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Changes in Muscle Activity and Kinematics of Highly Trained Cyclists During Fatigue

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Abstract

Muscle fatigue may alter kinematics and contribute to repetitive strain injuries. This study quantified how both localized muscle fatigue and movement kinematics change over time during exhaustive cycling. Seven highly trained cyclists rode a stationary bicycle ergometer at 100% of their VO_2max until voluntary exhaustion. Cycling kinematics and EMG activity from select lower extremity muscles were recorded. Cross-correlations were computed to quantify how EMG median frequencies (MDF) changed with changes in movement kinematics. All athletes maintained both cadence and power output for ~90% of the trial duration. Significant sustained muscle fatigue occurred in 18 of 28 muscles tested, most prominently in the biceps femoris ($p = 0.020$) and gastrocnemius ($p = 0.018$). Kinematics and MDF both fluctuated non-monotonically as subjects fatigued. Changes in MDF significantly preceded changes in mean trunk lean ($p = 0.009$) and hip angles ($p = 0.025$), and trunk lean range of motion ($p = 0.029$). Fluctuations in MDF were positively correlated with fluctuations in mean trunk lean ($p = 0.009$) and knee splay angles ($p = 0.011$), and with trunk lean ($p = 0.002$) and ankle ($p = 0.001$) range of motion. These results therefore establish a direct link between changes in muscle fatigue state and subsequent changes in movement kinematics during cycling..

Keywords

Cycling; Muscle Fatigue; Kinematics; EMG; Coordination; Cross-Correlation

INTRODUCTION

Increased popularization of both recreational and competitive cycling [1] has led to an increase in cycling-related injuries, primarily repetitive strain injuries (RSI's). Knee pain is the most common RSI associated with cycling [1–3]. Between 42–65% of recreational cyclists may experience overuse knee pain [4,5]. The prevalence of all non-traumatic injuries among cyclists may be as high as 85% [6]. Many of these injuries may result from biomechanical alterations associated with fatigue. As fatigue sets in, cyclists may change their muscle activation patterns to maintain performance. This may in turn cause changes in kinematics that could possibly lead to mal-adaptive joint loading.

The development of acute fatigue in cycling is a complex process involving energetic, cardiovascular, neuromuscular, biomechanical, and psychological factors [7–9]. While several studies have measured fatigue-related changes in performance related to mechanical and

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cardiovascular output, factors like muscle activity and joint kinematics have been less studied. Very short bouts (5–30 sec.) of *maximal* effort cycling altered the torque produced by trained cyclists [10–12]. However, links from these global performance measures to changes in muscle EMG or joint kinematics were not assessed. Moderate duration bouts (5–10 min.) of exhaustive cycling caused declines in cardiac output, muscle blood flow, O₂ delivery, and O₂ uptake [13,14]. However, changes in muscle EMG or kinematics were not assessed. Mildly fatiguing cycling for 30 min. altered muscle EMG parameters extracted from wavelet analyses that were *non-monotonic* [15]. However, changes in kinematics were not assessed. Long bouts (1–5 hrs.) of sub-maximal (55–65% of VO_{2Max}) cycling altered both EMG central drive and peripheral fatigue mechanisms [16–18]. Both muscle activation levels and twitch contractile properties changed non-monotonically over time [17]. Despite these fluctuations, trained subjects generally maintained consistent power output throughout the trials [18]. However, these authors did not examine how these physiological and/or EMG changes affected cycling kinematics. Optimization studies have predicted how muscle activations produce biomechanical movement patterns during *non-fatigued* cycling [19,20], but have not examined how fatigue affects cycling biomechanics. Thus, it remains unclear how, or if, cyclists alter their kinematics in response to progressive muscle fatigue.

Muscle fatigue alters the coordination of muscle activation patterns and joint kinematics in a variety of multi-joint tasks [21–27]. Most of these studies employed “pre. vs. post” experimental designs [21–25,27]. This inherently assumes that one kinematic strategy exists prior to fatigue and a second exists following fatigue. However, in one upper extremity task, both muscle activation patterns and movement kinematics changed *non-monotonically* throughout the progression of fatigue [26]. Changes in muscle fatigue state systematically preceded changes in joint kinematics, indicating that subjects continually changed their movement strategies to maintain task performance [26]. Such biomechanical changes resulting from fatigue may indicate an increased potential for injury [25,27] or may alternatively reflect protective strategies used to decrease injury risk [23].

Similarly, the non-monotonic changes observed in muscle activation levels and twitch contractile properties in trained cyclists [17] suggest that these changes are likely also accompanied by changes in movement kinematics. Therefore, this study quantified how changes in muscle fatigue associated with acute systemic fatigue were related to changes in kinematics in highly trained cyclists throughout a moderate-duration (~5–10 min.) exhaustive cycling bout on a stationary bicycle. We tested the hypothesis that these athletes would exhibit varying and non-monotonic changes in both movement kinematics and muscle activity as fatigue progressed. We hypothesized that EMG median frequencies would decrease indicating the progression of muscle fatigue. We also hypothesized that specific decreases in median frequency would be followed by correlated changes in movement kinematics, similar to previous upper extremity experiments [26].

METHODS

Ten highly trained (USCF Category 3 or higher level of competition) male cyclists, age 18–45 yrs, participated. The kinematic data obtained from 3 subjects could not be analyzed due to technical difficulties. Therefore, results from 7 subjects (Tables 1 & 2) are presented here. All subjects gave written informed consent before participating. No subject had any history of prior injuries or were currently taking any medications that might interfere with their ability to complete the experiment.

Each experiment consisted of 2 sessions. In Session 1, participants completed a VO₂ max test and familiarization trial (cycle to fatigue). Session 2 occurred at least 1 week later. Here, participants completed a 10 minute warm-up prior to data collection. The athletes then cycled

to exhaustion on a Lode Excalibur Sport bicycle ergometer (Lode Medical Technology, Groningen, The Netherlands) at a work load equivalent to 100% of their VO_2 max. Subjects were given vigorous verbal encouragement throughout the trials. Participants were instructed to cycle at ~90 revolutions per minute (RPM) and were allowed to recover if their RPM fell below 90. Trials were terminated when cadence dropped below 70 RPM or when the subject felt they could no longer continue.

Each subject was marked with reflective markers (Fig. 1) to record sagittal and transverse plane kinematics. Kinematic data were sampled at 120 Hz continuously throughout each trial using an 8-camera Vicon Motion Analysis system (Oxford Metrics, Oxford, UK). Bipolar single differential surface EMG electrodes (DE-2.1, Delsys Inc., Boston, MA, USA) were placed on the left leg over the vastus lateralis (VL), biceps femoris (long head; BF), gastrocnemius (lateral head; GAS), and tibialis anterior (TA), according to accepted recommendations [28]. The electrodes were 99.9% Ag, 10mm×1mm bars, spaced 10 mm apart. The Delsys signal amplifier had an input impedance of $> 10^{15}\Omega$, common mode rejection ratio of -92dB, and overall noise of $\leq 1.2\mu\text{V rms}$ (Delsys, Inc.). EMG data were sampled at 1080 Hz throughout each entire trial and were synchronized with the kinematic data. All data were processed and analyzed using custom MATLAB software (The MathWorks, Natick, MA). Final statistical analyses were performed in Minitab (Minitab, State College, PA).

Raw marker data were filtered with a 4th order zero-lag Butterworth filter with a cutoff frequency of 15 Hz. Sagittal plane trunk lean, hip, knee, and ankle angles were calculated for the left side of the body (Fig. 1A). Knee splay angle (i.e., thigh abduction/adduction) in the transverse plane was also calculated (Fig. 1B). Our goal was to examine how the general kinematic patterns of these joints changed as fatigue progressed during each trial (Fig. 2). A marker on the center of the pedal was used to define the beginning (top-dead-center), middle (bottom-dead-center), and end (return to top-dead-center) of each pedal revolution. Two dependent measures were computed for each revolution for each of the joint angles measured. Mean joint angles (MA) were calculated as the average joint angle during each pedal revolution. Ranges of motion (ROM) were calculated by subtracting the minimum from the maximum angle within each revolution (Fig. 2B). These MA and ROM measures quantified how the kinematics changed across consecutive pedal revolutions during each experiment (Fig. 2C).

EMG signals were band-pass filtered from 30 Hz to 400 Hz using a 4th order zero-lag Butterworth filter and band-stop filtered from 57–63 Hz to remove any residual 60 Hz noise [29]. The same time points defining the beginning, middle, and end of each revolution (see above) were used to split each EMG signal into down stroke and up stroke segments (Fig. 3B). EMG median frequencies, a common measure of local muscle fatigue [29–31], were calculated for each down and up stroke separately, then averaged to yield a single median frequency (MDF) for each crank revolution across each trial [26,32–34]. This “short-time” Fourier transform (STFT) method was previously validated for tracking slowly varying changes in muscle fatigue across multiple dynamic contractions [32–34]. MDFs were computed during phases when muscles were both active and inactive. We did not compute MDFs from only those phases when each muscle was “active” because of the difficulty in consistently defining “onset” and “offset” times for EMG signals, where the optimal criteria for defining these events would vary between different muscles within each subject, and also between subjects. Furthermore, since a signal’s frequency is independent of its amplitude, modulation of EMG amplitude does not substantially affect the calculation of MDFs using this STFT technique [32,33]. These MDF values thus quantified how the local fatigue state of each muscle changed across consecutive pedal revolutions during each experiment (Fig. 3C).

To determine which muscles exhibited *continued* and *sustained* fatigue across the entire experiment, linear regressions were performed. Localized muscle fatigue would cause the

EMG median frequencies to decrease (7), yielding a negative regression slope. These linear trends did *not* identify *transient* periods of muscle fatigue that were followed by periods of recovery (e.g., Fig. 7A), but only those trends that were sustained over the *entire* time course of the task. They thus represent very *conservative* estimates of the fatigue that was actually observed (see Discussion). Significance levels (p-values) of individual regressions were tabulated. Regression slopes for each muscle group were pooled across subjects and subjected to a 1-tailed student's t-test to determine if they were significantly less than zero.

Having obtained time series of MA, ROM, and MDF values for each crank cycle across each trial, these time series were then custom box-car filtered, twice, with a moving window length of 9 frames (Figs. 2C and 3C). These smoothed MA, ROM and MDF data represented the slowly varying local trends for each signal [32,33]. This procedure only captured those trends that were sustained for ≥ 17 consecutive cycles and thus effectively averaged out the majority of random cycle-to-cycle variations. It is possible there may have been some remaining fluctuations in the moving average trends for either the MDF or kinematic measures that were caused by random statistical fluctuations that just happened to be sustained for more than 17 consecutive cycles. However, any such fluctuations in the smoothed MDF trends that were random would not be expected to be correlated with changes in kinematics, or vice versa.

We then computed cross-correlations for each combination of MA and ROM with each muscle MDF for each subject (Fig. 4). It may not be immediately obvious that each muscle (e.g., tibialis anterior) should affect each kinematic variable (e.g., trunk lean angle). However, in general, the movements of all limb segments are mechanically coupled. Thus, each muscle in the body has the *potential* to affect (i.e., accelerate) any limb segment in the system, even remote limb segments to which that muscle is not attached [35,36]. This principle is the basis for the “induced acceleration analyses” that have become increasingly common in biomechanics (e.g., [36–40]).

To compare across subjects, cross-correlation magnitudes were normalized by dividing by the standard deviation of the correlation magnitudes at all time lags (Fig. 4C) [26]. This normalizing allowed us to determine which individual correlation values were “significantly greater than” the pool of all correlation values computed across all time lags for each comparison so we could make valid comparisons *between* cross-correlations. The sign of each peak cross-correlation (PCC) value determined the direction of the correlation. Time lags (τ) at each PCC were used to determine how changes in localized muscle fatigue either affected, or were affected by, changes in kinematics. A negative τ indicated that MDF shifts *preceded* kinematic changes. Positive τ values indicated that kinematic changes *preceded* shifts in MDF. To avoid spurious findings, the search for PCC's was restricted to time lags within ± 120 crank cycles (Fig. 4C).

For each MA and ROM variable, the PCC's and τ values (Fig. 4C) for each subject for each muscle MDF were subjected to a single-factor repeated measures analysis of variance (ANOVA). To determine if there were general trends across muscles, the data were pooled across muscles. One-tailed t-tests (mean < 0) were applied to the τ values to determine if changes in muscle fatigue state *preceded* changes in MA or ROM. Two-tailed t-tests (mean $\neq 0$) were applied to the PCC values to determine if changes in muscle fatigue state were positively or negatively correlated with kinematic changes.

RESULTS

Time to fatigue varied from ~5 to ~10 min across subjects (Table 2). While all subjects were “highly trained” (i.e., USCF Cat. 3 or higher), they still exhibited a wide range in body anthropometrics (Table 1) and training histories (Table 2). We ran several regression analyses

to determine if any of these characteristics, either together or combined, predicted time-to-fatigue. Greater body mass weakly predicted longer time to fatigue ($r^2 = 52.3\%$), but this was not quite significant ($p = 0.066$). None of the other variables came close ($r^2 \leq 14.4\%$; $p \geq 0.401$). These time-to-fatigue results were consistent with those obtained from two other recent studies using a similar protocol in a similar population [13,14].

Despite differences in time-to-fatigue, each athlete maintained both the required cadence and power output for ~90% of the duration of their trial (Fig. 5). The precipitous drop-off in cadence and power output during the last ~10% of each trial indicates that these athletes had reached their physical limits. These results are supported by similar recent findings in longer duration cycling fatigue tests [18].

Across each trial, kinematics varied both among joint angles and between subjects. The most prominent changes appeared in trunk lean angles (Fig. 6A). Visible changes were also often found in ankle (Fig. 6B) and knee splay angles (Fig. 6C). While mean hip and knee angles remained relatively constant, ROM for all angles changed non-monotonically (Fig. 6C). Overall, all subjects exhibited nonstationary and non-monotonic changes in joint kinematics with progressive fatigue.

EMG median frequencies (MDF) also exhibited nonstationary and non-monotonic changes (Fig. 7A). While the pattern of muscle fatigue changes varied between subjects, every subject exhibited significant *sustained* fatigue in at least 1 muscle and every muscle exhibited significant *sustained* fatigue in at least 3 subjects (Table 3). In many cases, MDF's began dropping almost immediately after the task began (e.g., Fig. 7A; VL, GAS, and TA muscles), indicating that these muscles began to fatigue right away. One-tailed t-tests (Fig. 7b) revealed significant muscle fatigue across *all* subjects in the BF ($p = 0.020$) and GAS ($p = 0.018$) muscles. However, these results only reveal those muscles that exhibited fatigue that was *sustained* across the *entire* trial. *Every* muscles in *every* subject exhibited *some* evidence of at least transient fatigue at some point during each trial (e.g., Fig. 7A; TA muscle).

Different muscle groups in different subjects exhibited different inter-relationships with joint kinematics (Fig. 8). Cross-correlation peaks were sometimes positive and sometimes negative, indicating that changes in muscle fatigue state were associated with both increases and decreases in either MA or ROM for different muscle/joint combinations. There were no significant differences between muscle groups for any of the 10 time lag (τ) comparisons ($0.18 < p < 0.89$) or for 9 of the 10 PCC comparisons ($0.24 < p < 0.84$). The only exception was for mean hip angle correlations ($p = 0.018$), where post-hoc comparisons showed that the GAS exhibited negative correlations in all subjects tested, whereas the other 3 muscles exhibited both positive and negative correlations.

On average, across subjects and muscle groups, pooled cross-correlation time lags (τ ; Fig. 9A) were significantly less than zero for mean trunk lean ($p = 0.009$), mean hip angle ($p = 0.025$) and trunk lean ROM ($p = 0.029$). Thus, changes in these joint kinematics were specifically *preceded* by changes in muscle fatigue. Time lags were not significantly less than zero for the remaining measures (Fig. 9A).

On average, across subjects and muscle groups, cross-correlation magnitudes (PCC; Fig. 9B) were significantly positive for mean trunk lean ($p = 0.009$), trunk lean ROM ($p = 0.002$), mean knee splay MA ($p = 0.011$), and ankle joint ROM ($p = 0.001$). Thus, increases (or decreases) in EMG median frequencies, corresponding to lesser (or greater) muscle fatigue, were significantly correlated with increases (or decreases) in mean trunk lean, trunk lean ROM, mean knee splay, and ankle joint ROM. Conversely, there was a trend for EMG median frequencies to be negatively correlated with hip joint mean angle ($p = 0.060$) and hip joint ROM ($p = 0.077$; Fig. 9B).

DISCUSSION

Repetitive strain injuries (RSI) are common among both recreational and competitive cyclists [1–6]. Many of these injuries may result from sub-optimal changes in kinematics and/or joint loading [25,27] that cyclists adopt as they change their muscle activation patterns to maintain performance [16–18] with increasing fatigue. To date, however, specific links between physiological and/or neuromuscular changes indicating fatigue and associated changes in cycling biomechanics have not been established. This study therefore quantified how acute fatigue affected changes in movement kinematics throughout a moderate-duration (~5–10 min.) exhaustive cycling bout.

Highly trained athletes displayed significant muscle fatigue during the prescribed protocol (Fig. 7, Table 3). In many cases, decreases in EMG MDF occurred almost immediately, similar to other dynamic multi-joint tasks [32,41]. Sustained muscle fatigue was particularly evident across subjects for BF and GAS muscles (Fig. 7B). Muscle fatigue states (Fig. 7A) and joint kinematics (Fig. 6) both varied non-monotonically over time during these trials. However, despite these temporal variations, subjects maintained overall task performance throughout the trial (Fig. 5). Comparably trained cyclists also maintained task performance over a 1 hour protocol despite similar fluctuations in neuromuscular fatigue [18]. Our results confirmed our hypothesis that cyclists would exhibit *non-monotonic* variations in both EMG fatigue states (Fig. 7) and joint kinematics (Fig. 6). Our results also confirmed our hypothesis that specific changes in EMG fatigue states would precede changes in movement kinematics (Fig. 9), extending previous findings [26]. To our knowledge, this is the first evidence demonstrating a direct link between muscle fatigue and subsequent alterations of movement kinematics in cycling.

Most subjects exhibited shifts toward greater trunk lean angles early in the fatigue protocol (Fig. 6A). Since the ergometer was configured to match each cyclist's own racing bicycle, it is unlikely these shifts resulted from discomfort. All subjects displayed decreasing ankle angles (Fig. 6B), possibly resulting from significant GAS fatigue, as exhibited by 5 of the 7 subjects (Table 3). The significant cross-correlation time lags (Fig. 9A) demonstrated that muscle fatigue (i.e., decreased MDF) *preceded* changes in trunk lean MA, hip MA, and trunk lean ROM. Thus, cyclists likely shifted their torso position specifically in response to muscular fatigue. This may have been to take advantage of the length-tension characteristics of muscle [42,43] to generate increased down-stroke force (hip extension) from the gluteus maximus. Likewise, changes in knee splay angle and ankle ROM were positively correlated with changes in muscle fatigue state (Fig. 9B) and occurred at approximately the same time (Fig. 9A).

Many differences existed across subjects in this study (Tables 1–2). These led to different responses between individuals (e.g., Table 3, Figs. 6–8). Psychological variations in motivation might have also contributed. However, since all participants were highly trained competitive athletes, it is unlikely these subjects gave sub-maximal effort and/or terminated participation prematurely. Nevertheless, the finding that different muscle/joint angle combinations exhibited different correlations in different subjects (Fig. 8) makes generalizing some of our results difficult. However, the inter-individual variations observed here were similar to those observed in previous studies in trained [13,14] and recreational cyclists [15], and for other multi-joint tasks [21,23,26,44]. These differences highlight the extensive motor redundancy available to the neuromuscular control system [45–47]. Despite these inter-individual differences, subjects still exhibited specific kinematic changes in response to changes in local muscle fatigue (Fig. 9). These changes may in turn indicate an increased risk of long-term injury.

People cycling at sub-maximal levels likely also exhibit non-monotonic kinematic fluctuations similar to those observed here, but *not* because of fatigue. However, if the subjects in the present

study had been doing this, we would not expect to find significant correlations between these kinematic shifts and shifts in MDF, which we clearly did find (Figs. 8 & 9). These correlations demonstrate that these subjects changed their kinematics specifically in response to changes in muscle fatigue and that these were not simply random cycle-to-cycle variations. To prove this conclusively, it would be tempting to include a “control” group/condition where subjects cycled for similar periods of time, but at “non-fatiguing” sub-maximal levels. Unfortunately, any context where subjects cycled, even with zero additional external load, would still require some level of muscle activation, so that *some* muscle fatigue would start to set in as soon as the activity began [32,41]. Thus, it would still not be possible to eliminate the possibility that any changes in muscle activity and/or kinematics observed in this “control” group/condition were not also due to (low level) fatigue. Instead, the study design adopted here was fully consistent with the majority of fatigue studies on cycling [7,10,11,16–18] and other multi-joint tasks [15,21–27,32,33,41,44] where each subject is used as their own control and their initial task performance (when fatigue is minimal) is compared to the their performance at the end of the task (when fatigue is maximal). Our results also extend this paradigm by demonstrating that typical “pre vs. post” comparisons do not adequately capture the *time course* over which these changes occur.

Continued and sustained localized muscle fatigue (Fig. 7A) was elicited in 18 of the 28 muscles tested in these athletes (Table 3). However, *sustained* fatigue was not observed in several muscles and results varied between subjects, especially in the VL and TA (Fig. 7B). Some muscles even displayed increasing MDF, which might have resulted from increases in intra-muscular temperature [48]. Extracting the slopes of linear fits to the clearly non-stationary trends in the MDF data (e.g., Fig. 7A) did not capture those transient periods of muscle fatigue that occurred in *every* muscle in *every* subject. To do this, we could instead have subjectively identified only those individual time windows from each muscle for each subject where the MDFs decreased the most. Had we done this, we certainly would have seen *highly* significant decreases in *all* muscles for *all* subjects. However, because the timing and duration of these periods varied across muscles and between subjects, we believed this would have unfairly biased our results. In the end, we felt the most objective way to characterize the progression of individual muscle fatigue states *over time* was to analyze the slowly varying moving average trends in the MDF data. This was the main focus of our cross-correlation analyses (Figs. 8–9). We believe those analyses effectively capture the time-varying fatigue processes that the linear slopes presented in Fig. 7 do not.

In spite of the kinematic and EMG differences observed between individual subjects, all subjects exhibited acute systemic fatigue, as evidenced by no longer being able to generate the power required to maintain task performance (Fig. 5). This confirms that localized muscle fatigue is only *one* component of the acute fatigue experienced during maximal effort exhaustive cycling, which also involves energetic, respiratory, and/or cardiovascular factors [7–9]. The present study only correlated changes in localized muscle fatigue and movement kinematics. Additional research is needed to determine if these other contributing components to acute fatigue may also induce specific changes in movement kinematics.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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A Sagittal View:

Angle Definitions:

θ_L -- Trunk Lean

θ_H -- Hip

θ_K -- Knee

θ_A -- Ankle

B Top View:

Angle Definition:

θ_S -- Knee Splay

Figure 1.

Schematic diagram of marker placement and joint angle definitions. **A:** Sagittal plane view showing marker placements and definitions of Trunk Lean (θ_L), Hip (θ_H), Knee (θ_K), and Ankle (θ_A) angles. **B:** Transverse plane view showing marker placements and definition of the Knee Splay (θ_S) angle. Lateral Knee Splay was defined as the displacement of the thigh away from a line drawn perpendicular to the L Hip – R Hip segment (i.e., the pelvis).

Figure 2.

Processing of Kinematic data. **A:** Continuous time series of Trunk Lean angles for a typical subject. **B:** Each continuous time series was divided into individual cycles. Dotted vertical lines indicate points of top-dead-center. The range of motion (ROM) for each cycle was defined as the maximum minus minimum joint angle during that cycle, as shown. **C:** The original ROM time series (thin line) and box-car filtered moving average trend (thick line). The same analyses were performed for all joint angle time series from all subjects.

Figure 3.

Processing of EMG data. **A:** Continuous time series of tibialis anterior (TA) EMG data for a typical subject. **B:** Each time series was divided into individual cycles. Dotted vertical lines indicate points of top-dead-center. The median frequency (MDF) of each EMG signal was computed for the down stroke and up stroke portions of each revolution and then averaged to obtain a single value of MDF for that revolution, as shown. **C:** The original MDF time series (thin line) and box-car filtered moving average trend (thick line). The same analyses were performed for all EMG time series from all subjects.

Figure 4. Cross-Correlation Analyses. **A:** Box-car filtered ROM (Fig. 2) and MDF (Fig. 3) data. **B:** Cross-correlation of the signals in **A**. Dashed horizontal lines indicate ± 2 standard deviations from zero. The peak cross-correlation (PCC) value is indicated by the circle. **C:** The same cross-correlations from **B** after normalizing to the number of standard deviations. Cross-correlation peak magnitudes (PCC; # of Std. Dev.) and time lags (τ ; # of cycles) at which these peaks occurred were extracted for statistical analyses. Similar cross-correlations were performed between each kinematic and muscle EMG signal obtained from each subject.

Figure 5.

Average power output (Watts) and cadence (revolutions per minute) as a function of the percentage of the trial completed for each subject. Symbols represent the average across subjects. Error bars represent between-subject standard deviations. All subjects maintained both power output and cadence at approximately constant levels until the final 10% of the fatigue trial, despite wide variations across subjects in time-to-fatigue (Table 2).

Figure 6.

A: Trunk Lean angles for Subjects 2 and 6 (randomly chosen). All subjects exhibited increased trunk lean. Early, abrupt shifts in trunk lean are evidence of non-monotonic changes in trunk lean angle. Nearly all subjects exhibited similar shifts in trunk lean. **B:** Ankle angles for Subjects 2 and 6. Nearly all subjects displayed decreases in ankle angle. **C:** Example sagittal plane Knee joint angles and transverse plane Knee Splay angles and associated ROM time series for a typical subject (#10). While the specific patterns of variations over time were subject specific, nearly all subjects exhibited greater nonstationary and non-monotonic variations in their joint ROM time series than in their joint angle data. Similar non-monotonic changes in kinematics were exhibited by most joint angle measures obtained from most subjects.

Figure 7.

A: Median frequency (MDF) results for the four muscles recorded from a typical subject. Thin lines show the original averaged median frequency values. Thick black lines show the boxcar filtered median frequencies. The thick straight lines represent linear regression lines computed from the original median frequency data. The slopes of these lines were used to assess general trends in muscle fatigue across the experiment. While the specific patterns of variations over time were highly subject specific, all subjects exhibited substantial nonstationary and non-monotonic variations in MDFs for all muscles recorded. **B:** Group means and 95% confidence interval upper bounds from one-tailed T-tests for the slopes of the EMG MDF plots. Negative slopes indicate muscle fatigue. Across subjects, slopes were significantly less than zero for BF

($p = 0.020$) and GAS ($p = 0.018$) muscles, but not for VL ($p = 0.701$) and TA ($p = 0.346$) muscles. Individually, every subject exhibited significant fatigue in at least 1 muscle group and every muscle exhibited significant fatigue in at least 3 subjects (Table 2).

Figure 8.

Peak cross-correlation (PCC) values vs. time lags (τ) for cross-correlations of EMG median frequencies (MDF) and Mean Joint Angle (MA) data for Hip, Ankle, Trunk Lean, and Knee splay angles. Similar results were obtained for Knee angles and for ROM data. Each symbol indicates the result for one subject. Different symbol types represent the four different muscle groups (VL, BF, Gas, and TA). Changes in coordination patterns in response to changing local muscle fatigue varied across both muscle groups and subjects. With the exception of PCC values for mean Hip joint angles ($p = 0.018$), there were no significant differences between the four muscles groups for either time lags, τ ($0.18 < p < 0.89$), or PCC values ($0.24 < p < 0.84$). These data were therefore pooled across muscle groups for further statistical analyses (see Fig. 9).

Figure 9.

Statistical analyses of cross-correlation results shown in Fig. 8. **A:** Means (circles) and one-tailed 95% confidence intervals for cross-correlation time lags (τ) for mean joint angles (MA) and joint angle ranges of motion (ROM). Changes in mean Trunk Lean angles, mean Hip angles, and Trunk Lean ROM were all significantly *preceded* by changes in muscle fatigue state. **B:** Means (circles) and two-tailed 95% confidence intervals for normalized peak cross-correlation (PCC) magnitudes for mean joint angles (MA) and joint angle ranges of motion (ROM). Increases (or decreases) in EMG median frequencies were associated with significant increases (or decreases) in mean Trunk and Knee Splay angles and in Trunk and Ankle ROM.

There was a trend for increases (or decreases) in EMG median frequencies were associated with decreases (or increases) in mean Hip angles ($p = 0.060$) and Hip ROM ($p = 0.077$).

Table 1

Basic characteristics for age (yrs), body mass (kg), and VO₂Max (mL/kg/min) for each cyclist.

Subject #	Age	Body Mass	VO ₂ Max
2	41	76.5	55.43
3	25	74.1	64.80
4	35	82.6	58.30
5	32	83.4	63.47
6	25	71.1	63.02
7	18	68.4	64.42
8	35	70.1	59.60
Mean ± SD:	30.14 ± 7.84	75.17 ± 5.97	61.29 ± 3.56
Range:	18–41	68.4–83.4	55.4–64.8

Table 2

Total years of cycling training (yrs), number of hours training per week during Low volume and High volume weeks (self-report), and time to fatigue (min:sec) for each cyclist.

Subject #	Years of Training	Training – Hrs/Wk		Time to Fatigue
		Low Vol.	High Vol.	
2	16	15	15	4:58
3	10	8	14	8:03
4	10	10	15	7:15
5	8	15	20	9:36
6	11	10	15	5:23
7	6	15	20	5:08
8	6	8	15	5:30
Mean ± SD:	9.6 ± 3.5	11.6 ± 3.3	16.3 ± 2.6	6:33 ± 1:47
Range:	6–16	8–15	14–20	4:58–9:36

Table 3

Muscles that exhibited significant evidence of sustained localized muscle fatigue, as indicated by a statistically significant negative slope of the median frequency (MDF) vs. pedal revolutions plots (e.g., Fig. 7A). While the patterns of muscle fatigue varied between subjects, every subject exhibited significant sustained fatigue in at least one muscle group and every muscle group exhibited significant fatigue in at least 3 subjects. In total, 18 of the 28 muscles measured (64.3%) exhibited significant fatigue.

Subject #	VL	BF	Gas	TA	Total
2	Y	Y	Y	Y	4
3			Y		1
4		Y	Y		2
5		Y	Y	Y	3
6	Y	Y			2
7	Y	Y		Y	3
8		Y	Y	Y	3
Total:	3	6	5	4	18