Dynamic Heart Rate Variability During Exercise in Athletes and Non-Athletes

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Abstract

Heart rate variability (HRV) is widely used to assess autonomic regulation. Recently, dynamical detrended fluctuation analysis (DDFA), a time- and scale-resolved extension of detrended fluctuation analysis (DFA), has shown promise for estimating metabolic thresholds and training intensity. Here, we apply DDFA to compare HRV dynamics between athletes and non-athletes during a standardized treadmill protocol including rest, acceleration, steady running (85% $\mathrm{HR}_{\max}$), deceleration, and recovery. Time-domain (SDRR, RMSSD) and non-linear metrics (SD1/SD2, DFA α_1) were computed in moving windows alongside DDFA $\tilde{\alpha}(t)$.

Athletes demonstrated higher resting variability, consistent with enhanced parasympathetic modulation. During exertion, time-domain measures declined similarly in both groups, whereas correlation-based metrics revealed clearer distinctions. Notably, athletes exhibited a more pronounced reduction in DFA α_1 and DDFA $\tilde{\alpha}(t)$ and a stronger rebound during recovery, indicating greater autonomic flexibility. These findings extend the application of DDFA to athlete–non-athlete comparisons and demonstrate its sensitivity for characterizing time-resolved changes in beat-to-beat correlation structure beyond conventional HRV measures.

1. Introduction

Heart rate (HR) variability (HRV), the beat-to-beat variation in cardiac rhythm derived from electrocardiography (ECG) or photoplethysmography (PPG) signals [1] is widely used in sports science to monitor recovery, training capacity [2, 3], cardiorespiratory fitness adaptations [4], and overtraining [5]. Beyond conventional time- and frequency-domain measures, dynamical detrended fluctuation analysis (DDFA), a time- and scale-resolved extension of detrended fluctuation analysis (DFA), characterizes evolving correlation properties under non-stationary conditions such as exercise [6], and has recently shown promise for estimating metabolic thresholds and exercise intensity [7, 8].

Training-related differences in resting HRV are well documented [4, 9], and aerobic training is known to enhance parasympathetic modulation, HR recovery, and metabolic efficiency [10–13]. However, it remains unclear whether these adaptations produce distinct time-resolved changes in beat-to-beat correlation dynamics across exercise and recovery.

Here, we apply DDFA alongside conventional time-domain and non-linear HRV metrics to compare autonomic dynamics between athletes and non-athletes across a standardized multi-phase treadmill protocol, evaluating whether correlation-based measures reveal group differences in transition kinetics and recovery that are not captured by conventional HRV metrics.

2. Data and Preprocessing

We utilized ECG data from the publicly available iAMwell dataset [14], comprising six elite Cyprus National Basketball League players (athlete group) and nine recreationally active individuals training fewer than three sessions or 4.5 h/week (non-athlete group), both male and female, aged 17–33 years, collected March–September 2016. High-resolution ECG (2000 Hz) was recorded via a BioNomadix system (BIOPAC MP150, BIOPAC Systems Inc., USA) with a three-electrode chest strap [14]. The study was approved by the Cyprus National Bioethics Committee, with all participants medically screened by a cardiologist prior to written informed consent. The protocol consisted of the following phases:

1. **Rest phase:** 5 min supine rest.
2. **Acceleration phase:** Running from 3 mph, increasing 0.6 mph every 2 min until 85% of age-predicted maximal HR [15].
3. **Steady phase:** 3 min running at 85% of maximal HR.
4. **Deceleration phase:** Speed decreased by 0.6 mph every 30 s to 3 mph.
5. **Recovery phase:** 5 min supine rest.

Each participant completed the protocol once. Total exercise duration (mean $\pm$ SD) was 27.3 $\pm$ 3.7 min for athletes and 21.0 $\pm$ 1.3 min for non-athletes, with acceleration-phase duration of 10.2 $\pm$ 3.2 min and 5.8 $\pm$ 1.2 min, respectively. RRIs were filtered using a rolling local median μ (31-RRI window), discarding values outside 0.9μ–1.1μ or with successive differences exceeding

200 ms; recordings with more than 10% of RRIs removed were excluded. This procedure excluded three non-athlete recordings (N06, N08, N12), yielding a final dataset of six athletes and six non-athletes.

3. Methods

Time-domain HRV metrics included SDRR (standard deviation of RR intervals) and RMSSD (root mean square of successive differences) [16]. Non-linear dynamics were characterized using the Poincaré ratio SD1/SD2, an index of autonomic balance [17], DFA α_1 [18, 19], and DDFA (20 logarithmically spaced scales between 5 and 64 RRIs) [6]). Both DFA and DDFA used maximally overlapping windows with second-order polynomial detrending [6]. The resulting DDFA landscape $\alpha(t, s)$, a two-dimensional representation of scaling behavior across time and scale, was averaged across scales in 1-second bins to yield the mean scaling exponent $\tilde{\alpha}(t)$, reflecting time-resolved correlation dynamics during exercise.

SDRR, RMSSD, SD1/SD2, DFA α_1, and HR (inverse of the mean RRI) were computed in 60 s sliding windows with 10 s steps (50 s overlap), and each value was assigned to the window midpoint. DDFA required no additional windowing, as $\alpha(t, s)$ is inherently time-resolved [6].

For group-level analysis, each exercise phase was independently rescaled to [0,1] per participant (MinMaxScaler) and concatenated sequentially. Within this normalized representation, SDRR, RMSSD, SD1/SD2, and DFA α_1 were binned into 20 equally spaced bins per phase (mean per bin), while $\tilde{\alpha}(t)$ was binned into 50 bins per phase to preserve DDFA's higher temporal resolution.

4. Results

Table 1 summarizes mean ± SD values of HR and HRV metrics across exercise phases. It shows that at rest, athletes exhibited lower HR (57 BPM) than non-athletes (69 BPM) and higher SDRR and RMSSD (76 ms, 73 ms vs. 68 ms, 58 ms). During Acceleration and Steady phases, HR increased similarly in both groups, reaching approximately 169–170 BPM during steady running, while SDRR and RMSSD declined markedly to a shared floor (3–6 ms) and did not return to baseline by the end of Recovery. The SD1/SD2 ratio was higher in athletes at Rest and Acceleration (0.67 vs. 0.53, 0.28 vs. 0.22), decreased in both groups during Acceleration, then rose again in athletes during Steady running before converging in Deceleration and Recovery. DFA α_1 and DDFA $\tilde{\alpha}(t)$ show the most consistent group separation. Both were higher in non-athletes across every phase except Rest, including Steady running, where SDRR, RMSSD, and HR had already converged (DFA α_1: 0.81 vs. 0.94, DDFA $\tilde{\alpha}(t)$: 0.67 vs. 0.89).

Table 1. HRV features across phases (mean ± SD) for athletes (N=6, top rows) and non-athletes (N=6, bottom rows, italic).

Measure	Rest	Acceleration	Steady Run	Deceleration	Recovery
HR (BPM)	57 ± 12	119 ± 10	170 ± 9	163 ± 10	106 ± 25
	69 ± 3	*115 ± 8*	*169 ± 7*	*164 ± 7*	*105 ± 16*
SDNN (ms)	76 ± 19	31 ± 11	5.19 ± 3.09	13 ± 3	24 ± 9
	68 ± 6	*36 ± 6*	*5.83 ± 2.61*	*16 ± 10*	*25 ± 9*
RMSSD (ms)	73 ± 14	14 ± 6	4.74 ± 1.19	6.01 ± 4.22	9.34 ± 4.71
	58 ± 14	*15 ± 6*	*3.49 ± 0.93*	*5.29 ± 1.20*	*11 ± 5*
SD1/SD2	0.67 ± 0.11	0.28 ± 0.05	0.67 ± 0.17	0.36 ± 0.17	0.23 ± 0.05
	0.53 ± 0.14	*0.22 ± 0.05*	*0.41 ± 0.09*	*0.36 ± 0.19*	*0.24 ± 0.06*
DFA α_1	0.95 ± 0.08	1.13 ± 0.11	0.81 ± 0.12	0.81 ± 0.16	1.57 ± 0.32
	1.13 ± 0.17	*1.44 ± 0.10*	*0.94 ± 0.18*	*1.05 ± 0.13*	*1.62 ± 0.22*
DDFA $\tilde{\alpha}(t)$	1.01 ± 0.08	1.13 ± 0.08	0.67 ± 0.09	0.82 ± 0.19	1.29 ± 0.22
	1.08 ± 0.14	*1.24 ± 0.01*	*0.89 ± 0.10*	*0.93 ± 0.15*	*1.28 ± 0.12*

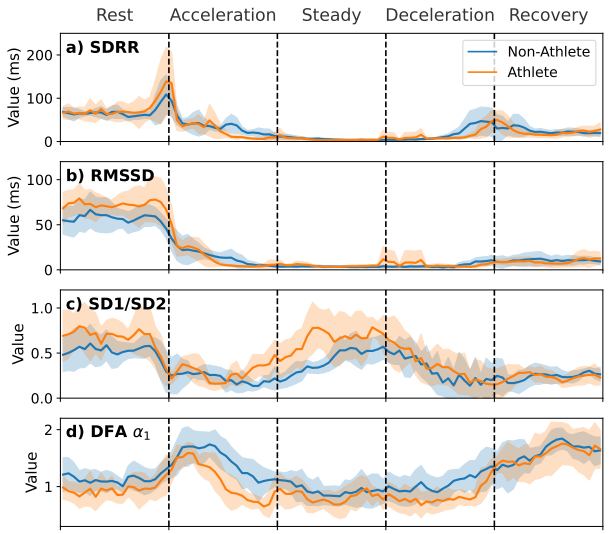

Figure 1. Temporally normalized HRV metrics aggregated across participants in the athlete (N=6) and non-athlete (N=6) groups. Shown are a) SDRR, b) RMSSD, c) SD1/SD2, and d) DFA α_1 as functions of normalized exercise time. Shaded regions represent group-level variability (mean ± SD).

Figure 1 shows these same metrics as continuous, temporally normalized trajectories. SDRR and RMSSD are elevated at Rest and decline steadily through Acceleration to a shared floor during Steady running, then only partially recover. SD1/SD2 (panel c) diverges specifically during Steady running, rising further in athletes before both groups converge again in Recovery. DFA α_1 (panel d) peaks in both groups at the onset of Acceleration before declining, with athletes settling at a lower plateau through Acceleration and Steady running despite comparable HR, then rising again during Deceleration and exceeding Rest baseline in Recovery. Figure 2 shows the corresponding DDFA landscape for non-athletes (panel a) and athletes (panel b). At Rest, $\tilde{\alpha}(t)$ is already lower in athletes despite similar HR of around 70 BPM, indicating that this base-

line difference in correlation structure is not explained by HR alone. This dissociation persists through Acceleration, where the decline in $\tilde{\alpha}(t)$ is concentrated in the long-scale band (16–64 RRIs) and is more pronounced in athletes.

5. Discussion

The convergence of SDRR and RMSSD outside Rest indicates that parasympathetic withdrawal dominates HRV once exercise intensity is high, largely independent of training status, and their partial post-exercise recovery agrees with previous findings [20]. The elevated resting values in athletes are consistent with enhanced parasympathetic modulation from regular aerobic training [21], but this advantage does not carry over once the autonomic system is actively engaged. The SD1/SD2 divergence appearing specifically under sustained load suggests athletes sustain more balanced regulation during exercise.

The correlation-based metrics yield the largest differences. Since scaling exponent values around 1.0 are commonly reported during moderate exercise [22], the elevated values sustained by non-athletes indicate more rigid beat-to-beat dynamics, whereas the lower values in athletes suggest greater adaptability of autonomic control under the same workload. A similar effect is seen in DDFA, where the decline in long-scale correlations (16–64 RRIs) during Acceleration was more pronounced in athletes, suggesting that suppression of these correlations under rising load is a marker of training adaptation rather than a generic exertion response. Athletes also sustained a longer Acceleration phase before reaching 85% HR_{max} (10.2 $\pm$ 3.2 min vs. 5.8 $\pm$ 1.2 min), together suggesting a more gradual, coordinated shift toward exercise-dominant regulation. In non-athletes, the higher exponent values under comparable HR may reflect less efficient cardiovascular-metabolic integration. The slightly higher Recovery values in athletes are consistent with post-exercise parasympathetic rebound [23] and suggest faster autonomic reactivation in the trained group. These findings imply that correlation structure, rather than variance-based HRV, most reliably distinguishes athletes from non-athletes during exercise.

Several limitations should be considered. The sample size was small, with six athletes and six non-athletes, and HRV metrics carry substantial inter-individual variability, so these results should be confirmed in larger cohorts. The non-athlete group, defined as fewer than three training sessions or 4.5 h of exercise per week, does not represent a fully sedentary population and may still possess moderate aerobic conditioning, potentially reducing observed group differences, while the athlete group consisted specifically of elite basketball players whose training profile may not generalize to other disciplines. Finally, exercise intensity was defined using age-predicted maximal HR, a measure with recognized limitations [15], and HR alone does not fully capture internal physiological load. Incorporating metabolic threshold assessments such as the first and second lactate thresholds could provide a more objective characterization of exertion in future work.

6. Conclusions

This study shows that HRV dynamics differ between athletes and non-athletes during progressive exercise and recovery. Time-domain measures (SDRR, RMSSD) were higher at Rest in athletes, consistent with enhanced parasympathetic modulation, but declined similarly in both groups during exercise, offering limited discriminatory value under load. SD1/SD2 indicated greater baseline cardiac complexity in athletes, though differences diminished during sustained exertion.

Correlation-based metrics revealed clearer group distinctions. Athletes showed a more pronounced reduc-

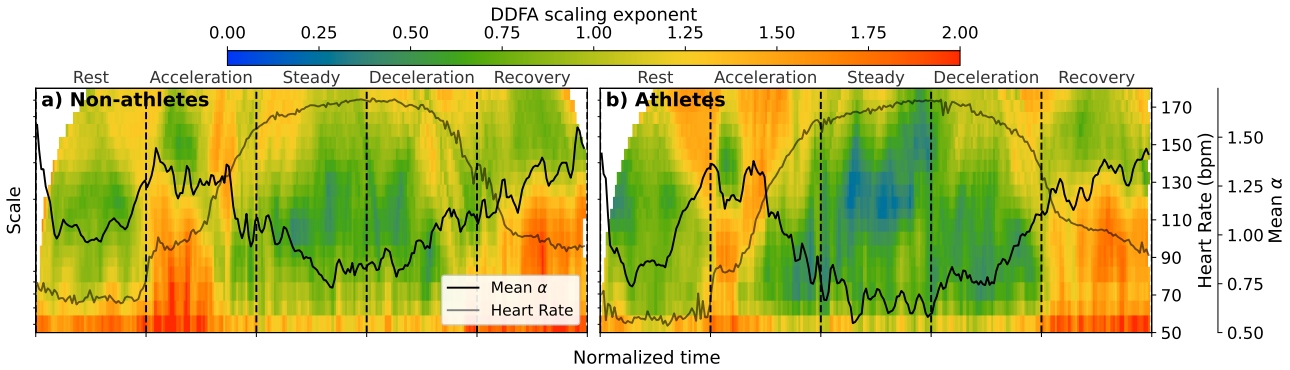

Figure 2. Aggregated temporally normalized DDFA results for **a)** the non-athlete (N=6) and **b)** athlete (N=6) groups. The heatmaps show the group-level mean scaling exponent $\alpha(t, s)$ as a function of normalized time and scale. The black curve represents the scale-averaged exponent $\tilde{\alpha}(t)$, and heart rate is overlaid for reference (grey).

tion in DFA α_1 and DDFA $\tilde{\alpha}(t)$ during Acceleration and Steady exercise despite comparable HR levels, suggesting more efficient autonomic adjustment to rising internal load, whereas non-athletes maintained higher, more rigid scaling exponents. During Recovery, exponents exceeded baseline in both groups, with a slightly stronger rebound in athletes.

These findings highlight the sensitivity of non-linear HRV measures, particularly DDFA, for characterizing time-resolved changes in beat-to-beat correlation structure during exercise, extending its established utility in threshold detection and supporting its potential as a complementary tool for monitoring training load and recovery.

Acknowledgments

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