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1 **How do the mechanical demands of cycling affect the information content of the EMG?**

2  
3 James M. Wakeling<sup>1</sup> and Emma F. Hodson-Tole<sup>2</sup>

4  
5 <sup>1</sup>Department of Biomedical Physiology and Kinesiology, Simon Fraser University, Burnaby, BC,

6 Canada

7 phone 1-778-7828444

8 fax 1-778 7823040

9 [wakeling@sfu.ca](mailto:wakeling@sfu.ca)

10  
11 <sup>2</sup>School of Healthcare Science, Manchester Metropolitan University, Manchester, United Kingdom

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13  
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18 **Abstract**

19 **Purpose:** The persistence of phase-related information in EMG signals can be quantified by its entropic  
20 half-life, EnHL. It has been proposed that the EnHL would increase with the demands of a movement  
21 task, and thus increase as the pedalling power increased during cycling. However, simulation work on  
22 the properties of EMG signals suggests that the EnHL depends on burst duration and duty cycle in the  
23 EMG that may not be related to task demands. This study aimed to distinguish between these alternate  
24 hypotheses. **Methods:** The EnHL was characterized for 10 muscles from nine cyclists cycling at a range  
25 of powers (35 to 260 W) and cadences (60 to 140 r.p.m.) for the raw EMG, phase-randomized surrogate  
26 EMG, EMG intensity and the principal components describing the muscle coordination patterns.  
27 **Results:** There was phase-related information in the raw EMG signals and EMG intensities that was  
28 related to the EMG burst duration, duty cycle pedalling cadence and power. The EnHLs for the EMG  
29 intensities of the individual muscles (excluding quadriceps) and for the coordination patterns decreased  
30 as cycling power and cadence increased. **Conclusions:** The EnHLs provide information on the structure  
31 of the motor control signals and their constituent motor unit action potentials, both within and between  
32 muscles, rather than on the mechanical demands of the cycling task *per se*.

33

34 **Key words:** Sample entropy, entropic half-life, principal component analysis, skeletal muscle,  
35 coordination, firing statistic

36

37

## 38 **Introduction**

39           The EMG signal represents the superposition of motor unit action potentials from activated  
40 motor units and is commonly assessed to identify characteristics such as firing rates of individual units  
41 (1) or recruitment of populations of units (2). Within an individual muscle, more generalized features of  
42 activity, such as the time of onset and offset and the magnitude of each burst, can also be determined  
43 from fluctuations in the intensity envelope of the EMG recorded during activities such as cycling.  
44 Additionally, the coordination of multiple muscles within each limb can also be assessed and has been  
45 shown to be a key determinate to cycling performance (3).

46           However, the structure (temporal organization of variability) within the EMG signal may also  
47 contain information on the challenge posed by a movement task and give us new insight to the motor  
48 control strategies that govern the muscles to meet these challenges. One way to determine the structure  
49 of a signal is to calculate its Entropy (4), and for EMG signals this can be done using a particular  
50 approach termed Sample Entropy. Sample Entropy (5) identifies how often small segments of data (with  
51  $m$  sample points) from a signal would be identified within the signal (within a specified tolerance)  
52 compared to segments that contained one more ( $m+1$ ) sample point. A low value of Sample Entropy  
53 reflects a high degree of structure in the signal, with higher Sample Entropy reflecting a more chaotic  
54 structure. This approach was further developed (6) and has been used to quantify the rate at which signal  
55 structure decays within EMG signals (7) using a measure termed the entropic half-life, EnHL.  
56 Calculation of EnHL involves resampling the original EMG signal at increasingly larger time steps, to  
57 identify the time-scale at which structure in the signal is lost as the resampled signal transitions to  
58 containing random fluctuations.

59 Muscle activity during cycling varies with both cadence and power (3, 8-15). When increasing  
60 the pedalling cadence the burst duration decreases (8) but not as much as the cycle duration, and so the  
61 duty cycle increases. All EMG signals recorded during cycling have structure reflecting the  
62 neuromuscular control of their motor units, and a theoretical analysis of the factors that shape surface  
63 EMG signals and their effect on EnHLs predicted that EMG signals at the fastest cadences (short burst  
64 durations and longer duty cycles) would result in shorter EnHLs (7). However, this is contrary to the  
65 view that high-cadence cycling represents a demanding task that would result in greater, or more  
66 persistent, structure to the neuromuscular control strategy. Whether EMG signal structure during cycling  
67 reflects structure to the neuromuscular control strategy is therefore not clear. We therefore aim to  
68 address this gap in knowledge by investigating structure of raw EMGs, EMG intensity and muscle co-  
69 ordination patterns and how EnHL changes in response to cycling demand. Below we provide a  
70 rationale for why each signal may be expected to be structured and the physiological responses to the  
71 cycling demand that may influence that structure.

72 Within the raw EMG each motor unit action potential occurs at a distinct time and leaves  
73 characteristic spectral components in the EMG signal (16). If the variability in the EMG signal is  
74 organized over time (i.e. it is structured) the action potential shapes, amplitudes and the relative phase  
75 between different motor units potentials' would be expected to influence the characteristics of the  
76 structure. As motor unit recruitment responds to the mechanical demands of cycling (17), changes in  
77 raw EMG signal structure would be predicted to occur in response to altered cycling mechanical  
78 demands.

79 The EMG intensity provides an envelope of the signal, smoothing out some of the time-  
80 dependent fluctuations from the raw EMG signal. It is therefore possible that EMG intensities from  
81 individual muscles may be more structured (i.e. fewer random fluctuations) than the raw EMG. This

82 may mean that burst parameters within each muscle, such as burst duration and duty cycle, are the main  
83 factors influencing the individual EnHLs. Cycling at higher cadences results in decreased burst duration  
84 (8), and higher duty cycles, that have led to predictions of shorter EnHLs (7). These predictions have yet  
85 to be tested on experimental data.

86 Muscle coordination patterns that consist of the EMG intensities from many different muscles  
87 may show greater variability due to the higher-dimensionality of the additional muscles, and so the  
88 EnHLs for coordination may be shorter than for the individual muscles as the more variable structure of  
89 the coordination may dissipate over shorter time scales. EnHLs from such multi-muscle coordination  
90 patterns may reflect the net response of the neuromuscular system and may therefore provide insight  
91 into the structure of variability of muscle recruitment patterns tolerated by the nervous system for  
92 different task demands. Enders et al. (18) showed an increase in EnHL from 9 ms to 16 ms between 150  
93 W and 300 W power conditions for cycling at 90 r.p.m. However, it is not known if this finding can be  
94 generalized across a range of cycling conditions particularly as the variability in and composition of the  
95 muscle coordination during cycling depends in a complex and non-linear fashion on both the power  
96 output and the cadence (8).

97 The purpose of this study was thus to explore the EnHL from the level of the raw EMG signal  
98 through to multi-muscle co-ordination patterns during cycling. We address the question of whether the  
99 EnHLs at these different signal levels vary with the opposing demands of the cycling task (high power  
100 output and cadence), or with EMG parameters (burst duration and duty cycle).

101

102 **Methods**

103 The entropic half-life, EnHL, was determined from a large cycling data set that has been  
104 described in a previous study (15). In brief, nine club to national level racing cyclists pedaled on an  
105 indoor ergometer at a range of cadences (60, 80, 100, 120 and 140 r.p.m.) at a low and fixed crank  
106 torque of 6.5 N m, and also cycled at a range of crank torques (12.9, 25.1, 32.4 and 39.9 N m) at the low  
107 cadence of 60 r.p.m. Cyclists pedaled for 5-10 seconds to reach a steady-state speed, and then data were  
108 recorded for a further 30 s for each trial. The cycle conditions were presented in a random order, and  
109 repeated in three blocks in order to minimize bias due to increasing fatigue and body temperature. A  
110 total of 6804 pedal cycles were analyzed (9 subjects x 3 blocks x 9 conditions x 28 cycles per condition).  
111 Pedalling cadence was maintained using visual feedback, and independently recorded with a pedal  
112 switch; post analysis showed it was on average 1.3 r.p.m. higher than the target velocity, and varied with  
113 a standard deviation of only 1.1 r.p.m. within each trial; there was a slight increase in variability in pedal  
114 cadence at the higher cadences, with the 140 r.p.m. trials having standard deviations of 1.8 r.p.m.. A 45  
115 s rest period was given between each condition. Each participant gave written informed consent in  
116 accordance with the Simon Fraser University's policy on research using human subjects.

117 Bipolar Ag/AgCl surface EMG electrodes (10 mm diameter, 21 mm interelectrode distance)  
118 were placed in the centre of the muscle bellies of the tibialis anterior (TA), medial gastrocnemius (MG),  
119 lateral gastrocnemius (LG), soleus (Sol), vastus medialis (VM), rectus femoris (RF), vastus lateralis  
120 (VL), biceps femoris long head (BF), semitendinosus (ST), and gluteus maximus (Glut) of the left leg  
121 and surface EMG was recorded at 2000 Hz. A pedal switch allowed the time of top-dead-centre to be  
122 identified.

123 *The entropic half-life, EnHL, was calculated for each signal using the following procedure.*  
124 Initially the EnHL was calculated from the raw EMG signals (as directly recorded from the EMG  
125 amplifiers). These signals were filtered (Butterworth, high-pass with 10 Hz cutoff) and standardized to

126 have a mean of zero and standard deviation of one. The SampEn was calculated using a freely available  
 127 software package (19).  $\text{SampEn}(m, r, N)$  quantifies the regularity of a time series of length  $N$ , reflecting  
 128 the conditional probability that two sequences of  $m$  consecutive data points, similar to one another  
 129 within a tolerance ( $r$ ), will remain similar when a consecutive data point is added (20). Values of  $m=0$   
 130 and  $m=1$ , were recorded with  $r=0.2$  for a range of reshape-scales from 1 ms to 1 s (6). For each reshape-  
 131 scale, the SampEn for  $m=1$  was normalized to the corresponding SampEn for  $m=0$  (which can be  
 132 interpreted as the negative logarithm of the probability of a match of length one (19)): this stage is  
 133 computationally faster but equivalent to normalizing to the random permutation of the signal as  
 134 described by Enders and co-workers (18). EnHL is the time-scale at which the normalized SampEn  
 135 (from across the reshape-scales) reached a value of 0.5 (18), indicating the time-scale that the time series  
 136 transitioned from ordered to random structure. It therefore provides a measure of the persistence of  
 137 structure in the EMG intensity envelopes from each individual muscle.

138 The intensity envelope for the EMG signals was calculated for each muscle, using an EMG-  
 139 specific wavelet analysis (21), where each wavelet  $k$  had a centre frequency  $f_c(k)$ , and the sum of the  
 140 intensities  $i_k$  over the frequency band 11 to 432 Hz ( $1 \leq k \leq 10$ ) generated the total intensity that was a  
 141 close approximation to the power within the EMG signal. The mean frequency  $f_m$  for the EMG intensity  
 142 (16) was:

143 
$$f_m = \frac{\sum_k f_c(k) i_k}{\sum_k i_k}$$

144 The EMG intensities for each muscle and trial were mean normalized and each intensity trace was  
 145 resampled at 1000 Hz. The burst durations for the normalized EMG intensities were taken as the  
 146 duration that the intensity was greater than 5% of the maximum for each pedal cycle, and the duty cycle  
 147 was the proportion of this burst duration relative to the period of each pedal cycle. The mean frequencies



148 of the EMG intensities were calculated for each pedal cycle. EnHLs were calculated for the EMG  
149 intensities in the same manner as describe above for the raw EMG signals.

150 The muscle coordination patterns were quantified by principal component analysis. For each  
151 cycling trial the coordination patterns for each time instant were generated from the normalized EMG  
152 intensities for all ten muscles, and placed in an  $p \times N$  matrix  $\mathbf{A}$  ( $p = 10$  muscles,  $N$  is number of time  
153 points for 28 pedal cycles at the 1000 Hz sample rate). The mean intensity vector (mean intensity for  
154 each muscle in  $\mathbf{A}$ ) was subtracted from  $\mathbf{A}$ , from which the covariance matrix  $\mathbf{B}$  was calculated. The  
155 principal components, PCs, of  $\mathbf{A}$  were described by Eigen analysis of  $\mathbf{B}$ : the PC loading scores were  
156 calculated from  $\xi' \mathbf{A}$ , where  $\xi'$  are the transpose of the Eigen vectors of  $\mathbf{B}$  and were ordered into  
157 decreasing Eigen values. The loading scores for the first six PCs explained 91 % of the variance within  
158 matrix  $\mathbf{B}$ , and were used as signals for the EnHL analysis, as above, providing a measure of the  
159 persistence of structure in the multi-muscle coordination patterns.

160 The EnHL was additionally calculated for phase-randomized surrogates (18) of the raw EMG  
161 signals and the EMG intensity envelopes for each muscle and each trial. Phase-randomized surrogate  
162 signals have the same power spectrum and auto- correlation as the original signal; however, the structure  
163 encoded in the phase is removed. The process of phase-randomization removes structure due to the  
164 bursting patterns of the EMG, due to regularity of firing or synchronization of the motor units, and from  
165 the shape of the individual motor unit action potentials (Fig. 1). Thus, the surrogate signals can be used  
166 as reference values for signals with no structure (18).

167 The factors influencing the EnHL values were tested using mixed model analyses of covariance  
168 (Minitab version 16, State College, PA): cadence, power, burst duration, duty cycle and EMG intensity  
169 were included as covariates, and subject was a random factor. ANCOVAs were evaluated for the EnHL

170 for the raw EMG and for the EMG intensities, and these used the muscle as an additional factor and  
171 muscle  $\times$  power, cadence  $\times$  power and muscle  $\times$  cadence as interaction terms. A third ANCOVA was  
172 evaluated for the EnHL for PC loading scores, and this used the PC number as an additional factor, and  
173 PC number  $\times$  power, cadence  $\times$  power and PC number  $\times$  cadence as interaction terms. For this  
174 ANCOVA the burst duration, duty cycle and EMG intensity values used as covariates were taken as the  
175 mean values across the ten muscles. Statistical effects were deemed significant at  $p < 0.05$ , and data are  
176 reported as mean  $\pm$  standard error of the mean.

177

## 178 **Results**

179 The cycling conditions encompassed a range of powers, of which a set was at a low-cadence of  
180 60 r.p.m. but at increasing crank torques, whilst a second set was at a low crank torque but with  
181 increasing cadence. There was a general increase in EMG intensity for each muscle with power, with  
182 greater EMG intensities occurring for the higher-cadence conditions for each given power (Fig. 2A).  
183 There was a cadence-specific effect on the burst durations with shorter burst durations occurring for  
184 faster cycling cadences (Fig. 2B). There was a general but small increase in duty cycle with power for  
185 all muscles (Fig. 2C).

186 The mean EnHLs for each muscle for the phase-randomized surrogate EMGs ranged between  
187  $5.23 \pm 0.04$  to  $9.05 \pm 0.05$  ms ( $N = 269$ ). These EnHLs showed a strong negative correlation with the  
188 mean frequency of the EMG intensities ( $r^2 = 0.94$ ; Fig. 3A), with even higher correlations occurring ( $r^2$   
189  $= 0.98$ ) when the EnHLs for the phase-randomized surrogate EMG intensities were correlated against  
190 the period of the mean frequencies ( $1 / \text{mean frequency}$ ) of the EMG intensities.

191 The mean EnHLs for each muscle for the raw EMGs were typically greater than their phase-  
192 randomized values and ranged between  $1.32 \pm 0.10$  and  $45.64 \pm 5.50$  ms ( $N = 28$ ) for the least  
193 demanding condition of 60 r.p.m. at 35 W. The mean EnHLs for each muscle for the raw EMGs showed  
194 a general decrease at the higher cadences when the torque was held constant (Fig. 4A). As the torque  
195 increased for the low cadence conditions there was an increase in EnHL for the raw EMG signals for  
196 VM, RF, VL and Glut, with a decrease for the remaining muscles (Fig. 4B). The EnHLs for each muscle  
197 for the raw EMGs neither correlated with the EnHLs for the phase-randomized raw EMGs ( $0.01 < r^2 <$   
198  $0.15$ ), nor with the mean frequency of the EMG intensities ( $0.01 < r^2 < 0.10$ ; Fig. 3B).

199 The EnHLs for the EMG intensities ranged between  $20.26 \pm 1.40$  and  $36.70 \pm 1.67$  ms ( $N = 28$ )  
200 for the least demanding condition of 60 r.p.m. at 35 W. There was a general decrease in the EnHLs to  
201 values between  $17.98 \pm 0.59$  and  $24.16 \pm 0.38$  ms as power output increased for both the increasing  
202 torque and increasing cadence conditions, with the EnHL being more sensitive to changes in cadence  
203 than crank torque (Fig. 5). However, the exception to this was the quadriceps muscles that increased  
204 their EnHLs for the increasing torque conditions to reach their maxima, between  $32.81 \pm 1.80$  and  $35.71$   
205  $\pm 1.44$  ms, at the 260 W power. The ANCOVA showed that decreases in EnHL were significantly  
206 associated with increases in both cadence and power, and the EnHL showed a significant negative  
207 association with EMG intensity and duty cycle, and a positive association with burst duration (Table 1).  
208 The mean EnHLs for the EMG intensities for each muscle were greater than the mean EnHLs for the  
209 intensities of the phase-randomized surrogate EMG and correlated with neither the mean frequencies of  
210 the EMG intensities ( $r^2 < 0.01$ ), nor with the EnHLs for the intensities of the phase-randomized signals  
211 ( $r^2 < 0.01$ ).

212

213 **Table 1. Statistical results for the ANCOVAs.** Columns show the effect of the covariate  
 214 (up/down arrow indicate positive/negative direction of effect; - indicates no significant effect), the  
 215 degrees of freedom (DF), *F*-value and *p*-value for the tests. Rows show the sources of variation:  
 216 factors (subject: random; PC number and muscle), covariates (cycling cadence and power; EMG  
 217 intensity; burst duration (BD) and duty cycle (DC)), interaction terms, and the error term.

	Raw EMG				EMG intensity				Muscle coordination			
	Covar.	DF	<i>F</i>	<i>p</i>	Covar	DF	<i>F</i>	<i>p</i>	Covar	DF	<i>F</i>	<i>p</i>
Subject		9	16.1	<0.00		9	10.29	<0.00		9	30.9	<0.00
			7	1				1			0	1
PC #										5	3.51	0.004
Muscle		9	53.2	<0.00		9	20.32	<0.00				
			6	1				1				
Cadence	-	1	1.29	0.256	↓	1	9.58	0.002	↑	1	6.17	0.013
Power	↑	1	8.43	0.004	↓	1	20.44	<0.00	-	1	0.21	0.648
								1				
Intensity	↑	1	304.	<0.00	↓	1	17.89	<0.00	-	1	0.29	0.587
			0	1				1				
BD	↑	1	14.1	<0.00	↑	1	52.97	<0.00	↑	1	4.59	0.032
			2	1				1				
DC	↓	1	22.3	<0.00	↓	1	195.3	<0.00	-	1	0.93	0.335
			7	1			9	1				
Muscle × Power		9	31.8	<0.00		9	41.45	<0.00				
			4	1				1				
Muscle × Cadence		9	24.6	<0.00		9	6.38	<0.00				
			5	1				1				
Cadence × Power		1	5.33	0.021		1	30.63	<0.00		1	7.10	0.008
								1				
PC # × Power										5	1.40	0.222
PC # × cadence										5	9.52	<0.00
												1
Error		26				263				157		
		06				6				9		

218

219 The EnHLs for the PC loading scores describing the muscle coordination patterns were shorter  
 220 than the EnHLs for the EMG intensities for the individual muscles (Fig. 6). The EnHLs for the muscle  
 221 coordination patterns ranged between  $14.81 \pm 1.22$  and  $21.80 \pm 1.81$  ms ( $N = 28$ ) for the least

222 demanding condition of 60 r.p.m. at 35 W. The ANCOVA (Table 1) showed significant interaction  
223 effects PC number, with the higher PCs resulting in lower EnHLs (Fig 5A). The EnHLs increased with  
224 burst duration, and a significant interaction between PC number  $\times$  cadence showed a greater cadence  
225 dependence for the higher PCs (Fig. 6A).

226

## 227 **Discussion**

228 *Is there information in the EMG signal related to EnHL?*

229 The entropic half-life analysis in this study shows that there is persistent and non-random structure in all  
230 levels of the EMG signals analysed, indicating that further investigation of structure in EMGs is  
231 warranted. The phase of a signal has been shown to contain important information (22). At the level of  
232 the raw EMG this may reflect the shapes of the MUAPs, variability (or lack-of) in the firing rates of the  
233 motor units and coherence between different motor units. The raw EMG signals typically had longer  
234 EnHLs than their phase-randomized surrogates (Fig. 3), so there is information in the phase properties of  
235 the EMG. The EnHL for the phase-randomized signals correlated with the mean frequency of the EMG  
236 and is of similar time-scale to the period of that mean frequency. This suggests that the fundamental  
237 presence of voltage fluctuations (from the motor unit action potentials that make up the raw EMG) that  
238 occur at distinct times and with distinct frequency properties, manifest as time-dependent structure to the  
239 phase within that signal. Each motor unit action potential occurs at a distinct time and leaves  
240 characteristic spectral components in the surface EMG signal (16) and these can also be resolved using  
241 time-frequency signal processing techniques such as wavelet analysis (21, 23). The raw EMG had more  
242 persistent structure (longer EnHLs) than the phase-randomized surrogate signals, but these EnHLs no  
243 longer correlated with the mean frequency of the EMGs (Fig. 3B). Thus the additional structure within

244 the raw EMG derived from other features that are likely to include variability in the discharge of  
245 individual motor units (24-25), synchronicity between motor units (26) and activation-deactivation burst  
246 duration and duty cycle. This persistent structure and thus information in the raw EMG (Fig. 4) was also  
247 found in the individual muscle EMG intensity envelopes (Fig. 5) and in the multi-muscle co-ordination  
248 patterns (Fig. 6), although the timescale over which structure persisted and the changes in response to  
249 cycling demand differed. The factors that may cause these differences are therefore considered below.

250 *Why does EnHL differ between the EMG signals analysed?*

251 The raw surface EMG signals are the superposition of motor unit action potentials in the  
252 underlying muscle. Frequency information in the raw signal is strongly correlated with EnHL, even  
253 when the EMG is phase-randomized, and this information reflects the time-varying voltage fluctuations  
254 of the constituent motor unit action potentials. The raw EMG signals and the EMG intensities  
255 additionally contain phase-related information seen by their EnHLs being longer than their phase-  
256 randomized surrogates. EMG signals are the convolution of the firing statistics and the time-varying  
257 properties of the individual MUAPs, and so additional information in the EnHLs for the EMG intensities  
258 likely derives from the structure and variability in the firing. This structure is related to the discharge of  
259 individual motor units and synchronicity between motor units, both of which vary with activation levels  
260 and the proficiency for doing tasks. Whilst the EnHL for the EMG intensities was related to the burst  
261 durations for the EMG, they were considerably shorter than for those burst durations, and so fluctuations  
262 in the firing statistics are more rapid than each burst of activity.

263 The EnHL values from the EMG intensities of individual muscles were generally longer than  
264 those for the raw signals, likely reflecting smoothing out some of the time dependent fluctuations in the  
265 raw EMGs when the envelope of the signal is calculated. This means that burst parameters, such as

266 duration and duty cycle, may dominate the structure of the EMG intensity signal and indeed ANCOVA  
267 revealed a negative associations between EnHL and duty cycle, and a positive association between  
268 EnHL and burst duration (Table 1); and this is consistent with changes in EnHL that were simulated  
269 across this physiological range (7). It is these changes in burst duration that appear to play the major role  
270 in affecting the EnHL in these EMG intensities. It is of note that the burst durations (177.4 – 526.8 ms)  
271 were an order of magnitude greater than the EnHL values (15.7 – 36.7 ms; Fig. 5), and thus the reason  
272 for the reduced EnHL is probably not limited by the actual burst duration.

273           In the quadriceps muscles, however, there were longer EnHLs in the raw EMGs than in the EMG  
274 intensities in some cycling conditions. Structure persisted over longer time periods within the raw  
275 signals from these muscles when compared to others (Fig. 4), and this structure must have been related  
276 to time dependent fluctuations in the raw EMGs that were removed when the intensity envelope was  
277 calculated. The significance of these differences is difficult to determine from the data available from  
278 this study, but it is interesting to note that these muscles were the only ones in which significant changes  
279 in EnHL occurred between cycling conditions (greater EnHL associated with increasing torque  
280 conditions). It could be suggested that differences in motor unit (e.g. size, spatial distribution) and  
281 muscle anatomical (e.g. size, fibre pennation angle) features could combine to influence the raw EMG  
282 signals and hence EnHLs. However, similar EnHL values occurred across all muscles for some of the  
283 cycling conditions (e.g. Fig. 4), suggesting that the raw EMG signal properties altered in response to  
284 task demand. Important time-varying differences in the neuromuscular drive across individual muscles  
285 may therefore occur in response to task demands. These may reflect differences in the neuromuscular  
286 control required to elicit different mechanical roles of each muscle over the time course of a task (e.g.  
287 power production, force transfer), the dynamics of which warrants further investigation.

288 No muscle works in isolation, however, and as such it is valuable to consider the amalgamated  
289 responses of multiple muscles to task demand. Here, this was done by combining the intensity traces  
290 into multi-muscle coordination patterns, quantified by their PC loading scores, which resulted in EnHLs  
291 that were shorter than the EnHLs for the individual muscles. This was as expected, because the more  
292 muscles that are amalgamated into coordination patterns the more ways in which those patterns can vary,  
293 or the greater the chance the signal structure will dissipate. The EnHLs for the coordination calculated  
294 from this study are generally longer than those reported by Enders et al. (18), however, the calculated  
295 EnHLs are sensitive to the filter cut-off frequencies used before the sample entropy analysis: in this  
296 study the data were low-pass filtered with a 10 Hz cut-off due to the pedal cadences reaching 140 r.p.m.,  
297 as opposed to the Enders et al. (18) study where a 2.5 Hz was used related to the cadence of 90 r.p.m..

298 *Why does EnHL differ across cycling conditions?*

299 Variability is a ubiquitous and fundamental characteristic of human movement (27), however  
300 variability may decrease with task constraints such as maximizing power output or pedalling velocity.  
301 Previous studies have shown that the dimensionality of muscle coordination patterns reduces for  
302 pedalling at greater power outputs (18, 28), and the variability of the muscle coordination patterns  
303 reduces at high cadences (8). However, the approaches used in those studies did not consider the  
304 temporal organization of variability that can be studied using the EnHL approach. Specifically, by  
305 analyzing the structure of signals over the time-course of the whole trial EnHL includes consideration of  
306 how one pedal cycle impacts subsequent cycles (i.e. effects of the order of data points across multiple  
307 pedal cycles is conserved in the analysis). The EnHL for the EMG intensities for the quadriceps muscles  
308 increased with power output for the low cadence, with increasing torque conditions. However, the EnHL  
309 for the remaining muscles, and for the increasing cadence conditions, showed general decreases with  
310 both power and cadence (Fig. 5). While the average variability of muscle coordination decreases with



311 greater task demand (8, 18, 28), the data presented here suggest that the time dependent structure of  
312 these coordination patterns has greater variability during more challenging movement tasks. The shorter  
313 EnHLs recorded may reflect greater interference, or more frequent adjustments, from the central nervous  
314 system or may suggest that during more challenging tasks the nervous system was more tolerant of time-  
315 varying fluctuations in coordination patterns; as has been suggested for postural balance tasks (29-30).  
316 Further assessment of the temporal structure of variation in muscle co-ordination patterns and changes in  
317 response to task demand are therefore warranted.

318         Previously, the EnHL for muscle coordination patterns has been shown to increase for cycling at  
319 higher power output (18). The muscle coordination patterns were calculated from the time-varying EMG  
320 intensities from seven lower extremity muscles that included three of the quadriceps, and it was found  
321 that the principal coordination pattern for the high-power condition was dominated by signal from the  
322 rectus femoris (18). In our current study the rectus femoris was one of the muscles that showed an  
323 increase in EnHL as crank torque increased (Fig. 5B). However, the coordination patterns determined  
324 here contain signals from 10 muscles, of which the majority did not show increases in EnHL with power  
325 (Fig. 5). Furthermore, due to the large number of different conditions that we tested, we calculated a  
326 common set of principal coordination patterns across all conditions: whilst such patterns are influenced  
327 by the variability in the EMG intensity from the rectus femoris, the other muscles still have substantial  
328 contribution across all principal patterns that are identified (8) and these muscles showed decreasing  
329 EnHL with increased power output. These methodological differences explain the finding in this study  
330 that the EnHL for the muscle coordination patterns did not increase with the increasing power (at a fixed  
331 cadence) conditions.

332         It is possible that the EnHLs (for both individual muscles and for the coordination between  
333 muscles) would have showed greater increases related to the increased demands of cycling at higher

334 power outputs than those tested in this study. It should be noted that the highest power tested in this  
335 study (260 W) is considerably less than the maximum powers that can be achieved by competitive  
336 cyclists (of over 1000 W; (31)) and so may have only been of limited challenge to the cyclists tested.  
337 Additionally, the relative intensities of the cycle conditions were not normalized to the maximum power  
338 achievable by each participant, and so the relative demands of the conditions may also vary between the  
339 cyclists tested.

340 We therefore conclude that there is structure at all levels of the EMG signals analysed here, with  
341 the persistence of this structure differing between muscles and in response to cycling task demand.  
342 Differences in structure relate to the underlying motor unit recruitment patterns and interacts with the  
343 electromyogram burst parameters. Further work is however required to determine the functional  
344 significance of the changes found here and to improve understanding of neuromuscular control of time  
345 dependent changes in muscle recruitment during dynamic tasks.

346

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353 The authors declare that the results of the study are presented clearly, honestly, and without fabrication,  
354 falsification, or inappropriate data manipulation.

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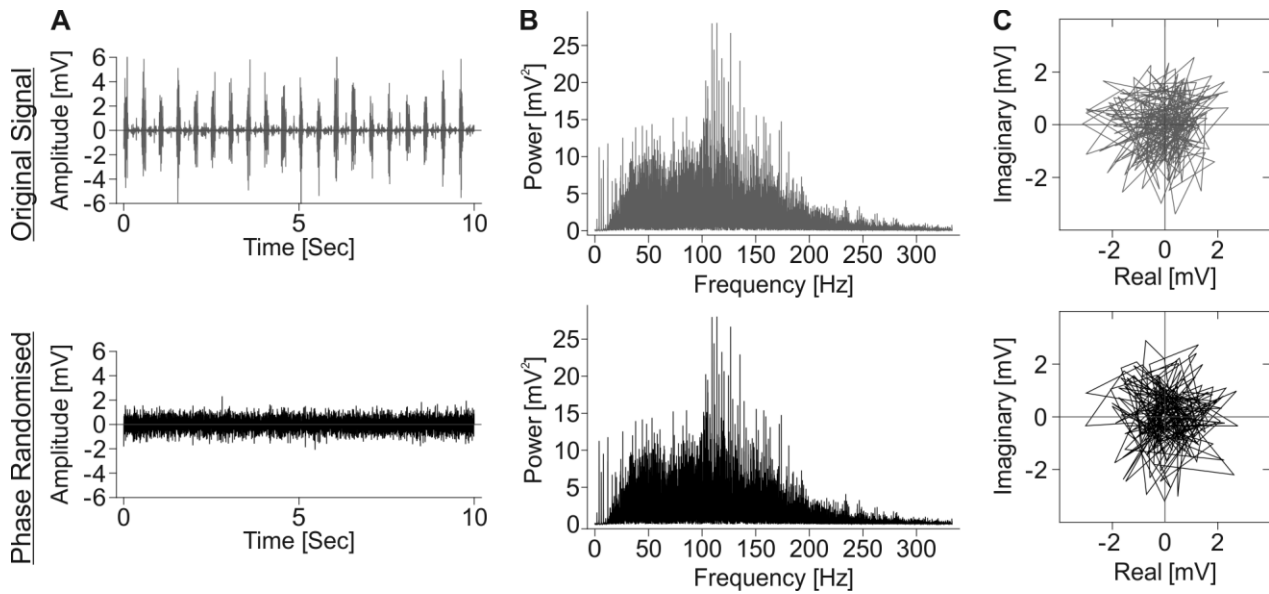
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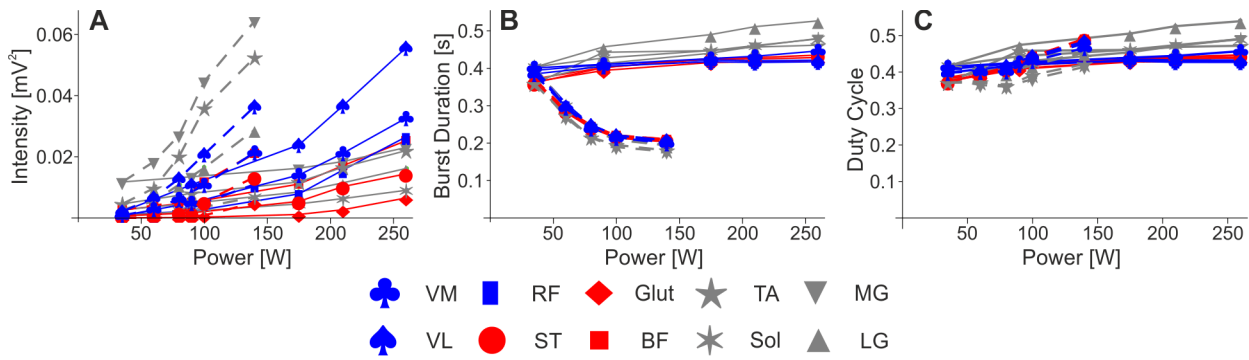
421 **Figures**



422

423 **Fig. 1.** Signal properties. Raw (top row) EMG shown in gray and phase-randomized surrogate (bottom  
424 row) signals shown in black. Time-varying signals (A), power spectra (B) and Argand diagrams  
425 showing the phase relations (C). Note that the power spectra (B) are the same for the raw signal and the  
426 phase-randomized surrogate. However, the signals have different phases (C) resulting in different burst  
427 characteristics as seen in the time-varying signals (A).

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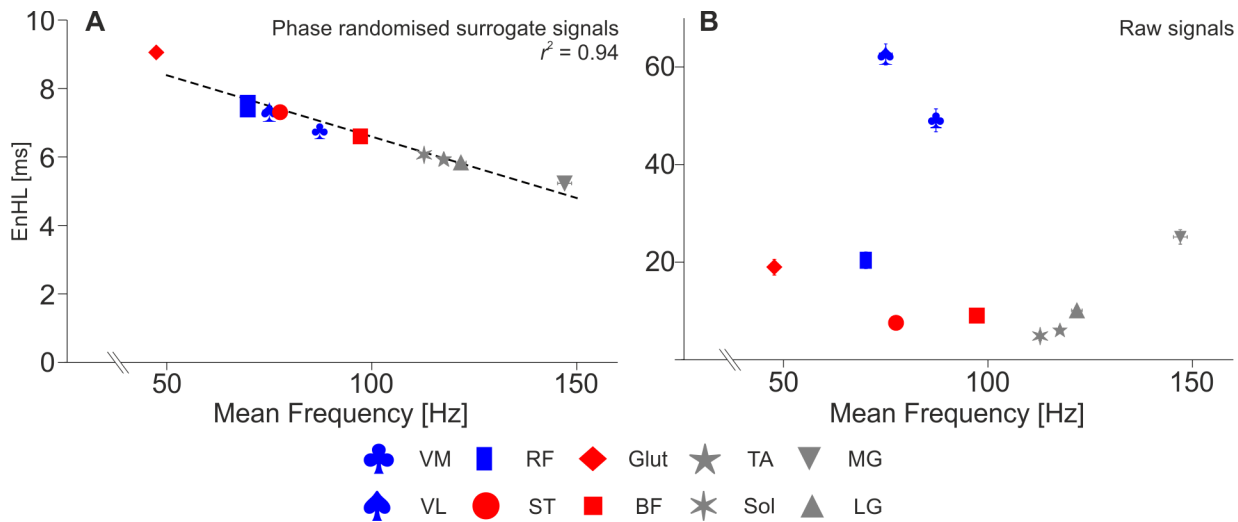
430 **Fig. 2.** EMG intensity (A), burst duration (B) and duty cycle (C) for the different cycling conditions.

431 Cyclists pedalled at a low crank torque but increasing cadences (dashed lines), and at a low cadence but

432 increasing torque (solid lines). Each point represents the mean from 84 steady pedal cycles for nine

433 subjects.

434



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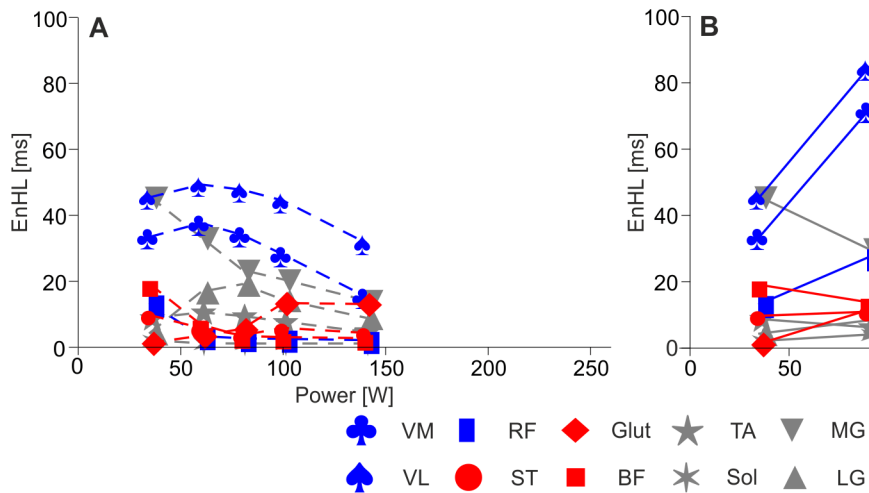
436 **Fig. 3.** Correlations of the entropic half-life EnHL with the mean frequency of the EMG for the phase-

437 randomized surrogate signals (A), and the raw EMG signals (B). Each point shows the mean  $\pm$  S.E.M.

438 calculated across all nine subjects, nine pedal conditions and 3 blocks.

439

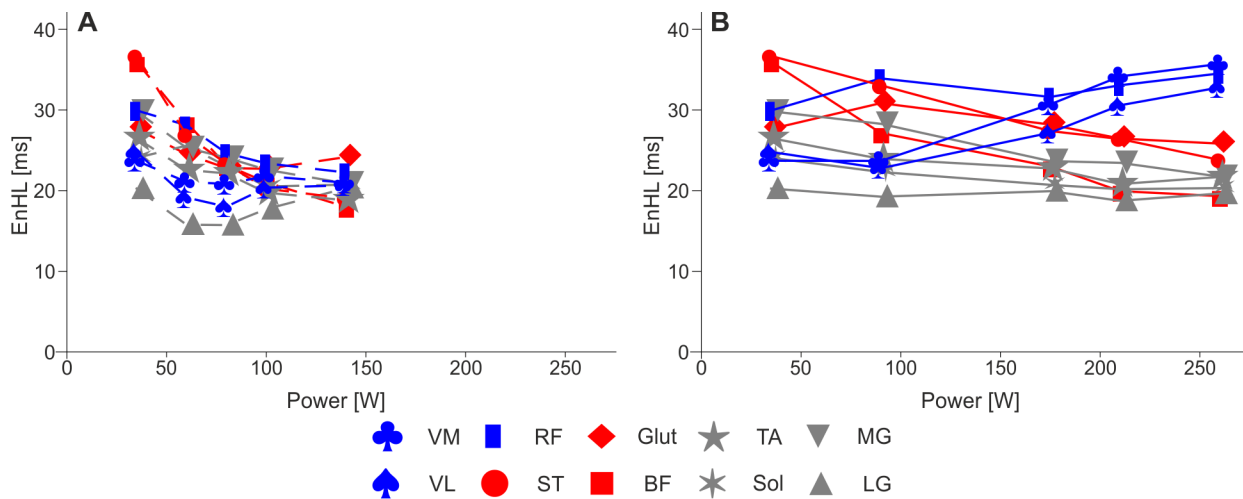




440

441 **Fig. 4.** Entropic half-lives EnHL for the raw EMG signals when cycling at a low crank torque but  
 442 increasing cadences (A), and at a low cadence but increasing torque (B). The results of statistical  
 443 analysis are shown in Table 1.

444

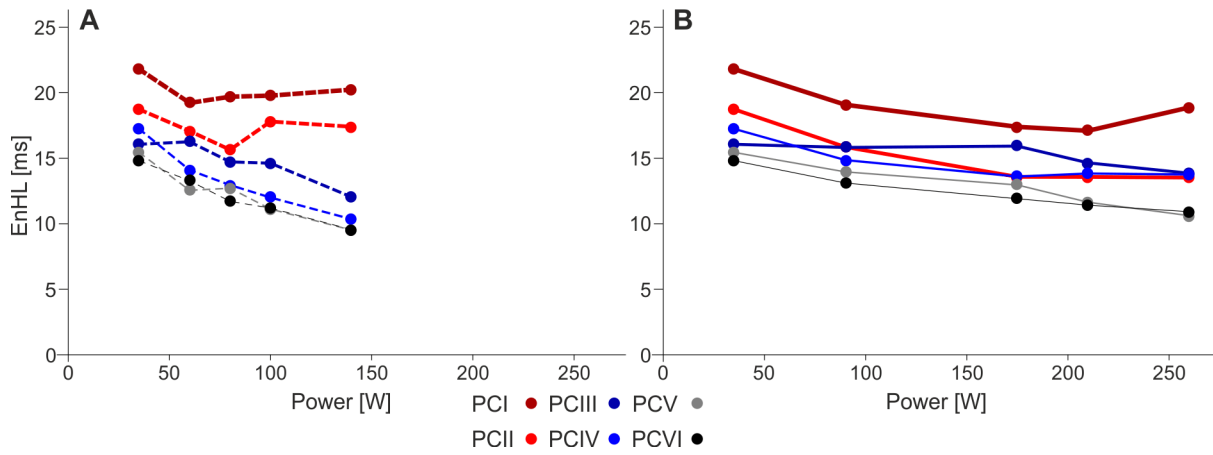


445

446

447 **Fig. 5.** Entropic half-lives EnHL for the EMG intensities when cycling at a low crank torque but  
 448 increasing cadences (A), and at a low cadence but increasing torque (B). The results of statistical  
 449 analysis are shown in Table 1.

450



451

452 **Fig. 6.** Entropic half-lives EnHL for the PC loading scores for the muscle coordination when cycling at a  
 453 low crank torque but increasing cadences (A), and at a low cadence but increasing torque (B). The  
 454 results of statistical analysis are shown in Table 1.