

The Effect of Endurance Exercise Modality on Markers of Fatigue

Andrew R. Moore^{a, b*}, Jasmin C. Hutchinson^a, Christa R. Winter^a, Paul C. Dalton^a, and Vincent J. Paolone^a

^aDepartment of Exercise Science and Athletic Training Springfield College 163 Alden Street Springfield, MA, USA, 01109

^bDepartment of Kinesiology Augusta University 1120 15th St, CFH-134 Augusta, GA, USA, 30912

Corresponding Author: Dr. Andrew Moore, E-mail: andmoore@augusta.edu

ARTICLE INFO

Article history

Received: May 15, 2021

Accepted: July 20, 2021

Published: July 31, 2021

Volume: 9 Issue: 3

Conflicts of interest: The authors declare that there are no conflicts of interest to report.

Funding: None

ABSTRACT

Background: Exercise power output, and resulting fatigue, is regulated based on central and peripheral sensory input. Whether exercise mode, specifically, contributes to this regulation remains unexplored. **Objective:** This study was designed to determine if differences in markers of fatigue would be present during two time trials of similar duration and intensity, as a result of exercise mode (cycling and rowing). **Method:** In a randomized crossover design, nine subjects completed the two 7-min time trials, on different days. Exercise power output, heart rate, rating of perceived exertion, and blood lactate measurements were analyzed using repeated-measures ANOVAs. **Results:** There was a significant interaction between mode and time for power output ($p = .02$), but no significant differences between matched time points were observed for any of the dependent variables used to assess fatigue ($p > .05$). **Conclusion:** Similar levels of heart rate, perceived exertion, and blood lactate for time trials on different modes, but with the same duration and directed intensity, suggest that in a laboratory environment, exercise is regulated more by physiological disturbance and sensory cues than by exercise mode. These findings support the sensory tolerance limit of exercise fatigue.

Key words: Ergometry, Rowing, Heart Rate, Lactate, Sensory Processing, Perception

INTRODUCTION

Neuromuscular fatigue is a symptom of physically demanding exercise that results in reduced muscle force production capability (Gandevia, 1992; Twomey et al., 2017). Exercise-related fatigue can originate from the central nervous system as central fatigue (a reduced neural activation of the muscle; Meeusen et al., 2006) and from sites at or distal to the neuromuscular junction as peripheral fatigue (i.e. biochemical impairments; Cè et al., 2020).

Central and peripheral fatigue are intertwined and influence each other through feedforward (Crapse & Sommer, 2008; Liu et al., 2005; Tucker & Noakes, 2009) and feedback (Laginestra et al., 2021; Taylor, Amann, Duchateau, Meeusen, & Rice, 2016) mechanisms. The main feedforward mechanism of note is corollary discharge in which signals to sensory areas of the motor cortex are produced when movement is initiated. In this way, effort perception and central fatigue are increased proportionately with exercise (Liu et al., 2005). Neural feedback mechanisms entail sensory information gathered from the peripheral contracting muscles (metabolic acidosis, increased heart rate, etc.) that is registered by sensory areas of the brain, resulting in greater exertion and fatigue (Taylor et al., 2016). Sensory information generated from central and peripheral locations throughout the body is integrated into an overarching sensation of fatigue which shapes the impairment

of muscle force output during exercise (Hureau, Romer, & Amann, 2018).

Current models of exercise-related fatigue propose that the purpose of integrating sensory information is to centrally regulate work rate during exercise in a way that prevents catastrophic fatigue and exercise failure (Hureau et al., 2018; Thomas, Goodall, & Howatson, 2018). Excessive sensory input beyond what is considered tolerable for the exercise task results in either cessation of the task, or a reduction in work output to cope with the demands of exercise while preventing harm. This concept was illustrated by Hureau, Ducrocq, and Blain (2016) who reported that subjects asked to complete a series of maximal 10-second sprints produced lower power output and experienced greater central and peripheral fatigue with each successive sprint, but only up to a certain point. After the sixth 10-second sprint, subjects displayed no additional reduction in power output, muscle activation, or electrically-evoked twitch force of the quadriceps muscles. The authors interpreted the fact that power output and indices of central and peripheral fatigue failed to decrease beyond a seemingly fixed amount, despite more maximal effort sprints, to mean that excessive sensory input halted further muscle activation to preserve homeostasis at a reasonable level (Hureau et al., 2016).

This concept of a fatigue threshold carries over to endurance exercise as well. Sensory markers of fatigue appear

to shape work rate during self-paced time trials (TTs), even when distinctly different pacing behaviors (work rate distribution patterns) are used. Azevedo et al. (2021) grouped cyclists of similar training status based on the natural spontaneous pacing behavior (U-shaped power output, J-shaped, and inverted-J-shaped) they displayed during a 4-km cycling TT. There were no differences between groups in the fatigue indices that were measured throughout the TTs. Rating of perceived exertion (RPE), heart rate (HR), and VO_{2max} all increased at a similar rate for the three groups despite widely varying power output patterns. Additionally, measures of fatigue (maximal voluntary isometric contraction, voluntary activation, and twitch force) compared before and 2 minutes after the TTs showed similar reductions between groups with unique pacing profiles. It was suggested by Azevedo et al. that sensory input was used to regulate work rate on a continuous basis throughout the TTs as a means to ensure a consistent level of homeostatic disturbance and fatigue, regardless of preferred pacing profile.

Whether this regulation of exercise based on sensory input is present in a given exercise task on different modes of exercise has not been determined. Unique exercise modes, such as cycling and rowing, require different physiological demands. For example, coordination of limbs (unilateral vs bilateral), active muscle mass, and movement cadence are all different and may alter sensory fatigue in different ways. The impact of exercise mode on changes in markers of fatigue during self-paced exercise has not been investigated.

Therefore, the purpose of this study was to determine if subjects who complete exercise tasks of similar duration and prescribed intensity on either rowing or cycling modalities exhibit differences in sensory markers of fatigue, reflected by RPE, HR, and blood lactate. For self-paced and self-adjusted exercise, we expect the exerciser to modify work rate in response to the level of perceived fatigue and sensory cues so as not to exceed a tolerable level, in accordance with the sensory tolerance limit model of exercise performance. We hypothesized that there would be no differences in HR, RPE, or blood lactate responses to maximal effort 7-min TTs on cycling and rowing modes of exercise.

METHOD

Participants and Study Design

The current study used a repeated-measures quasi-experimental design to examine the differences in physiological variables in subjects completing cycling and rowing TTs. All methods and procedures were reviewed and approved by the Institutional Review Board of Springfield College prior to any data collection. The dependent variables that were measured in this study were HR measured in beats per minute, RPE measured using the Borg 6-20 scale (Borg, 1982), lactate measured in mmol/L, and power output measured in Watts (W). The independent variable was mode of exercise (cycling or rowing).

A power analysis (G*Power version 3.1) indicated that for a two-way repeated measures ANOVA ($\beta = .20$, $\alpha = .05$, $\eta_p^2 = .34$), a minimum estimated sample size of 10 subjects

would be needed to observe a significant interaction effect in any of the dependent variables. Men age 18-40 years old from the greater Western Massachusetts area were recruited for this study. Potential subjects were screened to exclude people who were sedentary for the two months preceding the experiment (not engaging in physical activity for 30 min/day at least once a week) and people who had more than one month of training for cycling or rowing competitions. Potential subjects were also excluded if they had a cardiovascular or musculoskeletal health condition that would put them at a greater risk for injury during exercise according to a modified PAR-Q completed before the first day of participation.

A total of 10 subjects were recruited for the study, yet one subject was excluded from analysis due to failure to follow TT instructions. Therefore, nine men (age = 22.67 ± 2.65 years; BMI = 26.55 ± 4.24) completed the study. Informed consent was completed before any participation in the study ensued.

Instrumentation

A Velotron cycle ergometer (CompuTrainer Inc., Seattle, WA) and Concept II Model C rowing ergometer (Concept2 Inc., Morrisville, VT) were used to measure power output for the cycling and rowing TTs, respectively. Equipment monitors were positioned so that only the researchers, not the subjects, were able to view the screen.

A 2 foot by 3 foot laminated poster with the Borg 6-20 RPE scale clearly typed was made visible to all subjects during all laboratory visits. A Biosen 5030 (EKF Industrie, Barleben, Germany) blood lactate analyzer was used to measure blood lactate concentration before and after each TT. A Polar FT7 HR monitor (Polar Electro, Lake Success, NY) was used to measure HR during all exercise sessions. A clock displaying elapsed time was visible to subjects throughout both TTs.

Exercise Protocol

All approved subjects visited the Human Performance Laboratory on three different occasions. Subjects were asked to abstain from alcohol, caffeine, and strenuous exercise in the 24 hr prior to all laboratory visits, and to maintain a food log to ensure consistent diet and caloric intake prior to each visit. First a familiarization session was completed on both the rowing and cycle ergometers to become acquainted with the equipment and accurate response using the Borg RPE scale. A light warm-up preceded a 7-min exercise session of progressively increasing intensity. The session was completed at a self-selected speed on the designated mode of exercise, wherein each subject was asked to work at a set RPE on the Borg 6-20 RPE scale for a given amount of time. The first 2 min were performed at an RPE of 10, the next 2 min at an RPE of 13, the next 2 min at an RPE of 17, and the last 1 min at an RPE of 19. Thus, subjects were exposed to the full range of perceived exertion defined by the Borg RPE scale and the self-paced nature of the experimental sessions. After completing the familiarization session on one modality, subjects rested passively for 15 min while drinking water

ad libitum and then began the second familiarization session on the remaining exercise mode.

At least 24 hours after the familiarization session, all subjects completed the two 7-min TTs on rowing and cycling equipment on two visits to the lab. The TTs were separated by 3 days and completed in a counterbalanced manner. Subjects were informed of the duration of the task and instructed to give a maximum effort in order to cover the greatest distance (i.e. the greatest power output) possible in 7 min. Pace and power output were free to vary at the will of the subject, but no performance or physiological feedback was provided at any time. No verbal encouragement or motivation was provided either. Data collection procedures were identical for both TTs. Power output and HR were recorded at the end of each minute. RPE was recorded at the end of minutes 1, 4, and 7. Blood lactate data was collected and recorded prior to and immediately following each TT.

Statistical Analysis

Two 2x7 repeated measures ANOVAs were used to analyze the effects of mode (cycling or rowing) and time (minutes 1 through 7) on power output and HR during each minute of exercise. Repeated measures ANOVAs were also used to determine the effects of mode and time on RPE (2x3 ANOVA; minutes 1, 4, and 7) and blood lactate (2x2 ANOVA; pre- and post-exercise). Alpha level was set at .05 for all analyses, which were completed using SPSS software, version 25. All data were screened for outliers, sphericity, and normality. Follow-up Bonferroni adjusted post hoc tests were used for significant ANOVA results.

RESULTS

No outliers (studentized residual ≥ 3 SD from group mean) were detected. There were no violations to the assumption of normality based on the Shapiro-Wilk test. If the assumption of sphericity for any ANOVA had not been met according to Mauchly's Test of Sphericity, the Greenhouse Geisser adjusted value was used. Complete ANOVA results are reported in Table 1. Descriptive statistics for all dependent variables and the significant main and interaction effects are represented graphically in Figure 1. Complete descriptive statistics are also available in a supplemental table, along with complete post hoc test results (DOI 10.17605/OSF.IO/NM3XS).

There was a significant interaction effect between mode and time for power output, $F(2.19, 17.55) = 4.52, p = .024$. There was a significant interaction effect for HR as well, $F(2.47, 19.75) = 4.45, p = .02$. However, Bonferroni-adjust-

ed post hoc tests did not reveal significant differences in HR between any of the matched time points ($p > .05$ for all comparisons). Therefore, this significant ANOVA result was deemed a Type I error (Chen, Xu, Tu, Wang, & Niu, 2018). There was no significant interaction effect between mode and time for RPE, $F(2, 16) = 1.07, p = .37$ or lactate, $F(1, 8) = 0.10, p = .77$. There were significant main effects of time for HR, $F(2.58, 20.61) = 147.24, p < .001$, RPE, $F(1.26, 10.08) = 67.09, p < .001$, and lactate, $F(1, 8) = 134.67, p < .001$, each of which increased with time. No main effects of mode were present for any of the variables.

DISCUSSION

The objective of this study was to determine if different exercise modes for a similar exercise task would result in differences in markers of fatigue. The main finding was that there were no differences in HR, RPE, or blood lactate responses to TTs of the same duration and directed, but self-adjusted, intensity when cycling and rowing modes were used. These similarities in sensory fatigue signals at matched time points were found despite the fact that power output was different between modes and varied throughout each TT. Specifically, power output was significantly higher in the rowing condition at minute 1 and 7 (the first and last minutes of the TTs). There were other differences between exercise modes as well, such as active muscle mass (greater for rowing than for cycling), coordination (unilateral cycling vs bilateral rowing), spatial displacement (stationary cycling vs head and upper body moving on rower), and position (upright vs seated). Work rate was modified throughout both trials, yet the resulting disruption in homeostasis and corresponding fatigue markers were similar between conditions at matched time points.

Our findings are similar to those reported by Azevedo et al. (2021) that in trained cyclists using different spontaneous pacing behaviors for a simulated TT, perceptual responses and neuromuscular markers of fatigue (HR, RPE, VO_2 , muscle voluntary activation, etc.) changed in a similar fashion throughout the trial. The authors attributed the similar fluctuations in these markers of fatigue throughout the TTs to the unique way in which each subject regulated work output in accordance with their individual sensory tolerance limit to complete the TT as fast as possible without succumbing to catastrophic collapse (Azevedo et al., 2021).

The results of this study represent a novel application of the sensory tolerance limit of exercise by showing that power output during exercise of different modes is regulated in such a way as to result in the same pattern of homeostatic

Table 1. ANOVA results for fatigue variables and power output

	Mode			Time			Mode x Time		
	<i>F</i>	<i>p</i>	η_p^2	<i>F</i>	<i>p</i>	η_p^2	<i>F</i>	<i>p</i>	η_p^2
Heart Rate	0.27	.62	.03	147.24	< .005	.95	4.45	.02	.36
RPE	1.56	.25	.16	67.09	< .005	.89	1.07	.35	.12
Lactate	0.06	.82	.01	134.68	< .005	.94	0.10	.77	.01
Power output	4.14	.08	.34	16.01	< .005	.67	4.52	.02	.36

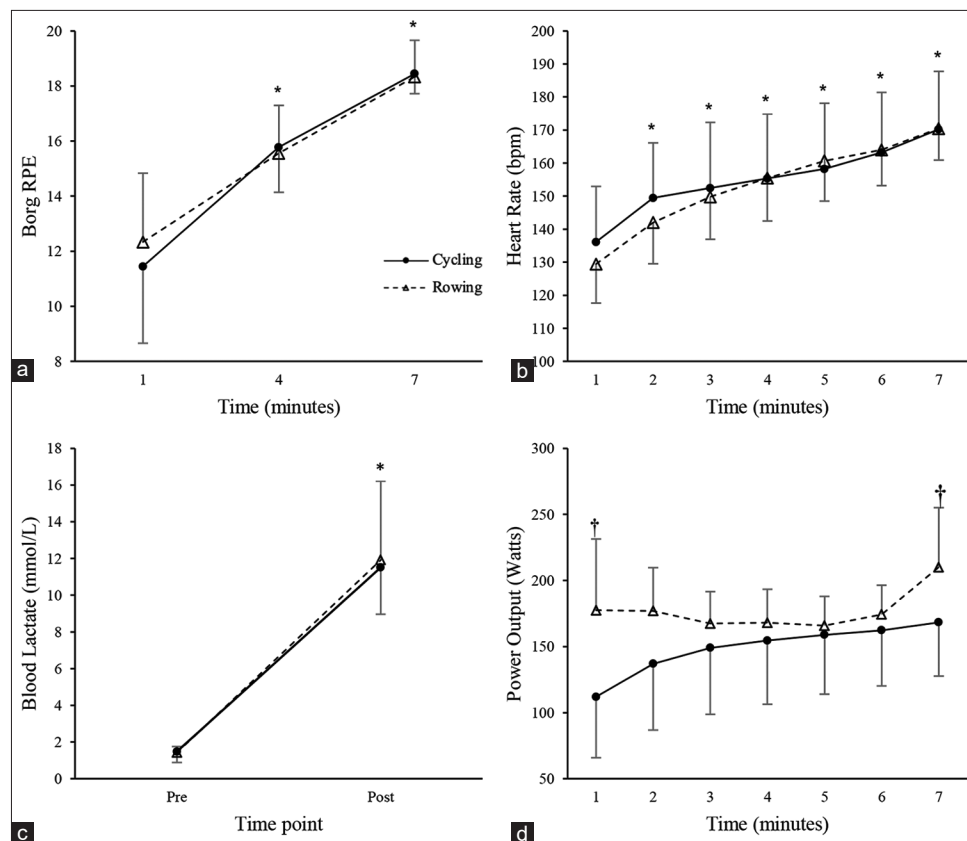


Figure 1. Change in fatigue variables and work rate throughout the cycling (solid line) and rowing (dashed line) TTs. (a) rating of perceived exertion (RPE); (b) heart rate (HR); (c) blood lactate; (d) power output. Values displayed are mean \pm SD. Bonferroni-adjusted post hoc test results are indicated using symbols.

* = main effect of time; greater than all previous time points, $p < .01$

† = interaction effect; rowing greater than cycling, $p < .05$

disturbance and sensory changes. That is, the level of fatigue that subjects considered “tolerable” appeared consistent between TTs despite the many differences between the exercise modes. The fact that exercise intensity was modified in both trials to ensure a consistent level of global fatigue markers (HR, RPE, and lactate) before the end of the task is in line with the sensory tolerance limit model of fatigue (Hureau et al., 2018).

Thomas et al. (2018) highlight the idea that performance fatigability (reduction in exercise performance) depends not only on the active muscle mass for a given exercise, but also on the ensuing disruption to homeostasis in other physiological systems. This concept is consistent with our findings, given that for modes of exercise which engage different amounts of muscle mass, the extent to which the musculature was used did not surpass the disturbance of HR, RPE, and lactate.

The existence of a sensory tolerance threshold beyond which fatigue does not increase is contradicted by Konings, Parkinson, Zijdwind, and Hettinga (2018) who noted that the inclusion of a virtual opponent in two 4-km cycling TTs resulted in different fatigue indices compared to when no opponent was used. During the TT for which there was an opponent programmed to have a fast start, subjects displayed a consistently higher HR, a higher power output, and a greater decrement in maximal voluntary contraction and potentiated

twitch force of the quadriceps muscles, compared to the TT for which there was no virtual opponent. RPE, however, remained similar between each of the TTs at each time point. Konings et al. (2018) underscore the idea that human-environment interactions influence central and peripheral fatigue beyond internal states and that sensory information may be interpreted differently in different environmental and psychological contexts. In this regard, future follow-up studies should seek to investigate how fatigue is impacted by exercise modes that result in different environmental interactions.

Mode familiarity may have an impact on exercise regulation, such that trained athletes may be able to exert themselves to a greater extent on a familiar mode of exercise, beyond what might be considered “tolerable” on an unfamiliar exercise mode. Future studies should investigate whether subjects trained in one mode of exercise exhibit similar increases in sensory and perceptual markers of fatigue for non-familiar modes given the same exercise duration and directive for intensity.

One limitation of this study was the small sample size which impacted the ability to determine if differences in variables were significant. Post hoc power analyses (G*Power version 3.1) revealed that based on the effect sizes recorded from RPE and lactate, minimum estimated sample sizes of 76 and 649, respectively, would have been required to observe adequately powered, significant ($\beta = .20$, $\alpha = .05$) interaction

effects between mode and time for these variables. Sample sizes of this magnitude were not feasible for this study. Bonferroni-adjusted post hoc tests for HR interaction effect between mode and time may have been detected with a larger sample size (minimum estimated 29 subjects for minute 2).

The results of this study are practically relevant to people interested in exercise who are concerned with the best form of endurance exercise for physical conditioning. Our results suggest that for a predetermined time and effort commitment, the same levels of physiological strain would be achieved on either rowing or cycling without explicit conscious adjustments to work rate needing to be made. One of the strengths of this study was that work rate was free to vary during both time trials which is similar to the manner in which exercise is completed in field settings. The findings are therefore more applicable to exercise experiences in the real world.

CONCLUSION

In conclusion, HR, RPE, and blood lactate responses to a self-paced exercise task in a laboratory environment were similar between cycling and rowing modes despite differences in power output. These findings support the idea that an individual sensory tolerance limit which regulates exercise intensity is consistent across exercise modes.

ACKNOWLEDGEMENTS

We would like to thank all subjects who donated their time and effort to complete the study.

REFERENCES

- Azevedo, R. d. A., Silva-Cavalcante, M. D., Cruz, R., Couto, P., Lima-Silva, A. E., & Bertuzzi, R. (2021). Distinct pacing profiles result in similar perceptual responses and neuromuscular fatigue development: Why different “roads” finish at the same line? *European Journal of Sport Science*, 1-11. doi: <https://doi.org/10.1080/17461391.2021.1922507>
- Borg, G. A. (1982). Psychophysical bases of perceived exertion. *Medicine & Science in Sports & Exercise*, 14(5), 377-381. doi: [10.1249/00005768-198205000-00012](https://doi.org/10.1249/00005768-198205000-00012)
- Cè, E., Longo, S., Limonta, E., Coratella, G., Rampichini, S., & Esposito, F. (2020). Peripheral fatigue: New mechanistic insights from recent technologies. *European Journal of Applied Physiology*, 120(1), 17-39. doi: <https://doi.org/10.1007/s00421-019-04264-w>
- Chen, T., Xu, M., Tu, J., Wang, H., & Niu, X. (2018). Relationship between omnibus and post-hoc tests: An investigation of performance of the F test in ANOVA. *Shanghai Archives of Psychiatry*, 30(1), 60-64. doi: [10.11919/j.issn.1002-0829.218014](https://doi.org/10.11919/j.issn.1002-0829.218014)
- Crappe, T. B., & Sommer, M. A. (2008). Corollary discharge across the animal kingdom. *Nature Reviews Neuroscience*, 9(8), 587-600. doi: <https://doi.org/10.1038/nrn2457>
- Gandevia, S. C. (1992). Some central and peripheral factors affecting human motoneuronal output in neuromuscular fatigue. *Sports Medicine*, 13(2), 93-98. doi: [10.2165/00007256-199213020-00004](https://doi.org/10.2165/00007256-199213020-00004)
- Hureau, T. J., Ducrocq, G. P., & Blain, G. M. (2016). Peripheral and central fatigue development during all-out repeated cycling sprints. *Medicine and Science in Sports and Exercise* 48(3), 391-401. doi: [10.1249/MSS.0000000000000800](https://doi.org/10.1249/MSS.0000000000000800)
- Hureau, T. J., Romer, L. M., & Amann, M. (2018). The ‘sensory tolerance limit’: A hypothetical construct determining exercise performance? *European Journal of Sport Science*, 18(1), 13-24. doi: [10.1080/17461391.2016.1252428](https://doi.org/10.1080/17461391.2016.1252428)
- Konings, M. J., Parkinson, J., Zijdwind, I., & Hettinga, F. J. (2018). Racing an opponent: Alteration of pacing, performance, and muscle-force decline but not rating of perceived exertion. *International Journal of Sports Physiology and Performance*, 13(3), 283-289. doi: <https://doi.org/10.1123/ijsp.2017-0220>
- Laginestra, F. G., Amann, M., Kirmizi, E., Giuriato, G., Barbi, C., Ruzzante, F., Schena, F. (2021). Electrically induced quadriceps fatigue in the contralateral leg impairs ipsilateral knee extensors performance. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 320(5), R747-R756. doi: <https://doi.org/10.1152/ajpregu.00363.2020>
- Liu, J. Z., Yao, B., Siemionow, V., Sahgal, V., Wang, X., Sun, J., & Yue, G. H. (2005). Fatigue induces greater brain signal reduction during sustained than preparation phase of maximal voluntary contraction. *Brain Research*, 1057(1-2), 113-126. doi: <https://doi.org/10.1016/j.brainres.2005.07.064>
- Meeusen, R., Watson, P., Hasegawa, H., Roelands, B., & Piacentini, M. F. (2006). Central fatigue. *Sports Medicine*, 36(10), 881-909. doi: <https://doi.org/10.2165/00007256-200636100-00006>
- Taylor, J. L., Amann, M., Duchateau, J., Meeusen, R., & Rice, C. L. (2016). Neural contributions to muscle fatigue: From the brain to the muscle and back again. *Medicine and Science in Sports and Exercise*, 48(11), 2294-2306. doi: [10.1249/MSS.0000000000000923](https://doi.org/10.1249/MSS.0000000000000923)
- Thomas, K., Goodall, S., & Howatson, G. (2018). Performance fatigability is not regulated to a peripheral critical threshold. *Exercise and Sport Sciences Reviews*, 46(4), 240-246. doi: [10.1249/JES.0000000000000162](https://doi.org/10.1249/JES.0000000000000162)
- Tucker, R., & Noakes, T. D. (2009). The physiological regulation of pacing strategy during exercise: A critical review. *British Journal of Sports Medicine*, 43(6), e1-e1. doi: [10.1136/bjism.2009.057562](https://doi.org/10.1136/bjism.2009.057562)
- Twomey, R., Aboodarda, S. J., Kruger, R., Culos-Reed, S. N., Temesi, J., & Millet, G. Y. (2017). Neuromuscular fatigue during exercise: Methodological considerations, etiology and potential role in chronic fatigue. *Clinical Neurophysiology*, 47(2), 95-110. doi: [10.1016/j.neucli.2017.03.002](https://doi.org/10.1016/j.neucli.2017.03.002)