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Effects of post-exercise cooling on heart rate recovery in normotensive and hypertensive men

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SUMMARY

Background: Post-exercise heart rate recovery (HRR) is determined by cardiac autonomic restoration after exercise and is reduced in hypertension. Post-exercise cooling accelerates HRR in healthy subjects, but its effects in a population with cardiac autonomic dysfunction, such as hypertensives (HT), may be blunted. This study assessed and compared the effects of post-exercise cooling on HRR and cardiac autonomic regulation in HT and normotensive (NT) subjects. Methods: Twenty-three never-treated HT (43±8 ys) and 25 NT (45±8 ys) men randomly underwent two exercise sessions (30 min of cycling at 70%VO_{2peak}) followed by 15 min of recovery. In one randomly allocated session, a fan was turned on in front of the subject during the recovery (cooling), while in the other session, no cooling was performed (control). HRR was assessed by heart rate reductions after 60 (HRR60s) and 300s (HRR300s) of recovery, short-term time constant of HRR (T30), and the time constant of the HRR after exponential fitting (HRRτ). HRV was assessed using time- and frequency-domain indices. Results: HRR and HRV responses in the cooling and control sessions were similar between the HT and NT. Thus, in both groups, post-exercise cooling equally accelerated HRR (HRR300s = 39±12 vs. 36±10 bpm, p≤0.05) and increased post-exercise HRV (lnRMSSD = 1.8±0.7 vs. 1.6±0.7 ms, p≤0.05). Conclusion: Differently from the hypothesis, post-exercise cooling produced similar improvements in HRR in HT and NT men, likely by an acceleration of cardiac parasympathetic reactivation and sympathetic withdrawal. These results suggest that post-exercise cooling equally accelerates HRR in hypertensive and normotensive subjects.

Keywords: heart rate variability, thermoregulation, autonomic nervous system, parasympathetic, hypertension, blood pressure

INTRODUCTION

Post-exercise heart rate recovery (HRR) is a non-invasive tool to assess cardiac autonomic function (Cole*, et al.* 1999; Imai*, et al.* 1994). HRR presents a biphasic behavior, with an initial fast decay determined by parasympathetic reactivation (Imai, et al. 1994) and a second slow decay that is determined by sympathetic withdrawal and further parasympathetic reactivation (Perini*, et al.* 1989).

A reduced HRR reflects autonomic dysfunction, and has been observed in several chronic diseases (Peçanha*, et al.* 2014) and is associated with poor prognosis (Cole, et al. 1999). Several strategies have been used to improve cardiac autonomic restoration after exercise and, thus, to accelerate HRR (Al Haddad*, et al.* 2010; de Oliveira Ottone*, et al.* 2014; Leicht*, et al.* 2009). Of note, Leicht et al. (2009) observed a faster HRR when subjects were exposed to a fan with water spray or ice pack application during the post-exercise period. Likewise, Al Haddad et al. (2010) observed similar effects using cold water immersion. These strategies are based on the recognized relationship between the autonomic control of HRR and thermoregulation (e.g., heat stress-induced impairments in HRR) (Peçanha*, et al.* 2017b). It should be noted, however, that these cooling studies were conducted with healthy subjects, and little is known regarding the effects of such strategies on HRR and its regulation in subjects with cardiovascular disorders usually associated with autonomic dysfunction and reduced HRR.

Hypertension is a highly prevalent cardiovascular disease characterized by the presence of autonomic dysfunction, i.e., a decrease in parasympathetic and an increase in sympathetic nerve activities (Mancia & Grassi 2014). This autonomic imbalance culminates with reduced HRR and increased cardiovascular risk in hypertensive (HT) when compared to normotensive (NT) subjects (Erdogan*, et al.* 2011). In light of the benefits of post-exercise cooling on HRR in healthy subjects, one could hypothesize that this strategy could be as effective in accelerating HRR in HT.

However, previous evidence suggests negative cardiovascular responses to cooling in hypertension. Greaney et al. (Greaney*, et al.* 2017) reported exaggerated increases in sympathetic nerve activity during whole body cooling in HT, and this response may mitigate the potential beneficial effect of post-exercise cooling on HRR in HT, which to the best of our knowledge has not been investigated yet.

Thus, this study was designed to assess the effects of post-exercise cooling with a fan on HRR in never-treated HT and normotensive (NT) men. The hypothesis was that the acceleration of HRR promoted by cooling would be blunted in HT compared with NT. To better clarify the mechanisms underlying the responses of HRR to post-exercise cooling in NT and HT, the effects of such a strategy on post-exercise heart rate variability (HRV) and blood pressure (BP) were also evaluated.

MATERIAL AND METHODS

Subjects

The participants were middle-aged (30–60 years) and physically inactive HT (systolic/diastolic BP between 140/90 and 159/99 mmHg) and NT (i.e. systolic/diastolic BP <120/80 mmHg) men. The exclusion criteria included smoking; established cardiovascular diseases; body mass index ≥35 kg/m^2; use of medications that could directly affect cardiovascular responses to exercise; and abnormal resting or exercise electrocardiogram. Additionally, HT subjects had never been treated with antihypertensive drugs and had no target organ damage or secondary hypertension. After a detailed explanation of the experimental procedures, subjects provided their written informed consent. This study was conducted in accordance with the Declaration of Helsinki and was approved by local Institutional research ethics committee (281.905/2013).

Preliminary assessment

Health status was investigated through a detailed anamnesis. Resting BP was measured by a mercury sphygmomanometer (Uniteq, São Paulo, Brazil), three times after 5-min seated rest in two distinct visits to the laboratory (Chobanian*, et al.* 2003). All HT subjects also underwent the routine screening of the Hypertension Unit of the General Hospital of the University of São Paulo to detect target organ damage, secondary hypertension, and/or other clinical conditions that preclude exercise participation.

On a separate day, subjects performed a maximal cardiopulmonary exercise test with assessments of resting and exercise electrocardiograms. This test was performed on a cycle ergometer (Computrainer, RacerMate, Seattle, USA) with an initial workload of 50 Watts and increments of 20 Watts every 3 min until volitional exhaustion. Immediately after exercise, workload was reduced to 50 Watts, and the subjects completed a 5 min recovery. During the test, ventilatory variables were continuously measured using a metabolic cart (CPX-Ultima, Medical Graphics Corporation, Minnesota, USA). Peak oxygen consumption (VO_{2peak}) and HR (HR_{peak}) were determined by the maximal values attained at the end of exercise (average of 30s). HRR was assessed through the calculation of the HRR60s index (i.e. HR_{peak} – HR at 60s of recovery) (Peçanha, et al. 2014).

Experimental protocol

All subjects randomly underwent two experimental sessions conducted in the morning of two separate days, with a minimum interval of 48h between sessions. Temperature and humidity of the room were kept constant across the sessions (20–22°C and 75–80%). Subjects were instructed to arrive at the laboratory in a fasted state, and to avoid alcohol and exercise for 24h and caffeine ingestion for 12h prior to the sessions. In each session, they received a standardized meal (two 25g cereal bars and 50ml of juice) and experiment began 30min after the ingestion of the meal. The experimental sessions started with resting measurements in the seated position for

10min and, then the subjects were submitted to 30min of cycle ergometer exercise (Computrainer, RacerMate, Seattle, USA) at 70%VO_{2peak}. After exercise, subjects immediately stopped pedaling and remained seated on the cycle ergometer for a 15min recovery. In one of the sessions, an industrial fan was turned on in front (~1 meter) of the subjects during all of the recovery period (cooling session), while in the other session, the recovery was performed without fanning (control session).

Experimental Measurements

T_c was measured in 10-s intervals via a telemetric temperature pill system (CorTemp®, HQInc., Palmetto, USA) ingested, at least, 2h before the experiments (Byrne & Lim 2007). HR was continuously measured using a three-lead electrocardiogram (EMG System, São Paulo, Brazil) and beat-by-beat BP was obtained using finger photoplethysmography (Finometer, FMS, Arnhem, The Netherlands). These signals were continuously acquired online (Windaq, Dataq Instruments, Ohio, USA; 500 Hz/channel). Mean T_c, HR and SBP were calculated for rest (5–10 min), exercise (25–30min), and immediate (0–5min) and late (10–15min) recovery periods. In addition, VO_2 was continuously measured during exercise with a metabolic cart (CPX-Ultima, Medical Graphics Corporation, Minnesota, USA).

Cardiovascular Autonomic Assessment

Preprocessing procedures. HR signals were exported to HeartScope software (A.M.P.S. LLC, New York, USA) for the generation of RR intervals (RRi) time series. These series were visually inspected and occasional misdetections were corrected. Likewise, ectopic beats were identified and replaced with interpolated RRi values (<2% of the total signal).

Heart Rate Recovery Analysis. HRR was assessed as previously reported (Peçanha, *et al.* 2016). The following indices were calculated: a) HRR60s and HRR300s, i.e. the absolute differences between peak HR (mean of the last 60s of exercise) and the HRs obtained, respectively, at 60 and 300s of recovery; b) T30, i.e. the short-term time

constant of HRR obtained from the negative reciprocal of the linear regression line between the log-transformed HR and the first 30 s of recovery (Imai et al., 1994) and; c) HRRt, i.e. the time-constant of HRR after exponential fitting of the HR during the entire 300s of recovery.

Heart Rate Variability. HRV was assessed at rest, during immediate recovery (0–5min) and during late recovery (10–15min) after exercise. For the immediate recovery period, given the non-stationary behavior of RRi, the assessment of HRV was performed using a time-varying approach (Goldberger*, et al.* 2006). Firstly, the RRi time series of recovery was filtered using a median filter operation. Then, HRV was assessed through the calculation of RMSSD (root mean square of successive differences in RRi) and RMS (root mean square residual of RRi) indices, on successive non-overlapped 30s segments, during the entire 5min immediate recovery.

At rest and late recovery, given the relative stationarity of the cardiovascular signals, HRV was assessed via spectral analysis using the Heart Scope software (A.M.P.S. LLC, New York, USA) and following international Task Force recommendations(1996). The power spectral density analyses of RRi (250-300 beats) were performed using the autoregressive method and the spectral components were calculated via the Levinson-Durbin recursion employing Akaike's criteria for choosing the order of the model (Malliani*, et al.* 1991). Low- (LF: 0.04–0.15Hz) and high-frequency (HF: 0.15–0.4Hz) components of RRi variability were expressed in normalized units (Task-Force 1996).

Statistical Analysis

Based on an expected Cohen's d effect size of 0.87 of post-exercise cooling on HRR60s (Al Haddad, et al. 2010), the sample size required to achieve a power of 90% and an α level of 5% was 32 subjects (i.e. 16 subjects per group) (G*Power v. 3.1.9.2, Universität Kiel, Germany).

Following the use of the Shapiro-Wilk test, the hypothesis of normality was rejected only for RMS and RMSSD. Thus, these variables were natural log-transformed (ln) and normality was achieved. T-tests were employed for comparing baseline characteristics between groups. Two- (group vs. session) or three-way (group vs. session vs. time) mixed ANOVAs were used for comparisons of responses between NT and HT groups. When a main effect or an interaction was significant, post-hoc comparisons were made using the Newman-Keuls test. Values of $p \leq 0.05$ were considered significant. Data are presented as mean ± 1 standard deviation.

RESULTS

Forty-eight men (HT=23 and NT=25) took part in the study. Their baseline characteristics have already been presented in a previous publication which assessed the influence of the metaboreflex on HRR (Peçanha, et al. 2016). The HT and NT groups were similar regarding age, body mass index, VO_{2peak} and HR_{peak} (Table 1; $p>0.05$ for all comparisons). By design, SBP and DBP were higher in the HT vs. NT group (Table 1; $p<0.01$ for both comparisons). Finally, HRR60s after the maximal test was reduced in the HT group (23±10 vs. 18±6 s; $p=0.04$).

Responses to exercise in the experimental sessions were also similar between groups. There were no differences in HR or VO_2 between groups and sessions. In both groups and sessions, exercise intensity corresponded to approximately 70% of VO_{2peak} (Table 2).

>>>>>>>>>>>>>>>>>>>>>>>>>>>TABLE 1>>>>>>>>>>>>>>>>>>>>>>>>>>>

Core Temperature and Hemodynamics

Figure 1 displays T_C, HR and SBP responses throughout the experimental protocol. There were no differences between the groups in T_c and HR at any time point,

whereas SBP was consistently greater in HT throughout the protocol ($p<0.01$ for main effect of group). Regardless of group, T_c increased significantly during exercise (~0.4ºC) with a further increase at 5 min of recovery (~0.3ºC in comparison with exercise). At 15 min of recovery, T_c decreased in the cooling session (~0.1°C in comparison with 5 min of recovery) and did not change in the control session. Consequently, T_c at 15 min of recovery was significantly lower in the cooling session ($p=0.01$ for session vs. time interaction; Fig1a). For HR, regardless of group, in both sessions, HR increased significantly from rest to exercise and, then, decreased progressively during recovery. The decrease in HR was greater in the cooling session and, consequently, HR at 5 and 15 min of recovery was lower in the cooling than the control session ($p<0.01$ for session vs. time interaction; Fig1b). Finally, regardless of group, in both sessions, SBP increased significantly from rest to exercise and, then, decreased during recovery. This decrease was reduced in the cooling session and, thus, SBP measured at 5 min of recovery was significantly higher in the cooling than the control session. In addition, only in the control session, SBP was significantly decreased at 15min of recovery in comparison with rest ($p<0.01$ for session vs. time interaction, Fig1c).

>>>>>>>>>>>>>>>>>>>>>>>>>>>FIGURE 1>>>>>>>>>>>>>>>>>>>>>>>>>>>

Cardiovascular Autonomic Variables during Recovery

Heart Rate Recovery. Regardless of group, T30 and HRR300s were, respectively, lower and higher in the cooling than the control session ($p=0.04$ and 0.01 for main effect of session, respectively, Figure 2). HRR60s and HRRt were similar between the groups and sessions ($p>0.05$ for main effects and interactions, Figure 2).

>>>>>>>>>>>>>>>>>>>>>>>>>>>FIGURE 2>>>>>>>>>>>>>>>>>>>>>>>>>>>

Heart Rate Variability. For HRV assessed during immediate recovery (Figure 3), regardless of group and time, both RMSSD and RMS were significantly higher in the cooling than the control session ($p=0.01$ and $p<0.01$ for main effect of session, respectively). In addition, regardless of group and session, both RMSSD and RMS were significantly increased from 60-300s of recovery in comparison with 30s of recovery ($p<0.01$ and $p<0.01$ for main effect of time, respectively).

>>>>>>>>>>>>>>>>>>>>>>>>>>>FIGURE 3<<<<<<<<<<<<<<<<<<<<<<<<<<<

For the HRV spectral indices (Figure 4), LF was higher and HF lower at rest in the HT than NT regardless of session ($p=0.04$ and 0.03 for group vs. time interaction, respectively). In addition, regardless of group, in both sessions, LF increased and HF decreased from rest to late recovery, and during this phase, LF was lower and HF was higher in the cooling than the control session ($p=0.02$ and 0.05 for time vs. session interaction).

>>>>>>>>>>>>>>>>>>>>>>>>>>>FIGURE 4<<<<<<<<<<<<<<<<<<<<<<<<<<<

DISCUSSION

The main findings of this study were: (1) post-exercise cooling with a fan induced a similar acceleration in HRR in NT and HT subjects; and (2) this effect was supported by a greater parasympathetic reactivation and sympathetic withdrawal induced by the cooling after the exercise.

To the best of our knowledge, this is the first study to assess the effects of post-exercise cooling on HRR and autonomic regulation in a population with autonomic dysfunction and increased cardiovascular risk, such as HT men (Mancia & Grassi

2014). The presence of autonomic dysfunction in the HT group was shown by their increased resting LF- and decreased HF-HRV as well as by the reduced HRR60s after the maximal exercise test in comparison with NT consistent with other reports (Aneni*, et al.* 2014; Best*, et al.* 2014; Erdogan, et al. 2011; Mancia & Grassi 2014; Pagani & Lucini 2001).

Based on recent evidence showing exaggerated sympathetic responses to cooling in hypertension (Greaney, et al. 2017), the hypothesis of the present study was that the previously reported benefits of cooling accelerating HRR would be blunted in HT compared with NT. On the contrary, the present results showed that cooling induced similar changes in HRR in both groups, suggesting that the presumed cooling-induced sympathetic overactivity in HT did not occur. This discrepancy may be due to the fact that in the present study cooling was used after exercise, i.e., a condition in which body temperature was elevated, while in the previous study cooling was employed at rest, when body temperature is normal. Thus, the present results suggest that HT do not have an exaggerated sympathetic response to cooling when this strategy is employed after exercise.

Based on HRR and HRV indices used in the present study, it is possible to suggest the autonomic mechanisms behind the improved HRR observed in both groups in the cooling session. T30, RMSSD and HF are indices accepted as mainly dependent on parasympathetic reactivation (Goldberger, et al. 2006; Hayano*, et al.* 1991; Imai, et al. 1994), while, despite some criticism (Goldstein*, et al.* 2011), HRR300s, RMS and LF indices are considered, at least in part, markers of sympathetic modulation (Malliani, et al. 1991; Ng*, et al.* 2009; Peçanha*, et al.* 2017a). Thus, the lower T30 and LF observed in the cooling session together with the higher RMSSD, HF, HRR300s and RMS suggest that the cooling strategy likely accelerated both cardiac parasympathetic reactivation and sympathetic withdrawal after exercise,

supporting both mechanisms as responsible for the cooling-induced faster HRR in both NT and HT subjects.

Besides accelerating HRR, the cooling protocol also increased SBP during recovery. In fact, cooling prevented the decrease in SBP below pre-exercise levels (i.e., post-exercise hypotension) that was observed at 15 min of recovery in the control session. The mechanisms behind this divergent BP response to the sessions are beyond the scope of this study, however, the existing literature might help to elucidate them. In the control session recovery, the thermoregulatory-induced skin vasodilatation alongside the interruption of the muscle pump might have reduced vascular resistance and stroke volume, promoting a decrease in BP (Carter*, et al.* 1999; Peçanha, et al. 2017b). So, the increased HR (i.e. reduced HRR) observed in the control session might partly reflect the baroreflex's attempt to counteract a BP decrease (Peçanha, et al. 2017b). On the other hand, in the cooling session, despite the interruption of the muscle pump, skin vasodilatation was reduced by the effect of the fan facilitating heat loss via convection (Barwood*, et al.* 2009), which might have limited the decrease in vascular resistance, keeping central blood volume and BP higher than in the control session. As a response, the baroreflex might have produced a greater reactivation of cardiac parasympathetic and withdrawal of cardiac sympathetic modulations, accelerating HRR. Therefore, it seems reasonable to suggest that the effect of post-exercise cooling on HRR and autonomic regulation probably involves its effects on BP.

Results of the present study may have relevant clinical impact. As HT subjects present impaired autonomic responses after exercise (Greaney*, et al.* 2014; Peçanha, et al. 2016), any strategy that could ameliorate this response has clinical importance, promoting a likely preventive effect on acute cardiac events (Cole, et al. 1999; Nishime*, et al.* 2000). In this sense, the present results showed the clinical applicability of post-exercise cooling with a fan in a population with recognized autonomic dysfunction and increased cardiovascular risks (Mancia & Grassi 2014). Other studies with healthy

subjects have also reported positive effects of different strategies for post-exercise cooling, such as ice packs (Leicht, et al. 2009) or cold water immersion (Al Haddad, et al. 2010). However, the use of a fan, may be more practical and safe, especially in subjects with cardiovascular disease and/or risk factors, since cold water immersion and ice use can trigger cardiac arrhythmia (Shattock & Tipton 2012) and considerably increase vasoconstriction and BP (Mawhinney*, et al.* 2017; Sramek*, et al.* 2000).

This study has some limitations. Firstly, it only involved men, and as thermoregulatory responses might differ between genders (Kaciuba-Uscilko & Grucza 2001), future investigations should be conducted with women. Other limitations include the absence of hydration control prior to the sessions. However, subjects in the present study presented similar pre-exercise body masses in both sessions (NT=87±11 vs. 86±11 kg and HT=91±15 vs. 92±16 kg for the cooling and the control sessions, respectively), which suggests no difference in hydration status. Finally, the results of the present study are limited to the first 15 min of the post-exercise period. Assessment of cardiac autonomic variables for a longer period of recovery might help to improve the comprehension of the benefits of cooling on cardiac autonomic restoration.

CONCLUSION

In conclusion, post-exercise cooling with a fan equally reduced core temperature and improved HRR in never-treated HT and NT men. The effects of post-exercise cooling accelerating HRR appeared to be promoted by accelerated post-exercise cardiac parasympathetic reactivation and sympathetic withdrawal, which might be partially related to the increased SBP with cooling. The positive effects of post-exercise cooling on HRR and post-exercise HRV in HT subjects highlight the clinical applicability of such a strategy for populations with increased cardiovascular risk and cardiac autonomic dysfunction.

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Conflict of interest

The authors declare no conflicts of interest.

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TABLES

Table 1. Baseline data

	NT (n = 25)	HT (n = 23)	*P*
Age (years)	45 ± 8	43 ± 8	0.29
Body mass index ($kg.m^{-2}$)	28.6±2.7	29.4±3.6	0.37
SBP (mmHg)	115±4	142±9	< 0.01
DBP (mmHg)	77±2	96±3	< 0.01
VO_{2peak} ($ml.kg^{-1}.min^{-1}$)	26.6±4.2	25.1±4.4	0.24
HR_{peak} (bpm)	169±11	169±16	0.93

Values are presented as mean ± SD. SBP = systolic blood pressure. DBP = diastolic blood pressure. VO_{2peak} = peak oxygen uptake achieved during the maximal exercise test. HR_{peak} = peak heart rate achieved during the maximal exercise test.

Table 2. Physiological variables measured during experimental sessions in normotensive (NT) and hypertensive (HT) groups.

	NT (n = 25)		HT (n = 23)		*P* (session)	*P* (group)	*P* (session*group)
	Control	Cooling	Control	Cooling			
HR (bpm)	128±13	129±12	130±13	129±13	0.97	0.73	0.73
HR (%peak)	76±6	76±5	77±7	77±7	0.99	0.65	0.70
VO_2 ($ml.kg^{-1}.min^{-1}$)	17.3±2.4	17.7±2.6	16.4±3.1	16.0±3.1	0.63	0.12	0.07
VO_2 (% peak)	71±4	72±6	69±5	68±5	0.95	0.07	0.30

Values are presented as mean ± SD. VO2 = oxygen uptake. HR = heart rate.

FIGURE LEGENDS

Fig. 1 Core temperature (T_c; 1a), heart rate (HR; 1b) and systolic blood pressure (SBP; 1c) measured at rest, during exercise and at 5 and 15 min of recovery in the cooling and control sessions in the normotensive (N; n = 25) and the hypertensive (HT; n = 23) groups. * = p ≤ 0.05 vs. rest. $ = p ≤ 0.05 vs. exercise. & = p ≤ 0.05 vs. 5 min of recovery. ‡ = p ≤ 0.05 vs. control session. On panel c, groups were different in all time points (i.e., main effect of group). The connecting lines were removed to improve visualization.

Fig. 2 Heart rate recovery indices assessed after the control and the cooling sessions in the normotensive (NT; n = 25) and hypertensive (HT; n = 23) groups. HRR60s – heart rate decrease at 60s of recovery (3a); T30 = short time-constant of heart rate recovery (3b); HRR300s – heart rate decrease at 300s of recovery (3c); HRRt = long time-constant of heart rate recovery (3d). ‡ = p ≤ 0.05 vs. control session

Fig. 3 RMSSD (4a) and RMS (4b) indices of heart rate variability assessed in segments of 30 s in the first 5 min of recovery after the control and the cooling sessions in the normotensive (NT; n = 25) and the hypertensive (HT; n = 23) groups. RMSSD = square root of the mean of the sum of the squares of differences between adjacent normal RRi, RMS = root mean square of residual of RRi. ‡ = p ≤ 0.05 vs. control session. # = p ≤ 0.05 vs. 30s

Fig. 4 Spectral indices of heart rate variability (HRV) assessed at rest and at the late recovery (Late Rec) of the control and the cooling sessions in the normotensive (NT; n = 25) and the hypertensive (HT; n = 23) groups. LF (nu) = low frequency component of HRV assessed in normalized units (5a); HF (nu) = high frequency component of HRV assessed in normalized units (5b). * = p ≤ 0.05 vs. rest. † = p ≤ 0.05 vs NT. ‡ = p ≤ 0.05 vs. control session. The connecting lines were removed to improve visualization

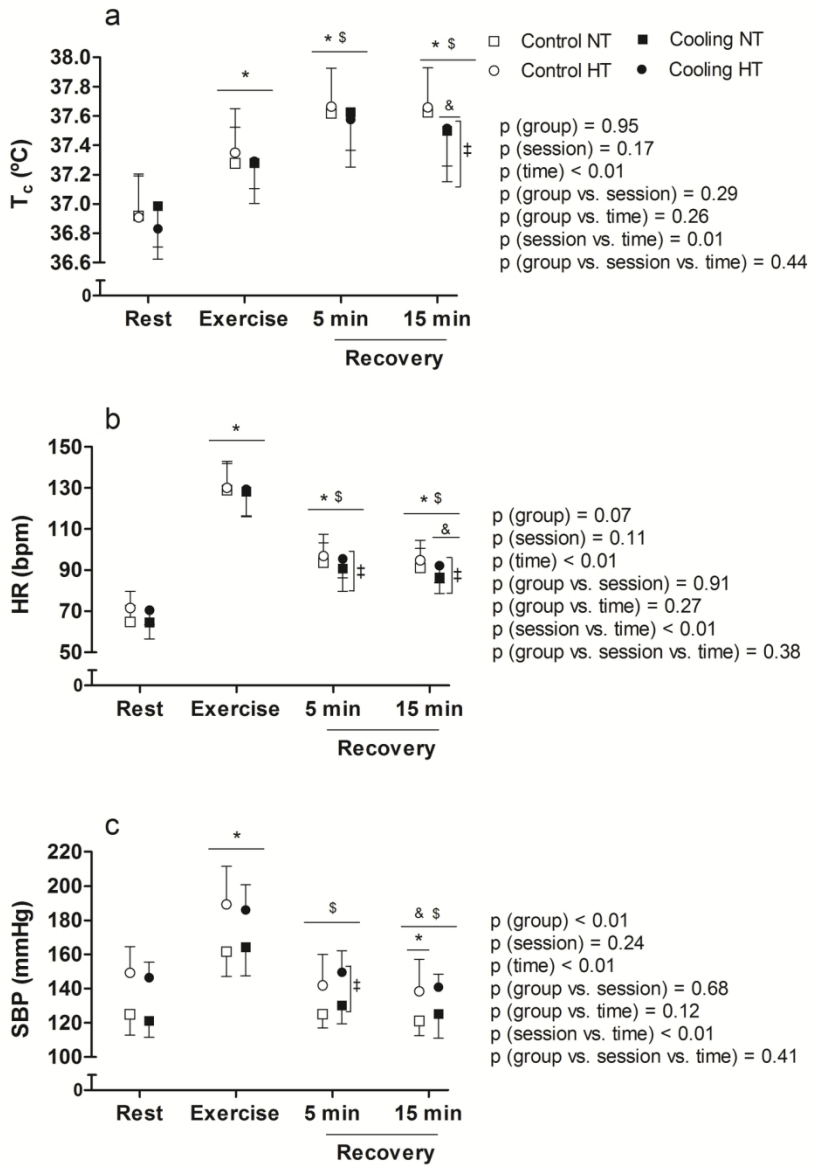

Figure 1

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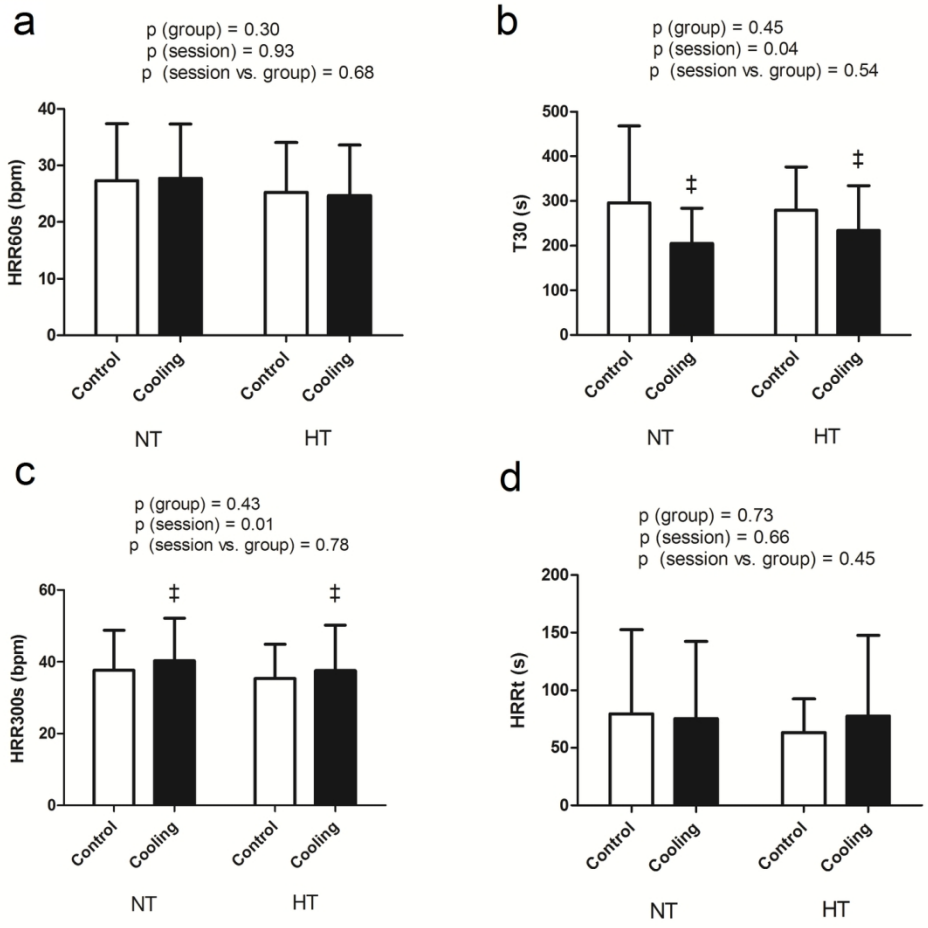

Figure 2

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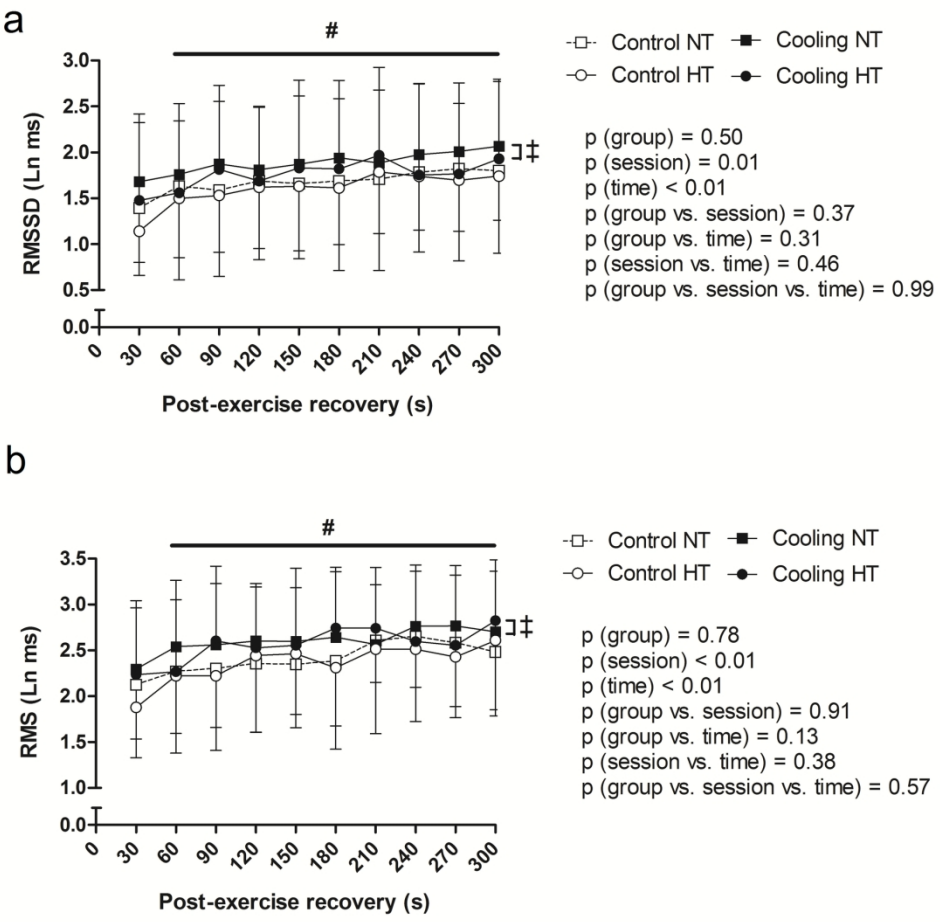

Figure 3

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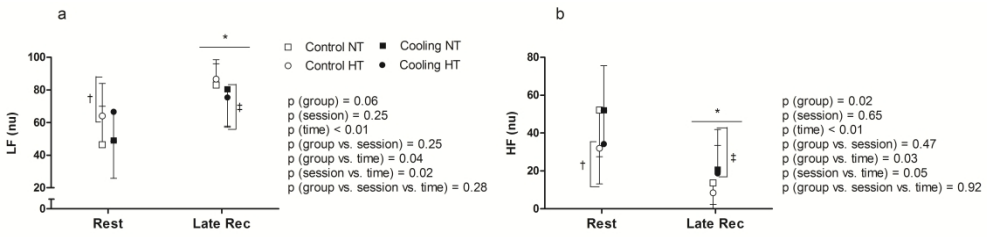

Figure 4

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