Neuromuscular Control and Exercise-Related Leg Pain in Triathletes

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ABSTRACT

CHAPMAN, A. R., P. W. HODGES, A. M. BRIGGS, P. J. STAPLEY, and B. VICENZINO. Neuromuscular Control and Exercise-Related Leg Pain in Triathletes. Med. Sci. Sports Exerc., Vol. 42, No. 2, pp. 233–243, 2010. Previous studies have shown that cycling can directly influence neuromuscular control during subsequent running in some highly trained triathletes. A relationship between this altered neuromuscular control of running and musculoskeletal pain and injury has been proposed; however, this link has not been investigated. Purpose: This study aimed to evaluate the influence of cycling on neuromuscular control during subsequent running in highly trained triathletes with and without exercise-related leg pain (ERLP). Methods: Participants were 34 highly trained triathletes: 10 triathletes with a history of ERLP and 24 training-matched control triathletes with no history of ERLP. Knee and ankle kinematics and leg muscle recruitment were compared between a baseline run (no prior exercise) and a transition run (preceded by cycling; i.e., run vs cycle run). Results: Knee and ankle joint kinematics were not different between baseline and transition runs for any triathletes: absolute mean difference (±95% confidence interval) was 1.49°±0.17°. However, muscle recruitment was different between baseline and transition runs, defined by absolute mean difference in EMG amplitude ≥10%, in 5 of 24 control triathletes (11/130 muscles exhibited altered recruitment) and in 5 of 10 triathletes with a history of ERLP (12/50 muscles exhibited altered recruitment). This represents a relative risk of 2.40 (0.89–6.50; P = 0.089) when defined by athletes and 2.62 (1.34–6.01; P < 0.01) when defined by muscles. The magnitude of change in muscle recruitment between baseline and transition runs was not different between control (14.10% ± 2.34%) and ERLP triathletes (16.31% ± 3.64%; P = 0.41). Conclusions: This study demonstrates an association between ERLP in triathletes and their neuromuscular control when running off the bike. Key Words: EMG, THREE-DIMENSIONAL KINEMATICS, TRIATHLON, MOTOR CONTROL, MUSCULOSKELETAL PAIN AND INJURY, TRANSITION

Musculoskeletal injuries occur frequently in triathletes; most recent data suggest the prevalence of lower extremity injury in recreational and highly trained triathletes is 37% to 91%, respectively (20). Exercise-related leg pain (ERLP), which encompasses the clinicopathologic features of several commonly used labels such as shin splints, medial tibial stress syndrome, periositis, stress fractures, tendinopathies, and compartment syndrome (37), is the most common injury in triathletes and causes the most disruption to their training (20,28) (Australian Institute of Sport, unpublished data). Improving our understanding of ERLP is therefore critical to improving the performance of elite triathletes and the participation, and subsequent general health, of recreational triathletes.

As success in triathlon competition depends primarily on triathletes’ ability to run immediately after cycling (run “off the bike”) (36), preparation for competition often involves running after cycling. However, most triathletes, even highly trained triathletes, suffer a decrease in run performance and report a perception of impaired coordination when running after cycling (5,22). Neuromuscular fatigue is likely to occur during training and to contribute to this perceived incoordination and decrease in run performance. However, the effects of cycling on running may also relate to interference with control of movement and muscle recruitment patterns (neuromuscular control), which can occur independent from, or in addition to, any fatigue effect (8,14). A previous study (8) reported that 5/14 of highly trained triathletes despite their considerable training history. This effect of cycling on muscle recruitment during subsequent running occurred independent of any neuromuscular fatigue and seemed to relate to the transfer or “carryover” of cycling muscle recruitment to running. Changes to running muscle recruitment were
evident throughout the stance phase of running. Put simply, it seems that cycling interferes with neuromuscular control during subsequent running in a proportion of highly trained triathletes because patterns of muscle recruitment during running became more like those used during the preceding cycling.

Evidence of the relationship between altered neuromuscular control and musculoskeletal injury (e.g., [18,23]) suggests this interference with neuromuscular control in triathletes may be related to injury. Altered neuromuscular control can have an immediate and a significant effect on tissue loading (e.g., [17,38]). Most instances of ERLP in triathletes are overuse in nature (19) and may be related to increased tissue loading due to altered neuromuscular control during running after cycling. However, the relationship between altered neuromuscular control during running off the bike and ERLP in triathletes has not been investigated. Therefore, the purpose of this study was to evaluate the effect of cycling on neuromuscular control of subsequent running in highly trained triathletes with and without ERLP.

**METHODS**

**Participants**

Participants were 34 highly trained triathletes, which included 10 triathletes (5 males and 5 females) with a history of ERLP (called the ERLP group) and 24 training-matched, control triathletes with no history of ERLP (16 males and 8 females; control group). Participants were included only after clinical diagnosis of ERLP. A history of ERLP was defined as musculoskeletal pain in one or both legs during exercise in the previous 12 months, which was experienced during running on two or more consecutive days and resulted in modification of training for ≥48 h but that was not currently provocative during submaximal running (37). ERLP included, but was not restricted to, pain associated with a medical diagnosis or evidence from bone imaging techniques of tibial osseous stress disorder (i.e., tibial stress fracture) or more diffuse tibial periosteal stress disorder (classically called “shin splints”); ERLP encompasses the clinicopathologic features of several commonly used labels such as shin splints, medial tibial stress syndrome, periositis, stress fractures, tendinopathies, and compartment syndrome (37). Triathletes who reported ERLP that was currently provocative during submaximal running were excluded to prevent confounding experimental effects of acute pain and because the study protocol may have exacerbated the pathologic findings of these triathletes.

Because training history has a significant influence on neuromuscular control of both running and cycling (9–11), triathletes were matched as closely as possible for triathlon training history to optimize sample homogeneity and to prevent confounding effects of training history. Training history was matched using weekly training distance, training intensity, number of training sessions, training duration, and years of previous training as well as longer-term training and competition history (estimates of training load of previous years, number of competitions per year), as described in previous triathlon studies (8–10). These triathletes were aged 29.1 ± 3.6 yr, had been competing in triathlon for 6.2 ± 1.9 yr, and had cycled (mean ± 95% confidence interval (CI)) 411.8 ± 40.2 km and ran 46.8 ± 8.4 km wk⁻¹ in 4.9 ± 0.4 and 5.2 ± 0.5 training sessions per week, respectively, during the previous 3 months. This participant sample was defined as highly trained in accordance with other published works (10,11,25,29). Statistical comparison of training history between control and ERLP groups revealed no difference for cycling training distance, running training distance, cycling training sessions per week, running training sessions per week, and years of competition (ANOVA; group (two levels: control vs ERLP) × training parameter (five levels as listed previously); P > 0.24). Furthermore, all triathletes specialized in Olympic distance, draft legal triathlon, and had experienced Australian national- or international-level competition or had qualified for the Triathlon World Championship representation in the year of testing.

Triathletes with any history of a) competition in sports other than swimming, cycling, or running at a competitive level on two or more occasions per week in the preceding 3 months or b) musculoskeletal or neurologic disorders affecting the spine or the lower limb other than ERLP were excluded to remove possible effects of cross-training or pain and injury not related to ERLP (5,8,14). Participants provided written informed consent. Procedures were approved by the institutional human research ethics committees and conducted in accordance with the Declaration of Helsinki.

**Pain and Disability Measures**

The pain and disability characteristics of triathletes from the ERLP group were collected using established procedures (18). ERLP group triathletes rated their worst pain during running (mean ± 95% CI rating = 6.2 ± 1.7 cm) and their typical pain during running while symptomatic in the preceding 12 months (3.4 ± 1.3 cm) on a 10-cm visual analog scale. The number of days for which training required modification during the past 12 months was also collected (42 ± 21 d).

**Protocol**

We replicated an established experimental protocol (8,14) to investigate the relationship between ERLP history and neuromuscular control during the bike–run transition. This protocol has been shown to be valid and repeatable for investigating the direct influence of cycling on neuromuscular control during running independent of the effects of a) neuromuscular fatigue, b) speed of running, c) variations in movement patterns, and d) changes in neuromuscular control that would occur with continued running irrespective of any prior cycling (14).
This protocol required triathletes to complete a 10-min baseline run and, after 60 min of recovery, 20 min of cycling followed immediately by a 30-min transition run, therefore allowing a comparison of run versus cycle run. A controlled period of 60 s was allowed between the bike and the transition run so as to standardize this transition period yet replicate as closely as possible the demands of training and competition. EMG measures of muscle recruitment and three-dimensional kinematics (i.e., movement patterns) were collected for the duration of the study. RPE were obtained at 1-min intervals during cycling.

Running trials were performed on a treadmill with no incline (Sportech, Australian Capital Territory, Australia) to facilitate control of running speed and continuous measurement of EMG and kinematics. Running speed remained constant and was the same for baseline and transition runs. After the transition from cycling to running, treadmill speed was increased to the required speed during the initial 15 s of the transition run. Running speed was nominated by the triathlete before testing as that speed comfortable for 30 min of constant speed running without causing what these highly trained and highly experienced triathletes’ perceived as fatigue or decline in running form. All participants had considerable previous experience of running on a treadmill and also of pacing their running. Therefore, similar to anchoring of RPE, selection of running speed was anchored to the requirement of 30 min of running (14). Triathletes wore their own footwear, which was checked in each case by the first author, an experienced physiotherapy clinician, to ensure it was in adequate condition for testing. Standardized footwear was not used because this would have created a novel movement condition for some individuals and a confounding experimental effect.

Cycling trials were conducted on the participant’s bike secured on a magnetic trainer (Minoura Mag-500 trainer; Minoura, Anpachi, Japan). All triathletes used clipless pedals. Cycling intensity, cadence, and body position were controlled.

RPE was used to control cycling intensity. Feedback to modify cycling intensity was provided until an RPE of 14 was reached. RPE were obtained from triathletes using a Borg 15-point (from 6 to 20) RPE scale. The reproducibility and the repeatability of this RPE scale have been established previously (e.g., [6,31]). All participants had received training and had extensive experience in the use of RPE to rate cycling and running intensity, and thus their ratings were adequately anchored, ensuring the validity of the ratings (for a detailed discussion, see Chapman et al. [14]). RPE was preferred to the regulation of power output because it provides a reliable measure of exercise intensity during cycling that is not influenced by variations in power profiles between triathletes and allows participants to use their own bicycles without modification (for a detailed discussion, see Chapman et al. [10]).

Cycling cadence has been identified as a variable that may influence subsequent running performance and a strategy of modifying cycling cadence before the bike–run transition is used by many triathletes in competition. However, experimental evidence to support this strategy is contradictory (3,21,35), and the relationship between cycling cadence and neuromuscular control during subsequent running has not been directly investigated. Therefore, we controlled cycling cadence. The triathletes cycled at their individual preferred cadence (IPC) for the first 5 min and final 3 min of the cycle period. The 4 × 3-min conditions of 1) IPC, 2) 55–60 rpm, 3) 75–80 rpm, and 4) 95–100 rpm were randomly ordered from the 6th to the 17th minute of the cycle period to simulate the varying cadences of normal cycling. These cadences were selected as representative typical for this population after collecting data of cadence from highly trained triathletes during training and during the International Triathlon Union World Cup Competition (10,14) (Australian Institute of Sport, unpublished data). Feedback of cadence was provided during controlled cadence conditions, but triathletes were blinded to their cadence during the IPC condition. When a change of cadence was required, participants were instructed to adjust their bicycle gearing to maintain a consistent level of exertion.

Body position was controlled because body position may influence cycling muscle recruitment (16). All triathletes remained in an upright body position, grasping handle bars or brake hoods, for the duration of the bike leg. Each triathlete used a bike that was set up for draft legal Olympic distance competition, which minimized differences in bicycle geometry between triathletes. Bicycle setup was not altered to a standardized setup for all participants because any change in bicycle setup would have created a novel movement condition for the individual and a possible confounding effect. It is noteworthy that although differences did exist between individuals in bicycle geometry, there were no systematic differences in bicycle geometry between groups, and thus bicycle geometry is very unlikely to have created a confounding effect.

**EMG**

EMG recordings were made from the tibialis anterior (TA), peroneus longus (PL), gastrocnemius lateralis (GL), gastrocnemius medialis (GM), and soleus (SOL) muscles. We used Ag/AgCl surface electrodes with circular, pre-gelled contact areas of 10 mm in diameter and a fixed inter-electrode distance of 20 mm (Nicolet Biomedical, Madison, WI). Electrodes were positioned parallel to the direction of muscle fibers (i.e., inclined relative to the vertical where applicable), at the center of the mediolateral dimension of the muscle, and relative to anatomical landmarks in the longitudinal dimension of the muscle, that is, a relative distance between two anatomical landmarks (Table 1); these placement guidelines are accepted in the literature (8,13,14), have been shown to be repeatable (13), and are consistent with recommendations from the literature regarding optimal signal quality and innervation zone location (32).
All electrodes were positioned by the first author to avoid intertester variability.

The skin was prepared by shaving and gentle local abrasion using abrasive paste and alcohol and in accordance with the International Society of Electrophysiology and Kinesiology (ISEK) and the European Surface Electromyography for the Non-Invasive Assessment of Muscles recommendations for skin preparation. The electrodes were fixed using Fixomull extensible dressing (Beiersdorf, New South Wales, Australia), and electrode position was confirmed using voluntary contractions. A ground electrode (3M electrosurgical plate; 3M, St. Paul, MN) was positioned over the anteromedial tibial plateau. EMG electrodes were not replaced between baseline and transition runs to maximize reproducibility of EMG recordings between baseline and transition runs.

EMG data were recorded with a Noraxon Telemyo with input impedance >10 MΩ and >85 dB common mode rejection ratio (Noraxon USA, Inc., Scottsdale, AZ). Double-differential myoelectric signals were amplified (×2000), band-pass filtered between 10 and 500 Hz to remove low-frequency movement artifact and high-frequency noise (first-order high pass with 6 dB per octave slope and sixth-order Butterworth low pass with 36 dB per octave slope), sampled at 3750 Hz using VICON Clinical Manager software (Oxford Metrics Ltd., Oxford, England), digitized with a 16-bit A-D converter (VICON Mezzanine; Oxford Metrics Ltd.), and analyzed with Matlab 6.5 (Mathworks, Natick, MA) in accordance with the established procedures (9,13,14) and the ISEK guidelines.

**Kinematics**

Three-dimensional motion of the legs was measured. Coordinates of 14-mm retroflective markers were sampled at 250 Hz using a VICON 620 eight-camera motion analysis system and a VICON Clinical Manager software (Oxford Metrics Ltd.). An anatomical coordinate system was defined by skin surface markers positioned as previously described in detail (4). All markers were positioned by the first author to avoid intertester variability and secured using both double-sided tape (3M) and Fixomull extensible dressing (Beiersdorf) to minimize movement relative to the skin. Retroflective markers were not removed between baseline and transition runs to maximize reproducibility of kinematic data.

**Data Management and Analysis**

Data management and analysis procedures are outlined in Figure 1, replicated established procedures (9–12), and are in accordance with the ISEK guidelines.

**Data screening.** All EMG and kinematic data were screened by visual inspection and assessed for quality of recording using criteria previously described (13). Data of inadequate quality (i.e., recordings that contained high levels of artifact, which could not be adequately removed with signal filters, may have masked the true EMG recording and thus may have led to a false interpretation of muscle activation or kinematic recordings with marker dropout requiring trajectory interpolation) were excluded from analyses.

**Initial signal processing.** EMG data were adjusted for DC offset, band-pass filtered between 10 and 500 Hz to remove low-frequency movement artifact and high-frequency noise, and full-wave rectified. Retroreflective marker trajectories were filtered using a generalized cross-validation spline algorithm to remove low-frequency movement artifact. Three-dimensional angular kinematics was calculated using the Plug-in-Gait lower limb model (version 1.8; Oxford Metrics Ltd.).

**Event detection.** Individual strides of running were defined using heel-strike events, and individual pedal strokes of cycling were defined using top-dead-center events (the most superior point of the pedal stroke when the crank is aligned with the vertical axis). Heel-strike and top-dead-center events were identified using the vertical and the horizontal displacements and velocity of retroreflective markers placed over the head of the second metatarsal, lateral malleolus, posterior calcaneum, and lateral aspect of the running and cycling shoes and also on the pedal crank of the bike during cycling.

**Signal normalization.** EMG and kinematic data for each stride were time normalized to 101 points (representing intervals from 0% to 100%). The 10 strides closest to each minute interval of the baseline and transition runs (i.e., the 10 strides closest to the 1-min mark, then the 10 strides closest to the 2-min mark, and so forth) were selected for analysis. EMG amplitude was normalized to the maximum measured EMG amplitude during the baseline run (for a detailed discussion of normalization, see Chapman et al. [13]). Kinematic data were normalized to represent joint angular position relative to that of the standing anatomical position.

**Data analysis.** On the basis of previous findings (8), the absolute mean difference (AMD) between baseline and transition runs in waveforms (i.e., time series data) of (i) joint angular position (kinematics) and (ii) EMG amplitude.
was used as the primary index for comparison of neuromuscular control in this study. Our analysis consisted of five questions:

Question 1: Was the baseline run pattern stable?

Comparisons of the baseline and transition runs were made only after it had been confirmed that stable baseline kinematic and muscle recruitment patterns had been achieved. The baseline measure was defined as unstable, and a participant’s data were excluded from analyses when (i) the AMD for data of the 8th, 9th, and 10th minutes of the baseline runs exceeded 3° (kinematics) or 10% EMG amplitude and (ii) the 95% CI for EMG or kinematic waveforms of the 8th, 9th, and 10th minutes of the baseline runs were discordant, that is, not overlapping, for ≥10% of the stride.
Question 2: Was cycling cadence different between control and ERLP groups?

Cycling cadence has been identified as a variable that may influence subsequent running. Therefore, we controlled this potential confounder, and comparisons of baseline and transition runs were made only after it had also been confirmed that cycling cadence was not different between groups, that is, only after it had been shown that this potential confounder had been controlled. Cycling cadences were compared between control and ERLP groups using ANOVA (group (two levels: control vs ERLP) × cadence condition (four levels: 57.5, 77.5, and 97.5 rpm and IPC)).

Question 3: How many triathletes exhibit altered neuromuscular control?

To determine changes in neuromuscular control between baseline and transition runs, EMG and kinematic data from

the 10th minute of the baseline run were compared with data from the 1st through 5th minute and every 5th minute thereafter of the 30-min transition run. The transition run measure was defined as different to that of the baseline run when (i) the AMD exceeded 2° (kinematics) or 10% EMG amplitude and (ii) the 95% CI for EMG or kinematic waveforms of baseline run 10th minute and transition run xth minute (x = 1–30) were discordant for ≥10% of the stride.

Question 4: Was a difference between baseline and transition run patterns more likely in triathletes with a history of ERLP?

Relative risk for the likelihood of altered neuromuscular control was calculated in two ways: a) defined by subject, by comparing the number of triathletes in each group who exhibited altered kinematics or activation of one or more leg muscles (i.e., x of 24 control group triathletes vs x of 10

FIGURE 2—A, EMG data of TA from a representative ERLP triathlete, which is plotted as example data of muscle recruitment, showing an effect of cycling on muscle recruitment during subsequent running. B, Comparison of the magnitude of change in muscle recruitment (pooled data) between the baseline and transition runs, which was found not to be different between control (14.10% ± 2.34%) and ERLP triathletes (16.31% ± 3.64%). Error bars in panels (A and B) represent the 95% CI. C and D, Ankle and knee joint angular kinematics from a representative ERLP triathlete, plotted as example data, and shows cycling did not influence sagittal plane kinematics at the knee or ankle during subsequent running. For panels A, C, and D, data on baseline run were defined as stable because AMD for baseline run on 8th, 9th, and 10th minutes is 3.81% ± 0.81% (EMG), 0.49° ± 0.04° (ankle), and 0.39° ± 0.05° (knee); neither ankle kinematics nor knee joint kinematics was different between baseline and transition runs because AMD for baseline run versus transition run is 1.17° ± 0.06° (ankle) and 1.64° ± 0.07° (knee); recruitment of TA was different between baseline and transition runs because AMD for baseline run versus transition run is 14.05% ± 1.08% EMG amplitude; data in black are from the 8th, 9th, and 10th minutes of the 10-min baseline run (total of 3 data series), whereas data in gray (see color scale provided) are from the 1st to the 5th minute and every 5th minute thereafter of the 30-min transition run (total of 10 data series); data are shown on the x-axis for one complete stride of running from heel strike (0%) to ipsilateral heel strike (100%), and data for each minute of running are the average of the final 10 strides from that minute of running; EMG amplitude is plotted as a percentage of the maximum measured EMG amplitude (MAX, 0–100%).
ERLP group triathletes); and b) defined by muscles, by comparing the total number of muscles of triathletes from each group exhibiting altered recruitment (i.e., x of 120 control group muscles vs x of 50 ERLP group muscles). Relative risk and CI for relative risk were calculated using the independent proportions method described by Armitage and Berry (1). We also conducted a sensitivity analysis to evaluate the effect of using thresholds of 5%, 10%, and 15% EMG amplitude to identify altered muscle recruitment on our findings of relative risk.

Question 5: Was the magnitude of change between the baseline and the transition runs different between control and ERLP groups?

AMD for muscles exhibiting an altered recruitment pattern (identified at the primary threshold of 10% EMG amplitude) was compared between groups using an unpaired t-test (control vs ERLP groups; AMD was of data pooled across muscles).

Whereas knee and ankle kinematics were three-dimensionally modeled, only sagittal plane kinematics were included in our analyses because caution must be used when interpreting transverse and frontal plane kinematic data. Caution is required because of the increased error associated with kinematic modeling of frontal and transverse plane motions. This is particularly the case for data of high-speed running because of the potential for increased marker movement artifact, as we have previously discussed in detail (14). Further improvements to kinematic modeling techniques may facilitate more extensive use of transverse and frontal plane data in the future (e.g., [4]).

The criteria used to define stability and to identify changes in neuromuscular control between baseline and transition runs were based on a) known replication thresholds of joint position and muscle activation levels, b) known effects of differences in muscle activation levels on myoelectric and mechanical characteristics of muscle activity (i.e., changes of ≥10% in the level of muscle activity are known to affect, e.g., time to fatigue), and c) known typical error associated with our measurement procedures, which have been reported previously (7,14,27). These criteria ensured that the identified differences in neuromuscular control were clinically meaningful, statistically significant, and exceeded measurement error.

![Figure 3](image-url)

**FIGURE 3**—A, Comparison of cycling cadences between control and ERLP groups. Note that error bars are not visible for 55–60, 75–80, and 95–100 rpm conditions because 95% CI ratings were <0.13 rpm. B, Prevalence of altered muscle recruitment in control and ERLP groups for each of TA, PL, GL, GM, and SOL muscles. Pooled data, defined by muscle (column 6) and subject (column 7), are also shown. Relative risk for control versus ERLP groups is shown as text in each column. C, Results of our sensitivity analyses conducted to evaluate the effect of using thresholds of 5%, 10%, and 15% EMG amplitude to identify altered muscle recruitment on our findings of relative risk. The dashed line represents a relative risk of 1, which implies no effect. Note that relative risk values for the default threshold in panel C correspond to the relative risk of 2.6 and 2.4 shown for data defined by muscle and group, respectively, in panel B.
Effect sizes (ES) were calculated using pooled SD. Data are presented as mean ± 95% CI throughout. Analyses were conducted using Statistica (version 6; StatSoft, Inc., Tulsa, OK).

RESULTS

Was the baseline run pattern stable? The baseline measures of kinematics and EMG were defined as stable for all triathletes. AMD for data of the 8th, 9th, and 10th minutes of the baseline run was 2.38% ± 0.11% EMG amplitude (see data in black in Fig. 2A; this represents the variation among the three data series in black), 0.56° ± 0.12° (ankle joint kinematics), and 0.41° ± 0.09° (knee joint kinematics; see data in black in Fig. 2C and D). Furthermore, the 95% CI for EMG and kinematic waveforms of the 8th, 9th, and 10th minutes of the baseline runs were never discordant for ≥10% of the stride. Therefore, no data were excluded on the basis of instability.

Was cycling cadence different between control and ERLP groups? Triathletes cycled at cadences of 57.5 ± 0.11, 77.5 ± 0.10, and 97.5 ± 0.13 rpm during the three controlled conditions and 84.1 ± 2.6 rpm during the IPC condition. Cycling cadences were not different between control and ERLP groups (ANOVA; $P = 0.67$, ES < 0.30; Fig. 3A), indicating adequate control of this potential confounder.

How many triathletes exhibit altered neuromuscular control? Sagittal plane kinematics of the leg and foot were not different between baseline and transition runs for any triathletes (illustrated by a comparison of data series in gray vs black in Fig. 2C and D): AMD was 1.26° ± 0.13° (ankle) and 1.52° ± 0.21° (knee), and 95% CI ratings for kinematic waveforms were not discordant for ≥10% of the stride. However, recruitment was different between baseline and transition runs for one or more leg muscles in 10 (29.4%) of 34 triathletes and in a total of 23 (13.5%) of 170 muscles tested. The comparison of data series in gray versus black in Figure 2A illustrates an example of altered muscle recruitment during the transition run.

Was a difference between baseline and transition run patterns more likely in triathletes with a history of ERLP? Five (50.0%) of 10 ERLP group triathletes versus 5 (20.8%) of 24 control group triathletes exhibited altered neuromuscular control, which represents a relative risk defined by subject of 2.40 (0.89–6.50; $P = 0.089$). Twelve (24%) of 50 muscles tested in ERLP group triathletes versus 11 (9.2%) of 120 muscles tested in control group triathletes exhibited altered neuromuscular control, which represents a significant relative risk defined by muscle of 2.62 (1.34–6.01; $P < 0.01$). Figure 3B illustrates the prevalence and subsequent relative risk of altered recruitment for each leg muscle tested.

Sensitivity analysis, illustrated in Figure 3C and outlined in full in Table 2, indicated that ERLP group triathletes were more likely to exhibit altered muscle recruitment regardless of the threshold used to define altered muscle recruitment; the default threshold of 10% ΔEMG amplitude provided the most conservative relative risk when defined either by subject (2.40 ($P = 0.089$) at 10% vs 3.36 ($P = 0.006$) at 5% and 4.00 ($P = 0.019$) at 15%) or by muscle (2.62 ($P < 0.001$) at 10% vs 2.95 ($P < 0.001$) at 5% and 3.60 ($P = 0.029$) at 15%).

Was the magnitude of change between the baseline and the transition runs different between control and ERLP groups? The magnitude of change in muscle recruitment between the baseline and the transition run patterns was not different between control (14.10% ± 2.34%) and ERLP group triathletes (16.31% ± 3.64%; $t$-test: $P = 0.41$, ES = 0.51; Fig. 2B).

DISCUSSION

We found that cycling can directly influence neuromuscular control during subsequent running in some highly trained triathletes, which is consistent with previous findings (8). A relationship between this altered neuromuscular control of running and ERLP has been proposed (8) but, before this study, had not been investigated. This study showed that triathletes with a history of ERLP are ≥2.4 times more likely to exhibit altered neuromuscular control when running off the bike and that recruitment of a given leg muscle when running off the bike is ≥2.62 times more likely to be influenced by cycling in triathletes with a history of ERLP.
Neuromuscular control and ERLP in triathletes. Triathletes must complete high training loads to be competitive, and overuse injury that interferes with training is common (20,28) (Australian Institute of Sport, unpublished data). Although the etiology of ERLP is likely multifactorial, models of overuse injury pathogenesis (2) and existing evidence of a relationship between altered neuromuscular control and musculoskeletal injury from other injury models (e.g., [18]) provide a theoretical link between altered neuromuscular control at the leg during running and ERLP in triathletes. For example, changes to muscle activation during running may result in altered bone and soft tissue loading (e.g., [17,38]). If there is an increase in loading that is of sufficient magnitude, cumulative load of running may quickly exceed the bone’s or soft tissue’s loading capacity, and microdamage will accumulate, which could contribute to overuse injury. This study demonstrated a link between a history of ERLP in triathletes and altered neuromuscular control when running off the bike. Our findings are consistent with preliminary evidence of a link between ERLP and altered neuromuscular control of running from a case–control study in running athletes (26). Our findings suggest that the ability of triathletes to complete large running volumes without injury is likely related to their ability to use neuromuscular control strategies that are optimal for running and not influenced by previous cycling.

Possible mechanisms for the effect of cycling on running. Because of the cross-sectional design of our study, it is not possible to determine whether the greater propensity for altered neuromuscular control in triathletes with a history of ERLP was present before the onset of pain and injury and could thus be seen as a causative factor or, conversely, if altered neuromuscular control occurs as a result of pain and dysfunction. It is also not possible to establish the mechanism of effect, that is, how and why cycling influences neuromuscular control of running. It is noteworthy that corrections to movement and muscle recruitment patterns during the switch from cycling to running are instantaneous in most highly trained triathletes. This finding is consistent with previous studies of triathletes (8) but in contrast to previous fundamental motor control studies of the effects of performing two tasks in sequence in the upper limb (24). Previous upper limb studies report large kinematic variations after the switch from one task to another. For example, Karniel and Mussa-Ivaldi (24) used patterns of movement and aftereffects observed during an upper limb task sequence to show that the CNS had a strong tendency to use a single control strategy, or movement plan, when performing a sequence of two previously learned tasks. That is, the CNS seemed to use a movement plan that was generalized to both tasks but not specific for either and used this single generalized movement plan in preference to the existing movement plan for each task. One difference between our study and these previous studies of the upper limb is that our triathletes had years of training. Therefore, one possible interpretation of the contrasting findings is that the ability to switch, or “transition,” from one movement plan to another is enhanced with ongoing training. Further studies are required to evaluate this hypothesis. Given the findings of our study, it is plausible that musculoskeletal pain and injury impede the triathletes’ ability to switch between movement plans for cycling and running. It is also plausible that a preexisting lesser ability to switch between movement plans for cycling and running is a predictive factor for injury and contributes to the etiology of ERLP via altered bone and soft tissue loading. Prospectively designed studies are required to test these hypotheses.

Implications for training design and clinical interventions. Because success in triathlon competition depends largely on the ability to run immediately after cycling (36), training often involves running after cycling. However, there is evidence that this multidiscipline training may influence both the adaptation of the neuromuscular system to training (i.e., learning) and the triathletes’ ability to use what they have learned (i.e., the ability to execute neuromuscular control strategies for running that have not been adversely influenced by cycling) (8,10). Combined, the results of this study and those of earlier studies (8,10,15) suggest that it is important when designing training interventions for triathletes to consider possible implications for neuromuscular learning and control and subsequent implications for both performance and injury.

We have shown that cycling can influence neuromuscular control of running in some triathletes independent of fatigue. This is not to say that neuromuscular fatigue does not occur, is not relevant, or does not affect running after cycling in triathlon. Rather, it emphasizes that effects of cycling on running may also relate to a direct interference effect of cycling on neuromuscular control of running and that interference effects may occur independent of, or in addition to, fatigue effects during training and competition. Most importantly for triathletes and their coaches, these findings suggest that training strategies used to improve triathletes’ fatigue resistance may not fully address the effects of cycling on running. At this time, neither fundamental laboratory studies nor studies of triathletes provide evidence for how best to design training to reduce the effect of cycling on neuromuscular control of running. However, the findings of this study do highlight the need for training and physiotherapy interventions that address neuromuscular control of running off the bike in triathletes with ERLP. This proposal is based on the clinical presumption that altered neuromuscular control during running off the bike is maladaptive in that it negatively influences performance, which is consistent with preliminary findings (15). It is worth noting that alterations in neuromuscular control that occur with injury have been shown to persist up to 66 months after injury (34). This finding suggests that it is likely that, without targeted interventions, altered neuromuscular control associated with ERLP in triathletes may persist well after the triathlete has returned to training and...
competition, which may have ongoing effects on performance and likely increases the probability of injury reoccurrence.

**Implications on performance.** Musculoskeletal injury of the leg and foot is arguably the greatest impediment to performance in triathletes via disruption to training and limited participation. An injury-induced restriction in training can result in detraining (i.e., a loss of training effects). Detraining may include adverse changes to the cardiorespiratory system as well as the neuromuscular system (30), all of which will be most pronounced in elite triathletes for whom even small decreases in performance capacity can have a significant impact on race outcome. In addition to performance decrements associated with detraining, altered neuromuscular control may directly influence running efficiency and therefore run performance (15). On this basis, the persistence of altered neuromuscular control after injury and return to training and competition noted previously (34) may have ongoing direct effects on performance. Although there is preliminary evidence that interventions such as plyometric training may improve running efficiency via improved neuromuscular control (33), further research is required in this regard.

**Sample size considerations.** As training history has a significant influence on neuromuscular control of both running and cycling (9–11), triathletes were matched as closely as possible for triathlon training history to maximize sample homogeneity and to prevent confounding effects of training history. Sample size was necessarily small to ensure sample homogeneity. Given the nature of participants required, that is, highly trained and high-performance triathletes, only small numbers of individuals were suitable for inclusion. Thus, larger participant samples could only have been used if inclusion criteria were compromised. Our data strongly suggest that the small participant samples used here do not compromise the merit of our conclusions.

**CONCLUSIONS**

This study showed that triathletes with a history of ERLP are >2.4 times more likely than training-matched control triathletes to exhibit altered neuromuscular control when running off the bike. When considered together, the results of this study and those of earlier studies (8,10,15) suggest that it is important to consider the possible implications for neuromuscular learning and control when designing training interventions for triathletes. This study also demonstrates the need for training and physiotherapy interventions that address neuromuscular control of running off the bike in triathletes with ERLP.

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**REFERENCES**