationship between fatiguing exercise and running mechanics has been observed only to report little change in mechanics, including ST, related to a decrease in RE. ST is usually most economical when it is self-selected although most elite runners train and compete at an SR between 180 and 200 steps/minute.[24] Our findings suggest that SR was not significantly altered following the submaximal LTR. Previous research that examined SR and mechanics produced equivocal findings.[1,3,7,14,23,25] Morgan et al, Kyröläinen et al, and Paschalis et al concluded that changes in RE could not be attributed to changes in submaximal mechanics.[3,7,23] The results of present study would agree with these findings. However, Johnston et al, Paavolainen et al, and Saunders et al suggested that RE changes might be due to mechanical factors.[1,14,25]

Previous research that has noted changes in RE due to muscle damage by commonly used modalities such as eccentric downhill running or plyometrics or increased exercise duration (i.e., marathon) to induce muscle damage. For example, Braun and Dutto designed a study to observe the specific effects of muscle damage caused by downhill running on RE.[6] It was concluded that RE was compromised during the period of DOMS and that this altered RE may have been due to the changes in mechanics and the general discomfort associated with DOMS. A subjective modification of gait was noted by Braun and Dutto.[6] In addition, it was suggested that the compromised RE may have been associated with the fact that more motor units were being recruited once force generating capacity was reduced, resulting in the concomitant increase in O_2 cost.[6] In an effort to quantify the muscle force–generating capabilities, we used a VJ test; however, no significant changes in VJ were noted. Therefore, despite the increased marker for muscle damage, no significant reductions in muscle power were noted. This may have been partly due to the sensitivity of the VJ as a power test.[26]

Further, it has been postulated that eccentric downhill running chiefly damages type II anaerobic fibers, meaning essentially that the type I aerobic fibers that play a greater role in determining the RE of an individual were not as damaged as they were thought to be. In addition, eccentric downhill running damages muscle fibers in the quadriceps muscle group, whereas it is the muscle fibers in the lower leg that are used to a greater degree in running. It has been suggested that the significant aerobic contribution from the lower leg during RE assessment may actually mask muscle damage in the quadriceps.[7] In contrast, the current investigation used an LTR with rolling hills that a runner might typically train on to ensure that only the muscles directly involved in running were affected and ultimately found that RE was not altered.

Research by Kyröläinen et al looked at the effect of a marathon on RE and kinematics in seven experienced triathletes who had previous marathon racing experience and determined that a worsened RE in the days following a marathon run could be explained by changes in substrate utilization and the regulation of increased thermal demands, increased neural input, as well as the acute effects of muscle damage.[3] The marathon distance used by Kyröläinen et al is a distance rarely performed during training. Our work used a distance more commonly run during endurance training. The discrepancy in results between the two studies was likely due to the extra distance of a marathon run.

Finally, Marcora and Bosio noted that (nonrunning) exercise-induced muscle damage (EIMD) brought on by a series of "35-cm drop-jumps" resulted in significant increases in CK and DOMS levels, but had a nonsignificant effect on RE or physiological responses to submaximal running.[20] However, the EIMD significantly reduced time trial performance 48 hours after the initial treatment and the authors speculated that this was a result of an increase in perceived exertion.[20] Again, our work used a typical training distance that resulted in similar CK and RE results, while the increased DOMS and performance times were most likely the result of different methodologies.

Conclusions

RE remained unchanged after a 26-km LTR in a group of runners training for a marathon. Despite a statistically significant rise in CK levels for 3 days post-LTR, RE, and muscle soreness data remained unaltered. Therefore, it can be argued that a long duration run performed at a submaximal intensity (<85% of HR_{max}) had no significant effect on the aerobic demand of submaximal running during the days immediately following the run.

Conflicts of interest

The authors declare that they have no conflicts of interest related to this research.

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