Effects of normobaric hypoxia and hyperthermia on ventilatory responses to high-intensity interval training bouts

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ABSTRACT

Purpose Respiratory frequency (f_R) and tidal volume (V_T) show distinct responses during high-intensity interval training (HIIT) bouts, yet the underlying mechanisms remain unclear. We investigated the effects of hypoxia and hyperthermia on ventilatory responses to HIIT bouts.

Methods Ten recreationally trained males (mean±SD: peak oxygen uptake: 3.98±0.62 L/min, age: 28±6 years) performed the same HIIT protocol to exhaustion in three randomized conditions: normobaric hypoxia (HYP, 15% O_2), hyperthermia (HOT, 35°C, 40% humidity), and control (CON, 18°C and 40% humidity). Work-recovery phases (30-30 s) were performed at 109% of peak power output from a prior incremental test and 50W, respectively.

Results Time-to-exhaustion (TTE) differed (P<0.05) across CON (18.0±4.4 min), HYP (9.4±2.8 min), and HOT (12.6±3.1 min) conditions. Iso-time f_R was associated with changes in TTE and positively correlated with perceived exertion (P<0.001; r=0.85) and aural temperature (P<0.001; r=0.58). On average, f_R increased during the work phase and decreased during recovery, primarily driving pulmonary ventilation ($\dot{V}_E$) during work–recovery alternations. Conversely, V_T showed a smaller and opposite response, with higher values during recovery. V_T plateaued in all conditions, but higher values were observed in HYP *versus* CON and HOT, accompanied by higher capillary blood lactate levels and lower blood oxygen saturation.

Conclusions f_R is associated with changes in exercise tolerance irrespective of the environmental conditions tested and is more closely related to perceived exertion than to aural temperature. f_R primarily drives the $\dot{V}_E$ response to high-intensity work-recovery alternations, while V_T appears fine-tuned on f_R levels and the magnitude of metabolic inputs.

Keywords

Ventilatory control, exercise hyperpnea, muscle afferent feedback, central command, breathing pattern.

INTRODUCTION

There is substantial evidence suggesting the existence of distinct and integrated mechanisms modulating respiratory frequency (f_R) and tidal volume (V_T) during exercise (Tipton et al. 2017; Nicolò and Sacchetti 2019). f_R appears to be substantially modulated by fast inputs, including muscle afferent feedback (especially during moderate exercise) (Girardi et al. 2021) and central command (especially during high-intensity exercise) (Thornton et al. 2001). Conversely, V_T appears to be primarily modulated by metabolic inputs; it is to a good extent associated with pulmonary carbon dioxide output ($\dot{V}CO_2$) and is sensitive to changes in arterial partial pressure of CO_2 ($PaCO_2$) and blood pH (Borison et al. 1978; Clark et al. 1980; Ahmad and Loeschcke 1982; Duffin et al. 2000; Mateika et al. 2004). There are also data suggesting that V_T might be fine-tuned based on f_R levels to promote appropriate matching between alveolar ventilation and metabolic requirements (Nicolò et al. 2018; Nicolò and Sacchetti 2023). The existence of distinct and integrated mechanisms modulating f_R and V_T during exercise is also suggested by their different exercise response dynamics observed during progressive (Clark et al. 1983; Gallagher et al. 1987), sinusoidal (Nicolò et al. 2018; Girardi et al. 2021), square-wave (Forster et al. 1986; Girardi et al. 2024), and intermittent exercise (Nicolò et al. 2014, 2017). These exercise protocols reveal that the V_T response is generally delayed compared to that of f_R. However, the mechanisms underlying these distinct behaviors remain elusive.

Due to the high degree of redundancy among the putative inputs driving pulmonary ventilation ($\dot{V}_E$) during exercise, it is very difficult to quantify their relative contribution to the modulation of f_R and V_T (Forster et al. 2012). A partial solution to this challenge is the use of exercise protocols to create conditions in which different inputs driving $\dot{V}_E$ act at different timings (Casaburi et al. 1977; Nicolò et al. 2017, 2018; Girardi et al. 2021, 2024). For instance, high-intensity interval training (HIIT) bouts have been proposed as a method to partially dissociate the contribution of fast-changing (e.g., central command and muscle afferent feedback) and slow-changing (metabolic stimuli) inputs modulating $\dot{V}_E$, leveraging the abrupt changes in work rate characterizing HIIT bouts (Nicolò et al.

2017). Using this approach, f_R was found to respond promptly to work–recovery alternations, while V_T was more closely associated with the dynamics of some metabolic input markers (Nicolò et al. 2017). The advantages of using HIIT bouts in studies investigating ventilatory control during exercise are enhanced when HIIT is combined with experimental interventions affecting the pattern of breathing. However, there is a lack of studies using this approach, and yet our understanding of the control of f_R and V_T during HIIT bouts is limited despite the widespread use of this exercise protocol in research settings and training practice.

A classical experimental intervention that affects f_R more than V_T is the increase in core body temperature during exercise. Hyperthermia exacerbates the increments in f_R during constant work rate exercise and reduces exercise tolerance (Hayashi et al. 2006; González-Alonso et al. 2023). The prompt variations in f_R during HIIT bouts may challenge the association between f_R and core body temperature, as the latter usually shows a delayed response compared to changes in power output (Todd et al. 2014). Hyperthermia alters brain activity and increases perceived exertion (Nielsen and Nybo 2003), a surrogate of central command (Enoka and Stuart 1992; Marcora 2009). However, whether hyperthermia during HIIT bouts influences the well-established relationship between f_R and perceived exertion remains unclear. On the other hand, acute hypoxia is an experimental intervention that reduces exercise tolerance but determines an increase in both f_R and V_T, with changes in the breathing pattern that are moderated by hypoxia levels and by the specific methodology used for inducing hypoxia (e.g., normobaric *vs.* hypobaric hypoxia) (Richard and Koehle 2012; Vinetti et al. 2025). Comparing conditions that impair exercise tolerance via different mechanisms may elucidate whether f_R is associated with variations in exercise tolerance and perceived exertion independent of environmental manipulation, or is influenced by the specific environmental stimulus (i.e. hyperthermia vs. hypoxia). However, to the best of our knowledge, the effects of hypoxia and hyperthermia on the responses of f_R and V_T during HIIT bouts have not yet been investigated.

The present study aimed to improve our understanding of the ventilatory control during HIIT bouts by testing whether hyperthermia and hypoxia would: (i) influence the association between f_R

and perceived exertion and exercise tolerance (measured as exercise duration) (ii) modify the responses of f_R and V_T to high-intensity work-recovery alternations. We hypothesized that: 1) f_R is associated with changes in perceived exertion and exercise tolerance irrespective of the environmental conditions tested; 2) regardless of the environmental condition tested, f_R primarily drives $\dot{V}_E$ response to high-intensity work-recovery alternations, with V_T being influenced by f_R levels and metabolic stimuli (i.e. a higher response under hypoxia).

METHODS

Ethical approval

The experimental protocols were approved by the Ethics Committee of the University of Kent School of Sport and Exercise Sciences (ID: 97_2017_18) and conformed to the standards set by the Declaration of Helsinki, with the exception of clinical trial registration. Prior to any experimental procedure, written informed consent was obtained from all participants.

Participants

Ten healthy males (mean ± SD: age 28 ± 6 years, stature 177 ± 7 cm, body mass 73 ± 8 kg) volunteered to participate in this study. They were non-smokers, recreationally trained cyclists or triathletes and were instructed to avoid strenuous exercise and caffeine or alcohol in the 24 h preceding each visit and to wear the same exercise outfit across all testing sessions.

Experimental overview

This was a randomized, single-blind, cross-over, controlled study. Blinding was applied only to the control and hypoxia conditions, as hyperthermia was easily recognizable and participants were able to identify this condition. Participants reported to the laboratory on four separate occasions over a period of 2 weeks, with a minimum of 48 h between visits. On the first visit, participants performed a preliminary ramp-incremental test until exhaustion to obtain peak power output (PPO) and peak pulmonary oxygen uptake ($\dot{V}O_{2peak}$). After an adequate recovery period, they were familiarized with the experimental procedures of the subsequent visits. On the three subsequent visits (2-4), participants performed the same HIIT protocol to exhaustion under three randomized environmental conditions: normobaric hypoxia (HYP), hyperthermia (HOT), or control (CON). All the protocols were performed on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands). The ergometer seat and handlebar position were adjusted for each participant during

the first visit, and the same settings were maintained for all subsequent visits. Cardiopulmonary, mechanical and perceptual variables were recorded throughout all the tests.

On the day before each experimental test, participants adopted the same diet and fluid intake to standardize hydration status. They consumed 10 ml water/kg body weight the night before the experiment and the same volume on the morning of the experiment. On the morning of the experimental tests, participants were asked to take in a light breakfast, and 1 h before the experiment they consumed 200–300 ml of water. To minimize the effects of circadian rhythm, each participant was tested at the same time of day across all visits.. Before each repeated testing session, participants were confirmed to have achieved adequate physical and cognitive recovery from the previous exercise session.

Ramp-incremental exercise test

Before the ramp-incremental test, participants were given standard instructions for overall rating of perceived exertion (RPE) using the 6-20 scale developed by Borg (Borg 1998).

After a standardized warm-up (5 min at 100 W + 5 min recovery), participants performed a ramp-incremental test (2 min at 20 W + 1 W increment every 2 s) until exhaustion, which was defined as a reduction in the pedaling cadence >10 rpm below the mean value maintained during the test, despite strong verbal encouragement. The peak values of oxygen uptake ($\dot{V}O_{2peak}$), f_R (f_{Rpeak}), V_T (V_{Tpeak}), and $\dot{V}_E$ ($\dot{V}_{Epeak}$) were defined as the highest value of a 30-s moving average reached during any phase of the ramp-incremental test, not necessarily at PPO; the PPO was computed as the highest power output achieved at exhaustion, recorded to the nearest 1 W. Participants were asked to report their RPE every minute and immediately after exhaustion by pointing the finger on a scale conveniently located on the handlebars of the ergometer. This procedure served as a familiarization with the RPE scale, and these RPE values were not included in subsequent data analysis.

Familiarization

After a minimum of 30 min of recovery from the ramp-incremental test (at visit 1), participants were familiarized with all the experimental procedures to be performed at the following visits. They completed an HIIT protocol to exhaustion (work–recovery cycle of 30s-30s), with the work and recovery intensities set at 110% of PPO and 50 W, respectively. This familiarization fulfilled three purposes: 1) to further familiarize participants with the RPE scale and the experimental procedures; 2) to adjust the work intensity for the experimental HIIT bouts in order to obtain a time to exhaustion (TTE) of approximately 20 min during the CON condition; and 3) to individualize each participant's preferred pedaling cadence for the subsequent exercise tests.

HIIT bouts

For each visit, before performing the HIIT bouts, participants underwent a standardized procedure inside the chamber lasting at least 30 min, which included 15 min of seated rest, 3 min of data recording under resting conditions, and 10 min of a standardized warm-up. Immediately after the resting measurements, aural temperature, blood oxygen saturation (SpO_2), and resting blood lactate concentration [La] were assessed.

The same HIIT protocol (30s-30s work-recovery alternations) to exhaustion was performed under different environmental conditions (HOT, HYP, and CON) on separate visits. All experimental conditions were performed in an environmental simulation chamber (Hypoxico Inc., New York, NY, USA).

The work intensity was set based on the TTE from the familiarization visit, with the aim that the CON condition would last approximately 20 min. During the CON visits, temperature and relative humidity were kept constant at approximately 18°C and 40%, respectively; under the HOT condition, values were approximately 35°C and 40%, respectively. Under the HYP condition, the oxygen concentration in the environmental simulation chamber was set to 15% (moderate normobaric hypoxia) balanced with nitrogen, whereas temperature and relative humidity were maintained constant at the same levels as in the CON condition.

Participants were instructed to maintain their preferred pedaling cadence within a range of ±5 rpm throughout each HIIT bout. Exhaustion was defined as the participant's pedaling cadence falling more than 10 rpm below the target cadence for more than 10 s. The participants were informed that a 10-s countdown would start when pedaling cadence fell outside the target cadence, and that no feedback or encouragement would be given during the test. Only completed bouts were considered valid. Given the environmental interventions used, participants were instructed to stop the test if unusual or unpleasant symptoms developed.

Measures

Cardiorespiratory variables. Pulmonary gas exchange, $\dot{V}_E$, f_R, V_T, P_{ETCO2}, and HR were measured breath-by-breath using a metabolic gas analysis system (MetaLyzer 3B; Cortex Biophysik GmbH, Leipzig, Germany) connected to an oro-nasal mask (7600 series; Hans Rudolph Inc., Kansas City, MO, USA). Calibration procedures were performed following the manufacturer's instructions.

Blood lactate concentration. Capillary blood samples were drawn from the earlobe and immediately analyzed for [La-] (Biosen; EFK Diagnostics, London, UK). During the experimental visits (2-4), [La-] was measured before the warm-up, every 2 min during the HIIT bouts, and 1 min after exhaustion.

Blood oxygen saturation. SpO_2 was measured every 2 min during the HIIT bouts using a pulse oximeter (Nonin Onyx Vantage 9500; Integrated Medcraft, Moodus, CT, USA).

Aural temperature. Aural temperature was measured every 2 min during the HIIT bouts using an aural infrared thermometer (Braun IRT6020 ThermoScan 5 Ear Thermometer; Braun Healthcare, Bethlehem, PA, USA).

Rating of perceived exertion. Perceived exertion, defined as "the conscious sensation of how hard, heavy, and strenuous exercise is" was measured using the 6-20 Borg scale immediately after each repetition during the HIIT bouts. Standardized instructions for the RPE scale were given to each participant before the warm-up at each visit. Briefly, participants were instructed to assess the overall

perceived effort. For instance, a rating of nine indicates a "very light" effort, while a rating of seventeen represents a "very hard" and demanding level of exertion.

Data analysis

Pulmonary gas exchange: Breath-by-breath data were edited for errant breaths (e.g., values resulting from sighs, swallows, or coughs) by deleting values greater than three standard deviations from the local mean (Lamarra et al. 1987). Subsequently, data were interpolated with a linear function and extrapolated every second. A detailed characterization of the physiological responses was then performed using multiple complementary approaches: i) the time course of second-by-second responses to high-intensity work-recovery alternations (only relevant phases are shown for clarity); ii) 60-s bin averages to describe the overall time course independent of fluctuations induced by work–recovery alternations, such that each work–recovery phase is represented by a single data point; iii) 10-s bin averages as a function of the work-recovery cycle yielding six data points per bout (three for the work phase and three for the recovery phase); and iv) analysis of associations between relevant physiological variables. To this end, the following analyses were used.

Individual isotime analysis: To compare responses across conditions at the same absolute time within each individual while minimizing data loss, thereby preserving sensitivity to detect true physiological differences (Nicolò et al. 2019). This analysis has been previously described in detail (Nicolò et al. 2019). Briefly, second-by-second data were averaged over 60-s intervals such that each work–recovery bout was represented by a single data point, and subsequently interpolated and extrapolated to obtain second-by-second time courses. The time course of physiological and perceptual variables was then compared across the three conditions (HOT, HYP, and CON), with each participant individually taken into consideration. For each participant, the shortest TTE test was segmented into ten equally spaced time points, which were then used to segment all the other experimental exercise tests of the same individual. For instance, if a participant completed the three TTE tests in 600 s, 660 s, and 720 s, the shortest test (i.e., 600 s) would have been selected to identify

the ten equally spaced time points (i.e., 60, 120, 180 s, etc., up to 600 s). These ten time points were then used as anchor points to extract the corresponding data from the other TTE tests of that participant, allowing direct comparison across conditions at matched isotime points. The inherent breath-by-breath variability might affect the comparison of single time points; however, this issue was greatly reduced by the approach adopted to reduce breath-by-breath noise (filtering + 60-s bin average). Thus, for each participant, all the three HIIT tests were segmented into ten time points based on the time points selected for the shorter HIIT test. This individual isotime approach is known to minimize the data loss that is commonly observed when other approaches are used to compare tests with different exercise durations (Nicolò et al. 2019). Peak responses, computed as described earlier (see '*Ramp-incremental exercise test*' section), were also compared across TTE tests. The code used to perform these analyses is available elsewhere (Nicolò et al. 2019).

Relative isotime analysis: To compare responses at the same relative percentage of each test's duration (i.e., normalized to the exercise duration of each TTE test), which avoids data loss but does not allow assessment of effects at the same absolute time (Nicolò et al. 2019). This analysis has been previously described in detail (Nicolò et al. 2019). Briefly, second-by-second data were averaged over 60-s intervals such that each work–recovery bout was represented by a single data point, and subsequently interpolated and extrapolated to obtain second-by-second time courses. Data were then segmented into ten equally-spaced timepoints based on the TTE of each test, irrespective of the length of the test. Thus, each exercise test to exhaustion is represented by 10 data points without data loss. With this approach, comparisons were performed at different absolute time points within each participant but at the same relative percentage of TTE across conditions (Nicolò et al. 2019). Individual participant data across relative isotime points were used for Bland-Altman within-subject correlation analysis (see '*Statistical analysis*' for details). Averaged responses across conditions were used to illustrate V_T responses at comparable $\dot{V}_E$ levels using the Hey plot (Hey et al. 1966).

Work-recovery cycle: To describe the time course of the physiological variables within the work-recovery cycle, a previously suggested analysis was performed (Nicolò et al. 2017). Briefly,

second-by-second data were averaged over 10 s, and each 60-s work–recovery cycle was thus represented by 6 data points. In analogy with the previously described individual isotime analysis, the test with the shortest TTE was identified for each participant and used to determine the total number of complete work–recovery cycles to be included in the analysis. Only the cycles completed in all three conditions for that participant (i.e., the number of cycles corresponding to the shortest test) were included, thereby avoiding unequal representation of early and late cycles across conditions. For each test, the work–recovery cycles selected (e.g., cycle 1, cycle 2, etc.) were temporally aligned (i.e. superimposed) and averaged. Finally, for each condition, data expressed as a function of the work-recovery cycle were averaged across participants and reported as mean ± SEM.

To further ensure an accurate description of the ventilatory response to the work–recovery transition across conditions, the time course of second-by-second f_R, V_T, and $\dot{V}_E$ was depicted for the first two isotime bouts, middle isotime bouts, last isotime bouts, and final bouts. This analysis is conceptually similar to the individual isotime approach, where work-recovery cycles were selected based on the shortest TTE completed by each participant. Briefly, the initial responses were characterized using the first two HIIT bouts for all conditions. The middle isotime bouts corresponded to those performed at half time of the shortest TTE for each participant. The last isotime bouts corresponded to the last two work-recovery cycles of the shortest TTE for each participant, and to the corresponding cycles of the other two conditions. The final bouts refer to the last two completed bouts in each condition, irrespective of total exercise duration. The same procedure was applied to $\dot{V}O_2$, $\dot{V}CO_2$, and HR.

Statistical analysis

Statistical analysis was conducted using IBM SPSS Statistics 22 (SPSS Inc., Chicago, IL, USA). All data were assessed for normality of distribution before analysis using Shapiro–Wilk test, histograms, Q–Q plots, and boxplots. A one-way repeated-measures ANOVA was used to compare the number of bouts completed across the three different conditions, and to compare the end-test values of

physiological and perceptual variables between the three conditions. Follow-up comparisons were performed using Bonferroni correction. A two-way repeated-measures ANOVA (condition × time) was used to compare the time course of physiological and perceptual variables across conditions, both when expressed as a function of time and as a function of the work-recovery cycle. In the case of a significant interaction, only pre-planned follow-up comparisons were performed (i.e., across conditions) using Bonferroni correction. When the sphericity assumption was violated, the Greenhouse–Geisser adjustment was performed. Partial eta squared (η_p^2) effect sizes were calculated; an effect of $\eta_p^2 \geq 0.01$ indicated a small, $\eta_p^2 \geq 0.059$ a medium, and $\eta_p^2 \geq 0.138$ a large effect (Cohen 1988).

Within-subject correlation coefficients (r) were computed for the correlations between RPE and f_R, RPE and aural temperature, and f_R and aural temperature, using a previously described method which adjusts for repeated observations within participants (Bland and Altman 1995). With this approach, all repeated observations obtained from the relative isotime analysis were entered into a single linear regression model for each investigated relationship (i.e., RPE *vs.* f_R, RPE *vs.* aural temperature, and f_R *vs.* aural temperature) in which "participant" was included as a categorical factor (using dummy variables), thereby accounting for the non-independence of repeated measurements within individuals. A p value of < 0.05 was considered statistically significant in all analyses. The results are expressed as mean ± SD in the text and as mean ± SEM in the Figures.

RESULTS

During the preliminary ramp-incremental test, participants reached a PPO of 353 ± 59 W, $\dot{V}O_{2peak}$ of 3.98 ± 0.62 L·min^{-1}, f_{Rpeak} of 59 ± 10 breath·min^{-1}, V_{Tpeak} 3.15 ± 0.63 L and $\dot{V}_{Epeak}$ of 164 ± 32 L·min^{-1}. Due to technical problems with the metabolic cart that occurred in one test, breath-by-breath data from one participant were excluded. The work phase power output of the HIIT test was 386 ± 65 W.

HIIT bouts

One-way ANOVA showed between-condition differences ($P < 0.001$; $\eta_P^2 > 0.840$) in TTE: CON (18.0 ± 4.4 min), HYP (9.4 ± 2.8 min) and HOT (12.6 ± 3.1 min) (Figure 1). Figure 1 depicts follow-up comparisons between conditions.

Work-recovery cycle. Figure 2 depicts the time course of f_R, V_T, and $\dot{V}_E$ during the first two isotime HIIT bouts, the middle isotime HIIT bouts, the last isotime HIIT bouts, and the final HIIT bouts across conditions. f_R showed a rapid increase at the onset of the work phase and a prompt decrease during recovery in all conditions, whereas V_T showed a comparatively smaller and opposite response pattern. Overall, f_R fluctuations across the work–recovery transitions predominantly determine changes in $\dot{V}_E$. Figure 3 depicts the time course of $\dot{V}O_2$, $\dot{V}CO_2$, and HR during the same high-intensity bouts.

Figure 4 shows the group's average response within the work–recovery cycle of relevant physiological variables during the HIIT bouts across conditions. A significant main effect of condition ($p < 0.05$; $\eta_P^2 > 0.395$) was found for all the variables reported in Figure 2, except for $\dot{V}CO_2$ ($p = 0.054$; $\eta_P^2 = 0.278$), while a significant main effect of time ($p < 0.05$; $\eta_P^2 > 0.351$) was observed for all the variables. A significant interaction ($p < 0.05$; $\eta_P^2 > 0.192$) was found for all the variables

reported in Figure 2 except for f_R ($p = 0.265$; $\eta_P^2 = 0.123$), V_T ($p = 0.532$; $\eta_P^2 = 0.091$), P_{ETCO2} ($p = 0.156$; $\eta_P^2 = 0.168$). Follow-up comparisons across conditions are shown in Figure 2.

Individual isotime analysis. Figures 5 and 6 depict the time course of the main physiological variables during the HIIT bouts across conditions. A significant main effect of condition ($p < 0.01$; $\eta_P^2 > 0.457$) was observed for all the variables reported in Figure 5, except for $\dot{V}CO_2$ ($p = 0.060$; $\eta_P^2 = 0.269$). A significant main effect of time ($p < 0.01$; $\eta_P^2 > 0.880$) was found for all the variables. A significant interaction ($p < 0.05$; $\eta_P^2 > 0.172$) was found for all the variables reported, except for $\dot{V}O_2$ ($p = 0.140$; $\eta_P^2 = 0.134$). A significant main effect of condition ($p < 0.05$; $\eta_P^2 > 0.392$) and time ($p < 0.01$; $\eta_P^2 > 0.744$) was observed for all the variables reported in Figure 6. A significant interaction ($p < 0.05$; $\eta_P^2 > 0.172$) was found for all variables, except for the aural temperature ($p = 0.735$; $\eta_P^2 = 0.079$). Follow-up comparisons are reported in Figures 5 and 6.

One-way ANOVA revealed a significant difference across conditions for all the end-test values showed in Figure 5 ($p < 0.04$; $\eta_P^2 > 0.307$), except for $\dot{V}CO_2$ ($p = 0.467$; $\eta_P^2 = 0.172$). No differences across conditions were observed for the peak values showed in Figure 6 ($p > 0.079$; $\eta_P^2 < 0.246$), except for SpO_2 ($p < 0.001$; $\eta_P^2 = 0.964$) and aural temperature ($p < 0.001$; $\eta_P^2 = 0.791$). Follow-up comparisons are reported in Figures 5 and 6.

Relative isotime analysis. Figure 7 displays the associations between RPE and f_R, RPE and aural temperature, and f_R and aural temperature using averaged responses across participants derived from the relative isotime analysis. Bland-Altman within-subject correlation analysis revealed a large positive association between RPE and f_R ($p < 0.001$; $r = 0.85$), RPE and aural temperature ($p < 0.001$; $r = 0.59$), and f_R and aural temperature ($p < 0.001$; $r = 0.58$) (Figure 7).

Figure 8 shows the relationship between $\dot{V}_E$, V_T, and f_R using averaged responses across participants derived from the relative isotime analysis. Note that V_T plateaus at a higher value under the HYP condition, even when matched for $\dot{V}_E$ requirements.

DISCUSSION

The present study aimed to deepen our understanding of the mechanisms underlying the control of f_R and V_T during exercise by investigating the effects of hypoxia and hyperthermia on ventilatory responses to HIIT bouts performed to exhaustion. Our main findings are as follows: 1) f_R changes according to hypoxia- and hyperthermia-induced variations in exercise tolerance and is more closely associated with perceived exertion than with aural temperature; 2) on average, f_R shows a prompt and large increase and decrease during work and recovery phases, respectively, while V_T shows an opposite and smaller response in all conditions; meaning that f_R primarily drives the $\dot{V}_E$ response to the work-recovery alternations; and 3) V_T plateaus in all three conditions but reaches higher values under hypoxia, even when matched for $\dot{V}_E$. We suggest that these findings provide novel insight into the control of f_R and V_T during HIIT bouts, as discussed below.

Modulation of respiratory frequency

Our data suggest that, in young healthy individuals, the f_R response is strongly associated with the variations in exercise tolerance and perceived exertion during HIIT bouts, irrespective of the underlying mechanisms impairing exercise tolerance (Figs. 6C and 7A). Exposure to a hypoxic environment compromises oxygen delivery to the exercising muscles and the brain (Rasmussen et al. 2010; Goodall et al. 2012) and increases respiratory muscle fatigue (Verges et al. 2010). On the other hand, hyperthermia affects exercise tolerance via different mechanisms, including changes in cardiac output and systemic blood flow (González-Alonso et al. 2008) and a greater development of central fatigue (Nybo and Nielsen 2001). Although hyperthermia typically induces a preferential increase in f_R while hypoxia increases both f_R and V_T, the specific levels of hypoxia and hyperthermia selected

421 in this study determined a steeper increase in f_R in the HYP than in the HOT condition. HYP showed,
422 on average, a greater reduction in TTE compared to HOT, which was associated with a more rapid
423 increase in RPE.

424 The similar time course of f_R and RPE observed in the three conditions can be partially
425 explained by two interlinked mechanisms. First, central command is thought to be the sensory signal
426 to generate perceived exertion (Marcora 2009, 2019; Zénon et al. 2015), and its magnitude is
427 proposed to influence exercise tolerance (i.e., greater central command reduces exercise tolerance)
428 (Marcora 2009, 2019; Nicolò and Sacchetti 2023). The magnitude of central command likely reflects
429 complex, integrated activity across motor and premotor regions (including primary motor, prefrontal,
430 and supplementary motor areas), subcortical structures (e.g., hypothalamus and periaqueductal gray),
431 and interactions between limbic and cortical networks, all of which contribute to perceived exertion
432 (Thornton et al. 2001; Williamson 2010; Zénon et al. 2015; Robertson and Marino 2016; Marcora
433 2019).. Assuming RPE represents a surrogate of central command magnitude, the close association
434 between f_R and RPE suggests that central command plays a crucial role in modulating f_R during high-
435 intensity exercise (Nicolò and Sacchetti 2023) and determining exercise tolerance (Marcora 2009,
436 2019). This is in line with a series of findings showing an association between the activity of cortical
437 and subcortical areas and the f_R response in human and animal models (Thornton et al. 2001; Green
438 et al. 2007; Homma and Masaoka 2008; Hérent et al. 2023). The second mechanism involves direct
439 neural inputs to the respiratory muscles that are believed to contribute to the respiratory effort, which
440 is a component of the overall perceived exertion (Marcora 2019). These interlinked mechanisms may
441 help explain the observed association between f_R, RPE, and changes in exercise tolerance.

442 While other studies have found a close association between f_R and RPE during high-intensity
443 exercise (Nicolò and Sacchetti 2023), this is the first study showing that this association is stronger
444 than the association between f_R and aural temperature. Indeed, for the same f_R values, a substantially
445 higher aural temperature was observed in the HOT *versus* the HYP condition (Figure 6). Conversely,
446 similar values of RPE were found across conditions for a relatively broad range of f_R values (Figure

7). While the relationship between f_R and core body temperature was found to be stronger in previous studies (Hayashi et al. 2006; González-Alonso et al. 2023), the weaker association observed herein can be attributed to different factors. First, core body temperature shows a delayed response to variations in work rate (Todd et al. 2014), while f_R shows rapid fluctuations. Second, exercise was performed at higher intensities and f_R values than in previous studies (Hayashi et al. 2006; González-Alonso et al. 2023). Third, the peak value of aural temperature was substantially lower in HYP versus HOT, despite similar peak values of f_R observed across conditions. It is also worth noting that the time course of perceptual and physiological variables depends on the a-priori selected levels of hypoxia and hyperthermia. Therefore, whether the observed patterns persist during HIIT bouts at different levels of hypoxia or hyperthermia warrants further investigation.

Overall, our findings suggest that aural temperature is not the primary factor determining the increase over time in f_R observed in the three conditions, and that the magnitude of central command might have played a significant role in this phenomenon. From a practical perspective, the fact that the link between f_R and RPE is preserved in hypoxic and hyperthermic environments strengthens the importance of monitoring f_R during exercise. Indeed, athletes, military personnel, and rescue operators can frequently be exposed to hypoxic environments, hot environments, or both (Girard et al. 2024). Whereas exposure to such extreme environments reduces the capacity to exercise or perform specific physical tasks, monitoring the f_R response can provide important information on environment-induced changes in exercise tolerance.

The prompt f_R response within the work and recovery phases, which was preserved across different environmental conditions, further supports the notion that fast-changing inputs (e.g., central command and the fast component of group III and IV muscle afferent feedback), rather than slower ones (e.g., metabolic inputs and core body temperature), primarily modulate the f_R response during HIIT bouts (Nicolò et al. 2014, 2017). Indeed, it is unlikely that the prompt f_R response to the work-recover transition was primarily modulated by changes in metabolic inputs or body temperature, which show a slow kinetic response to work rate variations (Casaburi et al. 1977; Yamazaki et al.

1996; Todd et al. 2014; Taylor et al. 2014; Nicolò et al. 2018; Girardi et al. 2021, 2024). However, we cannot exclude that the fast component of group III and IV muscle afferent feedback (hereinafter muscle afferent feedback) may have influenced f_R. Indeed, abrupt, f_R-mediated changes in ventilation at exercise onset have been demonstrated in humans during passive exercise and decerebrated animal preparations following electrically-induced exercise (Agostoni and d'Angelo 1976; Bell et al. 2003; Girardi et al. 2021). Muscle afferent feedback can be activated by changes in local mechanical pressure, muscle temperature, tissue inflammation, and metabolic by-products of muscle contractions (e.g., $[H^+]$, $[K^+]$, and [La-]) (Forster et al. 2012). The repetitive and rapid changes in muscle contractions characterizing HIIT bouts can affect these factors, ultimately stimulating muscle afferent feedback. HYP and HOT conditions may have further increased the contribution of muscle afferent feedback to f_R modulation, secondary to the increase in metabolic and thermal stress, which likely accelerated muscular fatigue and limited exercise tolerance. Although controversial data exists (Iannetta et al. 2024), muscle afferent feedback may have a lower relative contribution to f_R modulation during high- versus moderate-intensity exercise (Dempsey et al. 2014), whereas central command shows a higher relative contribution to f_R during high-intensity exercise (Nicolò et al. 2018; Girardi et al. 2021). Besides, we cannot exclude that lung vagal afferent feedback, likely from mechanoreceptors (volume feedback), might have partially contributed to modulating the f_R response observed in this study (Guz et al. 1966a), although this speculation is currently unsupported. Further studies are required to better understand the role of muscle and lung afferent feedback in modulating f_R.

While the time course of [La-] resembled to some extent that of f_R in this study, we suggest caution in attempting to make any causal inference on the potential role of blood lactate on f_R modulation. Indeed, different experimental interventions/conditions show a large dissociation between f_R and blood lactate, including muscle glycogen depletion (Busse et al. 1991), hyperthermia during submaximal exercise (Hayashi et al. 2006), exercise-induced muscle damage (Davies et al. 2011), the comparison between continuous exercise and different HIIT protocols (Nicolò et al. 2014),

and incremental exercise in patients with McArdle's disease (Voduc et al. 2004). It is also unlikely that metabolic acidosis has contributed to modulating the fast f_R response to the abrupt changes during work-recovery alternation, given the transit time between the exercising muscles, pulmonary circulation, and main sites of chemosensitivity (e.g., carotid bodies and central chemoreceptors). Nevertheless, metabolic acidosis enhances peripheral chemoreceptor activity (Kumar and Prabhakar 2012), and thus differences in the degree of metabolic acidosis induced by the environmental conditions may have contributed to the steeper f_R response observed during HYP (Figure 6) and the higher f_R offset within the work-recovery cycle under the HYP condition (Figures 2 and 4) (Guz et al. 1966a, b; Honda 1985). Body temperature is another physicochemical factor that stimulates carotid body activity (Eyzaguirre and Zapata 1984; Kumar and Prabhakar 2012), but its relative contribution to the overall ventilatory thermal response is low, thus making its contribution to the f_R response observed during HOT unlikely (Fujii et al. 2008).

Modulation of tidal volume

A higher V_T was observed in the HYP condition compared to the other two conditions, even when matched for ventilatory requirements (Figure 8), which is consistent with previous studies showing higher V_T values under hypoxia. For instance, intermittent hypoxia at rest increases V_T, but not f_R, compared to a control condition (Mateika et al. 2004). Similarly, an increase in V_T under hypoxia has been observed during running at 65% of $\dot{V}O_{2peak}$ (Hill et al. 2020). The higher [La^-] found in HYP suggests that metabolic acidosis may have contributed to the increase in V_T under this condition. This finding, consistent with prior studies (Nicolò and Sacchetti 2023), suggests that metabolic inputs, such as low PaO_2 and higher metabolic acidosis, modulated the V_T response and contributed to the increase in V_T observed during the HYP condition.

Importantly, our findings extend previous observations on the increase in V_T under hypoxia to high-intensity exercise conditions where a V_T plateau is attained. It is commonly believed that the V_T plateau occurs as a result of mechanical constraints that prevent further V_T expansion, thus leading

to tachypnea (Sheel and Romer 2012). However, despite the attainment of a V_T plateau in the HOT and CON conditions, significantly higher V_T values were found in HYP, thus arguing against V_T being constrained by mechanical limitations in HOT and CON. Our findings are consistent with those of previous studies showing that the values at which V_T plateaus out during high-intensity exercise may increase if potent metabolic stimuli are superimposed upon exercise. For instance, studies have reported that the peak value of V_T reached during incremental exercise can be increased when breathing hypercapnic gas mixtures (Martin and Weil 1979; Clark et al. 1980; Fan et al. 2012). Similar results have been observed when adding an extra dead space at the mouth during exercise, which is known to increase alveolar P_{CO2} (Jones et al. 1971). These data suggest that the V_T plateau can be affected by the magnitude of metabolic inputs and that it may be a regulated rather than an obligatory phenomenon, even during high-intensity exercise. It is also worth noting that the V_T plateau under CON, HYP, and HOT conditions occurred at values well below the V_T peak observed during the ramp-incremental test (V_{Tpeak} 3.15 ± 0.63 L), suggesting that higher V_T values were possible but were not reached.

Data suggest that the V_T plateau may be a regulated phenomenon even when mechanical limitations occur. Guenette and colleagues (Guenette et al. 2007) suggested that EFL is a physiological condition in which compensatory strategies are available rather than an all-or-none physiological condition. For example, when EFL appears, a small reduction in V_T, with concomitant increments in f_R, was observed and proposed to limit further EFL [see Figure 2 by (Guenette et al. 2007)]. In this perspective, the V_T response during exercise is continuously regulated by the ventilatory control system rather than mechanically pre-determined. This phenomenon is certainly interesting, even though the underlying mechanisms remain to be determined.

The fact that V_T was fine-tuned by the ventilatory control system rather than mechanically constrained in this study is further suggested by the evaluation of the V_T time course to work-recovery alternation. V_T did not show fixed values at the plateau (Figure 8, V_T plateaus at a higher value during HYP), but it was slightly lower during the work phase and slightly higher during the rest phase (see

Figures 2 and 4). The opposite response to that of f_R may suggest a mechanistic link between the two components of pulmonary ventilation. While the ventilatory control system regulates f_R and V_T to match alveolar ventilation with metabolic requirements, it is plausible that V_T is modulated based on f_R values to achieve this goal. This is consistent with the notion that f_R is primarily modulated by fast inputs, while V_T by the magnitude of metabolic inputs (Nicolò and Sacchetti 2023). The alternative explanation – i.e. f_R depends on V_T – is less convincing as discussed in a recent review (Nicolò and Sacchetti 2023). Indeed, the fluctuations in f_R to work-recovery alternation modify the time available for lung inflation, which may, at least in part, account for the reduction in V_T as f_R increases and for its increase as f_R decreases. On the other hand, the breathing cycle results from neural and mechanical factors (Younes and Remmers 1981), and it is not possible to exclude that the f_R–V_T relationship was partially influenced by mechanical factors in this study. We did not assess factors such as lung volumes, thoracic and intra-abdominal pressure changes, drive to breathe, static and dynamic pressure-volume relationships of the respiratory system, and work of breathing, which all may play an important role in modulating the relationship between f_R and V_T. Thus, the exact mechanisms underlying this relationship remain elusive, warranting further investigation.

Study limitations, methodological considerations, and future directions

The use of high-intensity work–recovery bouts was intended to help differentiate fast-changing from slow-changing inputs contributing to the f_R, V_T and $\dot{V}_E$ responses, with HYP and HOT further manipulating these inputs. However, it is important to note that no direct measures of the proposed drivers of ventilation—including chemoreflex sensitivity, central command, respiratory system mechanics, blood gases, serum potassium, or acid–base balance—were obtained. As such, mechanistic interpretations are presented as physiologically plausible hypotheses rather than definitive causal conclusions. Further studies involving direct assessment of the proposed mechanisms are needed.

No formal kinetic modelling of ventilatory and pulmonary gas exchange dynamics was performed. The behavior of f_R, V_T, and $\dot{V}_E$, as well as pulmonary gas exchange responses, during HIIT bouts is known to be markedly non-linear, making it difficult to apply conventional linear, mono- or multi-exponential models. To our knowledge, no established model currently exists that adequately captures breathing pattern dynamics during intermittent high-intensity exercise in a manner that allows direct inference of underlying physiological mechanisms dictating response dynamics. Accordingly, we avoided implementing a formal modelling approach. To address this limitation, we provided a detailed characterization of the ventilatory responses, including second-by-second data, 10-s bin averages across the work-recovery cycle, and 60-s bin averages to describe the overall time course independent of fluctuations induced by work-recovery alternations.

A further limitation concerns the use of aural temperature to estimate core body temperature. Infrared aural thermometry reflects a combination of tympanic and aural canal temperature, and its approximation to core body temperature depends on local blood flow (Kirk et al. 2009; Masè et al. 2021). Because facial and ear canal perfusion may vary with exercise duration and ambient heat exposure, the difference between aural and core body temperature cannot be assumed to remain constant between conditions. Accordingly, aural temperature should be interpreted as an index of peripheral thermal strain rather than a direct surrogate of core body temperature. Moreover, the measurement site (e.g., skin, esophageal, rectal, or aural) is known to influence estimates of core body temperature and its response to exercise and environmental stress (Taylor et al. 2014). Further studies involving direct assessment of core body temperature (esophageal or rectal) are needed to confirm the present findings.

Breath-by-breath pulmonary gas exchange was measured at the mouth level without accounting for inter-breath changes in lung gas stores (due to the unavailability of raw gas flow, O_2, and CO_2 traces required for alveolar correction). During step-wise high-intensity work–recovery alternations, abrupt changes in ventilation and lung volumes transiently affect lung gas stores, such that the O_2 and CO_2 volumes measured at the mouth over a given breath may not accurately reflect

602 true alveolar-capillary gas transfer (Capelli et al. 2011; Girardi et al. 2023). Future studies accounting
603 for dynamic lung gas store variations would allow a more accurate characterization of alveolar gas
604 exchange during high-intensity intermittent exercise. Such investigations would also benefit from the
605 use of standardized definitions of the breathing cycle (Girardi et al. 2025).
606 Future studies should investigate whether matching exercise tolerance between HYP and HOT
607 conditions by adjusting the severity of hypoxia and hyperthermia results in similar ventilatory
608 responses. Such an approach would help isolate the effects of the environmental stressors from those
609 of exercise duration and provide a more direct comparison of their influence on breathing pattern
610 regulation. In addition, inclusion of a combined hypoxia–hyperthermia condition would allow
611 evaluation of potential interactions between these environmental stressors and determine whether
612 their effects on ventilatory responses are, for example, additive or hyperadditive.
613

614 **CONCLUSION**

615 We found that f_R reflects perceived exertion irrespective of the environmental manipulation tested
616 and is generally associated with hypoxia- and hyperthermia-induced changes in exercise tolerance
617 during 30-30s work-recovery HIIT bouts. f_R responds promptly to work-recovery alternations,
618 primarily driving the well-established increases and decreases in $\dot{V}_E$ during HIIT work-recovery
619 cycles. The stronger association between RPE and f_R compared to aural temperature suggests that the
620 typical increase in f_R observed during high-intensity exercise is more closely related to RPE than body
621 temperature, suggesting that the magnitude of central command plays an important role in this
622 phenomenon. Overall, the present findings suggest that fast-changing inputs (e.g., central command
623 and the fast component of muscle afferent feedback), rather than slow-changing inputs (e.g., blood
624 O_2 and CO_2, and pH), modulate the f_R response to work-recovery alternations. V_T, on the other hand,
625 shows a smaller and opposite response to abrupt changes in work rate during HIIT and appears to be
626 fine-tuned based on f_R levels and the magnitude of metabolic inputs. Even when matched for $\dot{V}_E$
627 requirements, V_T plateaus at higher values during HYP versus CON and HOT conditions, and its

plateau value remained lower than that reached during the ramp-incremental test. This suggests that the V_T plateau is a regulated rather than a mechanically constrained phenomenon, at least under some of the conditions tested in this study (i.e. HOT and CON).

Author contributions

Conception or design of the work: M.G., A.N., S.M.M. and M.S. Acquisition, analysis or interpretation of data for the work: M.G., AN., I.B., F.F., J.D., S.M.M., M.S.. M.G. and A.N. wrote the first draft of the manuscript. All the Authors revisited the work critically for important intellectual content. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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Competing interest

The authors declare that they have no conflict of interest

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800

Figure captions

Figure 1. Number of bouts completed at exhaustion during CON, HOT and HYP conditions. Letters indicate the results of follow-up tests. (a) HYP vs. HOT ($P < 0.05$); (b) HYP vs. CON ($P < 0.05$); (c) HOT vs. CON ($P < 0.05$).

Figure 2. Second-by-second responses of respiratory frequency (f_R, top panels), tidal volume (V_T, middle panels), and minute ventilation ($\dot{V}_E$, bottom panels) during high-intensity interval bouts across conditions (CON, HYP, HOT). Data are presented as mean (blue, red, and back lines) ± SD (shaded areas). The time course is shown for four phases: first isotime bouts (first two work–recovery cycles), middle isotime bouts (two cycles performed at ~50% of the shortest TTE), last isotime bouts (last two cycles of the shortest TTE and corresponding cycles in the other conditions), and final bouts (last two completed cycles in each condition irrespective of total duration). Vertical dashed lines indicate transitions between work and recovery phases.

Figure 3. Second-by-second responses of pulmonary oxygen uptake ($\dot{V}O_2$, top panels), carbon dioxide output ($\dot{V}CO_2$, middle panels), and heart rate (HR, bottom panels) during high-intensity interval bouts across conditions (CON, HYP, HOT). Data are presented as mean (blue, red, and back lines) ± SD (shaded areas). The time course is shown for four phases: first isotime bouts (first two work–recovery cycles), middle isotime bouts (two cycles performed at ~50% of the shortest TTE), last isotime bouts (last two cycles of the shortest TTE and corresponding cycles in the other conditions), and final bouts (last two completed cycles in each condition irrespective of total duration). Vertical dashed lines indicate transitions between work and recovery phases.

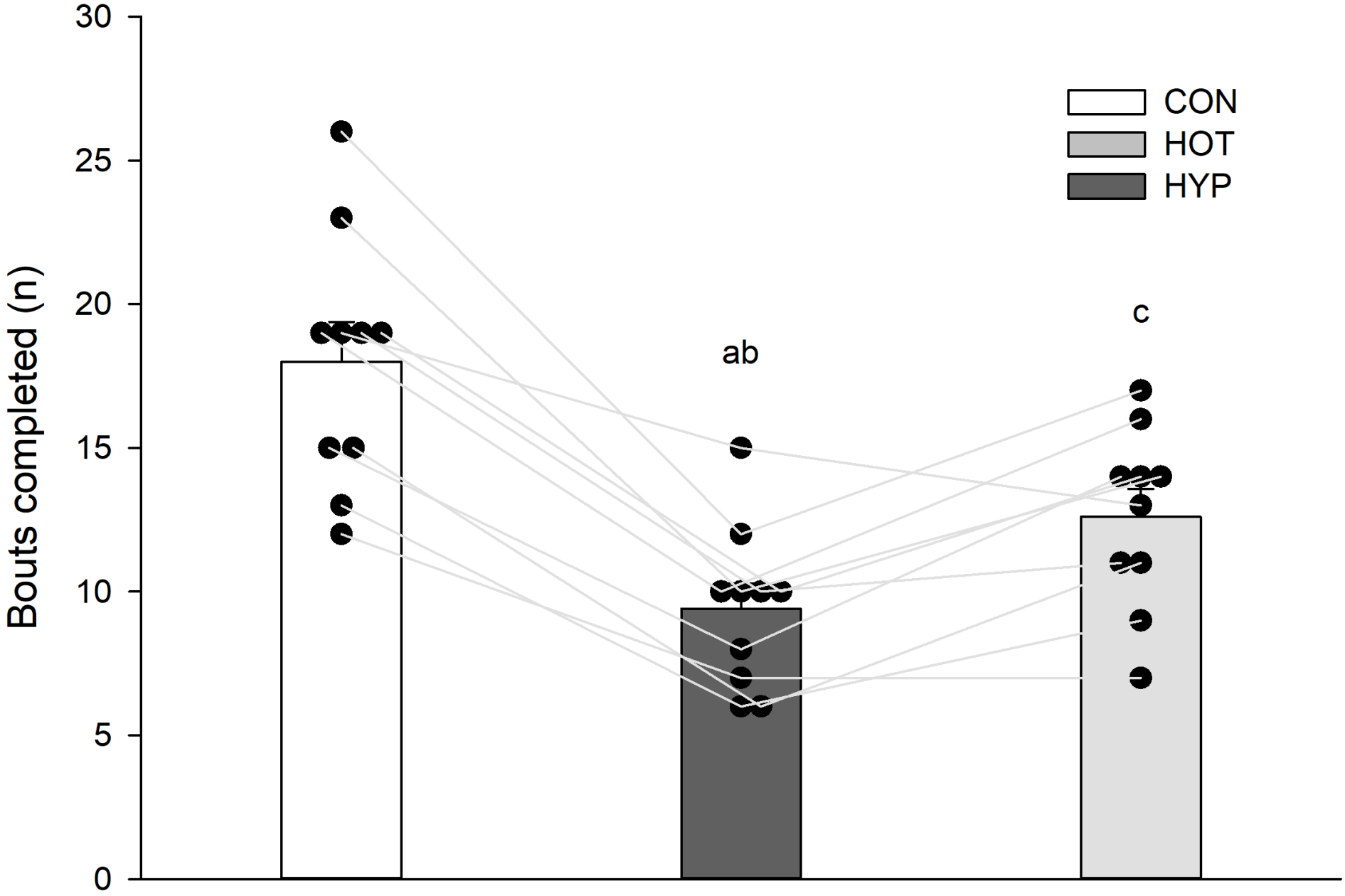
Figure 4. Work-recovery cycle response of respiratory frequency (A), heart rate (B), tidal volume (C), carbon dioxide output (D), minute ventilation (E), oxygen uptake (F), and end-tidal partial pressure of carbon dioxide (G) during the three experimental conditions. † Significant main effect of time ($P < 0.05$). § Significant main effect of condition ($P < 0.05$). # Significant interaction ($P < 0.05$). Letters indicate the results of follow-up tests. (a) HYP vs. HOT ($P < 0.05$); (b) HYP vs. CON ($P < 0.05$); (c) HOT vs. CON ($P < 0.05$).

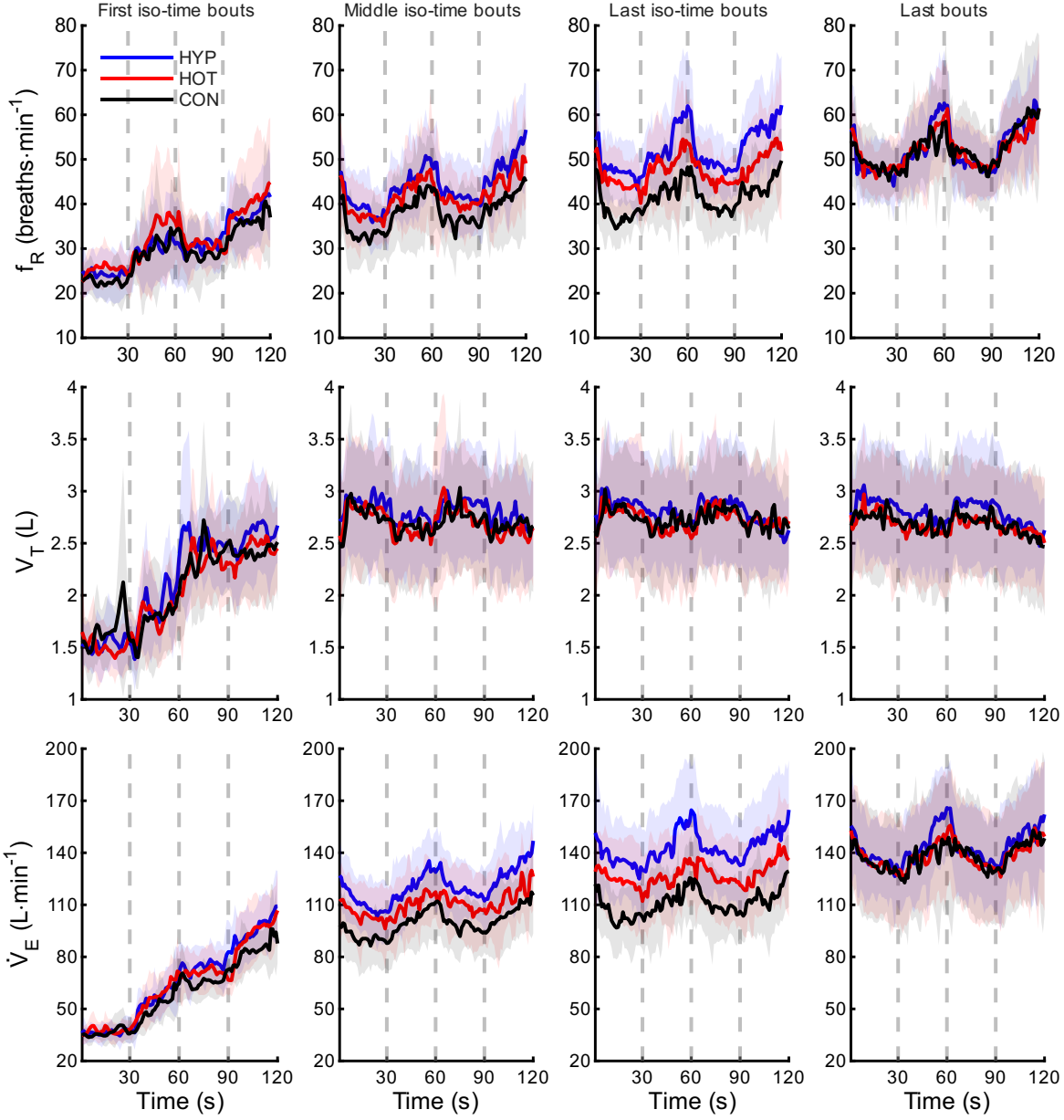
Figure 5. Time course of oxygen uptake (A), carbon dioxide output (B), heart rate (C), end-tidal partial pressure of carbon dioxide (D), and blood lactate (E) during the HYP (filled circles), HOT (open circles) and CON (open triangles) conditions. † Significant main effect of time ($P < 0.05$). § Significant main effect of condition ($P < 0.05$). # Significant interaction ($P < 0.05$). Letters indicate the results of follow-up tests. (a) HYP vs. HOT ($P < 0.05$); (b) HYP vs. CON ($P < 0.05$); (c) HOT vs. CON ($P < 0.05$).

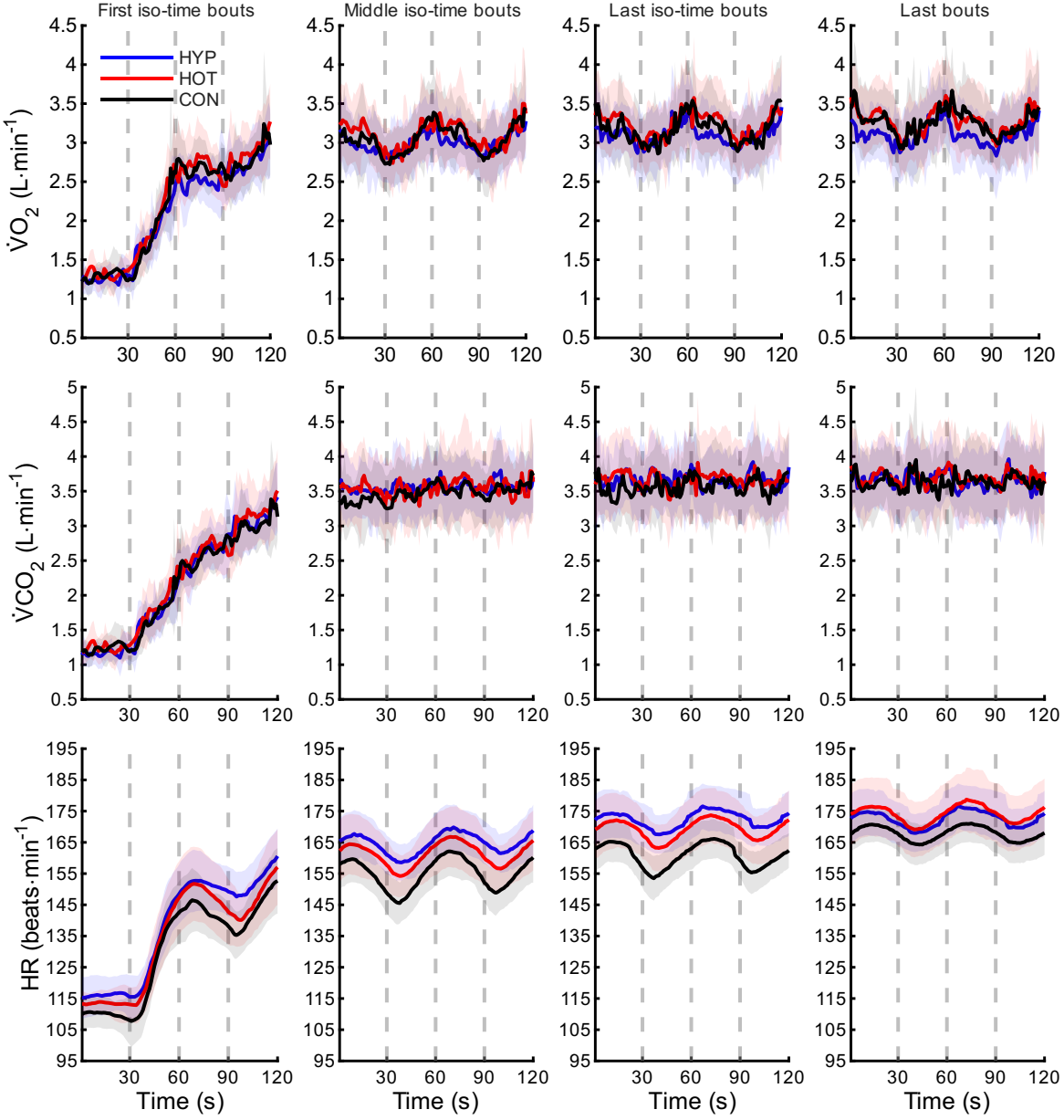
Figure 6. Time course of minute ventilation (A), SpO2 (B), respiratory frequency (C), rating of perceived exertion (D), tidal volume (E) and aural temperature (F) during the HYP (filled circles), HOT (open circles) and CON (open triangles) conditions. † Significant main effect of time ($P < 0.05$). § Significant main effect of condition ($P < 0.05$). # Significant interaction ($P < 0.05$). Letters indicate the results of follow-up tests. (a) HYP vs. HOT ($P < 0.05$); (b) HYP vs. CON ($P < 0.05$); (c) HOT vs. CON ($P < 0.05$).

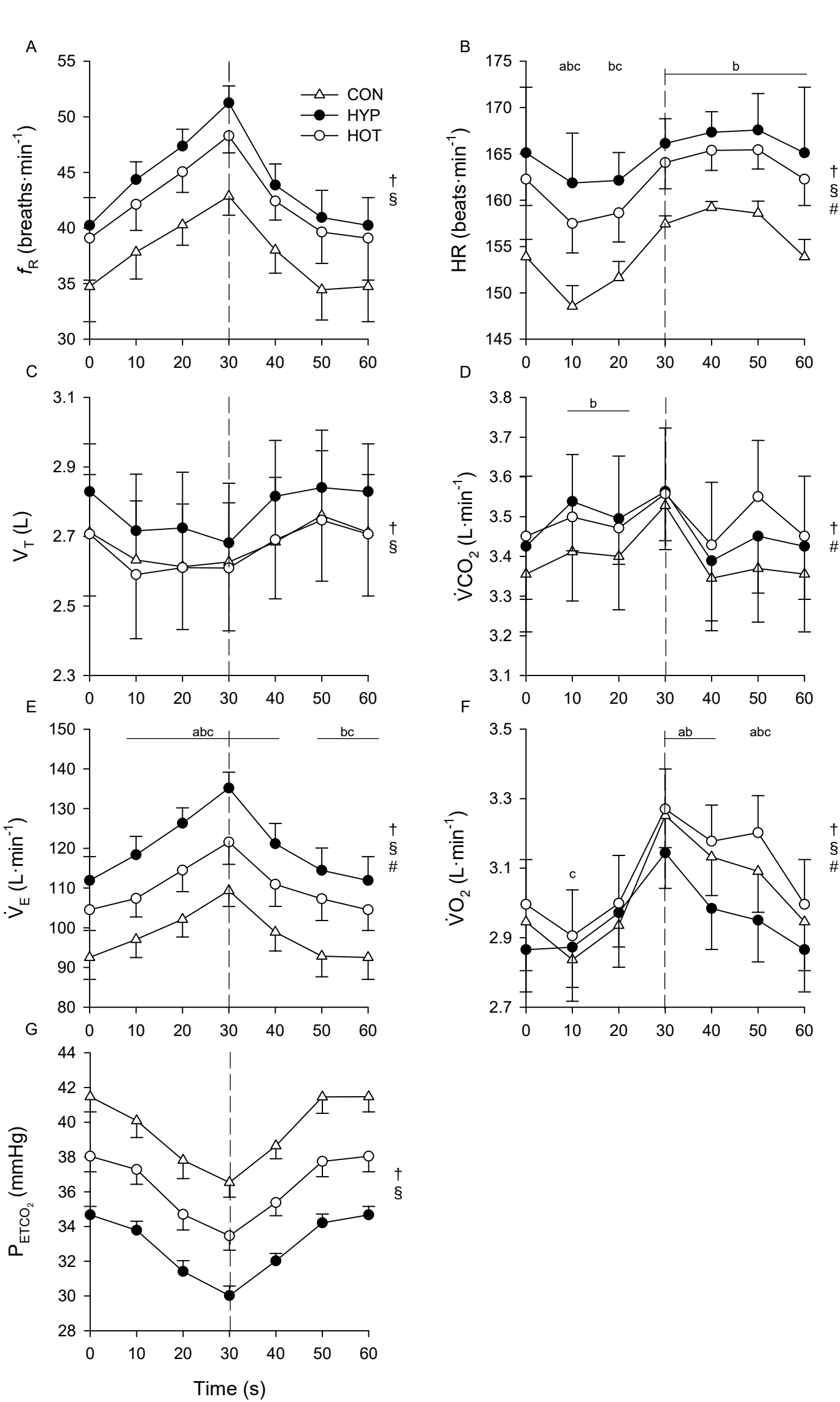
Figure 7. Correlation between rating of perceived exertion (RPE) and respiratory frequency (f_R) (A), aural temperature and f_R (B) and aural temperature and RPE (C). Each symbol represents the mean value of all participants every 10% of the TTE during the HYP (filled circles), HOT (open circles) and CON (open triangles) conditions.

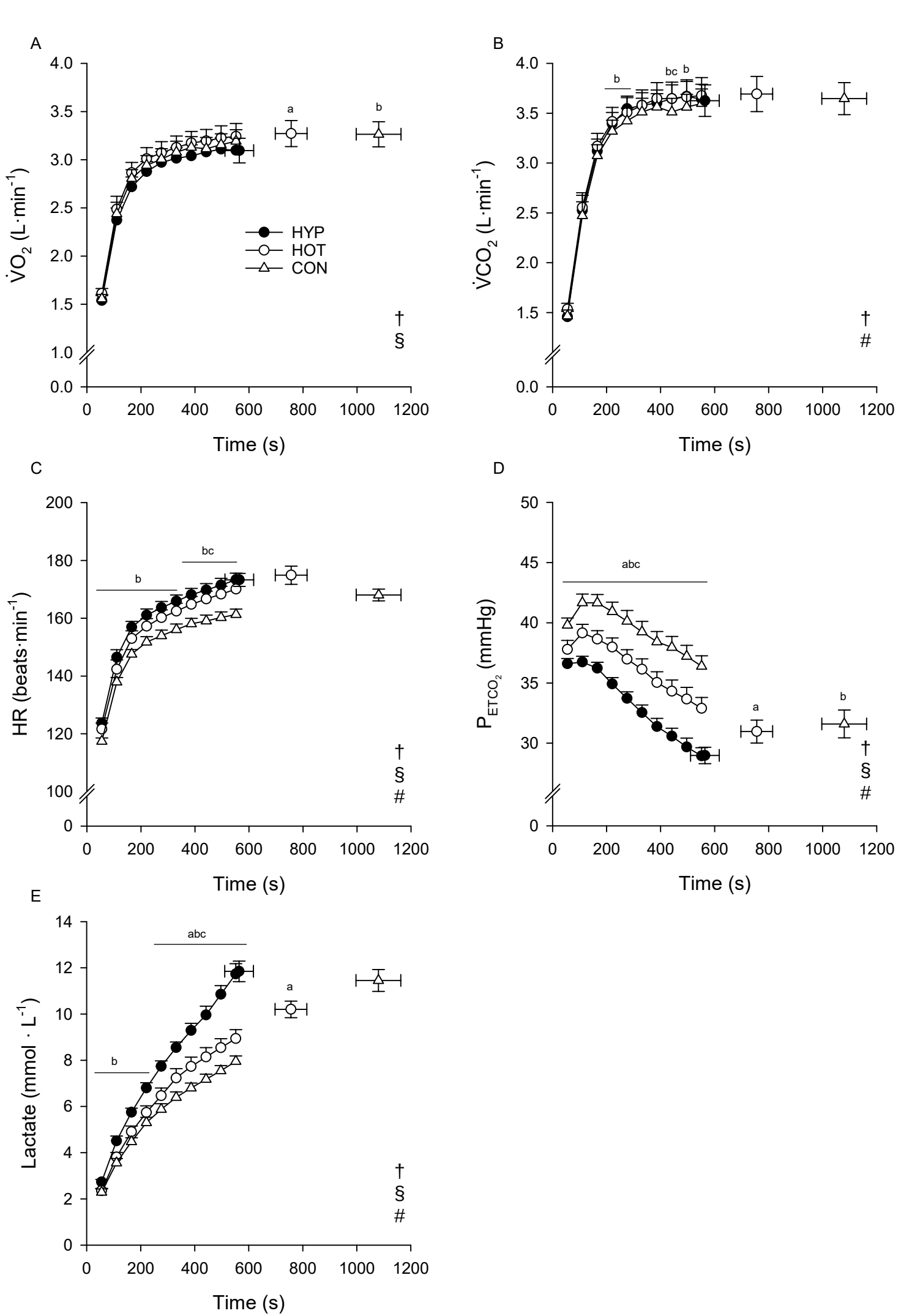
Figure 8. 'Hey plot' illustrating the relationship between minute ventilation ($\dot{V}_E$), tidal volume (V_T), and respiratory frequency (f_R) during the HYP (filled circles), HOT (open circles) and CON (open triangles) conditions. Dashed grey lines represent iso-f_R values ranging from 10 to 60 breaths per minute (bpm).

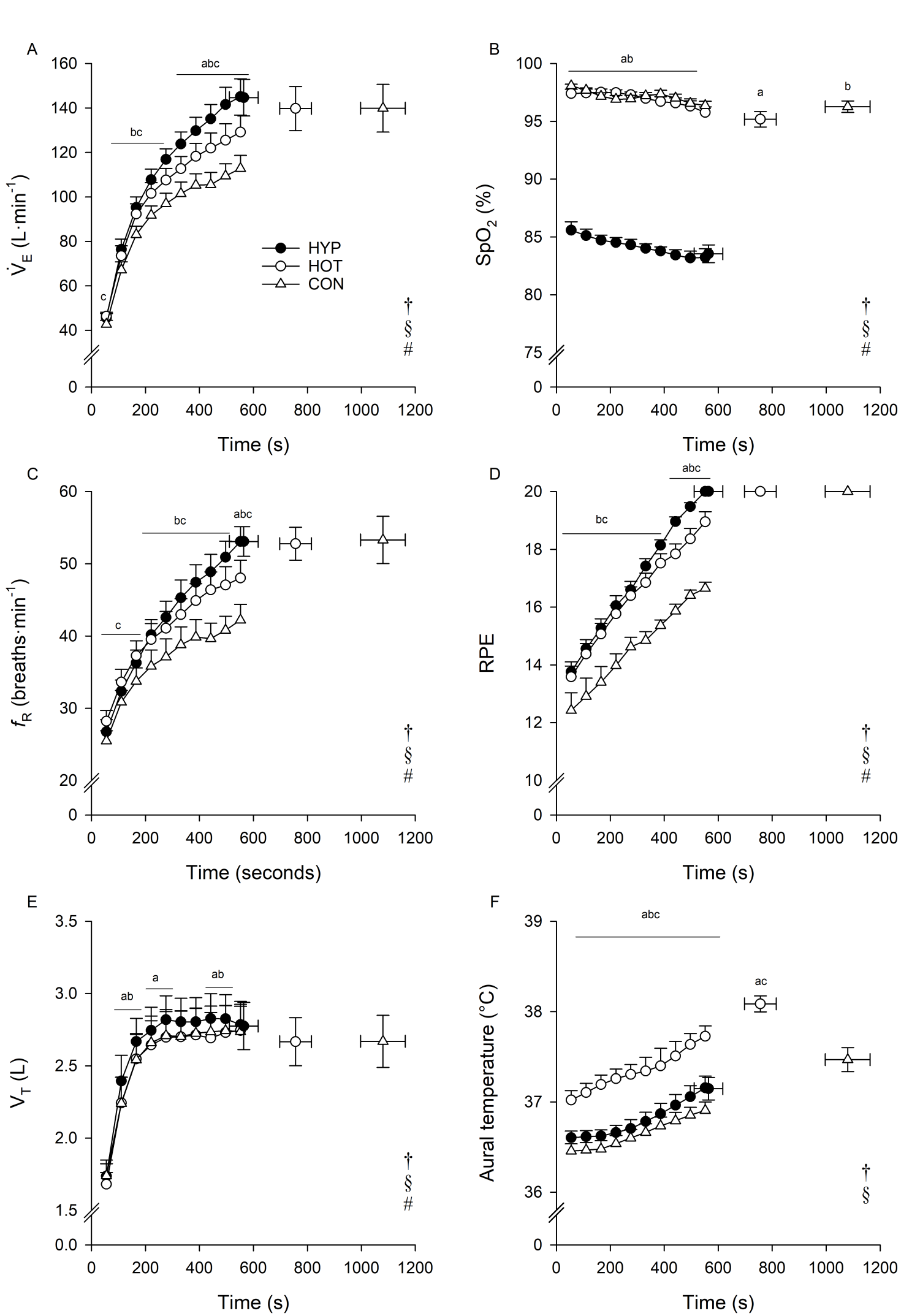

30
25
20
15
10
5
0
Bouts completed (n)
CON
HOT
HYP
ab
c

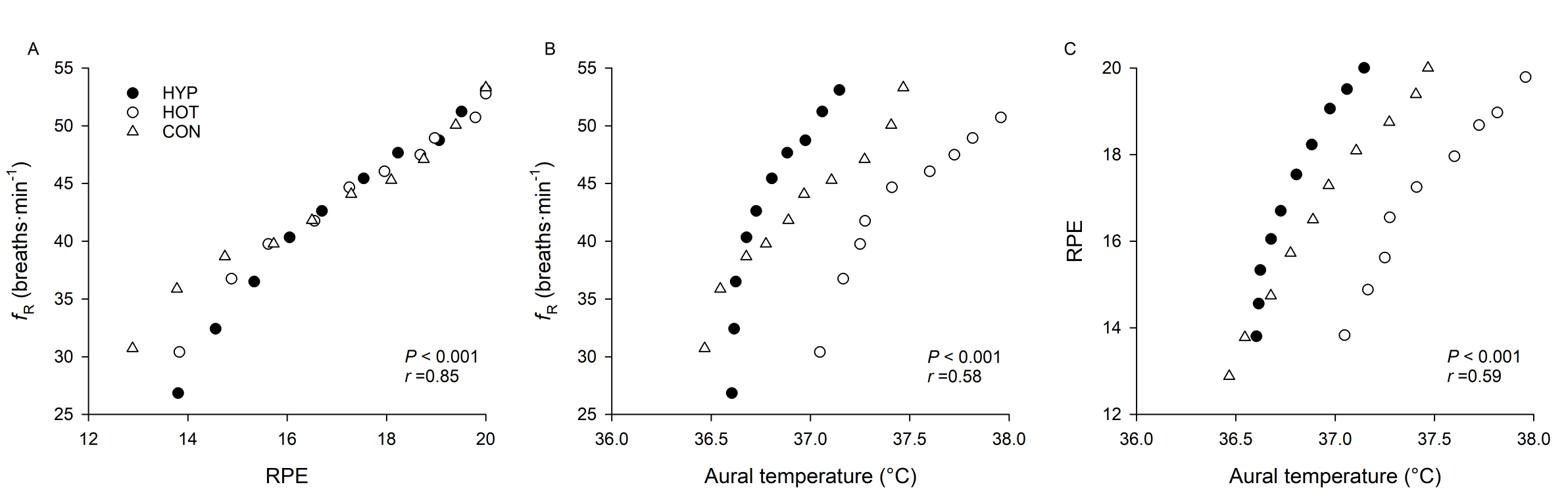

First iso-time bouts
Middle iso-time bouts
Last iso-time bouts
Last bouts
HYP
HOT
CON
f_R (breaths·min^{-1})
V_T (L)
$\dot{V}_E$ (L·min^{-1})
Time (s)

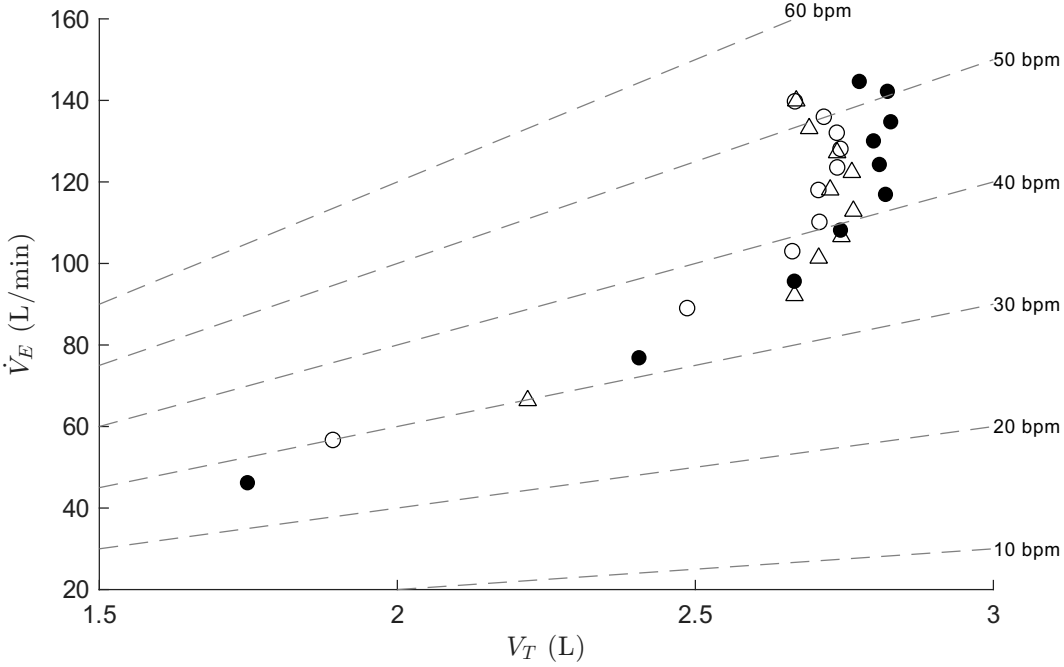

First iso-time bouts
Middle iso-time bouts
Last iso-time bouts
Last bouts
HYP
HOT
CON
V̇O2 (L·min-1)
V̇CO2 (L·min-1)
HR (beats·min-1)
Time (s)


A
B
C
D
E
F
G
CON
HYP
HOT
f_R (breaths·min^{-1})
HR (beats·min^{-1})
V_T (L)
$\dot{V}CO_2$ (L·min^{-1})
$\dot{V}_E$ (L·min^{-1})
$\dot{V}O_2$ (L·min^{-1})
P_{ETCO_2} (mmHg)
abc
bc
b
ab
c
†
§
#
Time (s)


A
B
C
D
E
HYP
HOT
CON
Time (s)
$\dot{V}O_2$ (L·min^{-1})
$\dot{V}CO_2$ (L·min^{-1})
HR (beats·min^{-1})
P_{ETCO_2} (mmHg)
Lactate (mmol · L^{-1})
a
b
bc
abc
†
§
#


A
B
C
D
E
F
HYP
HOT
CON
Time (s)
Time (seconds)
$\dot{V}_E$ (L·min^{-1})
SpO_2 (%)
f_R (breaths·min^{-1})
RPE
V_T (L)
Aural temperature (°C)
a
ab
abc
ac
b
bc
c
†
§
#


A
B
C
HYP
HOT
CON
f_R (breaths·min^{-1})
RPE
Aural temperature (°C)
P < 0.001
r =0.85
P < 0.001
r =0.58
P < 0.001
r =0.59


160
140
120
100
80
60
40
20
$\dot{V}_E$ (L/min)
1.5
2
2.5
3
V_T (L)
60 bpm
50 bpm
40 bpm
30 bpm
20 bpm
10 bpm