1    **Title Page**

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3    **Title:** A non-linear analysis of running in the heavy and severe intensity domains

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**Abstract**

*Purpose*

Altered movement complexity, indicative of system dysfunction, has been demonstrated with increased running velocity and neuromuscular fatigue. The critical velocity (CV) denotes a metabolic and neuromuscular fatigue threshold. It remains unclear whether changes to complexity during running are coupled with the exercise intensity domain in which it is performed. The purpose of this study was to examine whether movement variability and complexity differ exclusively above the CV intensity during running.

*Methods*

Ten endurance-trained participants ran at 95%, 100%, 105% and 115% CV for 20 min or to task failure, whichever occurred first. Movement at the hip, knee, and ankle were sampled throughout using 3D motion analysis. Complexity of kinematics in the first and last 30 s were quantified using sample entropy (SampEn) and detrended fluctuation analysis (DFA-α). Variability was determined using standard deviation (SD).

*Results*

SampEn decreased during all trials in knee flexion/extension and it increased in hip internal/external rotation, whilst DFA-α increased in knee internal/external rotation. SD of ankle plantar/dorsiflexion and inversion/eversion, knee internal/external rotation, and hip flexion/extension and abduction/adduction increased during trials. Hip flexion/extension SampEn values were lowest below CV. DFA-α was lower at higher velocities compared to velocities below CV in ankle plantar/dorsiflexion, hip flexion/extension, hip adduction/abduction, hip internal/external rotation. In hip flexion/extension SD was highest at 115% CV.

*Conclusions*

Changes to kinematic complexity over time are consistent between heavy and severe intensity domains. The findings suggest running above CV results in increased movement complexity and variability, particularly at the hip, during treadmill running.

**Key Words:** Running; exercise; non-linear dynamics; complexity; variability

**List of Abbreviations**

| | |
|---|---|
| CV | Critical velocity |
| CWR | Constant work rate |
| D′ | Curvature constant of velocity relative to time |
| DFA | Detrended fluctuation analysis |
| DFA-α | Detrended fluctuation analysis α exponent |
| MAV | Maximal aerobic velocity |
| SampEn | Sample entropy |
| SEE | Standard error of the estimate |
| $T_{lim}$ | Time to task failure |
| V | Velocity |

| vGET | Velocity evoking gas exchange threshold |
| $\dot{V}O_2$ | Oxygen uptake |

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70 **Introduction**

71 Running is comprised of a series of repeated and rhythmic actions. The cyclic nature of running
72 suggests that motion is regular, with low variability (Jordan et al. 2006). Previous studies examining the
73 variability of movement during running have traditionally used linear measures such as standard
74 deviation, coefficient of variation, and root mean square (Belli et al. 1995; Cher et al. 2017). Haken,
75 Kelson and Bunz (1985) proposed that increased variability in movement during locomotion is indicative
76 of a loss of stability and diminished ability to respond to external perturbations. Linear measures merely
77 quantify the magnitude of variability, whereas non-linear measures quantify dynamic and temporal
78 aspects of time series and provide greater insight into the complexity of underlying motor control
79 (Stergiou and Decker 2011). Complexity is considered to be temporal regularity or randomness of a
80 time-series (Pincus et al. 1994) and can be quantified using measures of entropy and fractal scaling.
81 For example, sample entropy (SampEn) quantifies the regularity of fluctuations in a time (Pincus 1991;
82 Richman and Moorman 2000), and long-range correlations and statistical persistence can be quantified
83 using fractal measures such as detrended fluctuation analysis exponent-α (DFA-α; Hausdorff et al.
84 1995).

85 Healthy and unfatigued systems are typically characterised by complex outputs which allow for
86 adaptability to external perturbations, but a loss of complexity is evident in ageing or diseased states
87 (Lipsitz and Goldberger 1992). Within the context of gait fractal dynamics, it has been shown that
88 healthy young adults exhibit statistical persistence in stride intervals (Hausdorff et al. 1998; Hausdorff
89 et al., 1997). Conversely, stride interval time series from healthy older individuals and individuals with
90 Huntingdon's (Hausdorff et al., 1997) or Parkinson's disease (Hausdorff et al. 1998) demonstrate anti-
91 persistence. Further to changes in gait dynamics caused by ageing and disease, fatigue has been
92 shown to alter temporal dynamics (Meardon et al. 2011; Schütte et al. 2015, 2018). During running

93    tasks performed to task failure at 5 km race pace on a track, runners exhibited decreased DFA-α of

94    stride time in the last lap compared to the first (Meardon et al. 2011). These findings were replicated by

95    Schütte et al. (2018), demonstrating decreased DFA-α of stride time alongside increased complexity of

96    stride length in the last lap compared to the first. Running-induced fatigue has also been shown to

97    increase step complexity and complexity of trunk accelerations (Schütte et al. 2015). Importantly, both

98    studies (Schütte et al. 2015; Schütte et al. 2018) quantified fatigue using rating of perceived exertion

99    scales, without reference to objective markers of fatigue.

100    In addition to fatigue, running velocity has been shown to affect complexity and variability of movement

101    (Jordan et al. 2006; McGregor et al. 2009; Murray et al. 2017). A quasi U-shape in standard deviation

102    and DFA-α was apparent with deviations from the preferred running velocity (Jordan et al. 2006; Mann

103    et al. 2015). McGregor et al. (2009) further demonstrated decreased control entropy of accelerometery

104    concomitant with increasing velocity during an incremental running test to exhaustion. When

105    considering angular kinematics, Estep et al. (2018) recently demonstrated greater complexity of hip and

106    knee movement during running when compared to walking. Importantly, Murray et al. (2017) showed

107    SampEn and root mean square of shank and waist accelerations were positively correlated with blood

108    [La] during a submaximal incremental test. Given the link between complexity and physiological

109    responses such as increased blood [La] (Murray et al. 2017), and such responses are determined by

110    physiological thresholds (Poole et al. 2016), changes in complexity during running may be defined by

111    physiological thresholds. To date, the link between physiological boundaries and the complexity of

112    movement during whole-body movement has not been investigated, it therefore remains unclear which

113    mechanisms mediate complexity during locomotion.

114    Previous work has shown the fatigue-mediated decrease in complexity of torque outputs exclusively

115    above critical torque (Pethick et al. 2016). The critical torque is analogous to critical velocity (CV) and

116    demarks the boundary between the heavy and severe exercise-intensity domains (Poole et al. 2016).

117    Exercise above CV is characterised by an inexorable increase in metabolic flux (e.g. inorganic

118    phosphate, $H^+$ ions, phosphocreatine) and systemic (e.g. pulmonary oxygen uptake) responses to

119    exercise, whereas exercise below CV allows for the attainment of a steady state (Jones et al. 2007;

120    Poole et al. 2016). Pethick et al. (2019) have recently demonstrated that muscle torque complexity

121    depends, in part, on metabolic rate, with greater intensities invoking lower complexity. However, this

122    phenomenon may not be evident during whole-body exercise, with greater movement complexity

123    exhibited at greater intensities (Jordan et al. 2006; Mann et al. 2015; Murray et al. 2017). At present,

124    no studies have examined changes to complexity of movement during whole-body exercise relative to

125    physiological thresholds.

126    A recent review has eloquently highlighted the potential implementation of non-linear methods,

127    including complexity, to forecast injury risk (Fonseca et al. 2020). However, without fundamental

128    understanding and exploration of movement complexity during locomotion, the successful

129    implementation of such methods in applied settings may be problematic and limited. The aim of this

130    study, therefore, was to explore the variability and complexity of lower limb angular kinematics above

131    and below CV. An additional aim was to examine whether there would be different temporal profiles of

angular kinematic complexity and variability above and below the CV. Since changes to motor control have been shown to be dependent on metabolic rate, and previous literature demonstrating greater complexity at increased velocities, it was hypothesised that compared to trials below CV decreased complexity would only be evident above CV. It was expected that greater decreases in complexity over time would be evident in trials above CV when compared to trials below CV.

**Methods**

*Participants*

Ten recreationally trained males (age, 29.3 ± 10.1 years; stature, 1.76 ± 0.04 m; body mass, 72.1 ± 9.6 kg; means ± SD) volunteered to participate in this study after giving written informed consent. The study was approved by the Health, Science, Engineering and Technology Ethics Committee of the University of Hertfordshire (protocol number: LMS/PGR/UH/03454) and adhered to the Declaration of Helsinki. All participants were male runners or multisport athletes e.g. triathletes, familiar with treadmill running, aged between 18 and 50 years, with a $\dot{V}O_{2max} \geq 45$ ml·min$^{-1}$·kg$^{-1}$. The level of the participants corresponded to performance level 2 in the classification of participants groups in sport science research (De Pauw et al. 2013). Exclusion criteria included vestibular or vision disorders, an observed $\dot{V}O_{2max} < 45$ ml·min$^{-1}$·kg$^{-1}$, pulmonary, neurological, or cardiovascular diseases, and current or recent (6 months prior to participation) musculoskeletal injuries to the lower extremity or back. Assessment of the exclusion criteria, with the exception of $\dot{V}O_{2max}$, was carried out by questionnaire during an initial screening on their first visit. Participants were instructed to arrive at the laboratory rested and were advised to avoid caffeine and food 3 h prior each test, and to avoid strenuous exercise and refrain from alcohol in the 24 h before each test. All tests were performed at the same time of day (±2 h) in laboratory conditions within a controlled environment (18–22°C; 45–55% relative humidity).

*Experimental Design*

Participants were required to visit the laboratory on seven occasions within a 6-week period, with all tests separated by a minimum of 48 hours (Figure 1). All testing was performed on a motorised treadmill (Mercury, h/p/cosmos Sport and Medical, Traunstein, Germany). The first session was used to familiarise participants with the procedures and to conduct an incremental running test to determine velocity evoking gas exchange threshold (vGET), maximal aerobic velocity (MAV), and the maximum oxygen uptake ($\dot{V}O_{2max}$). During the second and third visits, a series of constant work rate tests (CWR) at different intensities were performed to determine CV. On visits four to seven, participants performed in randomised order four experimental trails consisting of constant velocity test to task failure at velocities ranging from 95 to 115 % of CV. During experimental trials lower limb kinematics were recorded throughout each trial.

Throughout all tests, pulmonary gas exchange was measured breath-by-breath using an online gas analyser (MetaLyzer 3B, Cortex Biophysik, Leipzig, Germany). The participants wore a face mask with low dead space (125 mL) and breathed through a low-resistance (<0.1 kPa·L$^{-1}$·s$^{-1}$ at 20 L·s$^{-1}$) impeller turbine, while $O_2$ and $CO_2$ were sampled at 50 Hz. The gas analyser was calibrated before each test with gases of known concentration and the turbine volume transducer was calibrated using a 3 L syringe (Hans Rudolph, Inc., Kansas City, MO). The analyser rise time and the transit delay for $O_2$ and $CO_2$

171     were <100 ms and 800–1200 ms respectively, allowing breath-by-breath calculation. Therefore, $\dot{V}O_2$,

172     $\dot{V}CO_2$, and $\dot{V}_E$ data were recorded breath-by-breath and exported as a 10-s moving average for data

173     analysis. Heart rate (HR) was recorded beat to beat using a heart rate monitor (Polar H7, Polar Electro,

174     Kempele, Finland). Blood samples were collected from the fingertip into a capillary tube (10 μL) for

175     analysis of blood [La] using an automated blood analyser (Biosen C-line, EKF Diagnostic, Barleben,

176     Germany).

177     **_Measurements_**

178     *Incremental exercise test*

179     To determine vGET and MAV, an incremental exercise test to exhaustion was performed. The incline

180     of the treadmill during this and all subsequent tests was set at 1% (Jones and Doust 1996). Following

181     the completion of a standardised warm-up at a velocity of 1.67 m·s⁻¹ for 5 min, the test started at a

182     velocity of 2.22 m·s⁻¹ and was increased by 0.14 m·s⁻¹ every minute until volitional exhaustion despite

183     strong verbal encouragement.

184     *Determination of Critical Velocity*

185     A minimum of four CWR trials to task failure were performed at intensities that corresponded,

186     approximately, to 60% Δ, 70% Δ, 80% Δ and 100% MAV; where Δ refers to the velocity difference

187     between vGET and the MAV. This range of velocities was selected so that each participant could

188     perform between 2-15 minutes before exhaustion (Muniz-Pumares et al. 2019). The CWR trials were

189     performed in randomised order across two separate sessions, although in all testing sessions the lower

190     intensity test always preceded the highest intensity test (Triska et al. 2017). On the second laboratory

191     visit, two CWR trials were performed, with the final two CWR trials performed on the third laboratory

192     visit. To avoid a possible priming effect in $\dot{V}O_2$ and CV (Burnley et al. 2006; Karsten et al. 2018), a

193     recovery period of 60-minutes was provided between CWR trials where participants were allowed to

194     drink water *ad libitum*. Before the start of each CWR trial, all participants completed a standardised

195     warm-up at a velocity of 1.67 m·s⁻¹ for 5 min, followed by a period of 3 min passive rest. The treadmill

196     was then set to the criterion velocity where participants were required to stand with their feet astride the

197     treadmill belt holding onto the handrails. The transitions from rest to running were performed by the

198     participants using the handrails to suspend their body above the belt while they developed the velocity

199     required with their legs. Timing for each trial began when the participant released the handrails and

200     terminated when the participant grasped onto the handrails to signal task failure. Strong verbal

201     encouragement was provided throughout, and participants were blinded to the velocity and elapsed

202     time.

203     *Experimental Trials*

204     During the final four visits runs were performed at velocities corresponding to 95%, 100%, 105% and

205     115% of the calculated CV (95% CV, 100% CV, 105% CV, and 115% CV). The velocities of

206     experimental trials were calculated outside of the SEE for CV estimates, so that the trial below CV (95%

207     CV) was 95% of CV minus 1 SEE, and trials above CV (105% CV and 115% CV) were the product of

208     the percentage of CV plus 1 SEE. The order of these trials was randomised and completed in the same

209　manner as the CWR trials, with the participant running until task failure despite strong verbal
210　encouragement or for 20 minutes, whichever occurred first. Kinematic data from the right leg were used
211　for analysis.

212　*Motion Analysis*
213　Kinematic measures of the lower extremity were obtained using a 14-camera high speed motion capture
214　system sampling at 200 Hz (10 Osprey cameras and 4 Raptor-E; Motion Analysis Corp., Santa Rosa,
215　CA). Prior to data collection the participants performed a static and dynamic calibration. The static
216　calibration was performed using an L-frame with 4 retroreflective markers affixed placed on the corner
217　of the force plate. The dynamic calibration was performed by waving a wand with 3 retroreflective
218　markers within the capture volume (approximately 5 x 2 x 3 m). A maximum standard deviation of 1.0
219　mm for the distance between markers was obtained. Data were inspected using Cortex software
220　(Cortex-64 6.13.1751, Motion Analysis Corporation, Santa Rosa, CA) before importing into Visual 3D
221　(Visual3D v6x64, C-motion, Germantown, MD).
222　A modified Helen Hayes marker set (Kadaba et al. 1990) was used to place 39 passive retroreflective
223　markers (diameter: 12 mm) on the skin bilaterally on the first metatarsals, between the second and third
224　metatarsals, fifth metatarsals, calcaneus, lateral malleoli, medial malleoli, lateral femoral epicondyles,
225　medial femoral epicondyles, iliac crests, anterior superior iliac spines and sacrum. Clusters of four
226　markers were affixed bilaterally to the lateral aspect of the shank and thigh and were secured with rigid
227　tape. To minimise interference effects and movement artifact during running, wands were not used
228　during marker placement. Definitions for hip, knee, and ankle joint centres remained consistent with the
229　Helen Hayes model (Kadaba et al. 1990), and additional markers were utilised to enable more
230　consistent marker tracking. Previous pilot work demonstrated excellent intra- and inter-session
231　reliability, evidenced by low coefficients of variation during dynamic movements using this marker set
232　in sagittal (0.33 – 0.39%), frontal (1.22 – 1.41%), and transverse 2.11 – 3.12%) planes. From an initial
233　static trial in the anatomical position, a kinematic model (pelvis, thigh, shank, and foot) was created for
234　each participant. Ankle and knee joint centres were defined as the midpoint of the medial and lateral
235　malleolus and the medial and lateral femoral epicondyle markers, respectively. The 3D coordinates of
236　the hip joint centres were approximated using the coordinates of the reflective markers at the left and
237　right anterior superior iliac spines and the marker at the sacrum (Bell et al. 1990). The foot, shank, and
238　thigh were modelled as frustra of right cones, whereas the pelvis was modelled as a cylinder. Segmental
239　anthropometric and geometrical properties were used based on the model by Hanavan (1964).
240　Following the static trial, medial knee and medial ankle markers were removed for subsequent dynamic
241　trials. Marker coordinates were smoothed using a 6 Hz fourth-order low-pass Butterworth filter. Joint
242　rotations were calculated based on a right-hand convention using Euler angles in a X
243　(flexion/extension), Y (adduction/abduction), Z (internal/external rotation) rotation sequence. Kinematic
244　data were exported for the right hip, knee, and ankle joint rotations in the sagittal, frontal and transverse
245　planes of motion.

246　**Data Analysis**
247　*Determination of MAV and vGET*

248    MAV was calculated as the velocity of the last stage of the incremental exercise test fully completed. If

249    the final stage was not completed in full, MAV was calculated using the following equation:

$$MAV = V_L + \left(\frac{t}{60\,s}\right) x\ 0.14\ \text{m} \cdot s^{-1} \qquad \text{[Equation 1]}$$

251    where $V_L$ represents the last completed stage (m·s$^{-1}$), $t$ is the time of the incomplete stage performed,

252    60 s refers to the step duration, and 0.14 m·s$^{-1}$ denotes the delta velocity from the previous stage. This

253    linear interpolation was based on the methodology used by Kuipers et al. (1985), where maximal

254    workload was computed instead of MAV. In the current study, velocity of last completed stage and delta

255    velocity from the previous stage were used in lieu of last workload completed and final load increment,

256    respectively. $\dot{V}O_{2max}$ corresponded to the highest $\dot{V}O_2$ average obtained over a 30-s rolling average.

257    vGET was established as the velocity that elicited the following criteria: i) the first disproportionate

258    increase in $\dot{V}CO_2$ from visual inspection of individual plots of $\dot{V}CO_2$ versus $\dot{V}O_2$; ii) an increase in

259    $\dot{V}_E/\dot{V}CO_2$ with no concomitant increase in $\dot{V}_E/\dot{V}O_2$; and iii) the first increase in end-tidal $O_2$ with no fall in

260    end-tidal $CO_2$ tension (Vanhatalo et al. 2016).

261    *CV Estimation*

262    The CV was estimated using three 2-parameter models: i) the hyperbolic v-T$_{lim}$ model, where the

263    velocity is plotted against time [Equation 2]; ii) the linear distance-time model, where the distance

264    covered is plotted against time [Equation 3], and iii) the linear inverse-of-time model, where the velocity

265    is plotted against the inverse of time [Equation 4].

266

$$T_{lim} = \frac{D'}{v - CV} \qquad \text{[Equation 2]}$$

268

$$D = D' + CV \times T_{lim} \qquad \text{[Equation 3]}$$

270

$$v = D' \times \left(\frac{1}{T_{lim}}\right) + CV \qquad \text{[Equation 4]}$$

272

273    where $T_{lim}$ is time to task failure, $D'$ is the curvature constant, $v$ is the velocity of the task, $D$ is the

274    distance performed, and $CV$ is the asymptote termed critical velocity. The SEE associated with the CV

275    obtained from the three models was calculated and expressed as coefficient of variation. A pre-

276    determined threshold for SEE was set at 5% for estimates of CV and 10% for $D'$ (Muniz-Pumares et al.

277    2019). Where four constant velocity trials did not result in estimations of CV and $D'$ falling with the SEE

278    thresholds, additional trials were performed until the SEE of the estimated CV was <5%. To minimise

279    error associated with modelling the velocity-duration relationship, the model (Equation 2, 3, or 4) with

280    the smallest SEE for CV and $D'$ was used to give the 'best individual fit' estimate (Black et al. 2015,

281    2017).

282    *Variability and Complexity*

283    Variability and complexity analysis were performed using MATLAB 2018b (Mathworks, Natick, MA).

284    First, the initial and final 5 s of each trial were discarded (Terrier and Olivier 2011). Prior to linear and

nonlinear analysis of the dependant variables time series were divided into 30 s epochs. Measures of complexity and variability were then performed on the steadiest 20 s of each epoch, identified as the consecutive 20 s with the lowest standard deviation (Pethick et al. 2016). The degree of complexity of dependant variables was quantified using SampEn (Eq. 5) in accordance with the algorithm detailed by Richman and Moorman (2000). SampEn ($m, r, N$) is the negative natural logarithm of the conditional probability that if two sequences of data of length $m$ are both within the same distance $r$, then two sequences of data of length $m+1$ are also within the same $r$ (Richman and Moorman 2000). The parameters ($m, r$) were set ($m = 2, r = 0.2$) to allow for good conditional probability estimates whilst maintaining sufficient system information (Pincus et al., 1994), and in accordance with several studies examining gait (Arshi et al. 2015; Schütte et al. 2015; Murray et al. 2017; Vieira et al. 2017). SampEn quantifies a positive number typically between 0 and 2, with lower values towards 0 reflecting a high system regularity and low complexity and high values towards 2 representing a low system regularity and high complexity (Richman and Moorman 2000; Ramdani et al. 2009).

$$\text{SampEn(m,r,N)} = -\log\left(\frac{\sum_{i=1}^{N-m} A_i(r)}{\sum_{i=1}^{N-m} B_i(r)}\right) = -\log\left(\frac{A}{B}\right) \qquad \text{[Equation 5]}$$

where $r$ is the tolerance threshold, $m$ is the template length, $N$ is the length of the time series. $B_i$ is the number of matches that remain similar, given the tolerance $r$, of the $i^{th}$ template of length $m$. $A_i$ is then given as the number of matches that remain similar for the $i^{th}$ template of length $m+1$. The quantity given as $A/B$ is therefore the probability that two sequences within a tolerance of $r$ for $m$ points, remain within $r$ of each other at the next point ($m + 1$). The fractal scaling of the time series was assessed using detrended fluctuation analysis (DFA; Eq. 6) as defined by Peng et al. (1994) . Briefly, this method integrates the time series $N$, and then divides it into boxes of length $n$. A least-squares line is then fitted $y_n(k)$ in each box of length $n$, representing the trend in each box. The $y$ coordinate of the straight line segments is denoted by $y_n(k)$. The integrated time series $y(k)$ is detrended, by subtracting $y_n(k)$ from the local trend in each box. A root-mean-square calculation is then used to compute the magnitude of fluctuation, $F(n)$, of $y(k)$, from the least square trend in each box. For a box of length $n$, the size of fluctuation for the integrated and detrended time series is given as:

$$F(n) = \sqrt{\frac{1}{N}\sum_{k=1}^{N}[y(k) - y_n(k)]^2} \qquad \text{[Equation 6]}$$

where $F(n)$ is the average fluctuation, $n$ is the box length, $N$ is the total number of data points, $k$ is the order of integration, $y(k)$ is the integrated time series, and $y_n(k)$ is the local trend in its respective box. This process is then repeated across a range of box sizes (56 box sizes ranging from 4 – $N$/4 data points) to provide a relationship between box size and F$(n)$. The slope between log F$(n)$ and log$(n)$ represents the scaling exponent α, which corresponds to the correlational properties of the signal (Hu et al. 2001; Seely and Macklem 2004). Variability of the time series were quantified using standard deviation. Coefficient of variation was not used due to it being overly sensitive for mean values close to zero (Abdi 2010) which would be expected of transverse and frontal kinematics.

321 *Statistical Analysis*

322 Kolmogorov–Smirnov tests for normality were conducted on the data. A two-way analysis of variance

323 was performed on the SampEn and DFA values to test the effects of velocity (95% CV, 100% CV, 105%

324 CV, and 115% CV) and time (Start and End) on the results. A one-way analysis of variance was

325 performed to test the effects of velocity (95% CV, 100% CV, 105% CV, and 115% CV) on time to task

326 failure ($T_{lim}$), end pulmonary $\dot{V}O_2$, and blood [La] responses. Differences in $\dot{V}O_2$ between CWR trials

327 incremental exercise test, as well as experimental trials and incremental exercise test, were assessed

328 using a one-way analysis of variance. Pairwise comparisons were conducted using Bonferroni

329 adjustments where main effects and interactions were significant ($P < 0.05$). The assumption of

330 sphericity was tested using Mauchly's test, with Huynh-Feldt corrections made for violations ($P <$

331 0.05). All data are presented as means ± SD, and results were deemed statistically significant when P

332 < 0.05. All statistical analyses were performed using SPSS (version 25.0; SPSS Inc, Chicago, IL).

333 **Results**

334 **Preliminary Measures and Physiological Responses**

335 The mean vGET and MAV recorded during the first visit were 3.20 ± 0.44 m·s$^{-1}$ and 4.88 ± 0.41 m·s$^{-1}$,

336 respectively. The mean $\dot{V}O_{2max}$ measured in the incremental exercise test was 3.77 ± 0.30 L·min$^{-1}$ (53

337 ± 5 ml·min$^{-1}$·kg$^{-1}$) and was not significantly different from the mean $\dot{V}O_2$ in CWR trials performed to

338 estimate CV (3.62 ± 0.37 L·min$^{-1}$) measured at $T_{lim}$ ($P = 0.696$). Estimates of CV derived from Equations

339 2-4 and "best individual fit" model are presented in Table 1. All participants were able to continue for 20

340 min without reaching task failure at 95% CV. Increased velocities resulted in significantly reduced $T_{lim}$

341 above CV (Table 2). There was a significant effect on pulmonary $\dot{V}O_2$ at $T_{lim}$ ($P = 0.022$) with post-hoc

342 analysis showing pulmonary $\dot{V}O_2$ at $T_{lim}$ was significantly greater in the trial at 105% CV (3.53 ± 0.39

343 L·min$^{-1}$) compared to trials performed at 100% CV (3.40 ± 0.35 L·min$^{-1}$) and 95% CV (3.27 ± 0.30 L·min$^{-1}$

344 $^{1}$) ($P < 0.05$). Trials performed at 115% CV resulted in a $\dot{V}O_2$ at $T_{lim}$ of 3.37 ± 0.40 L·min$^{-1}$ which was

345 not significantly different from other velocities. The $\dot{V}O_2$ measured during the incremental exercise test

346 differed from $\dot{V}O_2$ at $T_{lim}$ in trials performed at 95% CV ($P < 0.001$), 100% CV ($P = 0.009$), and 115%

347 CV ($P = 0.012$), but not 105% CV ($P = 0.847$). Similarly, a significant effect was evident on HR at $T_{lim}$

348 ($P < 0.001$; Table 2). Trials performed at or above CV resulted in significantly higher blood [La] when

349 compared to the trial performed at 95% CV ($P < 0.001$; Table 2).

350 **Linear Analysis**

351 The standard deviation of each running velocity for dependent variables in the first and last epoch is

352 presented in Table 3. There were no significant velocity x time interactions ($P > 0.05$). There was a

353 significant effect for time on standard deviation for ankle plantar/dorsiflexion ($P = 0.032$) and

354 internal/external rotation ($P = 0.006$), knee internal/external rotation ($P = 0.001$), and hip

355 flexion/extension ($P = 0.001$) and abduction/adduction ($P = 0.002$), with greater variability during the

356 last 30-second epoch. There was a significant main effect for velocity on hip flexion/extension standard

357 deviation with greater variability observed at higher velocities ($P < 0.001$, Figure 2). There were

358 significant main effects for velocity on knee flexion/extension SD ($P = 0.015$) and hip

359 abduction/adduction standard deviation (P = 0.044) but no significant differences were observed using
360 Bonferroni correction.

**Nonlinear Analysis**

362 The SampEn and DFA-α values for the first and last epoch for all conditions are presented in Table 4
363 and Table 5, respectively. No significant velocity x time interactions were evident in SampEn (P > 0.05).
364 There was a significant effect for time on SampEn in knee flexion/extension, with greater regularity in
365 the last epoch compared to the first (P = 0.014). Conversely, hip internal/external rotation demonstrated
366 more complex fluctuations in the last epoch when compared to the first during trials at or above CV (P
367 = 0.001, Figure 4). There was a significant effect for velocity on SampEn for hip flexion/extension (P =
368 0.001). There was also a significant effect for velocity on SampEn of hip internal/external rotation (P =
369 0.049), but subsequent pairwise comparisons showed no significant differences between velocities.
370 There were significant velocity x time interactions of DFA-α evident in knee adduction/abduction (P =
371 0.049) and hip flexion/extension (P = 0.003, Figure 2). DFA-α in knee adduction/abduction was lower
372 in trials performed at 115% CV (P = 0.001). DFA-α was lower in ankle plantar/dorsiflexion during trials
373 at 115% CV, indicating less statistical persistence, than at 105% CV and 100% CV (P = 0.003). Trials
374 performed at higher velocities resulted in reduced statistical self-similarity at the hip, with lower DFA-α
375 values in hip flexion/extension (P < 0.001, Figure 2), hip adduction/abduction (P = 0.008, Figure 3), and
376 hip internal/external rotation (P = 0.001, Figure 4). In trials at or below CV, DFA-α in knee
377 internal/external rotation increased over time (P = 0.042).

**Discussion**

379 The purpose of the current study was to characterise changes to the pattern of variability and complexity
380 of lower limb joint kinematics during running at different velocities relative to CV. The distinct
381 physiological profile observed during the experimental trials (Table 2) suggests CV was approximated
382 correctly (Ozyener et al. 2001). Data observed in the sagittal plane at the hip suggest that there was an
383 increase in complexity and variability of proximal lower limb joint kinematics above the CV . Significant
384 decreases in DFA-*α* observed at 115% CV in ankle plantar/dorsiflexion, knee abduction/adduction, hip
385 abduction/adduction, and hip external/internal rotation indicated increased complexity above CV. The
386 hypothesis that lower complexity would be evident above the CV when compared to trials at or below
387 the CV is therefore refuted. Variability increased in a number of variables from the first epoch to last
388 epoch. Demonstrable changes as a result of time were not evident across complexity of all variables
389 and DFA-*α* and SampEn values behaved consistently between velocities below, at, and above CV.
390 Changes in locomotor behaviour in some variables were evident only through the use of non-linear
391 methods, suggesting these may be complimentary to non-linear methods. Ultimately, this may enable
392 better understanding on running biomechanics in research and clinical practice. The current study has
393 elucidated phenomena, herein discussed, which form the basis of further research in the application of
394 non-linear analyses in running.

**Effect of running velocity on variability and complexity**

396 The findings of the current study suggest that greater complexity, as demonstrated by higher SampEn
397 values and lower DFA-*α* values, and variability of angular kinematics, are evident at 115% CV compared

398  to lower velocities higher velocities. This was observed alongside similar metabolic conditions, as
399  inferred by comparable blood [La], between 105% CV and 115% CV, which suggests that complexity
400  and variability of movement during running may be more strongly mediated by velocity rather than
401  metabolic rate *per se.* It has previously been suggested that increases to complexity and variability at
402  greater velocities of locomotion may be a protective mechanism against injury, allowing for greater
403  accommodation of external stressors (Estep et al. 2018). Indeed, running at greater velocities has been
404  shown to increase the magnitude of potentially injurious variables including: ground reaction force
405  (Keller et al. 1996), and accelerations acting on the body in the vertical, mediolateral, and
406  anteroposterior planes (Sheerin et al. 2019). Risk of injury as a result of external perturbations may be
407  increased at greater velocities due to increased forces and, if there is a resultant angular excursion,
408  greater external joint moments. Furthermore, the time in which the system has to detect, identify, and
409  adapt to external perturbations when moving at greater velocities is decreased (Biewener and Daley
410  2007). Indeed, runners with a history of medial tibial stress syndrome exhibited decreased complexity
411  towards the end of a 3.2 km run (Schütte et al. 2018). Previous literature has expounded the relationship
412  between runners with overuse injuries and diminished coordinative variability (Miller et al. 2008; Hamill
413  et al. 2012; Schütte et al. 2018). It could therefore be argued that the decrease in complexity may
414  predispose runners to injury due to a diminished ability to adapt to environmental threats. Future lines
415  of inquiry may wish to examine the role of complexity in mitigating injury risk and overcoming external
416  perturbations. Indeed, a recent review proposed a greater understanding of non-linear systems may
417  lead to better injury forecasting in athletes (Fonseca et al. 2020).

418  It is unclear whether complexity and variability of angular kinematics change proportionately with
419  increased velocity of running over a range of different intensity domains. Both Murray et al. (2017) and
420  McGregor et al. (2006) have demonstrated proportional changes in complexity in relation to velocities
421  ranging from moderate to severe intensities using accelerometers. There has been evidence to suggest
422  that critical torque, and by inference CV, may be a phase transition rather than a sudden threshold,
423  whereby some physiological responses associated with the severe intensity domain are apparent 2
424  standard errors below the estimate (Pethick et al. 2020). Although the current study used velocities
425  outside the 95% confidence intervals associated with CV estimates (95% confidence limits 3.86 to 4.08
426  m·s$^{-1}$), the velocities used for 95% CV (3.71 ± 0.38 m·s$^{-1}$) and 105% CV (4.21 ± 0.42 m·s$^{-1}$) were close
427  to CV. This may have resulted in less pronounced differences in motor control between heavy and
428  severe domains. Further research should examine changes to variability and complexity of lower limb
429  angular kinematics over a greater range of intensities.

430  Previous works have demonstrated increased complexity of movement, as quantified by
431  accelerometers (McGregor et al. 2009; Murray et al. 2017) as well as through the use of stride-to-stride
432  measurements (Jordan et al. 2006) with greater running velocities. When considering the regularity of
433  angular kinematics, greater complexity has been demonstrated during running when compared to
434  walking (Estep et al. 2018). The entropy values shown by Estep et al. (2018) during running at a self-
435  selected velocity are lower than the current study. The authors used approximate entropy which should
436  result in similar values when similar data lengths are inputted to SampEn (Pethick et al. 2015). During
437  running, Estep and colleagues (2018) reported an average velocity of running (2.56 ± 0.27 m·s$^{-1}$) was

438 much lower when compared velocities in the current study which may account for the discrepancy
439 between studies. No previous investigators have examined changes to the complexity of angular
440 kinematics at different velocities of running using non-linear analysis, limiting direct comparisons to
441 existing literature.

442 **Effect of time on variability and complexity**
443 Alterations to the complexity of lower limb kinematics during exercise were only evident in few variables
444 at the hip and knee as a result of time (Table 4; Table 5). When changes to complexity and variability
445 were apparent over time, these occurred irrespective of intensity. In line with previous studies examining
446 variability of spatiotemporal gait parameters including stride intervals (Mo and Chow 2018; García-
447 Pinillos et al. 2020), a greater number of changes were evident in variability, demonstrated by increases
448 in standard deviation between the first epoch and last epoch (Table 3). To the best of our knowledge,
449 changes to the complexity of lower limb angular kinematics over time have not previously been
450 explored. In line with previous studies using accelerometers (Schütte et al. 2018), it was anticipated
451 that complexity of movement would decrease with time. It was expected that greater decreases in
452 complexity in trials above CV would be evident. Increased regularity was evident in internal/external
453 rotation at the hip at velocities equal to or above the CV. Similarly, when running at or below CV, knee
454 internal/external rotation exhibited greater self-similarity in the last epoch when compared to the first.
455 Changes as a result of time in this study are likely to be attributed to two mechanisms: fatigue or
456 habituation. Habituation in this case would be changes to variability and complexity in order to adapt
457 the neuromuscular system to the running environment (e.g. surface) and running demands (e.g.
458 velocity).

459 Fatigue has been implicated with the onset of maladaptive movement patterns at the hip and has been
460 implicated with numerous lower limb injuries (Dierks et al. 2008; Powers 2010; Schütte et al. 2018).
461 The increased complexity of hip movement in the transverse plane over time in this study may indicate
462 a protective mechanism which allows for greater adaptability to external stressors during increased
463 fatigue. The increase in complexity observed in the current study suggests that the ability of the system
464 to explore solutions to perturbations affecting the transverse plane improves over time at velocities
465 equal to or above CV. This may be a strategy which mitigates the increased risk of injury by rendering
466 the system more able to stabilise joints and decrease loading on passive structures. Conversely, at the
467 knee during velocities at or below CV, DFA-$\alpha$ values increase over time indicating greater self-similarity.
468 A similar finding was found at 95% in knee flexion/extension, with SampEn values decreasing indicating
469 greater regularity. These phenomena may be due to habituation and a lack of external stressors acting
470 about the knee joint at low velocities allowing it to 'switch off'. The roles of the knee during running are
471 to act as an intermediary between hip and ankle as well as contributing to lower extremity stiffness to
472 ensure efficiency of movement. Extraneous complexity at lower velocities may be inefficient and this
473 may be a method of maintaining efficiency during running at lower velocities. A similar strategy may
474 have not been necessitated at the hip due to the importance of hip musculature in maintaining lower
475 limb stability (Powers 2010), and the ankle due to its ability to respond rapidly to proprioceptive feedback
476 (Voloshina and Ferris 2015).

477 A common feature of performing sustained running is a reduction in the force generating capacity of
478 musculature over time (Nicol et al. 1991; Boullosa et al. 2011; Girard et al. 2012). Previous research
479 has consistently demonstrated increased variability concomitantly with a decrease in force production
480 (Pethick et al. 2015). Briefly, this phenomenon has been attributed to alterations in muscle activation
481 including increased motor unit synchronisation (Taylor et al. 2003), changes to the physiological
482 organisation of motor units (Missenard et al. 2009), and possible changes to common drive (Farina and
483 Negro 2015). Whether these central mechanisms are independent of, or are adjustments to, peripheral
484 mechanisms remain unclear. Given that changes to standard deviation were observed above and below
485 CV, our findings support the notion that changes to variability are controlled centrally (Missenard et al.
486 2009). Due to increased motor unit synchronisation associated with fatigue (Taylor et al. 2003), the
487 observation of changes to variability alongside minimal changes to the temporal structure of kinematics
488 was unexpected. This may be due to temporal constraints on movement placed upon the participants
489 by the use of a motorised treadmill (Lindsay et al. 2014). When considering whole body movements the
490 variability of ground reaction force and knee moments decreased and time-dependent measures of
491 variability (SampEn) increased with fatigue during a side stepping task (Cortes et al. 2014). The contrary
492 findings of the current study may be explained by differences in task. Cortes et al. (2014) measured
493 variability of a side-stepping task which had greater degrees of freedom than running on a treadmill. It
494 has previously been suggested that excessive variability of movement as a result of running fatigue
495 may represent a lack of coordination, leading to an increased energy expenditure (Le Bris et al. 2006).
496 The increased variability in this study may be either be viewed as a loss of system control (Stergiou
497 and Decker 2011), or as a response to increase adaptability in a temporally constrained environment
498 to mitigate increased injury risks associated with fatigue.

499 In the current study, fatigue was not measured directly and so changes to complexity or variability
500 between the first and last epochs cannot be solely attributed to the effects of fatigue. However, it is
501 likely that task failure observed in velocities above CV coincided with a marked decrease in the force
502 generating capacity of the lower limb musculature (Boullosa et al. 2011). It is likely that due to the lack
503 of distinctive changes of complexity between velocities above and below CV, changes to variability
504 during running are mediated by both central and peripheral fatigue. Pethick et al. (2019) showed
505 decreases in the complexity and variability of torque output was inextricably linked to the accumulation
506 of metabolites in isolated movements. It could be that due to greater cognitive demands of whole-body
507 movement that the complexity and variability of lower limb kinematics are not wholly determined by
508 accumulation of metabolites. Indeed, previous research examining dual-task gait has demonstrated
509 changes to variability in walking with the addition of cognitive tasks (Beauchet et al. 2005; Yogev-
510 Seligmann et al. 2008). These results alongside the current findings suggest that executive function
511 and finite attentional resources affect variability and complexity of locomotion. This may explain the lack
512 of clear differences between changes to movement complexity and variability throughout trials between
513 velocities above and below CV. Moreover, the mechanisms by which fatigue occurs may be different in
514 single joint isometric exercise when compared to running. The decline of torque production during single
515 joint isometric exercise is likely to be due to events occurring at the muscle (Place et al. 2009; Burnley
516 et al. 2012). In addition to locomotor muscles, whole-body exercise is associated with greater demands

517  on the pulmonary system (Amann 2012), and is usually terminated with the attainment of $\dot{V}O_{2max}$
518  (Burnley and Jones 2018). Further research may seek to expound on the relationship between central
519  and peripheral fatigue and complexity during whole body movement.

**Proximal changes to movement variability and complexity**

521  Differences in the amount of variability and complexity as a result of manipulating velocity occurred
522  more frequently at the hip joint when compared to the knee and ankle. This may be explained by the
523  morphology and neurology of the lower limb musculature. Previous studies have examined the
524  respective contribution of the hip, knee and ankle muscular function when running velocity is increased
525  (Lemaire and Robertson 1989; Belli et al. 2002; Hanon et al. 2005). According to the findings of the
526  previous authors, the relative contribution of the hip musculature increases concomitantly with
527  increased running velocity. During running, the knee and ankle maintain high joint stiffness before and
528  during the contact phase to allow for economical force production and elastic energy savings (Biewener
529  and Roberts 2000). The largely unchanged complexity and variability observed at the knee and ankle
530  may be a control strategy to preserve running efficiency at higher velocities (Schütte et al. 2017).
531  Furthermore, it has been suggested that muscles that act around the ankle rely on higher gain
532  proprioceptive feedback from ground contact, in contrast to the hip which is primarily feed-forward
533  controlled (Daley and Biewener 2006). Increases in complexity as a result of higher velocities seen
534  primarily at the hip may increase the adaptability to external stressors within proximal musculature,
535  which is relatively insensitive to changes during stance (Daley et al. 2007). This could act to preserve
536  stability of the centre of mass during running at greater velocities. An increase in complexity of ankle
537  movement at greater velocities may not be necessary to accommodate external stressors, due to higher
538  gain proprioceptive feedback regulation and enhanced capability to respond to perturbations (Voloshina
539  and Ferris 2015). Given the movement about the knee is largely dependent on the force balance
540  between hip and ankle (Daley et al. 2007), the velocity mediated alterations at proximal and distal joints
541  may not be large enough to alter knee dynamics.

**Limitations**

543  A motorised treadmill was used to maintain a controlled environment between testing sessions and to
544  enable the collection of large kinematic datasets using 3D motion analysis. The velocity of running is
545  more variable overground when compared to treadmill running, even when pacing is controlled (Riley
546  et al. 2008). Furthermore, when compared to overground running, treadmill running has been shown to
547  result in greater regularity through greater constraint and increased voluntary control (Lindsay et al.
548  2014). Therefore, the dynamics of lower limb kinematics exhibited in this study may not be fully
549  representative of lower limb kinematics of overground running. Moderately trained endurance runners
550  participated in this study, so comparisons with other level groups may be limited since biomechanical
551  differences have been reported between endurance runners of different performance levels (Ogueta-
552  Alday et al. 2018).

**Conclusion**

554  The findings of this study demonstrate changes to kinematic complexity and variability over time are
555  consistent between heavy and severe intensity domains during running. The findings suggest the CV

does not demark a boundary above which there are changes to complexity or variability in kinematics during treadmill running over time. Decreases in DFA-$\alpha$ and increased SampEn in a number of kinematic variables at 115% CV suggest that running at a velocity substantially above CV results in increased complexity of movement. Changes over time to angular kinematics during running on a motorised treadmill may be limited to fluctuations in variability in lieu of changes to complexity due to temporal constraints. Furthermore, changes to movement strategies adopted may be more pronounced at the hip when compared to more distal joints.

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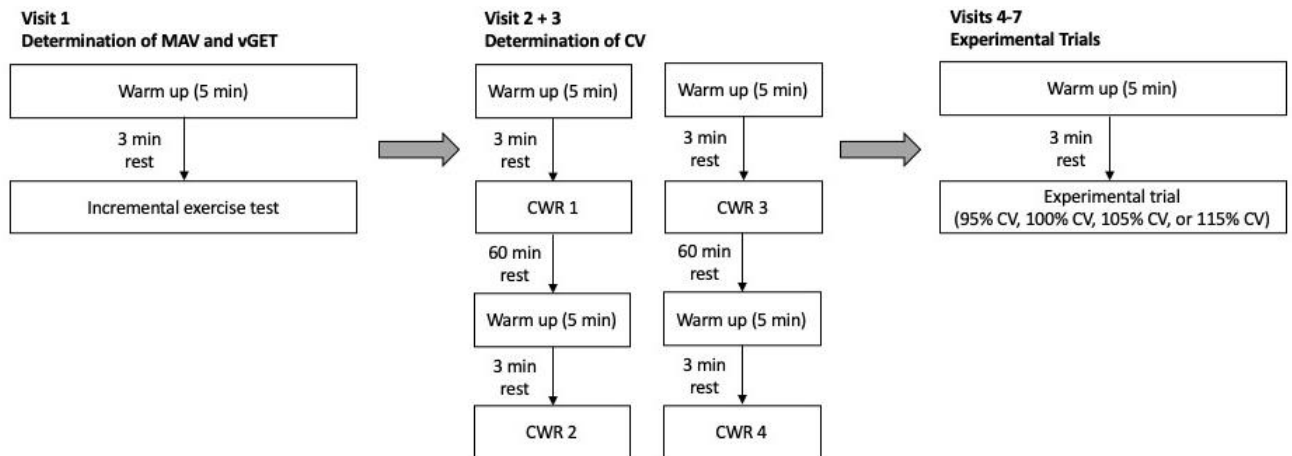783

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**Figure 1.** Schematic of experimental design. CWR, constant work rate trials performed in a randomised order. Visits 2 and 3 were identical apart from the velocity at which the four CWR trials were performed (60% Δ, 70% Δ, 80% Δ and 100% MAV; where Δ refers to the velocity difference between vGET and the MAV). The numbers denote the order and not the velocity at which they were performed. Each of the visits 4-7 differed only in the velocity at which trials were performed (either 95%, 100%, 105%, or 115% CV in a randomised order).

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**Figure 2.** Changes to hip flexion/extension sample entropy (A), DFA-α (B), and standard deviation (C) over the course of trials performed at 95% CV (open circles), 100% CV (black circles), 105% CV (open squares), and 115% CV (black squares). For clarity error bars (± SD) have been omitted for all but the final data point. *Different from first epoch $P < 0.05$; [a]Different from 115% CV $P < 0.05$; [b]Different from 105% CV $P < 0.05$.

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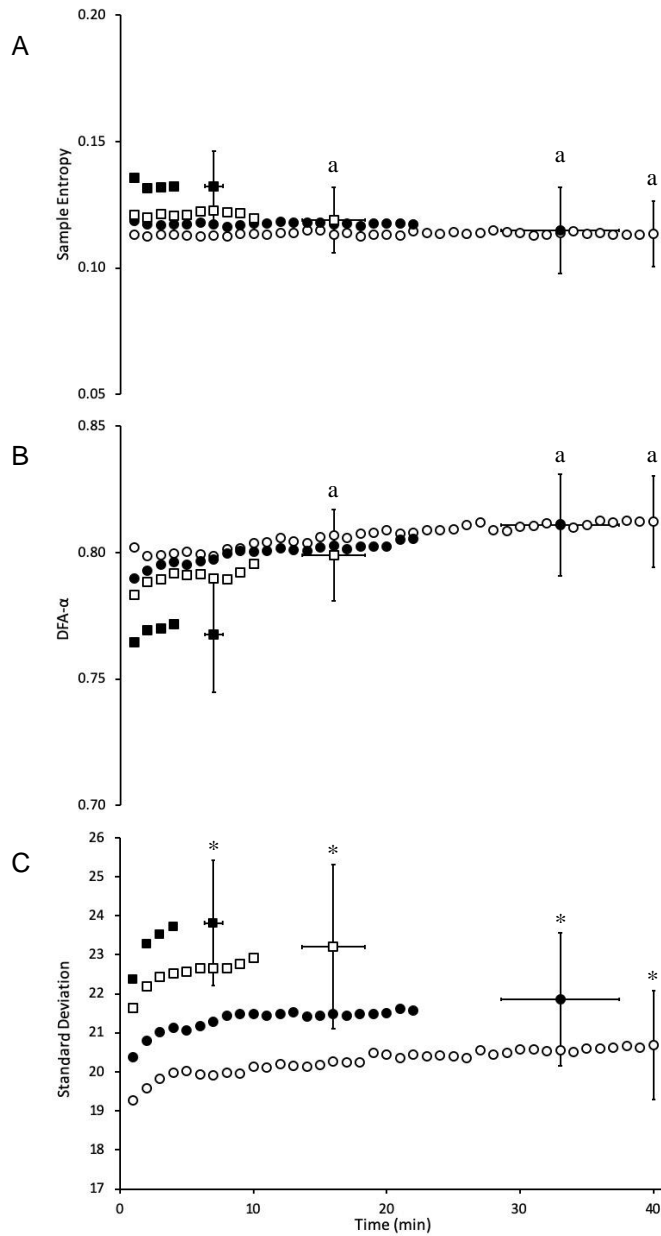925
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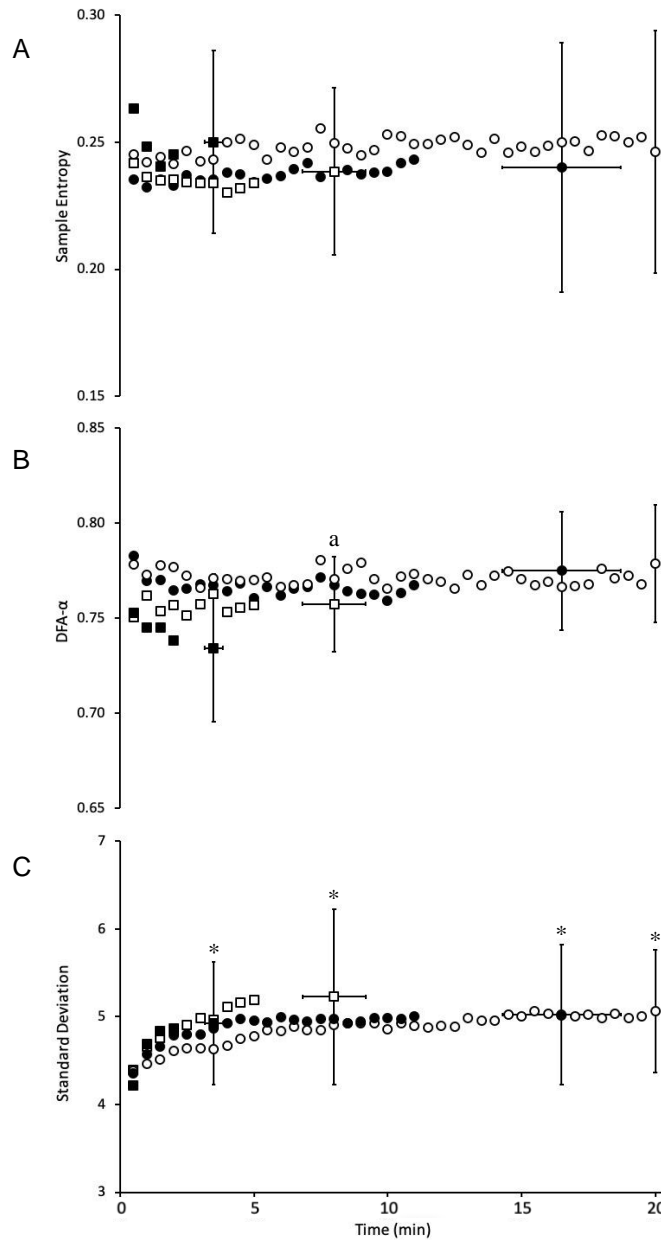**Figure 3.** Changes to hip adduction/abduction sample entropy (A), DFA-α (B), and standard deviation (C) over the course of trials performed at 95% CV (open circles), 100% CV (black circles), 105% CV (open squares), and 115% CV (black squares). For clarity error bars (± SD) have been omitted for all but the final data point. *Different from first epoch $P < 0.05$; [a]Different from 115% CV $P < 0.05$.

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**Figure 4.** Changes to hip internal/external rotation sample entropy (A), DFA-α (B), and standard deviation (C) over the course of trials performed at 95% CV (open circles), 100% CV (black circles), 105% CV (open squares), and 115% CV (black squares). For clarity error bars (± SD) have been omitted for all but the final data point. *Different from first epoch $P < 0.05$; [a]Different from 115% CV $P < 0.05$.
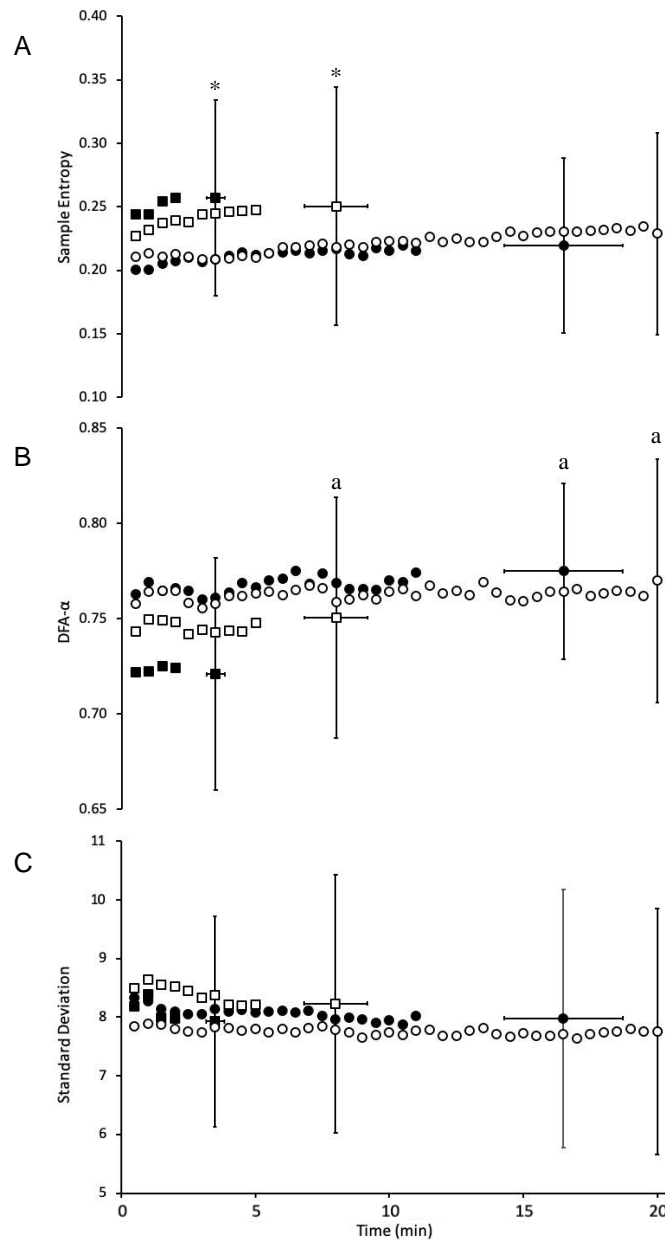
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997 **Table 1.** Parameter estimates derived from Equations 2-4 and the "best individual fit" model

| | $R^2$ | CI 95% (m·s$^{-1}$) | CV (m·s$^{-1}$) | CV SEE (m·s$^{-1}$) | CoV% | $D'$ (m) | $D'$ SEE (m) | CoV% |
|---|---|---|---|---|---|---|---|---|
| $v$-$T_{lim}$ model | 0.885 - 1.000 | 0.27 ± 0.18 | 3.94 ± 0.39 | 0.04 ± 0.03 | 1.05 ± 0.69 | 144 ± 49 | 10 ± 7 | 6.64 ± 3.68 |
| D-$T_{lim}$ model | 0.987 – 1.000 | 0.27 ± 0.15 | 3.97 ± 0.41 | 0.04 ± 0.02 | 1.07 ± 0.61 | 141 ± 44 | 10 ± 7 | 6.71 ± 3.84 |
| 1/$T_{lim}$ model | 0.913 – 1.000 | 0.31 ± 0.15 | 3.99 ± 0.41 | 0.05 ± 0.02 | 1.22 ± 0.58 | 138 ± 41 | 10 ± 6 | 7.06 ± 3.87 |
| BIF | 0.987 – 1.000 | 0.22 ± 0.12 | 3.94 ± 0.42 | 0.04 ± 0.02 | 0.92 ± 0.70 | 141 ± 44 | 9 ± 6 | 6.34 ± 3.32 |

Values are expressed as Mean ± SD. $R^2$ values are presented as the range. SEE, standard error of estimate; CV, critical velocity; CI 95% CoV, coefficient of variation; $v$-$T_{lim}$, hyperbolic velocity-time; D-$T_{lim}$, linear distance-time; 1/$T_{lim}$, linear inverse-of-time; BIF, 'best individual fit'.

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1002 **Table 2.** Trial parameters, pulmonary $\dot{V}O_2$, blood [La], and heart rate responses during trials
1003 performed below (95% CV), at (100% CV), and above (105% CV and 115% CV) critical velocity

| | 95% CV | 100% CV | 105% CV | 115% CV |
|---|---|---|---|---|
| Velocity (m·s$^{-1}$) | 3.71 ± 0.38 | 3.94 ± 0.42 | 4.21 ± 0.42 | 4.64 ± 0.49 |
| $T_{lim}$ (min) | 20.00 ± 0.00[a,d,f] | 16.68 ± 4.41[b,e,f] | 8.03 ± 2.36[c,d,e] | 3.27 ± 0.67[a,b,c] |
| $\dot{V}O_2$ at $T_{lim}$ (L·min$^{-1}$) | 3.27 ± 0.30[a,b] | 3.40 ± 0.35[b] | 3.53 ± 0.39[a] | 3.37 ± 0.40 |
| B[La] (mmol.L$^{-1}$) | 4.45 ± 1.38[a,c,e] | 7.25 ± 1.84[b,d,e] | 9.03 ± 1.98[c,d] | 9.48 ± 1.52[a,b] |
| HR at $T_{lim}$ (bpm) | 169 ± 9[a,d] | 177 ± 7[b,d] | 184 ± 7[a,b,c] | 176 ± 10[c] |

Values are expressed as Mean ± SD. $T_{lim}$, time to task failure; HR, heart rate. Mean values in the same row sharing the same superscript letters are significantly different from each other $P < 0.05$.

**Table 3.** Standard deviation values of lower limb kinematics in the first and last epoch of trials performed below (95% CV), at (100% CV), and above (105% CV and 115% CV) critical velocity

| | 95% CV | | 100% CV | | 105% CV | | 115% CV | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| | Start | End | Start | End | Start | End | Start | End |
| **Ankle** | | | | | | | | |
| Plant/Dorsi | 12.4 ± 1.2 | 13.1 ± 1.6 | 12.8 ± 0.8 | 13.8 ± 2.1 | 12.7 ± 1.1 | 13.8 ± 1.8* | 12.8 ± 1.4 | 13.9 ± 1.8* |
| Ever/Inv | 4.8 ± 1.6 | 5.0 ± 1.2 | 4.4 ± 1.5 | 4.6 ± 1.0 | 4.6 ± 1.0 | 4.7 ± 0.9 | 5.1 ± 1.2 | 5.2 ± 1.2 |
| Int/Ext Rot | 5.6 ± 1.3 | 6.9 ± 1.7* | 5.7 ± 1.5 | 6.3 ± 1.6 | 5.4 ± 1.3 | 6.0 ± 1.2* | 6.6 ± 1.3 | 7.1 ± 1.3* |
| **Knee** | | | | | | | | |
| Flex/Ext | 28.3 ± 3.5 | 30.7 ± 3.4 | 29.9 ± 5.1 | 31.5 ± 4.2 | 30.1 ± 4.2 | 31.8 ± 3.9 | 32.3 ± 4.4 | 32.7 ± 4.1 |
| Ab/Add | 8.6 ± 2.7 | 9.0 ± 3.0 | 9.0 ± 3.5 | 9.0 ± 3.3 | 8.7 ± 2.6 | 8.8. ± 2.7 | 8.9 ± 2.7 | 8.8 ± 2.5 |
| Int/Ext Rot | 10.5 ± 1.6 | 12.1 ± 1.6* | 10.7 ± 3.1 | 11.4 ± 2.4* | 9.8 ± 1.8 | 10.6 ± 2.4 | 11.3 ± 3.4 | 11.9 ± 3.5* |
| **Hip** | | | | | | | | |
| Flex/Ext | 19.3 ± 1.5[a,d] | 20.7 ± 1.4[a,d]* | 20.4 ± 1.9[b,e] | 21.8 ± 1.7[b,e]* | 21.6 ± 1.6[d,e] | 23.2 ± 2.1[d,e]* | 22.4 ± 1.7[a,b,c] | 23.8 ± 1.6[a,b,c]* |
| Ab/Add | 4.4 ± 0.5 | 5.1 ± 0.7* | 4.4 ± 0.6 | 5.0 ± 0.8* | 4.4 ± 0.6 | 5.2 ± 1.0* | 4.2 ± 0.7 | 4.9 ± 0.7* |
| Int/Ext Rot | 7.8 ± 2.0 | 7.8 ± 2.1 | 8.3 ± 2.5 | 8.0 ± 2.2 | 8.5 ± 2.4 | 8.2 ± 2.2 | 8.2 ± 1.6 | 7.9 ± 1.8 |

Values are expressed as means ± SD; Plant, plantarflexion; Dorsi, dorsiflexion; Flex, flexion; Ext, extension; Ever, Eversion; Inv, Inversion; Int, internal rotation; Ext, external rotation; Ab, abduction; Add, adduction; *Different from first epoch $P < 0.05$; Mean values in the same row sharing the same superscript letters are significantly different from each other $P < 0.05$.

**Table 4.** Sample entropy values of lower limb kinematics in the first and last epoch of trials performed below (95% CV), at (100% CV), and above (105% CV and 115% CV) critical velocity

| | 95% CV | | 100% CV | | 105% CV | | 115% CV | |
|---|---|---|---|---|---|---|---|---|
| | Start | End | Start | End | Start | End | Start | End |
| **Ankle** | | | | | | | | |
| Plant/Dorsi | 0.130 ± 0.027 | 0.140 ± 0.024 | 0.134 ± 0.009 | 0.136 ± 0.017 | 0.147 ± 0.022 | 0.138 ± 0.017 | 0.162 ± 0.344 | 0.146 ± 0.033 |
| Ever/Inv | 0.296 ± 0.065 | 0.270 ± 0.080 | 0.311 ± 0.057 | 0.307 ± 0.060 | 0.309 ± 0.046 | 0.312 ± 0.077 | 0.336 ± 0.067 | 0.335 ± 0.066 |
| Int/Ext Rot | 0.346 ± 0.070 | 0.319 ± 0.076 | 0.332 ± 0.060 | 0.317 ± 0.087 | 0.339 ± 0.068 | 0.335 ± 0.056 | 0.375 ± 0.125 | 0.363 ± 0.131 |
| **Knee** | | | | | | | | |
| Flex/Ext | 0.185 ± 0.021 | 0.163 ± 0.025* | 0.178 ± 0.030 | 0.168 ± 0.025 | 0.174 ± 0.032 | 0.167 ± 0.030 | 0.180 ± 0.024 | 0.172 ± 0.025 |
| Ab/Add | 0.125 ± 0.038 | 0.135 ± 0.066 | 0.146 ± 0.040 | 0.144 ± 0.042 | 0.154 ± 0.050 | 0.162 ± 0.065 | 0.172 ± 0.066 | 0.175 ± 0.077 |
| Int/Ext Rot | 0.294 ± 0.056 | 0.272 ± 0.057 | 0.291 ± 0.051 | 0.281 ± 0.047 | 0.274 ± 0.068 | 0.273 ± 0.071 | 0.322 ± 0.039 | 0.327 ± 0.070 |
| **Hip** | | | | | | | | |
| Flex/Ext | 0.113 ± 0.022[a] | 0.119 ± 0.012[a] | 0.119 ± 0.021[b] | 0.115 ± 0.017[b] | 0.121 ± 0.018[c] | 0.119 ± 0.013[c] | 0.136 ± 0.020[a,b,c] | 0.132 ± 0.014[a,b,c] |
| Ab/Add | 0.245 ± 0.054 | 0.246 ± 0.048 | 0.235 ± 0.047 | 0.240 ± 0.049 | 0.242 ± 0.033 | 0.239 ± 0.033 | 0.263 ± 0.045 | 0.250 ± 0.036 |
| Int/Ext Rot | 0.211 ± 0.062 | 0.229 ± 0.080 | 0.200 ± 0.066 | 0.220 ± 0.069* | 0.227 ± 0.85 | 0.251 ± 0.094* | 0.243 ± 0.079 | 0.257 ± 0.077* |

Values are expressed as means ± SD; Plant, plantarflexion; Dorsi, dorsiflexion; Flex, flexion; Ext, extension; Ever, Eversion; Inv, Inversion; Int, internal rotation; Ext, external rotation; Ab, abduction; Add, adduction; *Different from first epoch $P < 0.05$; Mean values in the same row sharing the same superscript letters are significantly different from each other $P < 0.05$.

**Table 5.** Detrended fluctuation analysis-α values of lower limb kinematics in the first and last epoch of trials performed below (95% CV), at (100% CV), and above (105% CV and 115% CV) critical velocity

| | 95% CV | | 100% CV | | 105% CV | | 115% CV | |
|---|---|---|---|---|---|---|---|---|
| | Start | End | Start | End | Start | End | Start | End |
| **Ankle** | | | | | | | | |
| Plant/Dorsi | 0.723 ± 0.047 | 0.741 ± 0.029 | 0.720 ± 0.033[a] | 0.740 ± 0.036[a] | 0.718 ± 0.040[b] | 0.736 ± 0.038[b] | 0.698 ± 0.048[a,b] | 0.702 ± 0.038[a,b] |
| Ever/Inv | 0.669 ± 0.050 | 0.688 ± 0.053 | 0.673 ± 0.044 | 0.660 ± 0.034 | 0.658 ± 0.037 | 0.664 ± 0.044 | 0.663 ± 0.054 | 0.651 ± 0.055 |
| Int/Ext Rot | 0.621 ± 0.102 | 0.619 ± 0.129 | 0.653 ± 0.061 | 0.640 ± 0.059 | 0.665 ± 0.105 | 0.645 ± 0.062 | 0.628 ± 0.080 | 0.624 ± 0.076 |
| **Knee** | | | | | | | | |
| Flex/Ext | 0.732 ± 0.029 | 0.756 ± 0.027 | 0.729 ± 0.026 | 0.745 ± 0.023 | 0.730 ± 0.031 | 0.739 ± 0.025 | 0.724 ± 0.040 | 0.726 ± 0.030 |
| Ab/Add | 0.754 ± 0.033 | 0.760 ± 0.043 | 0.744 ± 0.040[a] | 0.756 ± 0.039[a] | 0.737 ± 0.040 | 0.735 ± 0.043 | 0.719 ± 0.058[a] | 0.715 ± 0.053[a] |
| Int/Ext Rot | 0.631 ± 0.071 | 0.660 ± 0.072* | 0.624 ± 0.066 | 0.640 ± 0.067* | 0.630 ± 0.073 | 0.639 ± 0.066 | 0.606 ± 0.075 | 0.617 ± 0.060 |
| **Hip** | | | | | | | | |
| Flex/Ext | 0.802 ± 0.027[a,d] | 0.810 ± 0.018[a,d] | 0.790 ± 0.024[b] | 0.811 ± 0.020[b] | 0.783 ± 0.029[c,d] | 0.799 ± 0.018[c,d] | 0.765 ± 0.035[a,b,c] | 0.768 ± 0.023[a,b,c] |
| Ab/Add | 0.778 ± 0.044 | 0.775 ± 0.031 | 0.783 ± 0.049 | 0.775 ± 0.031 | 0.751 ± 0.049[a] | 0.758 ± 0.025[a] | 0.753 ± 0.062[a] | 0.735 ± 0.039[a] |
| Int/Ext Rot | 0.758 ± 0.067[a] | 0.765 ± 0.064[a] | 0.763 ± 0.048[b] | 0.775 ± 0.046[b] | 0.743 ± 0.060[c] | 0.751 ± 0.063[c] | 0.722 ± 0.061[a,b,c] | 0.721 ± 0.061[a,b,c] |

Values are expressed as means ± SD; Plant, plantarflexion; Dorsi, dorsiflexion; Flex, flexion; Ext, extension; Ever, Eversion; Inv, Inversion; Int, internal rotation; Ext, external rotation; Ab, abduction; Add, adduction; *Different from first epoch $P < 0.05$; Mean values in the same row sharing the same superscript letters are significantly different from each other $P < 0.05$.