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Effects of active versus passive recovery in sprint cross-country skiing

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Abstract

Purpose: Investigate the effects of an active and a passive recovery protocol on physiological responses and performance between two heats in sprint cross-country skiing. **Methods:** Ten elite male skiers (22 ± 3 yrs, 184 ± 4 cm, 79 ± 7 kg) undertook two experimental test sessions which both consisted of two heats with 25 min between start of the first and second heat. The heats were conducted as a 800-m time trial (6º, > 3.5 $m\cdot s^{-1}$, ~ 205 s) and included measurements of O_2-uptake and ΣO_2-deficit. The active recovery trial involved 2 min standing/walking, 16 min jogging (58 ± 5 % of VO_{2peak}) and 3 min standing/walking. The passive recovery trial involved 15 min sitting, 3 min walk/jog (~ 30% of VO_{2peak}) and 3 min standing/walking. Blood lactate concentration (La^-) and heart rate (HR) were monitored throughout the recovery periods. **Results:** The increased 800-m time between the Heat 1 and Heat 2 was trivial after active recovery (Effect Size; ES = 0.1; $P = 0.64$) and small after passive recovery (ES = 0.4, $P = 0.14$). The 1.2 ± 2.1% (mean ± 90% CL) difference between protocols was not significant (ES = 0.3, $P = 0.3$). In Heat 2, peak and average O_2-uptake was increased after the active recovery protocol. **Conclusions:** Neither passive recovery nor running at ~ 58% of VO_{2peak} between two heats, changed performance significantly.

Key Words: *Accumulated oxygen deficit, cross-country skiers, elite athletes, lactate reduction, repeated sprint,* VO_{2max}.

Introduction

Sprint cross-country (XC) skiing consists of one time-trial (prologue; interval start) and three knock-out heats (quarter-final, semi-final and final races). The 30 fastest skiers qualify from the prologue and thereafter, 6 skiers compete in each heat. Semi-final and final heats contain the two fastest racers from each quarter- and semi-final heat. In addition, the two overall fastest “lucky losers”, that did not directly qualify from each quarter and semi-final heat, progress to the next round. The typical heat duration for World cup races the past 4 seasons was ~ 180 s (range 130-210 s), whereas the recovery periods were 15-25 min between semi-final and final races.[1] During these recovery periods, skiers use different strategies and intensity workouts with the explicit goal of maintaining performance. Such ability to perform repeated sprints is likely to be influenced by the nature of the recovery strategy.[2,3] Although several studies have described changes in kinematic and physiological variables with repeated, simulated sprint heats in XC skiing,[4-8] surprisingly little information exists about the effect of different recovery protocols on performance.

A number of studies from other sports, such as swimming and cycling, have demonstrated a beneficial performance effect of active recovery on subsequent exercise bouts lasting < 5 min.[2,3,9] Further, it is generally accepted that active recovery after strenuous exercise leads to a faster reduction of blood lactate as well as muscles lactate compared to passive recovery. [10-12] More specifically, Menzies et al. [12] showed that reduction of blood lactate was more effective at the lactate threshold (LT; ~65% of maximal oxygen uptake: VO_{2max}) than 60% and 40 % of LT. Hence, they concluded that blood lactate reduction during active recovery displays a dose-response relationship and that intensity at or close to LT are preferable. However, active recovery and subsequent La^- reduction has not necessarily been associated with improved subsequent performance in laboratory settings and the importance of La^- reduction during repeated trials is not clear.[13-18]

Notably, active recovery protocols compared to passive recovery have also been reported to impair performance during high-intensity exercise with various duration and very brief recovery. [14,16-18] McAinch et al.[14] found no beneficial effect of active vs. passive recovery on cycling performance (total work over 20 min) in well-trained athletes. In addition, they found that active recovery does not alter either muscle glycogen content or lactate accumulation in the muscle. McAinch et al. [14] therefore concluded that there is no rationale for active "warm-down" after intense aerobe intervals.

Given the paucity of data relating recovery protocols in XC sprint skiing, we examined the effect of passive versus active recovery protocols from a performance perspective. In addition, when investigating the effect of recovery protocols, the main determinants of performance in the respective event as well as in a sport specific exercise should be addressed. Recently, it has been shown that performance in elite sprint XC skiing is highly related to the peak aerobic power (VO_{2peak}) and the anaerobic capacity as estimated from the accumulated oxygen deficit.[4,19] Therefore, the aim of this study was to investigate differences between an active versus a passive recovery on VO_{2peak}, anaerobic capacity and performance during two simulated heats on a roller ski treadmill using the skating technique. The main hypothesis of the present study was that an active recovery would maintain performance better during the subsequent sprint heat compared to passive recovery.

Method

Subjects

Ten national elite senior male XC skiers (22 ± 3 yrs, 184 ± 4 cm, 79 ± 7 kg) volunteered to participate in the study. All subjects had regularly participated in rollerski treadmill testing and were, therefore, familiar with this mode of exercise and the test procedures. The study was approved by the Regional Ethics Committee of Southern Norway and the subjects gave their written informed consent before study participation.

Design

All subjects had one familiarization of the 800-m time-trial before undertaking the main protocol which consisted of three sessions over 3 different days with at least 1 day rest between tests and performed at the same time of day (± 2 hrs). All tests were conducted in June –September. The subjects were instructed to avoid hard training the day before tests and keep normal competition routines before the tests. During day 1, steady-state oxygen uptake was measured at four to five submaximal workloads followed by a VO_{2peak} test. During day 2 and 3, two 800-m time-trials (hereafter called “Heat 1” and “Heat 2”) were performed each day with 25 min between the start of Heat 1 and Heat 2. Between the heats, either an active recovery or a passive recovery was performed. The order of recovery protocol was randomized in a counterbalanced manner. All tests were conducted on a rollerski treadmill with the use of the V2 ski skating technique (also called G3 and double dance; poles are used on every leg push-off), which is a technique appropriate for the speeds and inclines used in the present study.[4] We included only two repeated heats instead of up to four used during competition. This was due to problems recruiting elite skiers to undertake four heats, as pilot testing showed that additional repetitions with this very high work rate was extremely exhaustive for the subjects and had large impact on their training on following days.

Methodology

Submaximal oxygen uptake and VO_{2peak}

Prior to the start, subjects warmed up for 15 min at 3° and 2.25 $m \cdot s^{-1}$ (~ 60-75% of peak heart rate; HR_{peak}). All submaximal tests were performed at 3 $m \cdot s^{-1}$, with 5 min duration and 2-min breaks. Oxygen consumption was measured between 3:00 and 4:30 min into the trials. The speed was set high enough to induce a relevant technique at moderate inclines, but low enough to ensure a steady state VO_2 (< 90% of VO_{2peak}). Subjects started at 3.5° and the incline was subsequently increased 4-6 times by 0.5° (depended on the skiers work capacity) every 5 minutes. Eight minutes after the last submaximal effort, the subjects performed a 1000-m time trial (6°, ≥ 3.5 $m \cdot s^{-1}$) to measure VO_{2peak} in the V2 technique.

Sprint performance

Prior to Heat 1, subjects warmed up for 15 min and included three speed increases (20 s) at 7th, 9th and 11th min, with speeds of 3.5, 3.75 and 4 $m \cdot s^{-1}$ at 4°, respectively. The remaining speed and incline during the 15 min warmup was 3° and 2.25 $m \cdot s^{-1}$. During the heats, the subjects skied as fast as possible over 800 m on a rollerski treadmill (protocol modified after Losnegard et al. [4]). The 800-m test was used based on pilot testing and the fact that the average finishing time from sprint heats in World-cup races (seasons 2010-2014) was 179 ± 26 s.[1]. Thus, the test simulated a long sprint race and was conducted on a steady incline as a more fluctuated profile would demand several familiarization trials. The incline was set to 6° and the speed was fixed at 3.5 $m \cdot s^{-1}$ for the first 100 m (30 s) to avoid over-pacing. Thereafter, the subjects controlled the speed (0.25 $m \cdot s^{-1}$ increases or decreases) by adjusting their front wheel's position on the treadmill relative to two laser beams (60-cm distance apart) situated in front of and behind the skier. The speed changes were conducted manually by the test leader. All data including speed changes and time were sampled and saved for subsequent analysis. Oxygen consumption and HR were measured continuously (5 s epochs) and the average over the 12 highest continuous VO_2, ventilation (V_E) and HR values (60 s) were taken as VO_{2peak}, VE_{peak} and HR_{peak}. The subjects wore a safety harness for all maximal trials in case of a fall.

<<Fig. 1 near here>>

Recovery protocol

The recovery protocols are illustrated in Fig. 1. The active recovery included 2 min rest, 16 min running (3°, 2.25 $m \cdot s^{-1}$) and ~ 3 min rest. The speed and incline was chosen to induce an O_2-demand of ~ 60% of VO_{2max} (~ 45 $mL \cdot kg^{-1} \cdot min^{-1}$) since this intensity is shown to maximize lactate clearance .[12] The same incline and speed was used for all subjects due to the relative homogeneous VO_{2max} values in this group of skiers (range 67-79 $mL \cdot kg^{-1} \cdot min^{-1}$). Running as a recovery mode was chosen based on a survey conducted before the study concluding that running was the most frequently used recovery mode during competitions for this group of subjects (8 out of 10 subjects). O_2-cost during running, measured between 8-12 min, was 42 ± 3 $mL \cdot kg^{-1} \cdot min^{-1}$ which corresponded to 58 ± 5% (range: 53-62%) of their ski skating VO_{2max}. Heart rate was 76 ± 4% (range 70-83%) of HR_{peak} measured during the 1000-m test. Since XC skiers reach ~ 3% higher VO_{2max} in running than ski skating, [20] the actual relative recovery intensity in running might be close to ~ 56% of running VO_{2max}. During the passive recovery, subjects rested for 15 min seated on a chair, before undertaking 3 min light walk/jog (3°, 1.5 $m \cdot s^{-1}$, estimated to ~ 30% of VO_{2max}) and 3 min rest. Blood lactate concentration and HR were measured after 30 s, 3, 6, 12 and 18 min in the active and passive recovery periods.

Calculations of ΣO_2-deficit

The ΣO_2-demand at the supramaximal speeds was estimated by extrapolation of the individual linear relationship between the work rate (W) and steady state O_2-cost from at least 4 trials between 3.5-6° for each subject individually. The calculations are based on the assumption that the ratio O_2-cost·watt^{-1} is constant with increasing speed with its possible limitations.[4] The ΣO_2-deficit was calculated as ΣO_2-demand minus ΣO_2-uptake. Power was calculated as the sum of the power against gravity (Pg) and the power against rolling friction (Pf), in a coordinated system moving with the treadmill belt at a constant speed. Power against gravity was calculated as the increase in potential energy per time ($Pg = m \cdot g \cdot \sin(\alpha) \cdot v$) and the power against friction was calculated as the work against Coulomb frictional forces at a given tangential speed ($Pf = \mu \cdot m \cdot g \cdot \cos(\alpha) \cdot v$), where μ is the coefficient of friction, m is the total mass of the skier and equipment, g is gravitational acceleration, v is the belt speed and α the treadmill incline.

Apparatus

Oxygen consumption was measured by an automatic ergospirometry system (Oxycon Pro, Jaeger Instrument, Hoechberg, Germany), heart rate was measured with a Polar S610i™ monitor (Polar electro Oy, Kempele, Finland) and blood lactate concentration was measured in unhaemolysed blood, from capillary fingertip samples (YSI 1500 Sport, Yellow Springs Instruments, Yellow Springs, OH, USA) and described in detail previously. [21] All testing was performed on a rollerski treadmill with belt dimensions of 3 x 4.5 m (Rodby, Sodertalje, Sweden). The treadmill inclinations and speed were checked before, during and after the testing period. Swix CT1 poles (Swix, Lillehammer, Norway) with a tip customized for treadmill rollerskiing were used (pole length 165 ± 4 cm, corresponding to 91 ± 1% of body height). Two different pairs of Swenor Skate rollerskis (Swenor, Sarpsborg, Norway) with wheel type 1 were used depending on the binding system the skiers normally used (NNN, Rottefella, Klokkarstua, Norway or SNS, Salomon, Annecy, France). The rolling μ of the skis was tested before, during and after the project using a towing test.[4] Prior to Heat 1, the skis were warmed-up with 15 min of treadmill rollerskiing and during the recovery period the skis were kept in a ski-box at 60°C for 15 min to stabilize the temperature (Swix, Warmbox T007680-110, Lillehammer, Norway). Both procedures acquired a friction coefficient of 0.020. The subject's body-mass was measured before each treadmill test (Seca, model 708 Seca, Hamburg, Germany).

Statistical Analyses

All data are presented as mean ± standard deviation (SD) unless otherwise stated. Paired t-tests were used for detecting statistical differences between recovery modes. Precision of estimation and magnitude-based inferences were conducted. Confidence limits (90%) for the true mean values for effects were estimated.[22] The magnitude of differences between exercise modes was expressed as standardized mean differences (Cohen's *d* effect size; ES). The criteria to interpret the magnitude of the ES were as follows: 0.0–0.2, trivial; 0.2–0.6, small; 0.6–1.2, moderate; 1.2–2.0, large; and >2.0, very large. The correlation values were obtained using Pearson's Product Moment Correlation Analysis. Criteria for interpreting the magnitude of correlation (r) were: < 0.1, trivial; 0.1 – 0.3, small; 0.3 – 0.5, moderate; 0.5 – 0.7, large; 0.7 – 0.9, very large; and 0.9 – 1.0, almost perfect.[23] A P value < 0.05 was considered statistically significant. Microsoft Excel (Redmond, WA) and SigmaPlot 12.3 software (San Jose, CA) were used for statistical calculations.

Results

The increase in 800-m time between the Heat 1 and Heat 2 after passive recovery was small (mean ± CL; 1.7 ± 1.9%, ES = 0.4, $P = 0.14$) and after active recovery trivial (0.4 ± 1.5%, ES = 0.1, $P = 0.64$). The differences in 800-m time between recovery modes were small and non-significant (Table 1). There was no significant difference in pacing strategy between Heat 1 and Heat 2 (active or passive) or between recovery modes. Hence, the trivial time difference between Heat 1 and 2 in passive recovery modes was due to a consistent lower speed during the test. In Heat 2, after active recovery protocol, average O_2-uptake, VO_{2peak} and HR_{peak} were significant increased and increased more than after the passive recovery. Both recovery modes induced a significant reduced ΣO_2-deficit (Table 1). VO_{2peak} (mean ± SD: 73.0 ± 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$) and VE_{peak} (196 ± 24 $L \cdot min^{-1}$) measured during the separate 1000-m test, showed trivial differences to the peak values measured during any of the 800-m heats.

The effect on the different recovery modes on La^- and HR can be seen in Fig 2. The active recovery had a significant effect on blood La^- reduction from 3-18 min compared to passive recovery. There was a very large correlation between blood La^- reduction and the change in 800-m time between Heat 2 and Heat 1 in the active recovery period (Fig. 3A). Further, a very large correlation was found between blood La^- reduction and the change in $\sum O_2$-deficit between Heat 2 and Heat 1 in the active recovery period (Fig. 3B).

<<Table 1 near here>>
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Coefficient of variation between test 1 day 2 and test 1 day 3 was for the 800-m time 3.6%, VO_{2peak} 3.6% and $\sum O_2$-defict 9.8%. The correlation between external load and O_2-cost during the submaximal loads was almost perfect ($r^2 = 0.998 \pm 0.002$). There were no significant differences in O_2-cost per watt between inclines when we subtract the individual Y-intercept (resting metabolism) with a maximal numerical differences < 0.7%.

Discussion

Performance was not significant reduced after either an active or passive recovery between two simulated sprint heats on a roller ski treadmill. However, the active recovery had a small and significant effect on the aerobic turnover compared to the passive recovery.

Independent of recovery protocols, small differences in 800-m time were found between the two heats and this seem consistent with previous results simulating XC sprint races during rollerskiing,[4-6] on snow [7,8] and in World-cup races on snow.[1] Maintaining the average speed with subsequent maximal trials is an overall goal for elite skiers during competitions. In the present study the active recovery had a significant effect on average and peak O_2-uptake compared to passive recovery while the ΣO_2-deficit was reduced after both AR and PR. These findings are in line with previous research and have previously been related to an increased blood flow to the working muscle.[9,24] Notably, the lower ΣO_2-deficit in Heat 2 seems to be related to the short recovery period. Recently, we reported similar VO_{2peak} and ΣO_2-deficit data during two subsequent sprint heats, but with a longer recovery period than in the present study (~ 42 vs. 22 min).[4] Hence, it could be assumed that recovery time is a significant component regarding the ability to achieve a high ΣO_2-deficit, and thus, performance. From a practical point of view this is important, since skiers that are in semifinal 1 have a slightly longer recovery time than skiers in semifinal 2. Therefore, reaching semifinal 1 might be beneficial in order to maintaining performance in the final.

In terms of lactate reduction the active protocol induced, as expected, a significant effect on blood lactate reduction. Notably, there was also a very large correlation between La^- reduction and changes in ΣO_2-deficit, and further, La^- reduction and changes in 800-m from Heat 1 to Heat 2. Hence skiers with a largest La^- reduction performed better in Heat 2 versus

Heat 1 after active recovery compared to the skiers with the lowest La^- reduction. Such ability to reduce La^- after high intensity work-outs has previously been linked to performance in cyclist.[25] Recently, Sandbakk et al.[19] concluded that better sprint skiers have a faster La^- reduction than slower skiers indicating that level of skiers should be taken into consideration. However, in the present study with a more homogeneous group of skiers, only a trivial correlation was found between 800-m time in absolute values and La^- reduction ($r = -0.15$) which may strengthen the idea that La^- reduction has a positive effect on the ΣO_2-deficit and thus performance. However, La^- production and reduction is debated in the literature[15] and the relation between La^- reduction on subsequent performance in the present study should be taken with cautions. Weltman et al.[13] found that elevated La^- before exercise has little effect on maximal effort durations of 5 min. Further, McAinch et al.[14] showed similar blood lactate response as in the present study during passive and active recovery (15 min), but found no differences in muscle La^- and no meaningful differences in 20-min cycling performance. The authors, therefore, suggested that low intensity exercise subsequent to intense exercise, does not influence the lactate content in the specific muscle and this is likely attributed to mode of exercise and muscle mass engaged. Importantly, the present study measured La^- in the blood, and not the actual muscle lactate concentration. During one-leg exercise, Bangsbo et al.[11] found that the higher La^- reduction in active recovery was due to an increased La^- metabolism within the muscle rather than a greater release of lactate from the muscle.

Practical Application

The overall goal for the competitive XC skier is to maintain performance during subsequent sprint trials. Choice of recovery protocol will be influenced by their experience, beliefs, and knowledge concerning the optimal recovery protocol. In the present study, no significant effect between recovery modes according to performance was found between two heats.

However, a significant effect was found for average O_2-uptake and VO_{2peak}. The present study only investigated recovery between two heats while a sprint competition consists of three heats after the prologue, with, on average, ~25 min breaks between the heats. It could be that the effects of passive versus active recovery would be different with 3 heats. For instance the small negative effects of passive recovery on ΣO_2-deficit could accumulate over the three heats. Thus we believe that an active recovery protocol is likely to be the best choice. Furthermore, this could also be argued since they compete in cold environments.[26] Importantly, in the present study, a relatively high recovery intensity (~ 58 % of VO_{2peak}) was used with the explicit goal of maximal lactate reduction. Such repeated high intensity recovery could negatively impair performance due to reduced muscle glycogen depletion over several heats. We therefore suggest an active recovery protocol with a lower intensity with the main goal to maintain an elevated muscle temperature.

Conclusion

Neither passive recovery nor running at ~ 58% of VO_{2peak} between two heats in a simulated sprint, gave statistically significant differences in performance.

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Table 1: Performance (800-m time) and physiological response at Heat 1 and Heat 2 with the active or passive recovery.

	Active recovery (AR)		**Passive recovery (PR)**		**AR - PR**	**Cohan´s *d* ES**
Variable	**Heat 1**	**Heat 2**	**Heat 1**	**Heat 2**	**% Diff; CL**	
800-m time (s)	206.4 ± 8.1	207.2 ± 5.7	205.0 ± 7.9	208.4 ± 9.1	-1.2 ± 2.1	0.30; Small
VO_{2peak} ($mL \cdot kg^{-1} \cdot min^{-1}$)	72.3 ± 3.5	73.9 ± 3.5#	72.5 ± 3.5	72.4 ± 4.1	2.5 ± 0.9*	0.50; Small
VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	59.9 ± 2.3	61.8 ± 2.2#	60.1 ± 2.0	60.8 ± 2.6	2.2 ± 1.7*	0.58; Small
HR_{peak} ($beat \cdot min^{-1}$)	184 ± 9	186 ± 8#	183 ± 8	183 ± 8	1.6 ± 1.0*	0.33; Small
V_{Epeak} ($L \cdot min^{-1}$)	193 ± 26	194 ± 23	192 ± 27	194 ± 26	0.4 ± 2.0	0.03; Trivial
La^{-}_{peak} ($mmol \cdot L^{-1}$)	9.0 ± 1.5	9.5 ± 1.4#	9.1 ± 1.3	10.5 ± 1.9#	7.8 ± 7.7	0.53; Small
ΣO_2-deficit ($mL \cdot kg^{-1}$)	91.2 ± 14.1	84.1 ± 11.8#	91.6 ± 12.7	86.3 ± 11.9#	2.0 ± 5.3	0.05; Trivial

Note: Data are mean ± SD. VO_2 = average O_2-uptake; HR= Heart rate; V_E = Ventilation; La^- = blood lactate concentration. % Diff is the differences (log transformed data) and CL is confidence limits (90%) between the active and passive recovery. #Significant differences between Heat 1 and Heat 2 ($P < 0.05$). *Significant differences between active and passive recovery ($P < 0.05$).

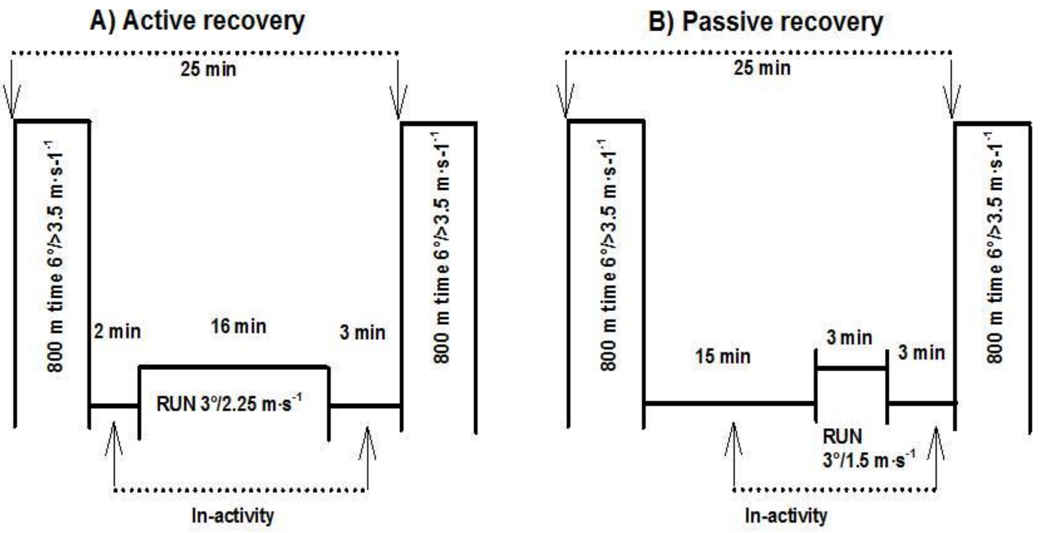

Fig. 1: A) Illustration of the active recovery protocol and B) the passive recovery protocol.

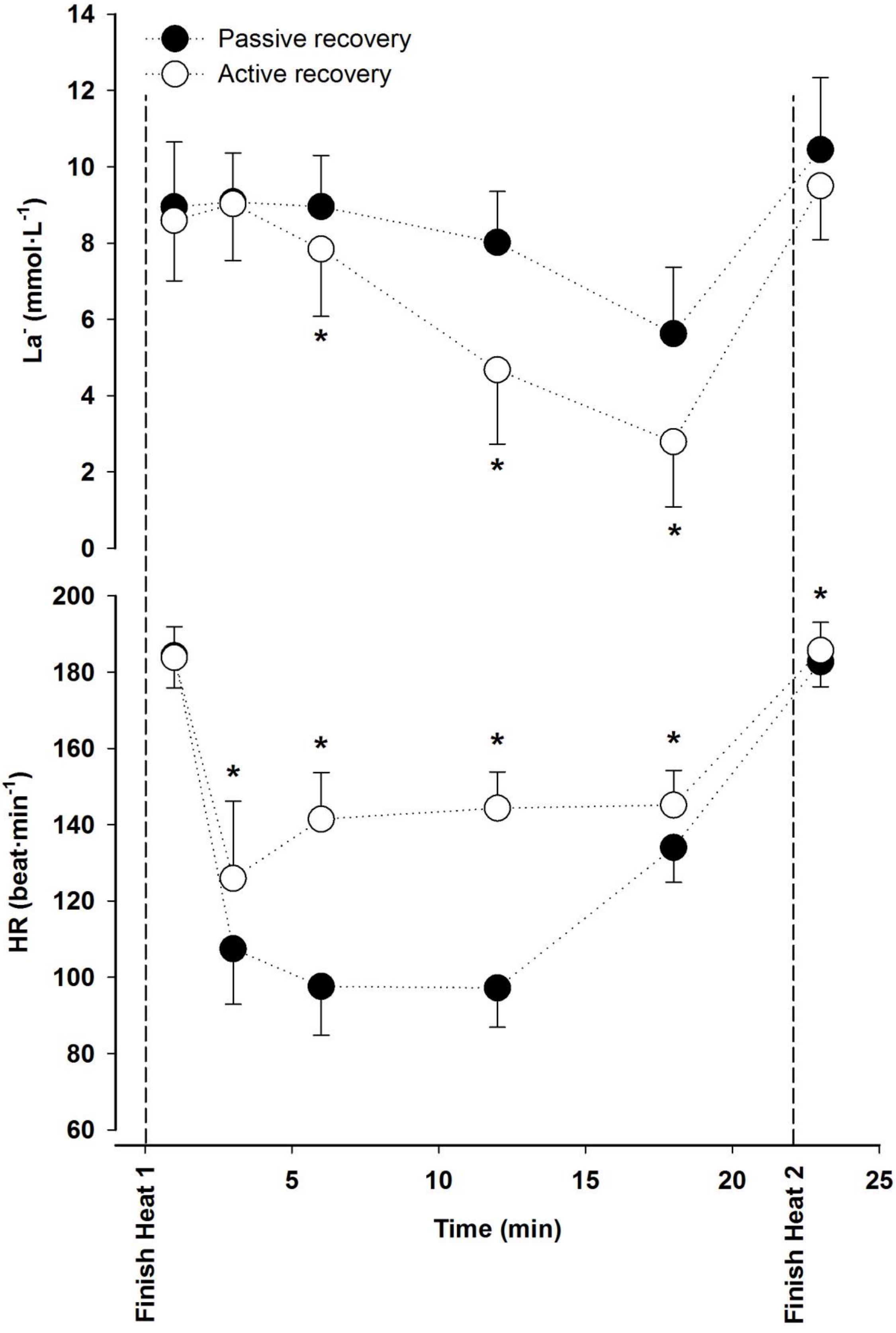

Fig 2: La$^-$ (upper panel) and heart rate (lower panel) after Heat 1 (first 800-m time), during passive or active recovery (3-6-12-18 min) and Heat 2 (second 800-m time). Values are mean ± SD. *Significant differences between active and passive recovery ($P < .05$).

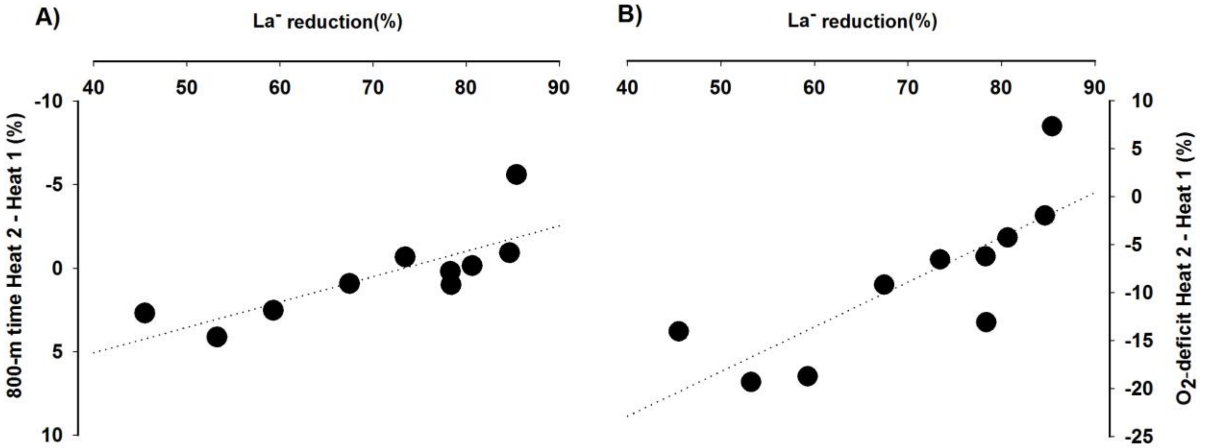

Fig 3: Relation between reduction in La$^-$ from 3 – 18 min in the active recovery (% of La$^-$ at 18 min) and A) change in 800-m time between Heat 2 and Heat 1 (% of Heat 1) and B) change in $\sum O_2$-deficit between Heat 2 – Heat 1 (% of Heat 1). Linear regression shown as dotted line (for A r = 0.86 and for B r = 0.88 and for both ES is very large, $P < 0.05$).