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Dynamics of locomotor fatigue during supra-critical power exercise

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Concept and design: CF, DTC. Data acquisition: ARS, BK, SY, JC, DTC. Data analysis: ARS, BK, SY, DTC. Data interpretation: ARS, BK, SY, JC, CF, DTC. Manuscript drafting: ARS, BK, DTC. Critical Revision: ARS, BK, SY, JC, CF, DTC. All authors approved the final version.

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Running title: Dynamics of locomotor fatigue

Abstract

PURPOSE We aimed to measure 1) the dynamics of locomotor fatigue during constant supra-critical power cycling, and 2) the magnitude of any reserve in locomotor power at intolerance to constant and ramp-incremental cycling in recreationally-active volunteers.

METHODS Fifteen participants (7 women and 8 men, 22±3 yr, 3.34±0.67 $L.min^{-1}$ $\dot{V}O_{2peak}$) completed ramp-incremental and very-heavy constant power (205±46 W) exercise to the limit of tolerance. Immediately following intolerance, the ergometer was switched into the isokinetic mode and participants completed a short (~5 s) maximal isokinetic effort at 70 rpm. The time course of locomotor fatigue during constant supra-critical power exercise was characterized with these short maximal isokinetic sprints at 30, 60, 120, 180 s and at the limit of tolerance. Each bout was terminated following the isokinetic sprint.

RESULTS Constant power exercise duration was 312±37 s. Isokinetic power production at 30, 60, 120, 180 s and the limit of tolerance (at 312±37 s) was 609±165, 503±195, 443±157, 449±133, and 337±94 W, respectively. Of the total decline in isokinetic power, ~36% occurred within the first minute of exercise and significant (p<0.05) reductions in isokinetic power occurred at all time-points vs the baseline maximal isokinetic power (666±158 W). Additionally, a significant power reserve of 132±74 W (64% of the task requirement) and 119±80 W (47%) was present at the limit of constant power and ramp-incremental exercise, respectively.

CONCLUSIONS Locomotor fatigue occurred rapidly during supra-critical power exercise with pseudo-exponential kinetics. Instantaneous isokinetic power production at the limit of tolerance exceeded that of the task requirement, regardless of the constant,

or ramp work rate profile. Thus, the perceptual and physiologic limits were dissociated at the limit of tolerance in recreationally-active volunteers.

Abstract word count: 275 (275 max).

Key words: isokinetic, cycling, fatigue, exercise tolerance

Abbreviations

CI_{95}, 95% confidence interval

P_{iso}, isokinetic power

$\dot{V}O_{2peak}$, peak oxygen uptake

Introduction

Exercise intolerance worsens quality of life and is a powerful predictor of mortality (1-3). Improving exercise tolerance has consistent positive impacts on healthy people and those with disease (4-6). Therefore, understanding the mechanisms of exercise intolerance is crucial for identifying effective prevention and rehabilitation strategies for chronic conditions that impact physical function.

Fatigue is a loss in the capacity for developing force and/or velocity that is reversible by rest (7). Fatigue plays a crucial role in determining exercise intolerance. Some of the mechanisms contributing to fatigue during dynamic, whole-body exercise include heightened perception of exertion or dyspnea, inhibitory afferent signals from the skeletal muscle environment, reduced membrane excitability, and local muscle metabolic and ionic factors that directly inhibit cross-bridge function (7-14). Collectively, the mechanisms are often grouped as ‘central’ and ‘peripheral’ according to the placement with the hierarchy of the neuromuscular system. Typically, central components are ‘north’ of the neuromuscular junction to include the motor and spinal nerves, spinal cord, brain stem, and brain.

The dynamics, or rate of accumulation, of locomotor fatigue may provide information about how the intracellular environment underpins intolerance, as the dynamic behavior of key fatigue-related metabolites is well established in the literature. For example, the time-course of inorganic phosphate (Pi) conforms to a pseudo-exponential profile with a half-time of approximately 30 s. Thus, for constant power exercise most of the change

89 in [Pi] occurs within the first 2-3 minutes of contraction (10, 15, 16). Pi elicits substantial
90 reductions in force, particularly when concentrations rise to ~10-20 mM during high-
91 intensity exercise (12, 13). During high-intensity contraction, reductions in muscle power
92 behave with similar dynamics to Pi (15, 17, 18). Muscle power also *recovers* with similar
93 dynamics to the intramuscular phosphates (17, 20). However, data from the literature on
94 the time-resolution for muscle power measurements during dynamic, large muscle mass
95 exercise is poor (19). Thus far, there are no data on the initial, rapid dynamics of
96 locomotor fatigue during whole-body, dynamic exercise. Therefore, the time-course of
97 fatigue during the first ~3 min of high-intensity exercise is unknown.
98
99 Ideally, measurement of locomotor fatigue is applicable to whole-body, large muscle
100 mass, dynamic exercise (walking, running, cycling). Additionally, it is critical that
101 contraction velocity be controlled for unless the majority of the power-velocity
102 relationship is measured (21, 22) - locomotor power is dependent on the parabolic
103 relationship of power and contraction velocity. Assessing fatigue using a combination of
104 cadence-independent cycling tasks followed immediately by a maximal isokinetic effort
105 allows the contraction velocity-specific decline in task-specific power to be measured in
106 accordance with these criteria. The recovery of skeletal muscle power following a bout
107 of high-intensity exercise is rapid, with most of the recovery occurring within the first two
108 minutes of recovery and nearly full recovery somewhere between 3 and 8 minutes (20,
109 23-25). In fact, even 5-10 s of rest results in substantial recovery when measuring
110 neuromuscular function (20, 25, 26). These data show the importance of rapidly
111 measuring locomotor fatigue (27), and highlight one of the key drawbacks of using

electrical stimulation, transcranial magnetic stimulation, or single-joint maximal voluntary contraction, particularly when following whole-body exercise. This approach requires, at minimum, a 1-2 min delay from the time of interest following cycling (28), save for the most recent innovation using a cycle ergometer capable of near-instantaneous measurement (27). Knee-extension exercise, of course provides near instantaneous assessment of neuromuscular fatigue (11, 24) with the drawback of being somewhat less taxing to the cardiopulmonary system.

Exercise intolerance should arise when the reduction in maximal evocable locomotor power is sufficient enough to constrain the exercise task (29). This is a contentious issue, however, during whole-body exercise even in healthy people. Some evidence shows there is a large 'reserve' in locomotor power (8), and some that there is little to no meaningful reserve at the limit of tolerance in healthy people (22, 23, 30). Further complicating the matter is the task profile (constant vs variable vs ramp) and task duration to intolerance appears to affect the size of the reserve (8, 31). Whether or not the limit of tolerance is consistently accompanied by a 'reserve' in locomotor power remains uncertain, and is likely variable across health and disease (30).

The purposes of this study are twofold. Firstly, we aimed to measure the dynamics of locomotor fatigue at the onset of high-intensity cycling. Second, we aimed to measure the reserve in locomotor power at intolerance to constant and ramp-incremental cycling.

Materials and Methods

Participants

Fifteen healthy, recreationally active volunteers took part in this study (7 women, 8 men, 22±3 yr, 173±12 cm, 66±12 kg, 3.34±0.67 L.min^{-1} $\dot{V}O_{2peak}$). Volunteers provided written informed consent and were screened for cardiovascular risks with the Physical Activity Readiness Questionnaire (PAR-Q) prior to beginning the study. The San Diego State University Institutional Review Board approved the protocol.

Exercise tests

Participants completed 6 laboratory visits, each separated by a minimum of 24 hours. Within each laboratory visit the participants completed two experimental phases: 1) short (~5 s) bouts of maximal effort isokinetic cycling at 70 rpm (measured in triplicate) to determine maximal isokinetic power at baseline (Figure 1A, Figure 2A); and 2) a ramp-incremental or constant power exercise test, terminated with a short (~5 s) maximal isokinetic effort at 70 rpm (Figure 1B).

Visit 1

The first laboratory visit included a ramp-incremental test to the limit of tolerance. The ramp test began with 1 min rest and a 2 min warm-up phase at 25 W. Following the warm-up, power was increased at 25 W.min^{-1} until intolerance. Each participant was instructed to maintain a cadence > 70 rpm. Failure to maintain > 60 rpm, despite strong verbal encouragement, marked the termination of the ramp-incremental phase. Immediately following intolerance, the cycle ergometer was switched from hyperbolic to isokinetic mode (70 rpm) and the participant was instructed to give a maximum effort for 5 s. Participants then completed a 5 min recovery phase at 20 W (Figure 1A).

Visits 2-6

During the second visit (Figure 1B), participants completed a constant power exercise test to the limit of tolerance. To assign each participant's constant power, 2 min worth of ramp incrementation (50 W) was subtracted from peak ramp power. This was a modified approach (32) to estimate an ~6 min duration exercise test, based on our pilot testing. Following completion of warm-up at 20 W, power was increased abruptly to the assigned constant power and participants were instructed to maintain the power until the limit of tolerance. Failure to maintain > 60 rpm, despite strong encouragement, marked the end of the constant power phase. The cycle ergometer was immediately switched from hyperbolic to isokinetic mode where participants were again strongly encouraged for 5 s of maximal effort cycling. Participants then completed a 5 min active recovery phase of light cycling at 20 W.

The remaining 4 visits were completed in random order and consisted of constant power cycling at the same work rate during visit 2. However, the durations of the final 4 visits were 30, 60, 120, and 180 s (Figure 1B). These tests were completed with essentially the same format, yet halted at the pre-determined times rather than continued to the limit of tolerance. Within the final ~5 s of the exercise bout, participants were instructed to decelerate to ~ 70 rpm to avoid an overcompensation of the flywheel braking action at the onset of the isokinetic cycling. At the completion of the pre-determined constant power cycling, the cycle ergometer was switched from hyperbolic

to isokinetic mode where participants gave a maximal effort for 5 s. A 5 min active recovery phase at 20 W followed, marking the end of the test.

Ergometry

The computer-controlled electromagnetically-braked cycle ergometer (Excalibur Sport PFM, Lode BV, Groningen, NL) is instrumented with force transducers in the bottom bracket spindle. Left and right torque (Nm) was measured independently (peak force 2000 N, < 0.5 N resolution and measurement uncertainty of < 3%). Instantaneous angular velocity of the crank ($rad.s^{-1}$) was measured with a resolution of 2° using three independent sensors sampling in series (measurement uncertainty of < 1%). During isokinetic efforts, power was calculated every 2° from torque and angular velocity measurements.

Cardiopulmonary Measurements

Respired gases and ventilation were measured breath-by-breath with a commercial metabolic measurement system (VMax Spectra, CareFusion, San Diego, CA USA). The system was calibrated immediately prior to each experiment. A 3 L syringe (Hans Rudolph Inc., Shawnee, KS, USA) was used to calibrate the mass flow sensor from ~0.2 to 8.0 $L.s^{-1}$, mimicking flow rates expected at rest and during exercise. The CO_2 and O_2 analysers were calibrated using gases of known concentrations (O_2 26.0% and 16.0%; CO_2 0.0% and 4.0%).

Statistical analyses

A Wilcoxon test was used to assess task power vs. isokinetic power at exercise intolerance. This was due to a violation in the assumption of equal variance in each attempt to use the paired t-test. A one-factor (time) repeated measures analysis of variance was performed to analyze the reduction in isokinetic power over time. In this case, all Bartlett's and Brown-Forsythe tests were non-significant. *Post hoc* t-tests were used to determine the location of differences in the event of a significant omnibus test. Statistical tests were considered significant at $p<0.05$. All data was analyzed using the Statistical Package for the Social Sciences (SPSS v22. SPSS Inc, Chicago, IL, USA). Results are reported as mean ± SD.

Results

Oxygen uptake during ramp-incremental and constant power exercise

$\dot{V}O_{2peak}$ during ramp-incremental and constant power exercise was 3.3 ± 0.7 L/min and 3.4 ± 0.7 L/min, respectively. $\dot{V}O_{2peak}$ was not different between ramp-incremental and constant power exercise ($p>0.05$).

Time course of locomotor fatigue during constant power exercise to intolerance

Constant power exercise duration was 312 ± 37 s. Maximal isokinetic power output declined ($F[5,84]=10.2$, $p<0.05$) throughout heavy constant power exercise (Figure 2B). P_{iso} at 30, 60, 120, 180 s, and the limit of tolerance was 609 ± 165, 503 ± 195, 443 ± 157, 449 ± 133, and 337 ± 94 W, respectively. Of the total decline in isokinetic power,

~36% occurred within the first minute of exercise and reductions (p<0.05) in isokinetic power occurred at the final 3 time-points vs the baseline P_{iso} (666 ± 158 W; Figure 2B).

The dynamics of locomotor fatigue were similar when the participants were split based on sex (Figure 2C, 2D). For men and women, the constant power tolerance was 323 ± 36 and 299 ± 38 s, respectively. The total decline in P_{iso} at the limit of tolerance was ~50% of the baseline power for both men and women. The dynamics appeared somewhat different for men as P_{iso} declined ($F[5,42]=25.6$, p<0.05) such that by 60 s ~30% of the total decline in P_{iso} had occurred (Figure 2C). For women, the same measurement in P_{iso} decline ($F[5,36]=19.7$, p<0.05) equaled ~48% (Figure 2D).

Isokinetic power and power reserve following intolerance

At baseline, participants generated a maximum isokinetic (70 rpm) power of 666 ± 158 W. Constant power (205 ± 46 W) exercise duration was 312 ± 37 s. At intolerance, participants generated an isokinetic power of 337 ± 94 W, and this was greater than the required constant power (p<0.05; Figure 3A). This resulted in a power reserve of 132 ± 74 W or (64 ± 35% of the constant power). During ramp-incremental exercise, participants achieved a peak power of 254 ± 47 W. At intolerance, participants generated a maximal isokinetic (70 rpm) power of 373 ± 101 W, and this was greater than peak ramp power (p<0.05; Figure 3B). Therefore, participants exhibited a reserve for instantaneous power of 119 ± 80 W (or 47 ± 31% of ramp peak power). Additionally, there was no difference (p>0.05) in the magnitude of the power reserve at intolerance

247 between ramp-incremental and constant power exercise (Figure 3). These same
248 findings were extended when the analysis was split based on sex (Figure 3C-F).
249
250 **Discussion**
251 We aimed to measure the dynamics of locomotor fatigue during high-intensity exercise
252 and the reserve in locomotor power at intolerance to constant and ramp-incremental
253 cycling. Our major findings are: 1) locomotor fatigue occurs pseudo-exponentially and
254 rapidly, with ~36% of total fatigue accumulating within the first minute of exercise, and
255 2) a 47 ± 31% and 64 ± 35% locomotor power reserve is present at the limit of tolerance
256 in ramp-incremental and constant power exercise, respectively. Therefore, locomotor
257 fatigue accumulates rapidly during high-intensity exercise, following an approximately
258 exponential profile. However, at the limit of tolerance, participants exhibited a reserve
259 for instantaneous power production that exceeds the task requirement.
260
261 *Locomotor fatigue within the first minutes of high-intensity exercise*
262 Similar to knee-extension exercise (24), approximately half of the locomotor fatigue
263 occurred early in the cycling bout. At the onset of high-intensity exercise, the finite
264 kinetics of oxidative phosphorylation necessitates that glycolysis and phosphocreatine
265 breakdown make substantial contributions to maintain ATP concentration (~8.2 mM),
266 with the greatest contribution at the start of the exercise bout (33). During the initial ~10
267 s of high-intensity exercise, [PCr] rapidly falls while [Pi] rises. Pi concentration is tightly
268 correlated to force production, such that high [Pi] decreases ATPase activity and power

production (13). During the first 30 s of exercise in our experiment, significant fatigue has already accumulated (Figure 2B).

In addition to depletion of the finite PCr stores, glycolysis contributes substantially to ATP resynthesis during high-intensity exercise, and is activated within seconds of exercise onset (34). While lactate is the preferred fuel of mitochondrial oxidative phosphorylation, the associated acidosis may be detrimental to muscle function (35). This occurs due to an acidic muscular environment reducing the number of high-force cross bridges in fast fibers, the force per cross bridge in both fast and slow fibers, and myofibrillar Ca^{2+} sensitivity (14, 36).

Continual depletion of the finite energy (PCr, glycogen) sources leads to raised concentrations of Pi, ADP, and lactate in working skeletal muscles. The additive effects of Pi and H^+ decrease power through cross-bridge disruption when elevated above resting concentrations (13). Locomotor fatigue accumulates with a similar time course to that of intramuscular metabolites. We feel this kinetic association suggestions the decline in voluntary power being explained by the accumulation of phosphate and fatigue-related intramuscular metabolites. However, there is clearly a complex interaction of many fatigue-inducing mechanisms in skeletal muscle and our study does not offer mechanistic insight into these mechanisms. In addition to the changes in high-energy phosphates, low pH, plasma membrane excitability through loss of intracellular K^+ (37), and cross bridge sensitivity to suboptimal $Ca2^+$ (35, 36) are critically important in the fatigue process. While the dynamics of increased interstitial K+ or reduced

292 intracellular Ca+ and pH may not match with the kinetics of locomotor fatigue, they likely
293 work synergistically with the phosphate changes to reduce force output. This is
294 particularly true in experiments completed at near physiologic temperatures (35).
295
296 We do want to highlight an important limitation in this section, in that the nature of the
297 exercise in our current experiment is substantially different to that of most experiments
298 where 31P MRS was used to measure instramuscular metabolites. In most examples,
299 knee extension is generally the exercise mode (15). Perhaps the closest design might
300 be that of Rodenburg et al., where the ergometer required hip and knee extension (38).
301 However, even in this case there appears to be a rapid increase in the Pi/PCr ratio with
302 increasing power output. This suggests that exercise similar to cycling should be no
303 different to isolated muscle experiments or single-leg knee extension.
304
305 *Total locomotor fatigue accumulation at limit of tolerance*
306 Constant power exercise was maintained for 312 ± 37 s. Progressive reduction in
307 maximal isokinetic power output is seen up until this point (Figure 2B). While women
308 exhibited lower P_{iso} generation at baseline, the relative reduction in P_{iso} at the limit of
309 tolerance was similar to men (~50% of baseline P_{iso}). The dynamics of locomotor fatigue
310 accumulation appeared to be somewhat faster in women, however we would need a
311 larger sample size to be sure about sex differences.
312
313 Critical power represents the highest work rate that will elicit a metabolic steady-state,
314 and therefore demarcates unpredictably-sustainable from predictably-unsustainable

315 exercise. While fatigue related metabolites reach a plateau at ~2-3 min during sub-CP
316 exercise, continual accumulation up to intolerance is seen in supra-CP exercise (15).
317 Similarly, participants performing sub-CP exercise were able to maintain voluntary
318 velocity-specific peak power between 3 and 8 min of exercise (19). Together, this lends
319 support to the close linkage between the intramuscular environment, locomotor fatigue,
320 and exercise tolerance (14). Our data show a continual accumulation of locomotor
321 fatigue during supra-CP exercise, and fit closely with this assertion that the
322 intramuscular events underpin the reduction in voluntary muscle power.
323
324 In addition to peripheral contributors to fatigue and the fall in isokinetic power
325 production, afferent feedback (including conscious perception of effort) and efferent
326 feed-forward mechanisms may modulate exercise tolerance. Muscle force production
327 falls progressively and evoked peak power reaches an approximate plateau (at ~40% of
328 time trial) during non-sustainable exercise (24). However, by intolerance, it appears as
329 though peripheral locomotor fatigue is 'regulated' to some maximal value (28, 39).
330 Additionally, early accumulation of peripheral fatigue is compensated for by increased
331 motor drive, and task failure therefore may be due, ultimately, to increased central
332 fatigue arising late in the exercise task (40). Therefore, in addition to intramuscular
333 fatigue-related metabolite buildup, central regulation and emotional responses to
334 exercise likely play a role in the development of locomotor fatigue (41). As we did not
335 measure contributions of 'central' and 'peripheral' mechanisms of fatigue, we can only
336 provide speculation as to the origin of locomotor fatigue in our data. Therefore, the close
337 dynamic association of our locomotor power data to the buildup of intramuscular

metabolites may only be coincidental, and not causal. A new approach is now available that may provide some insight into the fatigue-mechanisms through instantaneous assessment of neuromuscular fatigue following cycling exercise, albeit isometric (27). Finally, our data only provide insight for a single contraction velocity. The shape of the power-velocity relationship (42) suggests that the magnitude of change will be larger at high contraction velocities, but the dynamics of the fatigue at those contraction velocities is unknown.

A reserve in locomotor power generating capacity at the point of intolerance

Brief maximum isokinetic power at the limit of tolerance (and at 70 rpm) was higher than the task power in both ramp-incremental and constant power exercise (Figure 3). This represents a temporary separation between perceptual and physiological limits of power production. It is important to note that there was no difference in $\dot{V}O_{2peak}$ in ramp and constant power exercise, showing individuals did attain systems limits in both exercise test formats. Additionally, the reserve in locomotor power represents a near-instantaneous (~5 s) capacity for power production. This is in contrast to a sustained (>30s) maximal isokinetic effort in which power output falls nearer to critical power immediately following intolerance (43). Finally, the contraction velocity likely influences the magnitude of the power reserve, as power generation is a parabolic function of velocity (42, 44). While the portion of the power-velocity relationship that encompasses cycling becomes flatter with muscle fatigue (45), the absolute isokinetic power generated at intolerance at 70rpm is likely to be less than that of higher contraction velocities. This is especially true for short bursts of sprint type exercise. Conversely,

during longer, incremental exercise protocols there are surprisingly negligible benefits across a range of 40-120rpm for $\dot{V}O_{2peak}$ or peak power achieved during the incremental test (42, 44). This is of course in contrast to higher selected pedaling frequencies in competitive cycling where both efficiency-velocity, and power-velocity relationships must be considered. The relevancy of the 'reserve' in locomotor power in our experiment, therefore, is that the power output is constrained to the contraction velocity similar to that used by our volunteers during the constant or ramp task. However, this design decision to make isokinetic measurements only at 70rpm is also a weakness of our study – a more complete measurement of the power-velocity relationship would clearly have been a more desirable choice. We did not choose this design due to the substantial burden on research volunteers, as it would require many-fold more visits to the laboratory.

Additional recruitment of motor units is responsible for the instantaneous increase in force production, suggesting that the limit of tolerance during the exercise task is not defined by a ceiling of motor recruitment and power production – this is in contrast to single limb exercise (29). Exercise tolerance may in some circumstances therefore be limited by perception of effort, rather than neuromuscular fatigue that 'caps' power production sufficient to continue the task (8). However, these perceptual effects may be dependent on the population studied (chronic cardiopulmonary disease vs healthy vs athlete). Contrary to our findings, no reserve was present following whole body exercise in men with VO_{2max} 4.2 ± 1.0 L/min (22). It is possible that highly-trained individuals maintain a close association of perceptual and physiologic limits and exhibit central and

384 peripheral fatigue mechanisms different to that of our recreationally-active participants -
385 and certainly to that of patients with cardiopulmonary disease (30). No research has
386 examined oxidative capacity as a modifier of the locomotor power reserve, or the
387 mechanisms that underpin this phenomenon.
388
389 **Conclusions**
390 We aimed to measure 1) the dynamics of locomotor fatigue at the onset of high-intensity
391 cycling, and 2) the reserve in locomotor power following constant and ramp-incremental
392 cycling to the limit of tolerance. Maximal voluntary isokinetic power fell progressively
393 during constant power exercise. However, ~36% of the total fatigue occurred in the first
394 60 s of exercise, showing rapid, approximately exponential kinetics.
395
396 The dynamics of locomotor fatigue were similar to the dynamics reported for primary
397 fatigue-related intramuscular metabolites, suggesting a close mechanistic link between
398 the intramuscular milieu and voluntary muscle power. Instantaneous isokinetic power
399 production at the limit of tolerance exceeded that of the task requirement, regardless of
400 the work rate profile. Thus, the perceptual and physiologic limits were dissociated at the
401 limit of tolerance in recreationally active volunteers. While the dynamics of locomotor
402 fatigue may reflect the disturbances in the intramuscular metabolic environment, the
403 limit of tolerance may be predominantly determined by mechanisms limiting voluntary
404 motor unit recruitment.

Competing Interests

Authors have no competing interests.

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Figure 1. Schematic of the exercise testing protocols. **Panel A:** Visit 1 consisted of ramp-incremental exercise terminated with a maximal isokinetic effort at the limit of tolerance. Filled and open symbols represent 5 s maximal isokinetic power (P_{iso}) measured either in triplicate at baseline (filled) or at the termination of the exercise test (open). **Panel B:** Visits 2-6 were variable duration constant power tests terminated with P_{iso}. Constant power tests were terminated with maximal P_{iso} at either predetermined times (30, 60, 120, 180 s; shaded symbols) or at the limit of tolerance (open symbol).

Figure 2. Construction of the dynamics of locomotor fatigue during constant supra-critical power exercise. **Panel A:** Representation of a single P_{iso}, in this case at baseline with no prior exercise. Dashed line and filled symbol represents mean during maximal effort. **Panel B:** The dynamics of locomotor fatigue are presented as the reduction in maximal P_{iso} during exercise as a function of time ($F[5,84]=10.2$, $p<0.05$). Solid circle is the baseline mean as measured in Panel A. Lines represents mean (solid) and SD (dashed) constant power task requirement. **Panel C:** The dynamics of locomotor fatigue, as in Panel B, however with only the men included ($F[5,42]=25.6$, $p<0.05$). **Panel D:** Only the women included ($F[5,36]=19.7$, $p<0.05$). *Different to baseline P_{iso} ($p<0.05$).

Figure 3. Comparison between task requirement and maximal isokinetic power (P_{iso}) at the limit of tolerance. **Panel A:** P_{iso} at intolerance to constant power exercise was greater ($p<0.05$) than the task requirement. **Panel B:** P_{iso} at intolerance to ramp

incremental exercise was greater (p<0.05) than the peak power achieved during the ramp. **Panel C:** P_{iso} at intolerance to constant power exercise was greater (p<0.05) than the task requirement in men only. **Panel D:** P_{iso} at intolerance to ramp incremental exercise was greater (p<0.05) than the peak power achieved during the ramp in men only. **Panel E:** P_{iso} at intolerance to constant power exercise was greater (p<0.05) than the task requirement in women only. **Panel F:** P_{iso} at intolerance to ramp incremental exercise was greater (p<0.05) than the peak power achieved during the ramp in women only.

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