International Journal of EXERCISE SCIENCE

Original Research

Accumulated Oxygen Deficit During Arm Cranking in Hypoxia: A Bayesian Perspective and Methodological Considerations

TRISTAN M. STARLING-SMITH†1, DAVID BOFFEY†1, MICHAEL B. LA MONICA‡2, JEFFREY R. STOUT‡1, and DAVID H. FUKUDA‡1

[1]Department of Kinesiology, University of Central Florida, Orlando, FL, USA; [2]Center for Applied Health Sciences, Canfield, OH, USA

†Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 14(3): 1090-1098, 2021. The purpose of this investigation was to observe the effects of normobaric hypoxia on accumulated oxygen deficit (AOD) with evaluation using both Bayesian and Frequentist analyses. Eighteen recreationally active men performed a graded exercise test (GXT) in normobaric normoxia (N; FiO2~20%) and normobaric hypoxia (H; FiO2~14%) to determine peak power output (PPO). Time to exhaustion trials were later conducted at 110% and 120% PPO under both N, and H. AOD and %AN (% anaerobic energy contribution) were calculated in three conditions: N, H, and H using the N regression equation (HN). Bayesian repeated measures ANOVA revealed differences in AOD and %AN between regression equations while Frequentist Repeated measures ANOVA revealed non-significant differences for AOD (p = .148) and %AN (p = .150). Using predicted oxygen consumption extrapolated from a normoxic environment during exercise in hypoxia may lead to overestimation of AOD and %AN with a Bayesian approach and contrasting results using frequentist statistics.

KEY WORDS: Altitude, energy system, upper-body exercise, ergometry, time-to-exhaustion

INTRODUCTION

Energy system contribution is of great importance when assessing the demand for a given activity (1). Accumulated oxygen deficit (AOD) during constant work-rate ergometry is a valid indirect measure of anaerobic energy system contribution to exercise (27). Aerobic contribution is determined by extrapolation of the linear relationship between intensity and oxygen uptake (17). This extrapolation is commonly performed using a traditional graded exercise test where oxygen uptake and intensity are naturally observed. Often, this baseline graded exercise test serves as the only reference point for extrapolation. When calculating aerobic contribution in this way, it may be inappropriate to use regression equations acquired in normoxia to attempt to calculate AOD in a hypoxic environment due to possible underestimation of values. However, a second graded exercise test performed in hypoxia may be necessary.

Moderate normobaric hypoxia inherently reduces aerobic capacity by limiting oxygen availability and transport, possibly leading to an earlier reliance on anaerobic metabolism during exercise. Upper-body ergometry consistently produces maximal VO2 values around 70% of those observed during lower-body cycling (19). While prior research has focused on lower body cycling or full-body exercise, upper body differences in muscle fiber type distribution (14) and diffusion distance (6) may require greater anaerobic energy provisions as reflected by AOD.

Decrements in VO2max observed in hypoxic environments depend on the severity of the imposed hypoxia. In severe acute hypoxia, most of the decrement in VO2max is caused by a decrease in arterial O2 content (5), while in moderate hypoxia, the decrement is attributed to tissue O2 extraction (24). These decrements in O2 tissue extraction may lead to a greater reliance on anaerobic energy systems (12), which might be reflected within the AOD. Therefore, the primary purpose of the current study was to observe the effects of normobaric hypoxia on AOD and anaerobic energy system contribution during different intensities of upper-body arm cranking exercise. The secondary purpose was to provide an example of how a Bayesian analysis may be used to supplement a traditional frequentist analysis in the exercise science field.

To more closely observe these effects, this investigation utilized a Bayesian analysis alongside an equivalent frequentist analysis in an attempt to describe the results from both approaches. This allowed for comparisons between the approaches and provides an example of what a Bayesian analysis looks like in the field of exercise science where this approach is becoming more prevalent (2). Bayesian statistics utilize probability to express the likelihood of an event (4). Through most of the 20th century, Bayesian statistics were not utilized due to the great difficulty in hand calculating results (9), and frequentist statistics became more commonly taught and accepted by the scientific community. Now that Bayesian analysis can be easily calculated using modern computer software it is a viable and possibly more appropriate method of describing data in some scientific fields (8).

METHODS

Participants

Eighteen recreationally active men (21.4 ± 1.4 yr.; 175.5 ± 5.7 cm; 84.8 ± 11.7 kg; 28.8 ± 4.6 l·min^{-1}) volunteered to participate in this study. Six were not included in this final analysis due to incomplete data sets (n = 2) or calculated AOD values falling below zero (n = 4). All participants met the physical activity recommendations of the American College of Sports Medicine to be classified as recreationally active exercising at least 3-5 times per week. To eliminate residual fatigue and soreness during testing, participants were asked to refrain from any strenuous activity within 48 hours prior to testing. Before enrolling in the study, participants completed a Confidential Medical and Activity Questionnaire, as well as a Physical Activity Readiness Questionnaire (PAR-Q), to determine if they had any physical limitations that would keep them from performing any of the study procedures. Participants were asked to maintain their normal diet and, if any, nutritional supplementation consistent throughout the study. Written informed consent was obtained from all participants prior to testing, and the procedures were approved

by the university's institutional review board. "This research was carried out fully in accordance to the ethical standards of the International Journal of Exercise Science" (21).

Protocol

An acute cross-over design was used in this investigation to examine the effects of normobaric hypoxia on upper body ergometry performance. Participants visited the laboratory on seven occasions consisting of a familiarization trial, two days of graded exercise testing (GXT) [one in normobaric normoxia (N) and one in normobaric hypoxia (H)], and four days of time to exhaustion trials (TTE) randomized for intensity and environment. N and H sessions were conducted at a simulated altitude of 500 m (F_iO_2 = 0.201, normoxic condition) and 3250 m (F_iO_2 = 0.14, hypoxic condition), respectively. Participants performed all exercise inside of a large transparent cubicle (At-Home Cubicle, Hypoxico, Inc., New York, NY, USA), which were connected to altitude generators (Everest Summit II Hypoxico, Inc., New York, NY, USA) and were blinded to their environment. Familiarization of the GXT was completed by all participants to aid in conformity between sessions. Cranking cadence was standardized at 50 revolutions per minute (RPM) for all protocols, and testing sessions were preceded by a five-minute warm-up at 50W. The testing environment was monitored via the metabolic gas analyzer's environmental sensor with average values of temperature (24.5 ± 1.87 ˚C), relative humidity (37.7 ± 6.6 %), and barometric pressure (756.8 ± 4.7).

GXTs were performed by all participants over two visits separated by at least 48 hours in both N and H. The GXT was performed on a cycle ergometer (891E, Monark Upper Body Ergometer, Vansbro, Sweden) to determine peak power output (PPO) in watts (W) and peak oxygen consumption ($\dot{V}O_2$peak) in liters per minute ($l \cdot min^{-1}$) for both environmental conditions. The GXT protocol consisted of an initial work-rate of 50W for three minutes with 20W increases every two minutes.

Four days of TTE trials, randomized by intensity and environmental conditions, were performed by all participants. Intensities were set at 110% and 120% of PPO achieved during the GXT and performed at least 48 hours apart and in both conditions. Oxygen saturation was measured to confirm environment acclimation following a three-minute warm-up at 50 watts and immediately before the trial. During the GXT and TTE trials, participants viewed their RPM to maintain the cadence but were blinded to the environment and their performance. TTE was determined to the second of volitional fatigue or failure to maintain a 50 RPM cranking cadence for more than five seconds.

Accumulated Oxygen Deficit ($L \cdot min^{-1}$) was calculated as the difference between predicted O2 consumption and measured O2 consumption during the TTE trials. Predicted O2 consumption was extrapolated from regression equations calculated using O2 demands at differing power outputs during the GXT tests in both normobaric normoxia and normobaric hypoxia (20). Anaerobic energy system contribution (%AN) was calculated as [1-(actual O_2 consumed / predicted O_2)] × 100. Both AOD and %AN were calculated in three conditions, normoxia using the normoxia GXT regression, hypoxia using the hypoxia GXT regression, and hypoxia using the normoxia GXT regression equation.

Statistical Analysis

Two separate Bayesian two-way repeated measures ANOVA were conducted to evaluate AOD and %AN using. The effect of intensity and regression equation formula (Intensity x Formula) on accumulated oxygen deficit (AOD) and percent anaerobic energy system contribution (%AN) were examined with the null hypothesis indicating no difference across intensities or between regression formulas, and the alternative hypothesis indicating a difference between intensities and formula. Bayes factors were evaluated using the criteria recommended by Kass & Raftery (16), including weak = 3 or below moderate = 4-10, and strong = 11-30+ . Bayesian inferences were interpreted according to recently developed guidance from (28). For comparison, frequentist/classical two-way repeated measures ANOVA was also used to evaluate AOD and %AN. Normal distribution of all dependent variables was confirmed using Shapiro-Wilk test. Mauchly's test of sphericity indicated that the assumption of sphericity was not violated for regression formulas $\chi 2$ (2) = 2.281, $p < .320$. A power analysis conducted with G*POWER 3.1 (Universitat Kiel, Germany) determined that 15 participants were needed in the present study for a power of .80, with an effect size of .8 and an $\alpha = 0.05$. An open-source statistical software (JASP Team, 2019) was used for all analysis with an alpha level of $\alpha = 0.05$ used for all frequentist analyses.

RESULTS

The Intensity + Formula interaction for AOD had a Bayes Factor of 4.705, meaning that the alternative hypothesis is more likely than the null hypothesis. Post hoc testing revealed an alternative hypothesis with a posterior odds of 191051.430 for intensity, indicating support for increases between 110% and 120% PPO. Post hoc testing for regression formulas revealed posterior odds of 11.698, indicating that the alternative hypothesis is about twelve times more likely. This states that there is a difference between AOD for hypoxia (1.217 ± .525 L·min^{-1}, CI; 0.982-1.541) and hypoxia using the normoxia equation (1.448 ± .551 L·min^{-1}, CI; .901-1.533).

The Intensity + Formula interaction for %AN had a Bayes Factor of 4.106, meaning that the alternative hypothesis is more likely than the null hypothesis. Post hoc testing revealed an alternative hypothesis with a posterior odds of 6.277e + 7 for intensity, indicating support for differences between 110% and 120% of peak power output. Post hoc testing for regression formulas revealed posterior odds of 5.233 or about five times the likelihood for the alternative hypothesis stating that there is a difference between %AN measured in hypoxia (20.781 ± 8.198%, CI; 15.341-26.221) and hypoxia using the normoxia equation (23.969 ± 7.123%, CI; 19.242-28.696).

Frequentist repeated measures ANOVA for AOD revealed a significant main effects for Intensity [$F(1,11) = 19.12$, $p = .001$] with 120%PPO being greater than 110%PPO (mean = .422; SE = .096; $p = .001$), but no main effect for Formula [$F(2,22) = 2.087$, $p = .148$] and no significant interaction [$F(2,22) = .087$, $p = .917$]. Repeated measures ANOVA for %AN revealed significant main effects for Intensity [$F(1,11) = 38.429$, $p = .001$] but not Formula [$F(2,22) = 2.067$, $p = .150$], or interaction [$F(2,22) = .582$, $p = .567$]. Post hoc analysis for Intensity revealed that 110% PPO

produced significantly lower values than 120% PPO (mean difference = -8.967 ± 1.446%; p = .001).

Multiple paired Cumming plots were generated for AOD and %AN using Estimationstats web application (15), allowing visual comparison of differences between formulas. See (Figure 1, Figure 2).

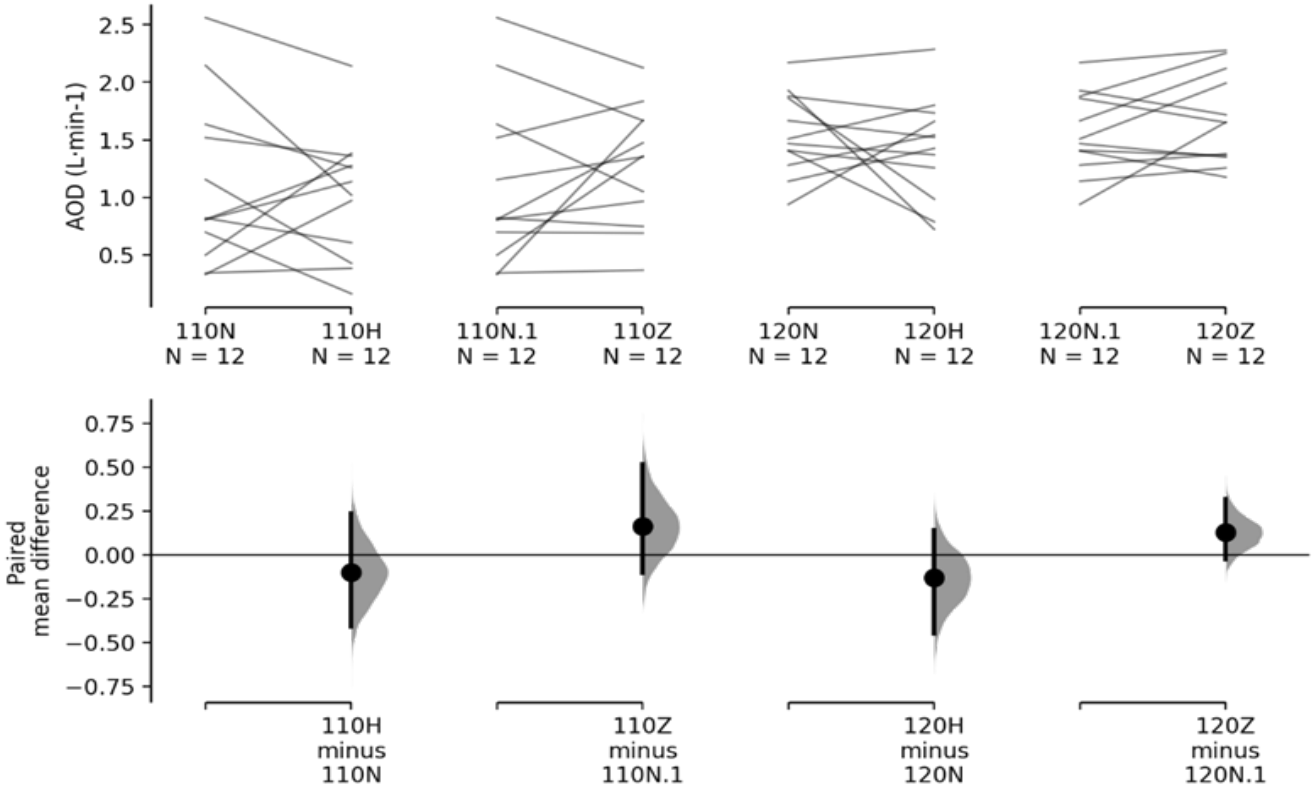

Figure 1. The paired mean difference for 4 comparisons is shown in the above Cumming estimation plot. The raw data is plotted on the upper axes; each paired set of observations is connected by a line. On the lower axes, each paired mean difference is plotted as a bootstrap sampling distribution. Mean differences are depicted as dots; 95% confidence intervals are indicated by the ends of the vertical error bars. AOD is represented in three conditions: N, H, and H using the N regression equation (Z), and two intensities 110% and 120%.

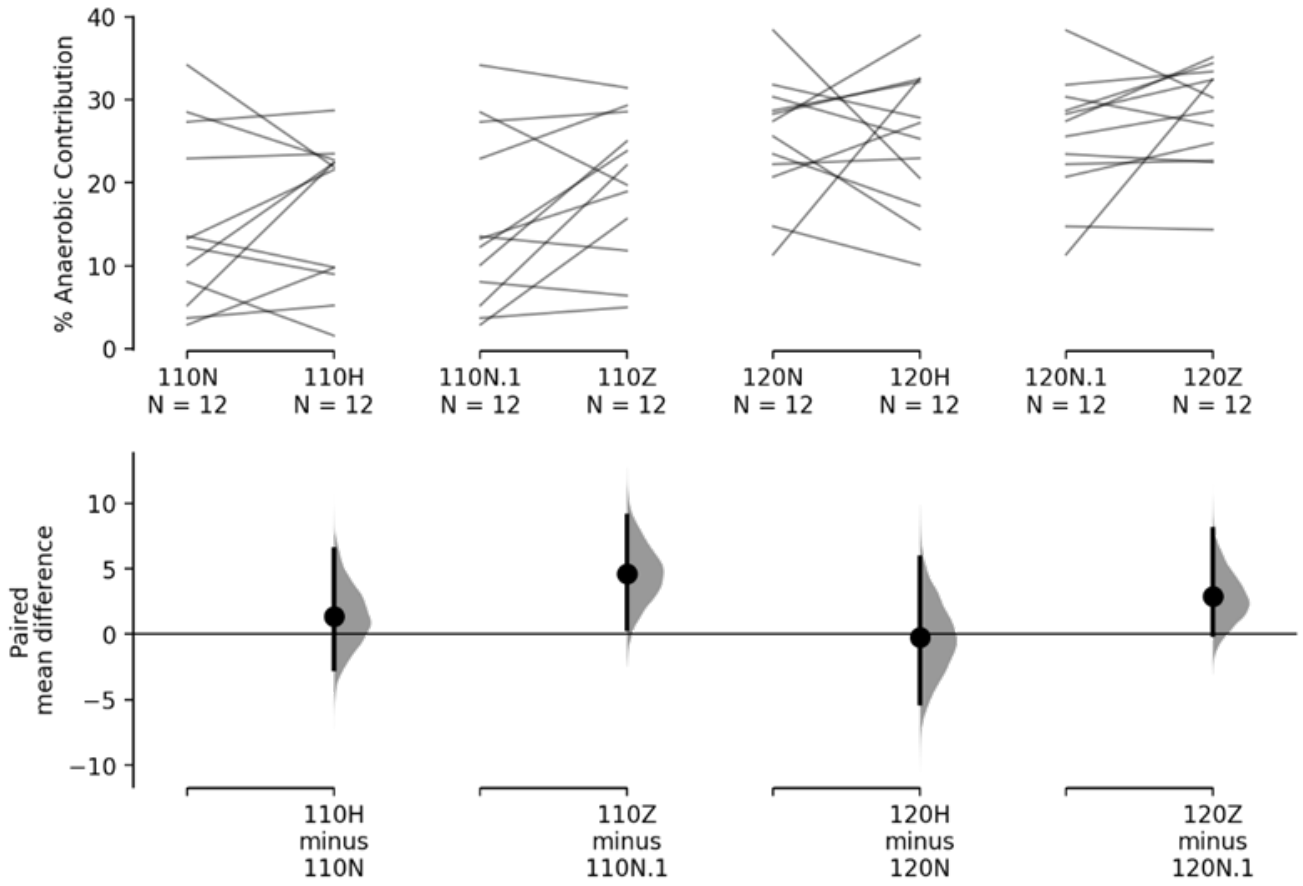

Figure 2. The paired mean difference for 4 comparisons is shown in the above Cumming estimation plot. The raw data is plotted on the upper axes; each paired set of observations is connected by a line. On the lower axes, each paired mean difference is plotted as a bootstrap sampling distribution. Mean differences are depicted as dots; 95% confidence intervals are indicated by the ends of the vertical error bars. Percent Anaerobic Contribution is represented in three conditions: N, H, and H using the N regression equation (Z), and two intensities 110% and 120%.

DISCUSSION

This investigation demonstrated different AOD and %AN values depending on if the predicted oxygen consumption was extrapolated from a normoxic or hypoxic environment. The results provide strong evidence that there were differences between intensities and moderate evidence that there was a difference depending on which regression equation was used to calculate both AOD and %AN. Using the regression equation calculated in normoxia to estimate AOD and %AN in hypoxia resulted in higher values for AOD and %AN compared to using the hypoxia regression equation during exercise in hypoxia. The Bayesian analysis provided moderate evidence that there are differences between regression formulas for both AOD and %AN (Table 1).

Table 1. Accumulated Oxygen Deficit and Anaerobic Percent Values per Regression Formula

	AOD (L·min-1)	% AN
N		
110	1.11 ± .69	15.2 ± 10.1
120	1.01 ± .53	16.5 ± 8.5
H		
110	1.28 ± .50	19.8 ± 8.3
120	1.55 ± .34	25.2 ± 7.1
HN		
110	1.42 ± .43	25.0 ± 7.9
120	1.68 ± .38	28.1 ± 5.9

AOD: Accumulated oxygen deficit (L·min-1); %AN: Anaerobic Energy System Contribution; N: Normoxia; H: Hypoxia; HN : Hypoxia using the Normoxia Regression Equation.

If these data were evaluated from on the frequentist perspective, a non-significant *p*-value for differences between AOD and %AN and the regression formulas would have been the primary finding, thereby limiting the identification of potentially meaningful methodological considerations.

This is the first investigation to look at the effects of hypoxia on AOD in the upper-body. These data provide conflicting results from what is observed in the lower-body where AOD is not affected by acute moderate hypoxia during treadmill exercise while using both normoxia and hypoxia regression equations (10, 11). The conflicting results may be driven by the propensity of the upper body to rely on anaerobic resources earlier during exercise compared to the lower-body (25). The upper body musculature has a greater amount of type II fibers when compared with the lower body (25), and the prime movers during upper-body ergometry are the arms and shoulders (26). Furthermore, significant correlations exist between arm lean body mass and anaerobic alactic energy (18). During high intensity upper-body exercise, the anaerobic lactic system reportedly contributes 15% more than during identical lower-body exercise (18). These differences and their influence on AOD during hypoxia provide an opportunity for further investigation.

Another methodological consideration that requires elucidation is whether the relationship between oxygen demand and work rate is linear or curvilinear at intensities greater than 90% during upper-body ergometry. Past investigation into this subject have determined that this relationship is upwardly curvilinear during running (13). Equivocal results are seen during lower body cycling where some report an exponential relationship between oxygen demand and exercise intensity (23), while more recently others report a linear relationship (13). More direct investigations into oxygen demand at supramaximal intensities during upper-body ergometry are needed to make determinations on this relationship.

The population used in this study consisted of recreationally trained men who were likely unaccustomed to upper-body specific training, particularly the use of an upper-body ergometer. Although these participants were familiarized with the equipment and protocol, future studies may want to utilize athletes from upper-body dominant sports (e.g., rowing, grappling, and

climbing). These athletes may be more economical with their movement leading to differences in energy system contribution (7). Finally, four participants were removed from this analysis due to AOD and/or AN% values falling below zero. It is possible that their training status, combined with the supramaximal nature of the exercise, contributed to this as indicated by others during lower body cycling (22).

Conclusion: Using predicted oxygen consumption extrapolated from a normoxic environment during exercise in hypoxia may lead to the overestimation of AOD and %AN. Accumulated O_2 uptake is significantly decreased in moderate hypoxia (11, 3), possibly influencing the differences between AOD and %AN calculations. Therefore, it may be inappropriate to use regression equations extrapolated from GXT in normoxia to evaluate AOD in hypoxia. However, further evaluation is needed to elucidate if this is true across populations and intensities. In some instances, data may be more appropriately described using a Bayesian analysis instead of a traditional frequentist methodology as demonstrated in this investigation. This investigation provides an example of when Bayesian statistics may be applicable in the field of exercise science.

ACKNOWLEDGEMENTS

No funding was provided to support the preparation of this manuscript. There are no relevant conflicts of interests for all authors included in this manuscript.

REFERENCES

1. Artioli G, Bertuzzi R, Roschel H, Mendes S, Lancha Jr. A, & Franchini E. Determining the contribution of the energy systems during exercise. JOVE J. Vis. Exp 61: 3413, 2012.

2. Austin P, Brunner L, & Hux J. Bayeswatch: an overview of bayesian statistics. J. Eval. Clin 8(2): 277-286, 2002.

3. Balsom P, Gaitanos G, Ekblom B, & Sjödin B. Reduced oxygen availability during high intensity intermittent exercise impairs performance. Acta Physiol. Scand 152(3): 279-285, 1994.

4. Bayes T. An essay towards solving a problem in the doctrine of chances. Biometrika 45(3-4): 296-315, 1958.

5. Calbet J, De Paz J, Garatachea N, Cabeza de Vaca S, & Chavarren J. Anaerobic energy provision does not limit wingate exercise performance in endurance-trained cyclists. J Appl Physiol 94(2): 668–676, 2003.

6. Calbet J, Holmberg H, Rosdahl H, van Hall G, Jensen-Urstad M, & Saltin B. Why do arms extract less oxygen than legs during exercise? Am J Physiol Regul Integr Comp Physiol 289(5): R1448–R1458, 2005.

7. Caputo F, Mello M, & Denadai B. Oxygen uptake kinetics and time to exhaustion in cycling and running: a comparison between trained and untrained subjects. Arch Physiol Biochem *111*(5): 461–466, 2003.

8. Earp B, & Trafimow D. Replication, falsification, and the crisis of confidence in social psychology. Front. Psychol 6: 621, 2015.

9. Eddy S. What is bayesian statistics? Nat. Biotechnol 22(9): 1177-1178, 2004.

10. Feriche B, Delgad M, Chirosa I, Miranda M, Ndez J, & Lvarez, J. The effect of acute moderate hypoxia on accumulated oxygen deficit during intermittent exercise in nonacclimatized men. J Strength Cond Res 21(2): 413-8, 2007.

11. Friedmann B, Frese F, Menold E, & Bärtsch P. Effects of acute moderate hypoxia on anaerobic capacity in endurance-trained runners. Eur J Appl Physiol 101(1): 67–73, 2007.

12. Gutierrez G. Cellular energy metabolism during hypoxia. Crit. Care Med. May; 19(5): 619-626, 1991.

13. Hill D, & Vingren J. Maximal accumulated oxygen deficit in running and cycling. Appl Physiol Nutr Metab 36(6): 831-838, 2001.

14. Janssen I, Heymsfield S, Wang Z, & Ross R. Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. J Appl Physiol 89(1): 81–88, 2000.

15. Joses H, Tayfun T, Sameer A, Hyungwon C, Adam C. Moving beyond P values: Everyday data analysis with estimation plots. Nat. Methods: 1548-7105, 2019.

16. Kass R, & Raftery A. Bayes factors. J. Am. Stat. Assoc 90(430): 773-795, 1995.

17. Li Y, Niessen M, Chen X, & Hartmann U. Method-induced differences of energy contributions in women's kayaking. Int J Sports Physiol Perform 13(1): 9–13, 2018.

18. Lovell D, Kerr A, Wiegand A, Solomon C, Harvey L, & McLellan C. The contribution of energy systems during the upper body wingate anaerobic test. Appl Physiol Nutr Metab 38(2): 216-219, 2013.

19. Martin T, Zeballos R, & Weisman I. Gas exchange during maximal upper extremity exercise. Chest 99(2): 420–425, 1991.

20. Medbo J, Mohn A, Tabata I, Bahr R, Vaage O, & Sejersted O. Anaerobic capacity determined by maximal accumulated O2 deficit. J Appl Physiol 64(1): 50-60, 1988.

21. Navalta J, Stone W, Lyons T. Ethical issues relating to scientific discovery in exercise science. Int J Exerc Sci 12(1): 1-8, 2019.

22. Özyener F, Rossiter H, Ward S, & Whipp B. Negative accumulated oxygen deficit during heavy and very heavy intensity cycle ergometry in humans. Eur. J. Appl. Physiol 90(1-2): 185-190, 2003.

23. Pugh L. The relation of oxygen intake and speed in competition cycling and comparative observations on the bicycle ergometer. J Physiol 241(3): 795-808, 1974.

24. Saltin B, & Calbet J. Point: In health and in a normoxic environment, V̇ O 2 max is limited primarily by cardiac output and locomotor muscle blood flow. J Appl Physiol 100(2): 744–748, 2006.

25. Sanchis-Moysi J, Idoate F, Olmedillas H, Guadalupe-Grau A, Alayon S, Carreras A & Calbet, J. The upper extremity of the professional tennis player: muscle volumes, fiber-type distribution and muscle strength. Scand J Med Sci Sports 20(3): 524-534, 2010.

26. Smith P, Chapman M, Hazlehurst K, & Goss-Sampson M. The influence of crank configuration on muscle activity and torque production during arm crank ergometry. J Electromyogr Kinesiol 18(4): 598-605, 2008.

27. Vandewalle H, Péerès G, & Monod H. Standard anaerobic exercise tests. Sports Med 4(4): 268–289, 1987.

28. van Doorn J, van den Bergh D, Bohm U, Dablander F, Derks K, Draws T & Wagenmakers E. The JASP guidelines for conducting and reporting a Bayesian analysis. PsyArXiv, 2019.