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Conference Abstract

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Acute Hemodynamic Responses to Three Work-Matched HIIT Training Protocols in Trained Cyclists

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Abstract

Current literature indicates that studies comprehensively examining acute central and peripheral hemodynamic responses to different high-intensity interval training (HIIT) modalities in trained cyclists remain limited. Therefore, this study aims to compare the acute central cardiac output (Q), stroke volume (SV) and peripheral arteriovenous O_2 difference ($a-vO_{2diff}$) responses elicited by three distinct HIIT models and to determine which modality provides a greater acute stimulus to the respective physiological components. Twelve trained male cyclists (Level 3-4) participated in this study (age: 29.7 ± 5.3 years; maximal oxygen uptake (VO_{2max}): 57.8 ± 4.1 $mL \cdot min^{-1} \cdot kg^{-1}$; body mass: 69.8 ± 6.0 kg). Acute hemodynamic responses were evaluated with three work- and effort-matched HIIT models administered in a randomised order: Long-HIIT (4×3 min work- 3 min recoveries), Short-HIIT (1 series of 15×43.5 sec work - 43.5 sec recovery), and Decreasing Power Output HIIT (DPO-HIIT; 6×114.5 sec work - 114.5 sec recovery). Q, SV, and $a-vO_{2diff}$ were measured using a non-invasive inert gas rebreathing method (N_2O_{RB}). Two-way repeated-measures analysis of variance revealed no significant main effect of the protocol for Q ($F(2.000, 22.000)=1.966$, $p=0.178$, $\eta p^2=0.152$), SV ($F(2.000, 22.000)=0.790$, $p=0.446$, $\eta p^2=0.067$), heart rate (HR) ($F(2.000, 22.000)=0.620$, $p=0.547$, $\eta p^2=0.053$), or $a-vO_{2diff}$ ($F(2.000, 22.000)=1.586$, $p=0.227$, $\eta p^2=0.126$). The Protocol × Time interaction was significant only for HR ($F(2.467, 27.132)=4.561$, $p=0.014$, $\eta p^2=0.293$). Consequently, while overall systemic stress remains matched, coaches and athletes can utilise these different HIIT structures interchangeably, which may allow for flexibility in endurance training prescription.

Keywords

arterio-venous O_2 difference; high-intensity interval training; cardiac output; performance; stroke volume

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1 Introduction

Cardiac output (Q), stroke volume (SV), and the arteriovenous O_2 difference ($a-vO_{2diff}$) together constitute the primary determinants of maximal oxygen uptake ($\dot{V}O_{2max}$), which is a critical physiological factor determining endurance performance (Rønnestad et al., 2020). SV and heart rate (HR), which together determine Q, represent the central component of $\dot{V}O_{2max}$, whereas the $a-vO_{2diff}$ constitutes the peripheral component (Faricier et al., 2025; Joyner & Dominelli, 2021). Individual differences in physiological and haemodynamic responses should therefore be considered when targeting cardiovascular and peripheral adaptations through HIIT (Buchheit & Laursen, 2013). Accordingly, for an athlete with a larger deficit in the central component, high-intensity interval training (HIIT) protocols that elicit and sustain higher peak Q and/or peak SV for longer durations may be considerably more effective. Current literature indicates that studies comprehensively examining acute central and peripheral hemodynamic responses to different HIIT modalities in trained cyclists remain limited. Therefore, this study aims to compare the acute central (Q or SV) and peripheral ($a-vO_{2diff}$) responses elicited by three distinct HIIT models and to determine which modality provides a greater acute stimulus to the respective physiological components.

2 Material and Methods

2.1 Participants

Twelve trained male cyclists (Level 3-4), (De Pauw et al., 2013) participated in this study (age: 29.7 ± 5.3 years; $\dot{V}O_{2max}$: 57.8 ± 4.1 $mL \cdot min^{-1} \cdot kg^{-1}$; body mass: 69.8 ± 6.0 kg). The study was conducted in accordance with the Declaration of Helsinki and received institutional ethics committee approval (Decision No: 24-11.1T/41; Date: 28 November 2024).

2.2 Design

A randomised repeated-measures design was used in this study. Acute hemodynamic responses were evaluated with three work- and effort-matched (RPE ≥ 19) HIIT models administered in a randomized order: Long HIT (4×3 min work - 3 min recovery; WR ~88% of peak WR (WR_{peak}); Recovery (REC) ~25% of WR_{peak}), Short-HIIT (1 series of 15×43.5 sec work - 43.5 sec recovery; WR ~110% of WR_{peak}; REC ~10% of WR_{peak}) and Decreasing Power Output HIIT (DPO-HIIT, 6×114.5 sec work – 114.5 sec recovery; WR starting at I_{HIGH} and decreased by 4 equal steps to the CP + 10%; Rec ~25% of WR_{peak}).

2.3 Methodology

Familiarisation sessions were conducted to adapt the participants to the electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) and cardiac measurement system (Innocor, COSMED, Italy).

$\dot{V}O_{2max}$ was determined via a ramp incremental test, with the WR increasing by 1 W every 2 s (30 $W \cdot min^{-1}$).

Critical power and the highest constant-intensity that permits the attainment of $\dot{V}O_{2max}$ (I_{HIGH}) were determined using multiple constant-work-rate testing sessions (Caputo & Denadai, 2008; Jones et al., 2010).

Q, SV, and $a-vO_{2diff}$ were measured using a valid and reliable non-invasive inert gas rebreathing method (N_2O_{RB}), (Fontana et al., 2009).

Throughout the sessions, variables were recorded at four time points (ΔTime: 25%, 50%, 75%, and 100% of each bout).

2.4 Statistical Analysis

Data were analysed using a two-way (Protocol × ΔTime) repeated-measures ANOVA with Greenhouse-Geisser corrections

where appropriate. Partial eta squared (ηp^2) was reported as the effect size for the overall effect of protocol in the repeated-measures ANOVA and was interpreted as small ($\eta p^2 \geq 0.01$), medium ($\eta p^2 \geq 0.06$), and large ($\eta p^2 \geq 0.14$) (Richardson, 2011) (Richardson, 2011).

Bonferroni-adjusted post-hoc tests were applied for pairwise comparisons. Cohen's d was reported for pairwise post-hoc comparisons to quantify the magnitude of standardised differences between individual HIIT protocols and was interpreted as small ($d \geq 0.20$), medium ($d \geq 0.50$), and large ($d \geq 0.80$) (Cohen, 1988). Statistical analyses were performed using JASP (Version 0.98.1; JASP Team, 2026).

3 Results

Two-way repeated-measures ANOVA revealed no significant main effect of protocol for Q ($F(2.000, 22.000) = 1.966$, $p = 0.178$, $\eta p^2 = 0.152$), SV ($F(2.000, 22.000) = 0.790$, $p = 0.446$, $\eta p^2 = 0.067$), HR ($F(2.000, 22.000) = 0.620$, $p = 0.547$, $\eta p^2 = 0.053$), or $a{-}vO_{2diff}$ ($F(2.000, 22.000) = 1.586$, $p = 0.227$, $\eta p^2 = 0.126$). The Protocol × Δ Time interaction was significant only for HR ($F(2.467, 27.132) = 4.561$, $p = 0.014$, $\eta p^2 = 0.293$). The Protocol × Δ Time interactions for Q ($F(2.476, 27.232) = 2.448$, $p = 0.095$, $\eta p^2 = 0.182$), SV ($F(2.330, 25.629) = 0.620$, $p = 0.569$, $\eta p^2 = 0.053$), and $a{-}vO_{2diff}$ ($F(3.043, 33.477) = 2.314$, $p = 0.093$, $\eta p^2 = 0.174$) were not significant. The Protocol × Δ Time effect sizes for Q and $a{-}vO_{2diff}$ were large. The temporal responses of Q, SV, $a{-}vO_{2diff}$, and HR across the three HIIT protocols are presented in Figure 1.

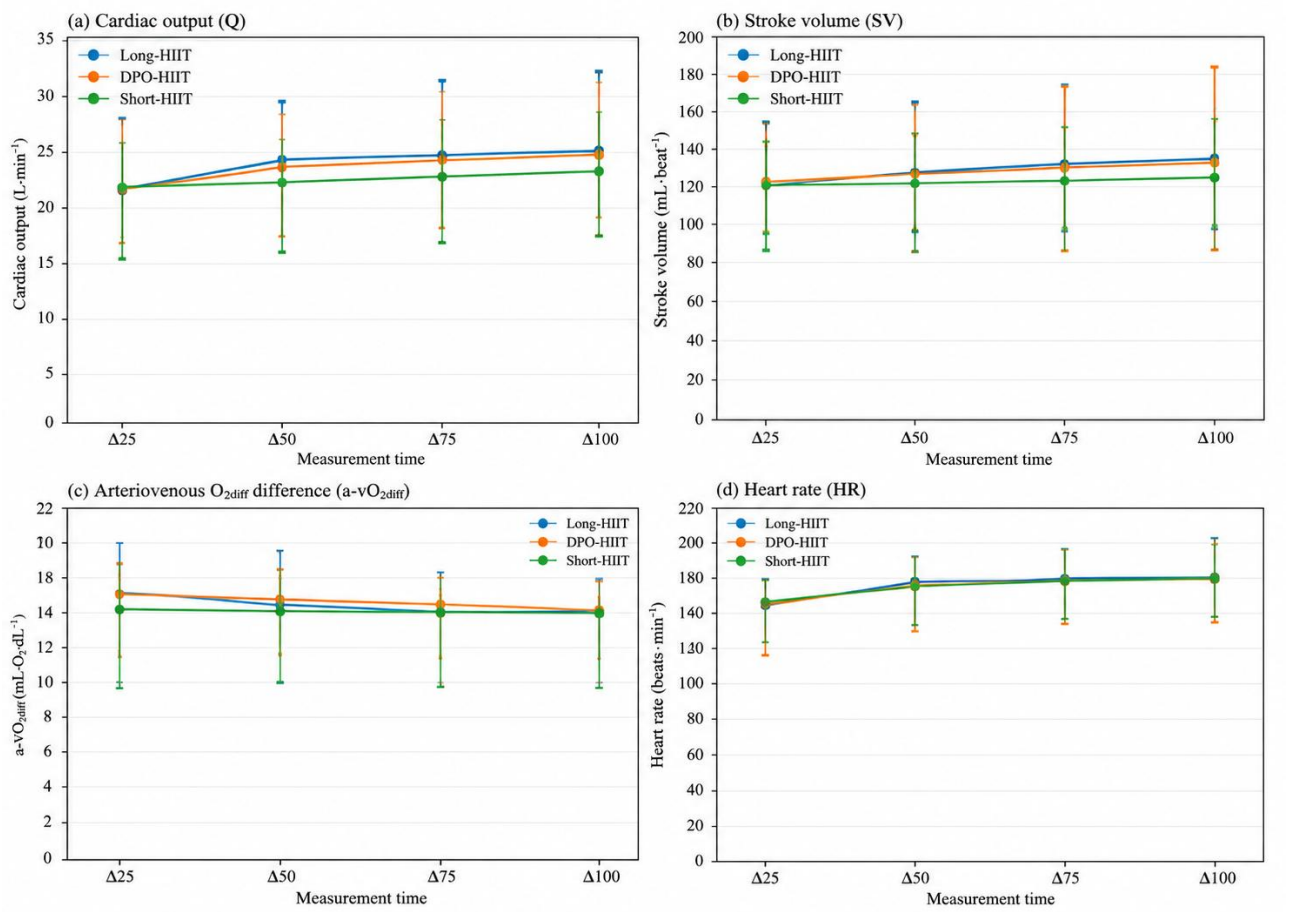

Figure 1. Acute hemodynamic responses during the three high-intensity interval training protocols: **(a)** cardiac output (Q); **(b)** stroke volume (SV); **(c)** arteriovenous O_2 difference ($a{-}vO_{2diff}$); and **(d)** heart rate (HR) at Δ25, Δ50, Δ75, and Δ100 of each bout. Data are presented as mean ± SD.

Q increased over time across all protocols (Figure 1a). Bonferroni-adjusted pairwise comparisons, averaged across protocols, showed an increase from Δ25 to Δ100 ($p = 0.001$).

SV showed no significant change from Δ25 to Δ100 when averaged across protocols (Figure 1b). The Bonferroni-adjusted pairwise comparison between Δ25 and Δ100 was not significant ($p = 0.187$).

The $a-vO_{2diff}$ showed no significant change from Δ25 to Δ100 when averaged across protocols (Figure 1c). The Bonferroni-adjusted pairwise comparison between Δ25 and Δ100 was not different ($p = 0.415$).

HR increased significantly from Δ25 to Δ100 across protocols (Figure 1d). No significant main effect of protocol was observed, indicating that overall HR responses were similar between HIIT protocols. However, the significant Protocol × ΔTime interaction suggests that the temporal pattern of HR increase was not identical across protocols.

4 Discussion

The absence of a main protocol effect for Q, SV, and $a-vO_{2diff}$, further supported by trivial effect sizes in pairwise comparisons (Cohen's d < 0.2), aligns with the observation that, despite differences in interval structure, the three work-matched models elicited broadly comparable average central and peripheral responses.

In our data, Q increased over time, while SV remained stable. Therefore, the increase in Q seems to be mainly related to the increase in HR. For $a-vO_{2diff}$, we did not observe a clear protocol-specific difference. This pattern suggests that central hemodynamic adjustments contributed more to the temporal rise in oxygen transport than peripheral extraction.

Previous studies provide useful but different perspectives. Stanley and Buchheit (2014) examined acute cardiovascular responses during HIIT in well-trained cyclists and reported that SV could be maintained during interval exercise, regardless of recovery intensity (Stanley & Buchheit, 2014). This is consistent with our finding that SV remained relatively stable across the three HIIT protocols.

Coe and Astorino (2024) compared different HIIT formats and showed that protocol structure can influence acute hemodynamic responses such as Q and SV (Coe & Astorino, 2024). However, their protocols differed in total duration and exercise volume. Therefore, it is difficult to separate the effect of interval structure from the effect of total work or duration.

In contrast, our study matched total work, total time, and effort across protocols. So, our design provides a more controlled comparison of how interval structure itself may influence acute central and peripheral hemodynamic responses.

Although the Protocol × ΔTime interactions for Q and a−vO2diff exhibited large effect sizes ($\eta p^2 = 0.182$ and 0.174, respectively), subsequent pairwise comparisons revealed only trivial effect sizes (Cohen's d < 0.2) between the HIIT protocols. Therefore, the practical superiority of one specific HIIT structure over another regarding acute central and peripheral hemodynamic responses cannot be conclusively established from the current data.

5 Practical Applications

Consequently, while overall systemic stress remains matched across the different HIIT intervals, manipulating the specific structure of work-matched HIIT (Long, Short, or DPO) does not appear to selectively target central or peripheral oxygen extraction kinetics. Based on

our data, we cannot make protocol-specific recommendations for cyclists with this training status. Overall, these findings should be interpreted as practical tendencies rather than clear evidence of protocol superiority.

6 Conclusions

When overall work and effort are strictly matched, different HIIT structures (Long, Short, and DPO) elicit comparable acute central (Q, SV) and peripheral (a−vO_{2diff}) hemodynamic stress. Therefore, manipulating work-effort match interval structure does not selectively isolate specific central or peripheral physiological responses. This may provide coaches and athletes with the flexibility to utilise any of these formats interchangeably to achieve a similar overall cardiovascular stimulus.

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