An Index of Nonlinear HRV as a Proxy of the Aerobic Threshold in Elite Triathletes

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Abstract: Background: A non-linear index of heart rate (HR) variability (HRV) known as alpha1 of Detrended Fluctuation Analysis (DFA a1) has been shown to change with increasing exercise intensity, crossing a value of 0.75 at the aerobic threshold (AT) in recreational runners defining a HRV threshold (HRVT). Since large volumes of low intensity training below the AT is recommended for many elite endurance athletes, confirmation of this relationship in this specific group would be advantageous for the purposes of training intensity distribution monitoring. **Methods**: Nine elite triathletes (7m, 2f) attended a training camp for diagnostic purposes. Lactate testing was done with an incremental cycling ramp test to exhaustion for the determination of the first lactate threshold (LT1). Concurrent measurements of cardiac beta-to-beat intervals were performed to determine the HRVT. **Results**: Mean LT1 HR of all 9 participants was 155.8 bpm (±7.0) vs HRVT HR of 153.7 bpm (±10.1) (p = 0.52). Mean LT1 cycling power was 252.3 W (±48.1) vs HRVT power of 247.0 W (±53.6) (p = 0.17). Bland Altman analysis showed mean differences of -1.7 bpm and -5.3 W with limits of agreement (LOA) 13.3 to -16.7 bpm and 15.1 to -25.6 W for HR and cycling power respectively. **Conclusion**: The DFA a1 based HRVT closely agreed with the LT1 in a group of elite triathletes. Since large volumes of low intensity exercise are recommended for successful endurance performance, fractal correlation properties of HRV shows promise as a low cost, non-invasive option to that of lactate testing for identification of AT related training boundaries.

Keywords: heart rate variability; lactate threshold; DFA a1; training intensity distribution; polarized training

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Introduction

Knowledge of exercise intensity boundaries is important for guidance in athletic training and diagnostics. Various models of training intensity distribution have been proposed (polarized, threshold and pyramidal) but all share one aspect in common, that the majority of training is to be done in a low intensity zone range.[1] In the typical 3 zone model, the upper low intensity limit is felt to be regarded as the aerobic threshold (AT), represented by either the first lactate (LT1) or ventilatory threshold (VT1).[2] Although an incremental ramp test with gas exchange monitoring can be done to determine the VT1 many athletic training centers utilize progressive constant cycling power intervals with lactate testing to determine the AT using the LT1. Although on first glance this would seem a simple endeavor, several issues make the concept more complex. Importantly, there does not appear to be universal agreement on what concept defines the LT1.[3] Some investigators have used a fixed value of 2 mmol/L while others use a fixed value above baseline of 0.5 or 1 mmol/L. Other options include logarithmic plotting of lactate against

cycling power or heart rate (HR).[4] This could lead to variable results of a training boundary obtained by one measure or another.

Besides the confusion surrounding test definition, lactate testing is invasive, relatively costly and generally requires additional personnel to perform the test while the athlete is exercising. Therefore, alternate means of determining the AT have been evaluated over the years including modalities related to various heart rate variability (HRV) indexes.[5,6,7] However, despite initial appeal, general use for the purpose of low intensity training guidance has not occurred for various reasons.[8,9] Recently, HRV monitoring during the exercise session has received a resurgence in attention as a method of measuring the AT.[8] In a group of recreational runners, the AT was found to closely match that of a HRV threshold (HRVT) derived from a non-linear HRV index of fractal correlation properties determined by Detrended Fluctuation Analysis termed alpha1 (DFA a1).[9] As exercise intensity rises the DFA a1 declines from values near 1, which represent well correlated fractal behavior of the cardiac beat to beat pattern, passing a value of 0.75 at the AT, then reaching uncorrelated, random behavior at intensities past the AT. Potential advantages of this approach to estimating the AT include cost, easy availability of HR monitoring devices and its non-invasive nature. However, since initial validation of this approach was performed by comparing the HRVT to the AT as represented by the VT1 derived from gas exchange during treadmill running, potential agreement with an alternative method such as the LT1 evaluated during an incremental cycling stage ramp is unknown and should be addressed.

In order to show that the HRVT is a robust, reliable surrogate for the AT, evaluation in certain population subsets would also be helpful. For instance, this marker may be applicable in a group possessing average fitness abilities but loses validity with elite endurance athletes. Since the elite endurance athlete typically performs large amounts of training in the low intensity zone, validation of the HRVT principle in these individuals would be especially beneficial. Recent observational analysis has shown that the volume of low intensity training done by competitive long-distance runners[10] as well as the high training volume done by recreational half marathon runners[11] is related to their future performance. Therefore, given the need for accurate delineation of the low intensity boundary in elite athletes for training and testing purposes, the intent of this report is to explore the relationship of the DFA a1 based HRVT with that of the LT1, obtained at an athletic team assessment camp.

Methods

Participants

Nine elite triathletes (7 male, 2 female) of various countries of origin were recruited from a triathlon assessment training camp. Current competition category ranged between national and international levels. Average age was 24 years (±4), body weight 69 kg (±9), height 175 cm (±10), weekly training volume 20 hours (±3) and $\dot{V}O_{2MAX}$ 67 ml/kg/min (±7). The study was conducted during routine diagnostic procedures in a training camp and no additional equipment was used for data collection and no further procedures were performed. Physiologic testing was done at the beginning of the camp. Athletes had not performed any recent high intensity exercise and were deemed well rested by the coaching staff. All participants were informed about the study procedures and objectives. They provided written informed consent according to the ethical guidelines in accordance with the institutional review board and the guidelines of the Helsinki World Medical Association Declaration.

Exercise testing protocol

An incremental cycling stage test until voluntary exhaustion was performed with a Cyclus2 ergometer (RBM elektronik-automation GmbH, Leipzig, Germany) with a freely chosen cadence between 80 and 90 rpm. The test protocol for men consisted of a starting

cycling power of 90 W (watts), with an incremental rise of 30 W every 3 minutes. For women, the starting cycling power was 75 W with an incremental rise every 3 minutes of 25 W. Machine calibration was done in accordance with manufacturer recommendations. Ambient temperature (14 to 17 °C), altitude of 45 meters and meal timing were similar for all participants. Both caffeine and alcohol consumption were avoided for 24 hours pre-testing. There was no tobacco usage in any participant.

Lactate testing

All lactate samples were measured with the Lactate Pro2 (Arkray KDK, Japan) between 30 and 40 seconds before the end of each ramp stage, except the last sample which was taken directly after completion of the test (10-15 seconds after cessation). Determination of the first lactate threshold was done using automated testing software[4] using logarithmic plotting of lactate vs either cycling power or HR.

RR measurements and calculation of DFA a1 derived HRVT

A Polar H10 (Polar Electro Oy, Kempele, Finland) HR monitoring device (HRM) was used to detect RR intervals in 7 individuals, a Pioneer HRM (Pioneer Electronics, USA) Inc was used in one individual and a Garmin HRM (Garmin Inc, Olathe, KS, USA) in another due to individual preferences. All RR data was recorded with a Garmin 530 cycling computer (Garmin Inc, Olathe, KS, USA) then imported into Kubios HRV Software Version 3.4.3 (Biosignal Analysis and Medical Imaging Group, Department of Physics, University of Kuopio, Kuopio, Finland). Kubios preprocessing settings were set to the default values including the RR detrending method which was kept at "Smoothn priors" (Lambda = 500).[12] DFA a1 window width was set to $4 \leq n \leq 16$ beats. The RR series was then corrected by the Kubios "automatic method" and relevant HRV parameters exported as text files for further analysis. Artifact levels measured by Kubios HRV were below 5%. This limit was previously shown to have minimal effect on the HRVT.[13] DFA a1 was calculated from the RR data series using 2-minute time windows with repeat computation every 5 seconds throughout the test (time-varying method - window width = 2 minutes, grid interval = 5 seconds). Two-minute time windowing was chosen based on the calculations by Chen et al.[14] For the detection of HRVT, a DFA a1 value of 0.75 was selected based on previous study in recreational athletes.[9] This value is also the midpoint between a fractal, well correlated behavior of the HR time series of 1.0 (seen with very light exercise) and an uncorrelated value of 0.5 which represents white noise, random behavior (seen with high intensity exercise).[8] Plotting of DFA a1 vs HR was then performed, generally showing a stable area above 1.0 at low work rates, a rapid, near linear drop reaching below 0.5 at higher intensity, then flattening without major change. The procedure used to indicate at what level of cycling intensity as HR the DFA a1 would cross a value of 0.75 has been detailed previously.[9] Cycling power at DFA a1 = 0.75 (cycling power at HRVT) was calculated from the 180 second average cycling power preceding the time DFA a1 reached 0.75.

Statistics

Statistical analysis was performed for: HR and cycling power at LT1 derived from lactate testing, HR and cycling power at HRVT derived from DFA a1. Standard statistical methods were used for the calculation of means and standard deviations (SD). Normal distribution of data was checked by Shapiro-Wilk's test. The agreement with LT1 parameters was assessed using linear regression, Pearson's r correlation coefficient, intraclass correlation coefficient (ICC), Lin's concordance correlation coefficient (CCC), standard error of estimate (SEE), and Bland Altman plots with limits of agreement.[15] All Bland Altman plots were assessed for proportional bias. The size of Pearson's r correlations evaluated as follows: $0.3 \leq r < 0.5$ low; $0.6 \leq r < 0.8$ moderate and $r \geq 0.8$ high,[16] The paired t-test was used for comparison of LT1 vs HRVT for both cycling power and HR. For all tests,

the statistical significance was accepted as p ≤ 0.05. Analysis was performed using Microsoft Excel 365 with Real Statistics Resource Pack (Release 6.8) and Analyse-it software (Version 5.66).

Results

Lactate threshold and HRVT comparison

Mean LT1 HR for all 9 participants was 155.8 bpm (±7.0) vs HRVT HR of 153.7 bpm (±10.1) (p = 0.52). Mean LT1 cycling power was 252.3 W (±48.1) vs HRVT cycling power of 247.0 W (±53.6) (p = 0.17). Regression plots are shown in Figure 1 for HR and cycling power comparisons. Bland Altman analysis for comparisons of HR and cycling power are shown in Figure 2. Mean differences were -1.7 bpm and -5.3 W with LOA of 13.3 and -16.7 bpm for HR and of 15.1 and -25.6 W for cycling power. Results were normally distributed. ICC (1,1) between LT1 HR and HRVT HR was 0.69 (95% confidence limits 0.14 to 0.92) and was 0.98 ((95% confidence limits 0.91 to .99) for cycling power. CCC between LT1 HR and HRVT HR was 0.66 (95% confidence limits 0.11 to 0.90) and was 0.98 ((95% confidence limits 0.92 to .99) for cycling power.
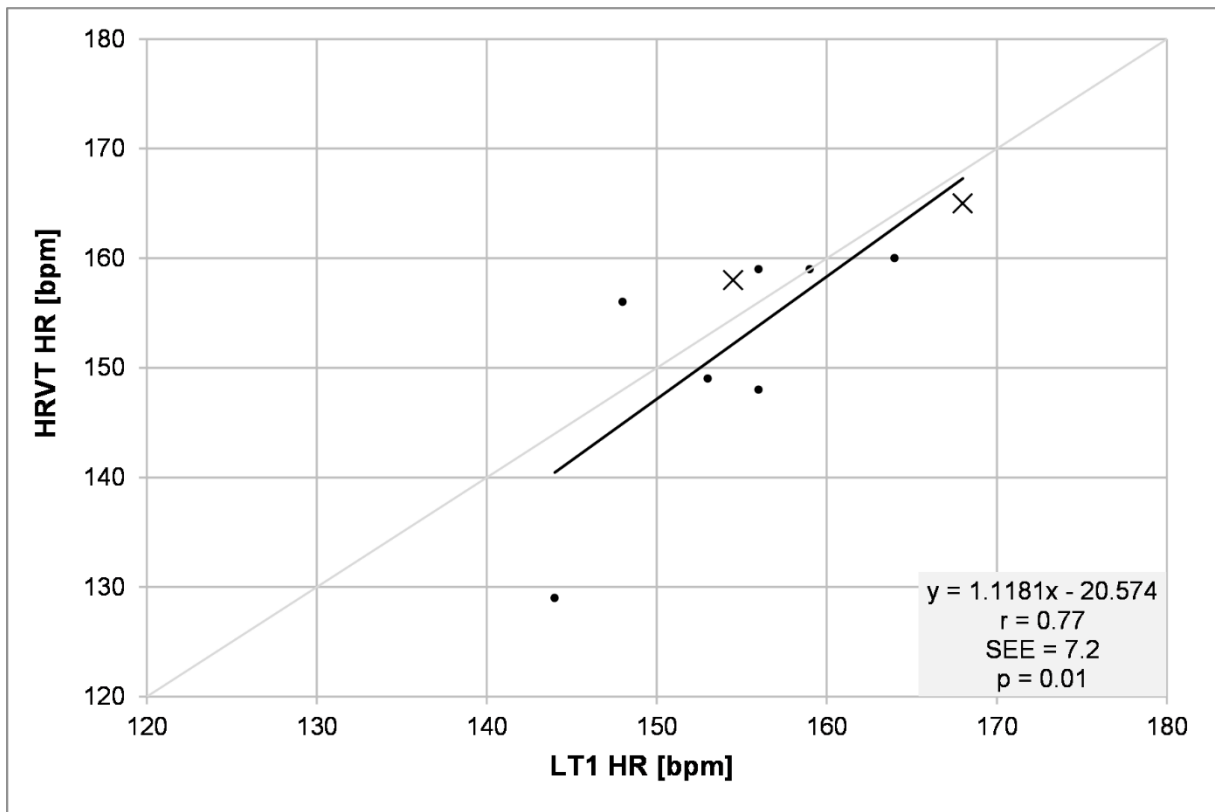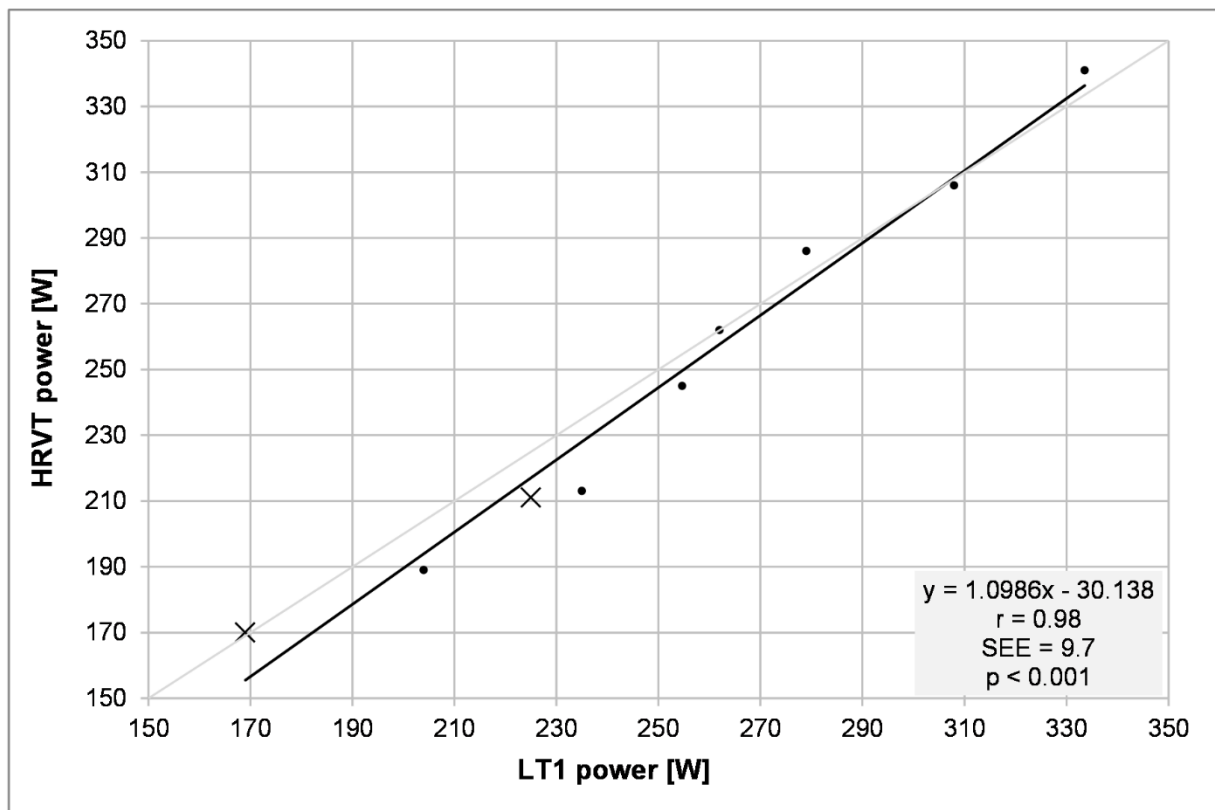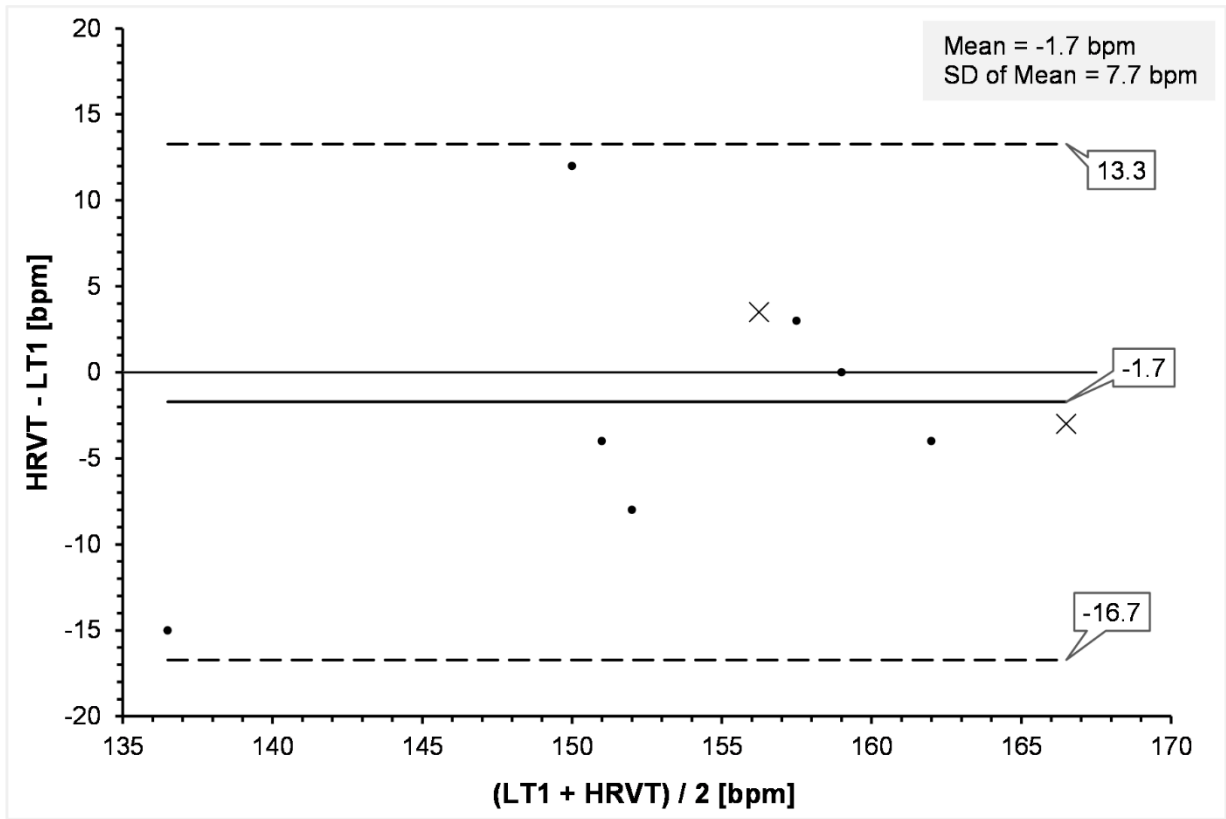
Figure 1. Regression plots for all participant data. A: Values of LT1 vs HRVT for HR in bpm; B: Values of LT1 vs HRVT for cycling power in W. Bisection lines in light gray. SEE: standard error of estimate; r: Pearson's r. Points symbolized by X represent female participants.
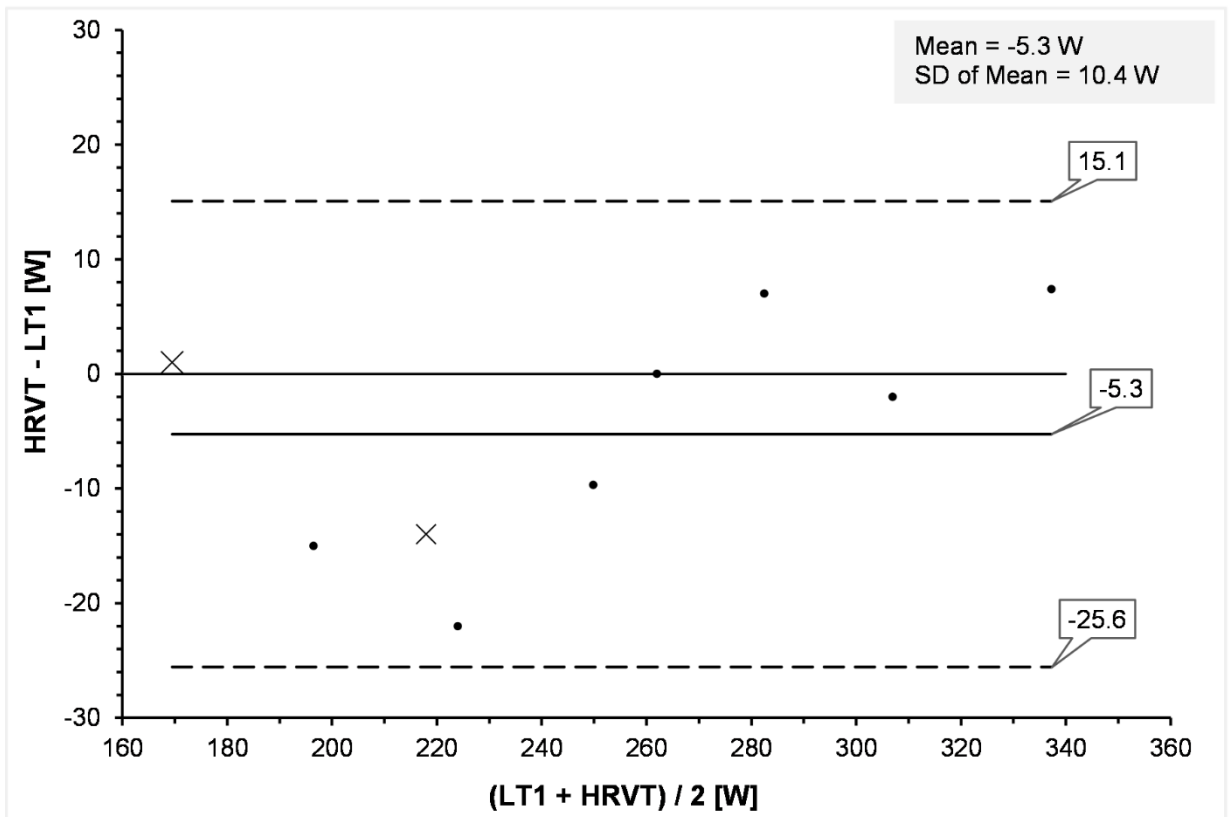
Figure 2. Bland Altman Plot of LT1 vs HRVT for all participants. A: Values of LT1 vs HRVT for HR in bpm; B: Values of LT1 vs HRVT for cycling power in W. Center line in each plot represents the mean difference between each paired value,

the top and bottom lines are 1.96 standard deviations from the mean difference. Points symbolized by X represent female participants.

Discussion

This study aimed to confirm the association of the DFA a1 based HRVT with that of the LT1, an established marker of the AT[3] in a demographic consisting of elite male and female triathletes using a cycling stage ramp test. The results show clear relationships between the LT1 calculated by the log-log method and HRVT for both HR and cycling power. This was supported by linear regression, with Pearson's r of 0.77 and 0.98 for HR and cycling power respectively. There was no difference between mean values of LT1 and HRVT by paired t testing. Bland Altman analysis showed minimal mean differences that were felt to be acceptable for the purpose of exercise and training prescription. Although a previous report found a good concordance between the HRVT and VT1 assessed by gas exchange, that study was done in recreational runners using a treadmill test with research grade ECG monitoring.[9] Given the potential value of an alternate non-invasive marker for the AT, further investigation into varied user populations, alternate recording devices and exercise modalities are warranted before widespread usage. The participants evaluated here did represent an elite class of triathlete including several national team members. Weekly training volumes and $\dot{V}O_{2MAX}$ measurements were in agreement with this and well above typical recreational levels.[17] Since elite endurance sport participants are frequently the subjects of training intensity distribution research, positive confirmation of the HRVT equivalence to the LT1 could be helpful with non-invasive zone boundary identification and enforcement.

The underlying mechanism for DFA a1 behavior during exercise appears to be related to the antagonistic behavior of the sympathetic and parasympathetic branches of the autonomic nervous system on the sinoatrial node[18] as well as other potential factors.[19] As work intensity rises there is a withdrawal of the parasympathetic component and enhancement of the sympathetic component with the net result being that of a decline of DFA a1 as well as other HRV parameters. However, as opposed to conventional HRV indexes that rely of a nadir to determine the AT, DFA a1 appears to pass a specific dimensionless value of 0.75. Therefore, asymptotic curve interpretation and calibration to metabolic parameters such as a lactate or ventilatory threshold is unnecessary. This has obvious advantages in both retrospective HRV analysis and real-time monitoring of exercise demands. The reasoning behind choosing a DFA a1 value of .075 as a focus for VT1 transition was based on several factors.[8,9] Inspection of prior DFA a1 incremental ramp studies appeared to indicate that the aerobic threshold was situated near this area[8]. In addition, this value represents the midpoint between correlated fractal patterns seen with low intensity loads (1.0) and uncorrelated random patterns seen at intensity domains past the aerobic threshold (0.5)[8]. Additional studies in other demographic groups also appear to support the 0.75 HRVT association with the gas exchange derived VT1.[20,21] With respect to DFA a1 activity in elite athletes, it is certainly plausible that this group could exhibit distinct differences from inactive or recreational cohorts based on factors such as cardiac remodeling[22,23] and/or altered vagal tone.[24] The concordance between the HRVT to that of the AT in both elite and non-elite populations is reassuring to the hypothesis that the DFA a1 threshold concept is widely applicable. The HRVT can also be considered as a separate concept within the theoretical framework[8] of different organismic regulation patterns.

Limitations and future directions

Although the derivation of the HRVT is relatively straightforward, both the definition and calculation of the LT1 are subject to numerous views (i.e.: rise of either 0.5 or 1.0 mmol/L, a fixed 2.0 mmol/L threshold etc.).[3,25, 26] As one of several established methods, even logarithmic transformation has interpretive options that could lead to slightly different results for cycling power or HR[25]. Therefore, comparison of the HRVT to the various LT1 concepts could be done in future investigations of larger sample size. Scant data on

DFA a1 behavior is available in female athletes and further study is needed in that population. Fortunately, in this study two participants were female and had results similar to the entire group. A possible limitation of this study is the heterogeneity of HRM devices. Although they are all share similar form factors, it is certainly possible that unique device bias could be present. A strength of this study is that artefact presence in the included data was below 5% which has been shown to have minimal effect on the HRVT.[13] In the future, devices with a chest belt form factor with single lead ECG functionality would be advantageous for both evaluation of artefact type, manual correction of artefact as well as arrythmia identification.[28] It is also feasible that the DFA a1 measurement be integrated into smartwatch or smartphone monitoring devices for real-time intensity and zone 1 enforcement. Several new implementations of DFA a1 monitoring via adapted open source python packages have recently emerged making this idea closer to attainment.[29] Although group sample size was somewhat less than optimal at 9 individuals, this is not unexpected given the exclusive nature of high level athletic training populations. Finally, while participant training status was felt not to be overreached, further investigations into using changes in DFA a1 behavior as a measure of endurance exercise fatigue appear promising based on recent data.[30]

Practical Applications

The intensity region where DFA a1 declines through a value of 0.75 (HRVT) can be used to delineate the AT in terms of HR and cycling power during an incremental cycling ramp. The agreement between the HRVT and LT1 was similar to previous VT1 and LT1 comparisons.[24] This information can be used for both measurement of current fitness status as well as for regulation and monitoring of low intensity training distribution. Further extension of the HRVT hypothesis was confirmed in an elite group of endurance athletes.

Conclusion

A heart rate variability threshold based on DFA a1, a non-linear measure of fractal correlation properties, was closely associated with the first lactate threshold in a population of elite male and female triathletes during a cycling ramp stage test. Given the importance of identifying the upper boundary for low intensity zone training for endurance type sports, the DFA a1 related threshold demonstrates an exciting low cost, non-invasive option to that of lactate testing. In addition, due to its dimensionless nature and agreement with the aerobic threshold, future use on a retrospective or real-time basis to enforce training intensity distribution guidelines is possible.

References

1. Stöggl TL, Sperlich B. Editorial: Training Intensity, Volume and Recovery Distribution Among Elite and Recreational Endurance Athletes. Front Physiol. 2019;10:592.

2. Mann T, Lamberts RP, Lambert MI. Methods of prescribing relative exercise intensity: physiological and practical considerations. Sports Med. 2013;43(7):613-625.

3. Faude O, Kindermann W, Meyer T. Lactate threshold concepts. Sports Med. 2009;39(6):469-490.

4. Newell J, Higgins D, Madden N, et al. Software for calculating blood lactate endurance markers. J Sports Sci. 2007;25(12):1403-1409.

5. Tulppo MP, Mäkikallio TH, Takala TE, Seppänen T, Huikuri HV. Quantitative beat-to-beat analysis of heart rate dynamics during exercise. Am J Physiol. 1996;271(1 Pt 2):H244-252.

6. Cottin F, Médigue C, Lopes P, Leprêtre PM, Heubert R, Billat V. Ventilatory thresholds assessment from heart rate variability during an incremental exhaustive running test. Int J Sports Med. 2007;28(4):287-294.

7. Karapetian GK, Engels HJ, Gretebeck RJ. Use of heart rate variability to estimate LT and VT. Int J Sports Med. 2008;29(8):652-657.

8. Gronwald T, Rogers B, Hoos O. Fractal Correlation Properties of Heart Rate Variability: A New Biomarker for Intensity Distribution in Endurance Exercise and Training Prescription? Front Physiol. 2020;11:1152.

9. Rogers B, Giles D, Draper N, Hoos O, Gronwald T. A New Detection Method Defining the Aerobic Threshold for Endurance Exercise and Training Prescription Based on Fractal Correlation Properties of Heart Rate Variability. Front Physiol. 2021;11:596567.

10. Casado A, Hanley B, Santos-Concejero J, Ruiz-Pérez LM. World-Class Long-Distance Running Performances Are Best Predicted by Volume of Easy Runs and Deliberate Practice of Short-Interval and Tempo Runs. J Strength Cond Res. 2019;online ahead of print.

11. Fokkema T, van Damme AADN, Fornerod MWJ, de Vos RJ, Bierma-Zeinstra SMA, van Middelkoop M. Training for a (half-)marathon: Training volume and longest endurance run related to performance and running injuries. Scand J Med Sci Sports. 2020;30(9):1692-1704.

12. Tarvainen MP, Niskanen JP, Lipponen JA, Ranta-Aho PO, Karjalainen PA. Kubios HRV--heart rate variability analysis software. Comput Methods Programs Biomed. 2014;113(1):210-220.

13. Rogers B, Giles D, Draper N, Mourot L, Gronwald T. Influence of Artefact Correction and Recording Device Type on the Practical Application of a Non-Linear Heart Rate Variability Biomarker for Aerobic Threshold Determination. Sensors. 2021;21(3):821.

14. Chen Z, Ivanov PC, Hu K, Stanley HE. Effect of nonstationarities on detrended fluctuation analysis. Phys Rev E. 2002;65(4 Pt 1):041107.

15. Bland JM, Altman DG. Measuring agreement in method comparison studies. Stat Methods Med Res. 1999;8(2):135-160.

16. Chan YH. Biostatistics 104: correlational analysis. Singapore Med J. 2003;44(12):614-619.

17. Stöggl TL, Sperlich B. The training intensity distribution among well-trained and elite endurance athletes. Front Physiol. 2015;6:295.

18. White DW, Raven PB. Autonomic neural control of heart rate during dynamic exercise: revisited. J Physiol. 2014;592(12):2491-2500.

19. Persson PB. Modulation of cardiovascular control mechanisms and their interaction. Physiol Rev. 1996;76(1):193-244.

20. Naranjo-Orellana, J.; Nieto-Jiménez, C.; Ruso-Álvarez, J. Non-linear heart rate dynamics during and after three controlled exercise intensities in healthy men. Physiol Int. 2021;107(4):501-512

21. Rogers B, Mourot L, Gronwald T. Aerobic Threshold Identification in a Cardiac Disease Population Based on Correlation Properties of Heart Rate Variability. J Clin Med. 2021;10(18):4075.

22. Brown B, Millar L, Somauroo J, et al. Left ventricular remodeling in elite and sub-elite road cyclists. Scand J Med Sci Sports. 2020;30(7):1132-1139.

23. La Gerche A, Heidbüchel H, Burns AT, et al. Disproportionate exercise load and remodeling of the athlete's right ventricle. Med Sci Sports Exerc. 2011;43(6):974-981.

24. Coote JH, White MJ. CrossTalk proposal: bradycardia in the trained athlete is attributable to high vagal tone. J Physiol. 2015;593(8):1745-1747.

25. Jamnick NA, Botella J, Pyne DB, Bishop DJ. Manipulating graded exercise test variables affects the validity of the lactate threshold and VO2peak. PloS One. 2018;13(7):e0199794

26. Pallarés JG, Morán-Navarro R, Ortega JF, Fernández-Elías VE, Mora-Rodriguez R. Validity and Reliability of Ventilatory and Blood Lactate Thresholds in Well-Trained Cyclists. PloS One. 2016;11(9):e0163389.

27. Goodwin ML, Harris JE, Hernández A, Gladden LB. Blood lactate measurements and analysis during exercise: a guide for clinicians. J Diabetes Sci Technol. 2007;1(4):558-569.

28. Hartikainen S, Lipponen JA, Hiltunen P, et al. Effectiveness of the Chest Strap Electrocardiogram to Detect Atrial Fibrillation. Am J Cardiol. 2019;123(10):1643-1648.

29. Gronwald T, Berk S, Altini M, Mourot L, Hoos O, Rogers B. Real-Time Estimation of Aerobic Threshold and Exercise Intensity Distribution Using Fractal Correlation Properties of Heart Rate Variability: A Single-Case Field Application in a Former Olympic Triathlete. Front Sports Act Living. 2021;28(3):668812.

30. Rogers B, Mourot L, Doucende G, Gronwald T. Fractal correlation properties of heart rate variability as a biomarker of endurance exercise fatigue in ultramarathon runners. Physiol Rep. 2021;9(14):e14956.