IMPACT OF WARM-UP INTENSITY ON SIMULATED TEAM-SPORT RUNNING PERFORMANCE

by

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Abstract

"IMPACT OF WARM-UP INTENSITY ON SIMULATED TEAM-SPORT RUNNING PERFORMANCE".

Background

Ideally, warm-ups optimise performance; however, most warm-ups are prescribed based on trial and error rather than applying an evidence-based approach (Fradkin, Zazryn, & Smoliga, 2010). The most appropriate warm-up strategy for optimal team-sport performance remains elusive. While the impact of warm-up intensity on repeated-sprint performance has been examined (Yaicharoen, Wallman, Bishop, & Morton, 2012), sprinting only constitutes a fraction of the total activity that occurs during a match. Optimising submaximal efforts (e.g. jogging, running) between sprints may impact performance by ensuring correct positioning of players and recovery between sprint efforts. There is little research investigating the impact of warm-up on team-sport running demands. The purpose of the study was to determine the impact of warm-up intensity on a self-paced, team-sport simulation on a non-motorised treadmill.

Methods

Thirteen male team-sport athletes (22.4 ± 3.0 y, 79.7 ± 9.3 kg, $\dot{V}O_{2peak}$ 55.3 ± 4.1 $mL \cdot kg^{-1} \cdot min^{-1}$) attended six testing sessions ($\dot{V}O_{2peak}$ + familiarisation; two additional familiarisation sessions; three experimental trials). In a randomised, cross-over design, the experimental trials consisted of a 6 min, constant-speed warm-up and 20 min passive recovery. Warm-up intensities were defined as severe (SEV) 70% of the difference between ventilation threshold and $\dot{V}O_{2peak}$ (70% Δ), heavy (HVY; 30% Δ), and control (CON; 4 $km.h^{-1}$).

Subsequently, a 30 min team-sport simulation (TSS) was performed, which contained deliberate changes in speed (i.e., Stand, Walk, Jog, Run, Sprint), including five repeated-sprint clusters, to simulate the time-motion demands of team-sport. The total distance covered and average speed of the trial, speed activities, and repeated-sprint clusters were assessed. Metabolic blood markers (pH, lactate, HCO_3, PO_2, PCO_2 and sO_2), T_c, RPE, $\dot{V}O_2$ and $\dot{V}_E$ were assessed throughout the TSS. Differences between conditions were assessed with a magnitudes based approach and considered practically important when the magnitude of the difference was >75% likely to have exceeded a threshold value. Effects with less certainty were categorised as trivial, and when the likelihood that the true value of the statistic could occur in both directions was >5%, the effect was reported as unclear (Batterham & Hopkins, 2006).

Results

There was an important difference in lactate (9.1 ± 3.7 v 0.9 ± 0.3 mmol·L^{-1}), T_c (37.7 ± 0.3 v 37.2 ± 0.1°C) (SEV > CON), pH (7.29 ± 0.08 v 7.42 ± 0.01) and HCO_3 (17.1 ± 4.0 v 26.0 ± 2.1 mmol·L^{-1}) (SEV < CON) between SEV and CON at the start of the TSS. However, these physiological differences dissipated across the TSS and all physiological parameters were similar at the end between conditions. The warm-up intensities had a trivial impact on the total distance covered and distance covered performing the speed activities throughout the TSS. However, the average speed and the average speed jogging during the 8-20 min time period were importantly decreased (~6-8%) between the SEV and CON. An important attenuation in T_c (0.6 ± 0.1 v 0.9 ± 0.2°C) was also detected in the SEV compared to the CON during the same time-period. Repeated-sprint ability during the 2nd and final repeated-sprint clusters (RSC), when compared to the average speed of the 1st sprint, was importantly

decreased within the HVY and SEV compared to the CON. HVY importantly increased speed in the initial sprint ~6% above CON.

Discussion

SEV and HVY warm-ups provided little performance benefit during a self-paced TSS. Despite initial differences in the internal milieu, submaximal and maximal activity profiles were similar between conditions. However, it was determined that an important decrease in average speed and average speed jogging occurred during the 8-20 min time period between the SEV and CON. It is possible participants adopted a pacing strategy during this time-period within the SEV to reduce the increase in T_c. Furthermore, the HVY and SEV reduced repeated-sprint performance as the TSS progressed. These data suggest that apart from the initial sprint, the examined warm-up intensities have a limited or negative impact on team-sport running performance.

STATEMENT OF SOURCES

"I, Grant Rowe, declare that the MExSc(Res) thesis entitled "Impact of Warm-Up Intensity on Simulated Team-Sport Running Performance" is no more than 24,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work. All research procedures reported in this thesis received the approval of the relevant Ethics/Safety Committees".

Signature: Date:

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ABBREVIATIONS

		Units
<	Less than	
>	Greater than	
µL	Microliter	
ADP	Adenosine Diphosphate	
ATP	Adenosine Triphosphate	
$b \cdot min^{-1}$	Beat per Minute	
CON	Control Warm-Up	
CV%	Co-Efficient of Variation Percentage	
$g \cdot kg^{-1}$	Gram per Kilogram	
HCO_3	Bicarbonate	$mmol \cdot L^{-1}$
HR	Heart Rate	$b \cdot min^{-1}$
hr	Hour	
HVY	Heavy-Intensity Warm-Up	
Hz	Frequency	
iEMG	Integrated Electromyography	Hz
K^+	Potassium	
$km \cdot h^{-1}$	Kilometre per Hour	
m	Meters	
$m \cdot s^{-1}$	Metre per second	
min	Minutes	
mL	Millilitre	
mmHg	Millimetre of Mercury	
$mmol \cdot L^{-1}$	Millimole per Litre	
MOD	Moderate-Intensity Warm-Up	
$mOsmol \cdot kg^{-1}$	Milliosmole per Kilogram	
N	Newton	
Na^+	Sodium	
P_B	Barometric Pressure	mmHg
PCO_2	Partial Pressure of Carbon Dioxide	mmHg
PO_2	Partial Pressure of Oxygen	mmHg
RH	Relative Humidity	%
RPE	Rating of Perceived Exertion	
rpm	Revolution per Minute	
RSC	Repeated-Sprint Cluster	
s	Second	
SEV	Severe-Intensity Warm-Up	
sO_2	Saturation of Blood Oxygen	%
T_{amb}	Ambient Temperature	°C

T_c	Core Temperature	°C
TD	Total Distance	m
T_m	Muscle Temperature	°C
TSS	Team-Sport Simulation	
v	Versus	
$\dot{V}CO_2$	Carbon Dioxide Production	$L \cdot min^{-1}$
$\dot{V}_E$	Minute Ventilation	$L \cdot min^{-1}$
$\dot{V}O_2$	Oxygen Consumption	$L \cdot min^{-1}$ and $mL \cdot kg^{-1} \cdot min^{-1}$
$\dot{V}O_{2max}$	Maximal Oxygen Uptake	$L \cdot min^{-1}$ and $mL \cdot kg^{-1} \cdot min^{-1}$
$\dot{V}O_{2peak}$	Peak Oxygen Uptake	$L \cdot min^{-1}$ and $mL \cdot kg^{-1} \cdot min^{-1}$
WU	Warm-Up	
Δ	Change or Difference	

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Chapter One: Introduction

A warm-up is a period of exercise and skill-practice performed by athletes immediately before training or competition. Ideally, warm-ups optimise performance, however, most warm-ups are prescribed based on trial and error rather than applying an evidence-based approach (Fradkin et al., 2010). One of the issues in interpreting warm-up research is the use of a wide range of protocols. Furthermore, the majority of studies examining the effect of warm-up on performance commonly use isolated performance measures, such as countermovement jump and time-trial performance, which may not be representative of a team-sport setting where performance includes a range of speed activities (Stolen, Chamari, Castagna, & Wisloff, 2005). There is little research investigating the impact of warm-up on team-sport running demands (e.g. Australian football, soccer, field-hockey). It is possible that the types of warm-up that positively influence isolated performance measures differ from those required to enhance the multiple capacities needed in team-sport.

Warm-ups have traditionally been performed using moderate-intensity exercise (Bishop, 2003b). A moderate-intensity warm-up (MOD) can generate favourable physiological effects, including increasing muscle temperature (T_m) while limiting the depletion of anaerobic energy stores and the production of fatigue-related metabolites (Bishop, 2003b). MOD also improves repeated-sprint performance more than a heavy-intensity warm-up (HVY) (Yaicharoen, Wallman, Morton, & Bishop, 2012).

However, the implication that a HVY and severe-intensity warm-up (SEV) would be less effective for prolonged exercise contrasts to previous findings. Specifically, increases in muscle acetylcarnitine concentration, a substrate for oxidative ATP production, are

positively related to warm-up intensity (Gray, De Vito, & Nimmo, 2002). The priming of $\dot{V}O_2$ kinetics are also intensity dependent, and subsequently occur by performing a HVY and SEV (Burnley, Doust, & Jones, 2006; Gerbino, Ward, & Whipp, 1996). While MOD has been shown to improve performance, there is some evidence to suggest that higher intensity warm-up protocols may in fact be more effective.

Warming-up can improve peak power output and work performed during a single-sprint, but may not improve prolonged intermittent-sprint performance (Yaicharoen, Wallman, Bishop, et al., 2012). Subsequently, the impact of warm-up on team-sport running performance may be negligible beyond the initial acute benefits. However, no one has examined how warm-up might alter submaximal activity profiles between sprints.

Distances covered at submaximal intensities, for example walking, jogging and running, may be critical in team-sports because athletes spend the majority of match-play performing these activities (Coutts, Quinn, Hocking, Castagna, & Rampinini, 2010; Spencer et al., 2004). With time-motion studies demonstrating that high-intensity activity profiles are unaltered during team-sport matches (Coutts et al., 2010), previous findings into the impact of warm-up on prolonged intermittent-sprint exercise may be less relevant than those including additional contributions from submaximal intensities. The data from this thesis will make a significant contribution by strengthening the evidence-base for warm-up protocols on team-sport athletes.

1.1 Aims

The aim of this thesis was to determine the impact of warm-up intensity on team-sport running performance. Specifically, the thesis aims to:

1. Assess the difference in activity profile measures during a simulated team-sport running protocol following the performance of a heavy-intensity warm-up (HVY), severe-intensity warm-up (SEV) and CON warm-up.

   It is expected the SEV will enable a greater total distance to be covered during a team-sport simulation (TSS) compared to the HVY and CON. The improvement in performance is expected to be observed in jogging and running activity profiles and during repeated-sprint clusters.

2. Assess the difference in metabolic and respiratory responses during a TSS following the performance of the warm-up conditions: HVY, SEV and CON.

   It is predicted the HVY and SEV will attenuate the drop in blood pH and bicarbonate (HCO_3), and reduce the elevation in lactate and oxygen consumption compared to the CON. These responses may demonstrate an enhanced aerobic metabolic response following the HVY and SEV.

1.2 Delimitations

This thesis is focused on expanding the knowledge of active warm-up methods on team-sport performance in thermoneutral ambient conditions. Because environmental conditions can confound metabolic responses, controlled laboratory conditions were selected (Febbraio, 2000). The population for the study was delimited to male, team-sport athletes with a $\dot{V}O_{2peak}$ ($\geq$50 $mL \cdot kg^{-1} \cdot min^{-1}$). These selection criteria are consistent with the populations targeted in previous team-sport running simulations (Sirotic & Coutts, 2008). The elevated fitness requirement of the participants may limit the application of results to

team-sport athletes, however, specificity of outcomes for athletes with reasonable aerobic fitness was deemed to be of high relevance for the thesis.

A further delimitation to the thesis lies in the use of treadmill-based testing to simulate field-based performance. The overall aim is to advance the understanding of the impact of different warm-up intensities on a self-paced simulated team-sport ***running*** protocol. Other factors influencing performance in competitive team-sport, such as multidirectional movement, jumping, and skill-level, have been omitted in this protocol.

1.3 Limitations

Athletes' nutritional status and motivation to participate were potential limitations to the thesis. Participants were required to complete a food diary, 24 hr preceding an experimental trial, to help maintain consistent eating patterns between trials. Nutritional status was a limitation because nutritional intake was not scrutinised, and if eating patterns were dissimilar between trials, participates were still allowed to be tested. To help reduce the influence of this limitation, participants were provided a carbohydrate drink 2 hr before data collection.

Chapter 2: Review of Literature

This review of literature begins with a definition and overview of warm-up. The impact of body temperature on physiological responses and performance will be explained, as well as the effects of pre-trial blood lactate accumulation and the changes in metabolism following a warm-up. Then, the importance of warm-up intensity towards subsequent muscle function and performance will be discussed. Finally, the validity of exercise modalities to replicate team-sport demands will be detailed.

2.1 Definition and Overview of Warm-Up

Warm-up has been defined as a period of preparatory exercise performed in order to enhance subsequent competition or training performance (Fradkin et al., 2010). Different warm-up strategies optimise different performance measures which can be categorised as short (maximal effort for ≤10 s), intermediate (maximal effort for >10 s, but <5 min) and long-term (fatiguing effort for ≥5 min) (Bishop, 2003b) efforts.

Warm-up protocols aimed at improving short-term performance are recommended to be performed at a moderate-intensity, with warm-up and recovery durations of 10-20 min and 5-15 min, respectively (Bishop, 2003b). These warm-up recommendations seek to maximise the increase in muscle temperature (T_m) and limit the impact of fatigue at subsequent exercise onset. The increase in T_m has been shown to positively affect the force-velocity relationship, the maximal shortening velocity and power output of the muscle (Bennett, 1984; Davies & Young, 1983), and the degradation of high-energy phosphates (Febbraio, Carey, Snow, Stathis, & Hargreaves, 1996).

In contrast, warm-up protocols aimed at improving long-term performance are also suggested to be prescribed at a moderate-intensity, but the warm-up (<10 min) and recovery durations (<5 min) should be shorter (Bishop, 2003b). These recommendations seek to elevate baseline oxygen consumption ($\dot{V}O_2$) at the commencement of subsequent exercise (Andzel, 1978; Andzel & Busuttil, 1982; Bishop, 2003b; Grodjinovsky & Magel, 1970). The elevation of baseline $\dot{V}O_2$ may improve performance by reducing the O_2 deficit, consequently sparing anaerobic energy stores and reducing the accumulation of fatigue-related metabolites, such as hydrogen ions, inorganic phosphates and extracellular potassium (Bailey, Vanhatalo, Wilkerson, DiMenna, & Jones, 2009; Bishop, 2003a).

However, the effectiveness of these recommendations has been questioned (Bailey et al., 2009). Specifically, the recommendations to improve long-term performance are based on previous research which did not investigate $\dot{V}O_2$, or confirm if an elevated baseline $\dot{V}O_2$ actually improved performance (Andzel, 1978; Andzel & Busuttil, 1982; Grodjinovsky & Magel, 1970). Instead, commencing subsequent activity with an elevated baseline $\dot{V}O_2$ may indicate incomplete phosphocreatine resynthesis, due to the strong relationship between $\dot{V}O_2$ and intramuscular phosphocreatine (Rossiter et al., 2002). Moreover, long-term exercise can be improved when baseline $\dot{V}O_2$ is restored, and negatively affected when $\dot{V}O_2$ is elevated (Bailey et al., 2009). Current warm-up recommendations which seek to elevate $\dot{V}O_2$ at the beginning of subsequent exercise may negatively affect long-term performance.

Within previous research, warm-up intensity has been prescribed based on a percentage of $\dot{V}O_{2max}$ or HR_{max}. However, this method of prescribing warm-up intensity may generate dissimilar metabolic profiles due to the variability of lactate threshold (Yaicharoen, Wallman, Morton, et al., 2012). It is likely when using a percentage of $\dot{V}O_{2max}$, some

participants will be exercising below, whereas others above, their lactate threshold (Bishop, Bonetti, & Dawson, 2001). To improve upon this shortcoming, warm-up intensity has more recently been prescribed based on a percentage difference within intensity domains.

The three intensity domains that will be discussed include the moderate, heavy and severe. The moderate-intensity domain comprises exercise intensities below lactate threshold. The heavy-intensity domain covers exercise intensities between lactate threshold and critical velocity. The severe-intensity domain describes exercise intensities between critical velocity and $\dot{V}O_{2max}$ (Burnley & Jones, 2007). The categorisation of warm-up intensity into these domains enables the normalisation of pulmonary responses and blood acid-base profiles between participants.

The methods used within warm-up protocols have also received considerable attention. One of the main practices that has been scrutinised is the effectiveness of stretching on performance (Behm & Chaouachi, 2011). Previously, static-stretching had been considered an essential component within warm-up practices to prepare for subsequent exercise (Behm & Chaouachi, 2011). However, it has been shown that warm-up protocols consisting of static-stretching may provide little benefit (Wallmann, Mercer, & Landers, 2008) or can negatively impact performance (Cramer et al., 2005; Fletcher & Jones, 2004; Marek et al., 2005; Pearce, Latella, & Kidgell, 2012).

At present, team-sport athletes likely follow a three-phase warm-up model beginning with brief aerobic exercise, followed by active, sport-specific stretches, and finishing with activities that simulate the movement and bioenergetic patterns of the upcoming exercise (Taylor, Weston, & Portas, 2013). In contrast to static-stretching, the implementation of active-stretching into warm-up protocols improves performance

(Manoel, Harris-Love, Danoff, & Miller, 2008; Turki et al., 2012) by increasing musculotendinous stiffness (Belli & Bosco, 1992; Fletcher & Jones, 2004) and activating post-activation potentiation (Gelen, 2010; Sale, 2002). However, a negative effect of post-activation potentiation is that it has been shown to coexist with fatigue (Tillin & Bishop, 2009). Inclusive of the active-stretching phase, if the prescription of intensity, volume and rest period within warm-up protocols are unsuitable, negative performance results may eventuate (Tillin & Bishop, 2009). For example, a higher volume active-stretching warm-up has been shown to negatively impact 20 m sprint performance (Turki et al., 2012).

Furthermore, a two-phase warm-up involving an initial cardiovascular phase followed by task-specific activities (submaximal shuttle runs and maximum sprint) provided the same repeated-sprint performance as a three-phase warm-up (Taylor et al., 2013). It is likely the performance benefits generated by a warm-up can be created without performing active-stretching. Whilst the current warm-up methodology is intended to enhance subsequent performance, preparatory exercise of this nature is also considered beneficial to reduce injuries and mentally prepare athletes for competition (Towlson, Midgley, & Lovell, 2013). Thus, warm-up expectations have broadened the interpretation of what should be included as part of a warm-up. Certain warm-up protocols may be better designed to reduce injury-incidence (Fradkin, Gabbe, & Cameron, 2006); although, it can be argued that they may lack the stimulus and specificity required to prime mechanisms associated with performance enhancement (Taylor et al., 2013).

In summary, currently accepted warm-up guidelines and practices may not improve performance as much as coaches and athletes are expecting. There is little agreement within the literature on the optimal organisation of warm-up protocols. More attention

needs to be focused on better understanding the mechanisms underlying the performance changes following a warm-up to enable a more scientific approach to determine a warm-up structure and methodology.

2.2 Temperature and Non-Temperature Effects of a Warm-Up

Passive and active warm-up techniques are used to induce temperature and/or non-temperature related effects. A passive warm-up involves increasing T_m and core temperature (T_c) passively via exogenous heat sources, including saunas, diathermy, heating pads, hot showers and baths (Bishop, 2003a). The advantage of performing a passive warm-up is the attainment of all the temperature related effects without depleting provisions of energy and accumulating fatigue-related metabolites (Bishop, 2003b). These physiological effects are likely to improve short-term performance measures (Dolan, Greig, & Sargeant, 1985).

An active warm-up involves metabolic heat production generated by muscle contractions, including calisthenic drills, cycling, running, swimming, etc. Independent of attaining the temperature related effects, an active warm-up can also provide greater cardiovascular and metabolic variation, as well as better cognitive and neuromuscular preparation for motor control precision (Ajemian, D'Ausilio, Moorman, & Bizzi, 2010). For this reason, it is likely that an active warm-up would impact more parameters associated with team-sport performance than a passive warm-up (Ajemian et al., 2010).

A fundamental outcome of performing a warm-up, which has been shown to positively influence performance, is an increase in T_m and T_c. For example, a 1°C increase in T_m has been associated with ~4-6% improvement in maximal dynamic strength, power

output, jumping and sprinting performance (Bergh & Ekblom, 1979). Other benefits of elevating T_m include increased anaerobic adenosine triphosphate (ATP) turnover, muscle fiber conduction velocity, and improved efficiency of cross-bridge cycling (Ferguson, Ball, & Sargeant, 2002; Gray, De Vito, Nimmo, Farina, & Ferguson, 2006); all of which reduce the time to peak tension and half-relaxation time (Asmussen & BØJe, 1945; Bennett, 1984).

The benefits of elevating the temperature of a working organism is the facilitation of work performance (Asmussen & BØJe, 1945). However, increased thermoregulatory strain can also adversely affect certain types of performance measures (González-Alonso et al., 1999). For example, elevating T_c to 38°C by an active and passive warm-up significantly reduced intermittent treadmill running performance compared to no warm-up (Gregson, Batterham, Drust, & Cable, 2005). During long-term, self-paced exercise, it is suggested that cerebral thermal centres interpret the rate in which heat has been stored, calculated against the actual T_c, to anticipate the effort required to execute the desired performance (Tucker, Marle, Lambert, & Noakes, 2006). In addition, increases in skin temperature are associated with voluntary reductions in exercise intensity during self-paced exercise (Schlader, Simmons, Stannard, & Mündel, 2011). The decrease in exercise intensity is seen as the 'first-line of defence' to regulate body temperature, which occurs well before body temperature reaches critical levels (Schlader, Stannard, & Mündel, 2011). Therefore, a pre-generated elevation in body temperature from a warm-up may modify subsequent exercise intensity to limit further heat accumulation.

The non-temperature related effects of a warm-up that can augment performance include a residual metabolic acidemia with a rightward shift in the oxyhaemoglobin disassociation curve (Wasserman, Hansen, & Sue, 1991) and increased supply of acetyl

groups before the onset of exercise (Gray et al., 2002). Furthermore, improved $\dot{V}O_2$ kinetics (Billat, Bocquet, Slawinski, Laffite, & et al., 2000; Gerbino et al., 1996) and reduced rate of lactate accumulation (Campbell-O'Sullivan, Constantin-Teodosiu, Peirce, & Greenhaff, 2002; Campbell, Constantin-Teodosiu, Lambourne, & Greenhaþ, 1999) are other non-temperature related effects which can improve subsequent performance. These findings provide evidence that increasing body temperature during a warm-up is not the most important effect for all performance tasks.

In summary, the positive effects of elevating body temperature on physiological function and performance following a warm-up have been described. However, the negative effects of elevating body temperature during a warm-up, i.e., reducing the 'heat-sink' of the body, need to be appreciated as performance may be impaired during long-term exercise. Lastly, the physiological benefits generated by a warm-up can be observed despite body temperature remaining or returning back to baseline levels.

2.3 The Role of Pre-Trial Blood Lactate Accumulation

The accumulation of lactic-acid and consequent acidification of skeletal muscle may contribute to muscle fatigue (Allen, Lamb, & Westerblad, 2008). Decreases in cellular pH are thought to inhibit muscle contractile mechanisms (Kristensen, Albertsen, Rentsch, & Juel, 2005). Therefore, the accumulation of lactate during a warm-up may lead to subsequent performance decrement, especially during intermediate or long-term exercise (Bishop, 2003b; Burnley, Doust, & Jones, 2005b). However, other researchers have since speculated that the nomenclature '*warm*-up' could be changed to '*acid*-up'. It is postulated that many of the proposed mechanisms which improve performance following a warm-up can be explained by acid-base changes rather than temperature changes.

A potential cause of fatigue during intermittent-sprint exercise is decreased muscle excitability associated with decreases in Na^+-K^+ pump activity (Allen et al., 2008; Bishop, 2012). The presence of lactic-acid can result in recovered force production in the presence of high extracellular K^+ levels, thereby enabling action potentials to still be propagated despite the muscle being depolarised (Nielsen, de Paoli, & Overgaard, 2001). These findings suggest that increased lactate accumulation during a warm-up may help maintain mechanical force production during subsequent periods of high-intensity intermittent-sprint exercise (Pedersen, Nielsen, Lamb, & Stephenson, 2004).

Second, the breakdown of lactic-acid, resulting in the accumulation of hydrogen ions in the extracellular compartment, plays an important role in maintaining or increasing capillary partial pressure of O_2 (Wasserman et al., 1991). The acidification facilitates O_2 diffusion into cells via a right-shift in the O_2 dissociation curve (Bohr Effect) (Wasserman et al., 1991). Additional acidification also vasodilates local microvasculature, promoting greater oxygen utilisation (Stringer et al., 1994). Therefore, it can be argued that lactic-acid serves to attenuate further anaerobic metabolism during high-intensity muscle contractions (Wasserman et al., 1991).

The findings from Wasserman et al. (1991), Nielsen et al. (2001) and Pedersen et al. (2004) demonstrate that lactic acidosis generated in a warm-up may enable subsequent performance improvements. However, when arm exercise was used to increase arterial lactate levels to 12 $mmol \cdot L^{-1}$ subsequent performance in a time-to-exhaustion trial using the legs was significantly reduced (Bangsbo, Madsen, Kiens, & Richter, 1996). It has also been argued that prior lactate exposure and the associated lower muscle pH, may indirectly cause a greater K^+ release into the interstitium, which in turn could increase muscle fatigue

(Bangsbo et al., 1996). The varying effects of lactate accumulation make it difficult to generalise its benefits to exercise during in vivo conditions (Lamb & Stephenson, 2006).

A possible pre-trial blood lactate range of ~3-5 mmol·L^{-1} has been suggested to support subsequent exercise. This range has also been associated with improved $\dot{V}O_2$ kinetics (Burnley et al., 2005b). Warm-ups that either do not change or excessively increase pre-trial blood lactate (≥5 mmol·L^{-1}) may not provide performance benefits. For example, an 'all-out' intermittent-sprint warm-up followed by a 15 min rest period, elevated pre-trial blood lactate to ~8 mmol·L^{-1}, primed $\dot{V}O_2$ kinetics but reduced performance by ~20% (Wilkerson, Koppo, Barstow, & Jones, 2004a). In contrast, a SEV followed by a 20 min rest period, elevated pre-trial blood lactate to 3.0 mmol·L^{-1}, primed $\dot{V}O_2$ kinetics and improved performance by 30% (Bailey et al., 2009).

Nevertheless, the correlation between changes in acidification and muscle function remain inconsistent. The restoration to baseline force occurs faster than the restoration of pH, suggesting the absence of a mechanistic link (Sahlin & Ren, 1989). The calculated relationship between pre-trial blood lactate and enhanced performance may have been observed coincidentally rather than as a result of lactate accumulation acting directly on muscle function.

2.4 Altered Aerobic Metabolism Contribution

An outcome of performing a warm-up, which is temperature-independent, is an improvement in the aerobic contribution to metabolism (Fukuba et al., 2012). A change in $\dot{V}O_2$ kinetics is one of the main indicators used to determine the influence of warm-up on metabolism (Jones, Koppo, & Burnley, 2003). $\dot{V}O_2$ kinetics refers to the rate at which

pulmonary (and by inference, muscle) oxygen uptake increases following the onset of exercise (Jones, DiMenna, et al., 2008). It can assist in determining the relative contribution of oxidative and non-oxidative metabolism to energy supply during exercise (Jones & Burnley, 2009).

The metabolic information derived from $\dot{V}O_2$ kinetic analysis is important to investigate because of the finite amount of anaerobic work that can be performed during exercise. Ideally, improvements in $\dot{V}O_2$ kinetics result in a reduced contribution from anaerobic metabolism and therefore accumulation of fatigue-related metabolites during exercise, improving exercise tolerance. For example, reductions in O_2 deficit, blood lactate accumulation, and improved performance have been observed when $\dot{V}O_2$ kinetics are accelerated (Bailey et al., 2009; Carter et al., 2005; Wittekind & Beneke, 2009).

HVY, but not MOD, can improve $\dot{V}O_2$ kinetics during subsequent supralactate exercise (Gerbino et al., 1996), suggesting the up-regulation of aerobic metabolism can occur following a warm-up, but only if the warm-up intensity is above lactate threshold. Other researchers have since reported that a supralactate warm-up can improve $\dot{V}O_2$ kinetics by reducing the slow component amplitude, not by changing the primary component time-constant (rate at which $\dot{V}O_2$ rises toward steady state) as initially reported (Burnley, Jones, Carter, & Doust, 2000). A reduced slow component is postulated to reflect a more rapid establishment of intracellular homeostasis due to the recruitment of fewer Type II muscle fibers (Burnley et al., 2000) and a reduced reliance on anaerobic pathways (Palmer, Jones, Kennedy, & Cotter, 2009).

Improvements in $\dot{V}O_2$ kinetics may not be the only beneficial effect of supralactate warm ups. For example:

A heavy-intensity dynamic plantar-flexion and leg-extension exercise improved phosphocreatine kinetics during subsequent exercise, resulting in less subsequent total phosphocreatine breakdown (Forbes, Raymer, Kowalchuk, Thompson, & Marsh, 2008; Rossiter et al., 2001). HVY improved muscle deoxygenation kinetics during subsequent heavy-intensity cycling exercise, demonstrating improved O_2 utilisation at the onset of subsequent heavy-intensity exercise (Marles et al., 2007). The execution of a high-intensity intermittent warm-up significantly increased the concentration of muscle acetylcarnitine, an oxidative substrate, before the commencement of subsequent exercise (Gray et al., 2002). The performance of a supralactate warm-up has been shown to blunt iEMG activity as subsequent high-intensity exercise is continued. It was suggested the reduced iEMG activity following a supralactate warm-up demonstrated an increased reliance on slow, oxidative Type I muscle fibers during subsequent exercise (Bailey et al., 2009; Burnley, Doust, Ball, & Jones, 2002b). A high-intensity knee-extension and plantar-flexion warm-up resulted in less muscle lactate and hydrogen ion production during subsequent exercise (Bangsbo, Krustrup, González-Alonso, & Saltin, 2001; Forbes et al., 2008).

Collectively, these findings imply that a high-intensity warm-up can decrease the reliance on energy derived from anaerobic sources during subsequent high-intensity exercise. A possible explanation for the augmented aerobic response following a supralactate warm-up may be due to the different concentrations of inorganic phosphates in the muscle (e.g. ADP, ATP). Altered phosphate-related concentrations may subsequently upregulate mitochondrial respiration, and hence $\dot{V}O_2$ to re-establish metabolic homeostasis (Mahler, 1980; Rossiter et al., 2001; Whipp & Rossiter, 2005).

In terms of applying these results in the real-world, a possible limitation of the abovementioned findings is that they were determined during constant work-rate exercise. However, it has previously been demonstrated that relative scores during a repeated-sprint test are significantly correlated with the primary phase for the $\dot{V}O_2$ off-kinetics (Dupont, McCall, Prieur, Millet, & Berthoin, 2010). This finding provides confidence that the ability to maintain sprint performance is associated with faster $\dot{V}O_2$ kinetics. Investigations into the impact of supralactate warm-up on submaximal and maximal activities during a TSS will help determine if the expected improvements in aerobic metabolism contribution can be harnessed by team-sport athletes.

In summary, various supralactate warm-up protocols, including intermittent-sprint and constant-work-rate, can improve the aerobic metabolism contribution during subsequent high-intensity exercise (Bangsbo et al., 2001; Billat et al., 2000; Wittekind & Beneke, 2009). What remains unexplored is the capacity for an enhanced aerobic metabolism contribution from a warm-up to influence submaximal and maximal activity profiles of team-sport athletes during a running TSS.

2.5 Prescription of Warm-Up Intensity

The prescription of warm-up variables governs the effectiveness of warm-up protocols. There is strong evidence to support that warm-up protocols provide a performance benefit to varying exercise efforts, including time-trial and time-to-exhaustion testing (Bailey et al., 2009; Ingham, Fudge, Pringle, & Jones, 2013; Jones, Wilkerson, Burnley, & Koppo, 2003; Palmer et al., 2009; Takizawa & Ishii, 2006). However, warm-ups can also adversely affect performance (Fradkin et al., 2010). A major limitation within the warm-up literature is the lack of understanding of how individual variables may impact performance

(Fradkin et al., 2010). While there are substantial amounts of research investigating the impact of warm-up intensity on *muscle function*, the diversity within warm-up protocols makes it difficult to interpret the impact of warm-up intensity on activities involving a variety of aspects.

Both HVY and SEV protocols have been shown to generate a priming effect, including improved muscle O_2 extraction fraction, $\dot{V}O_2$ kinetics and motor unit recruitment, as well as the sparing of intramuscular phosphocreatine and reduced blood lactate accumulation (Billat et al., 2000; Burnley, Doust, Ball, & Jones, 2002a; Burnley et al., 2006; Gerbino et al., 1996; Raymer, Forbes, Kowalchuk, Thompson, & Marsh, 2007; Rossiter et al., 2001). Furthermore, both HVY and SEV protocols have been shown to improve intermediate and long-term, high-intensity exercise (Bailey et al., 2009; Ingham et al., 2013; Jones, Wilkerson, et al., 2003). However, there is no evidence to draw any conclusions about the effectiveness of HVY and SEV on the multiple capacities required in team-sport exercise.

There are also potential disadvantages of implementing a supralactate warm-up, especially within the severe-intensity domain. Severe-intensity exercise can cause significant reductions in phosphocreatine and pH, increased accumulation of inorganic phosphates (Jones, Wilkerson, DiMenna, Fulford, & Poole, 2008) and blood lactate, and the failure of the $\dot{V}O_2$ slow component to plateau (Burnley & Jones, 2007). Collectively, these responses are known for their negative effects on muscle function and are associated with performance decrements (Carter et al., 2005). Therefore, warm-ups performed in the severe-intensity domain could negatively affect subsequent exercise.

Knowing that a supralactate warm-up can activate both positive and negative mechanisms, a possible explanation for a positive performance result is the relationship between warm-up intensity and rest period. SEV can significantly preserve primed $\dot{V}O_2$ kinetics for greater than 30 min (Burnley et al., 2006). Furthermore, anaerobic work capacity can approximate baseline levels within 15 min after a severe-intensity, exhaustive exercise bout (Ferguson et al., 2010). Therefore, it is argued that a SEV, coupled with a rest period between 15-30 min, may provide an optimal opportunity for primed effects to govern the performance outcome without the impedance of an impaired anaerobic work capacity.

Bailey et al. (2009) clearly demonstrated the interplay between warm-up intensity and rest period on performance. In their study, a SEV followed by a 3 min rest period significantly reduced severe-intensity time-to-exhaustion cycling performance by 16% compared to CON; whereas, the same SEV followed by a 20 min rest period significantly improved performance by 30% (Bailey et al., 2009). Based on this finding, previous reports opposing SEV protocols may not be accurate because their conclusions may be founded on a shorter rest period. Prolonging the rest period is expected to nullify the negative effects of severe-intensity exercise, while a short rest period is likely to restrict the restoration of anaerobic work capacity before subsequent exercise. SEV followed by a 20 min rest period may provide performance benefits greater than currently recommended warm-up protocols for team-sport athletes.

2.6 The Validity of Exercise Modalities to Replicate Team-Sport Demands

Previous studies investigating the impact of warm-up on team-sport demands have frequently prescribed varying exercise modes. For example, cycling has been used to

replicate the running demands of team-sport (Yaicharoen, Wallman, Bishop, et al., 2012). However, it is argued that varying modes may lack the specificity to activate task-specific mechanisms (Enoka & Stuart, 1992). Many researchers justify the prescription of a cycle ergometer in place of a running-based test because of an established strong relationship between intermittent-sprint cycling and running performance (Bishop, Spencer, Duffield, & Lawrence, 2001). However, the mechanisms governing the performance outcomes during cycling and running may differ (Billat, Richard, Binsse, Koralsztein, & Haouzi, 1998; Carter et al., 2000), limiting any attempts to interpret the results.

Examples of the lack of specificity in metabolic responses between cycling and running have been observed in the $\dot{V}O_2$ kinetic slow component response. A significantly larger slow component is generated during cycling compared to running when exercising at the same relative intensities (Carter et al., 2000). The disparity in the slow component between cycling and running is also observed in triathletes with similar abilities between modalities (Billat et al., 1998). The larger slow component observed during high-intensity cycling is speculated to be caused by higher intramuscular tension which may recruit more Type II muscles fibres. Running also utilises eccentric contractions which may lead to better metabolic economy and a smaller slow component response (Carter et al., 2000).

In contrast to cycling, studies investigating the impact of warm-up using a motorised treadmill are more likely to activate mechanisms that are team-sport specific. However, the use of a motorised treadmill has its own limitations in terms of simulating team-sport running demands. During competitive matches, team-sport athletes change speed approximately every 5 s, and on average, maximal sprints tend to last ~4 s (Carling, Bloomfield, Nelsen, & Reilly, 2008; Spencer et al., 2004). Previous attempts to simulate

team-sport demands on a motorised treadmill have used a protocol in which change of speed occurred infrequently (Drust, Reilly, & Cable, 2000). The time required for a motorised treadmill to change speeds restricts any attempt to simulate the short-intermittent demands of team-sports. While the use of field-based testing protocols, such as the Loughborough Intermittent Shuttle Test, are likely to off-set the limitations confronted during motorised treadmill testing they have their own limitations. These include, the standardisation and quality of the simulation, they are often externally paced, and there is a reduction in the amount and quality of physiological and performance data that can be collected.

More recently, the development of the non-motorised treadmill may have enabled laboratory-based testing to better simulate field-based running performance. Albeit that no research has compared running on a non-motorised treadmill to overland running, it is possible the non-motorised treadmill enables similar kinematic and kinetic outputs, increasing the likelihood that running-specific fatigue, inherent in team-sports, is generated (McKenna & Riches, 2007). The use of a non-motorised treadmill also enables participants to self-regulate their speed throughout a team-sport simulation (TSS), including accelerations and decelerations. The eccentric loading required to decelerate at high sprinting speeds may provide a greater understanding of the metabolic demands of team-sport athletes (Spencer et al., 2004).

Ideally, examining physiological changes in-vivo during team-sport matches would provide the clearest picture of athletic demands. However, as many mechanistic physiological tests are laboratory-dependent and time-consuming, field-based duplication is problematic (Sirotic & Coutts, 2008). The use of the non-motorised treadmill to investigate

the impact of warm-up on team-sport running demands provides confidence that the results are ecologically valid.

2.7 Summary

A number of studies have determined the impact of warm-up on physiological function and performance. However, these findings were determined during exercise which may not be representative of a team-sport setting where performance includes a range of speed activities. Furthermore, SEV coupled with a prolonged rest period has been shown to improve performance greater than currently suggested warm-up recommendations. Therefore, the priming effects of a SEV, followed by a prolonged rest period, may reveal a potential warm-up methodology to optimise team-sport performance.

Chapter Three: Methods

3.1 Participants

Thirteen male, recreationally trained, team-sport athletes (Australian football, soccer and rugby union) were recruited for the study. The participant characteristics were as follows (mean ± SD): age = 22.4 ± 3.0 yr, body mass = 79.7 ± 9.3 kg, stature = 181.7 ± 8.8 cm and $\dot{V}O_{2peak}$ = 55.3 ± 4.1 mL·kg^{-1}·min^{-1}. Prior ethics approval was obtained from the Australian Catholic University Research Ethics Committee.

3.2 Experimental Design

Participants attended the laboratory on six occasions (T_{amb} = 21.6 ± 0.9°C, RH = 41.5 ± 12.0%, P_B = 751.5 ± 5.7 mmHg); three familiarisation sessions and three experimental trials. During each familiarisation session, participants performed the team-sport simulation (TSS) to become accustomed to running and changing speeds on the non-motorised treadmill (Woodway Curve 3, Waukesha, Wisconsin). In addition, during the first familiarisation session, participants also completed a graded exercise test.

During each of the experimental trials, participants performed one of the three different warm-up conditions prior to the TSS. The order of the warm-up conditions was determined using a block randomised design (i.e. 1,2,3 : 1,3,2 : 2,1,3 : 2,3,1 : 3,1,2 : 3,2,1) with all experimental trials held at least one week apart.

Experimental trials were conducted at approximately the same time of day (maximum 2 h variance) for each participant to avoid any circadian variations in performance (Racinais, Blonc, & Hue, 2005). Participants were instructed to arrive at the

laboratory in a euhydrated state, having avoided structured exercise and alcohol consumption for 24 h, and caffeinated drinks for 6 h (Sawka, Burke, & Eichner, 2007). Participants submitted a completed food diary for the 24 h preceding their initial experimental trial, and were subsequently provided a photocopy of this food diary to follow for the 24 h prior to the remaining experimental trials.

3.3 Experimental Protocol

3.3.1 Familiarisation

Previous work from our laboratory has demonstrated that participants require two familiarisation sessions to become adept at running on a non-motorised treadmill (Tofari et al. In Press). This skill proficiency included responding to visual and audio cues to accelerate or decelerate to various activity speeds: standing, walking, jogging, running or sprinting. Therefore, participants in the current study performed three familiarisation sessions before undertaking the experimental trials. The data collected from familiarisation trials two and three were used to determine the reliability and smallest worthwhile change of the performance variables (Table 1).

In the first familiarisation session participants performed a graded exercise test followed, after a 15 min break, by the TSS. Subsequently, the data collected from the TSS performed during the first familiarisation session was discarded. The second and third familiarisation sessions involved a standardised warm-up prior to undertaking the TSS.

The standardised warm-up consisted of an initial 3 min jog at a self-selected pace on the non-motorised treadmill. This was followed by a 5 min active stretching routine which emphasised range of motion of the major lower body muscles including hamstrings,

quadriceps, gluteals and calves. Lastly, the participants performed a 3 min replication of the TSS. This involved responding to automated, synchronised visual and auditory cues to either stand, walk, jog, run or sprint; however, participants were instructed to reduce the intensity of these efforts to a level they felt appropriate for a warm up. The TSS commenced approximately 3 min after the completion of the standardised warm-up.

3.3.2 Graded Exercise Test

Participants completed a modified version of a graded exercise test on a motorised treadmill (HP COSMOS Pulsar, Nussdorf-Traunstein, Germany). The treadmill slope was kept at 4% and the test started at a speed of 4 $km \cdot h^{-1}$ for 4 min, and then increased by 1 $km \cdot h^{-1}$ every minute until volitional fatigue (Davies, Daggett, Jakeman, & Mulhall, 1984).

Participants were fitted with a two way respiratory valve (Hans Rudolph, Shawnee, Kansas) and oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) determined in 10 s increments using a metabolic gas analyser (Parvomedics, Sandy, Utah). The gas analyser and flowmeter were calibrated immediately before each test following the manufacturer's guidelines. Heart rate was measured continuously using a chest strap (Polar, Kempele, Finland).

Participants were considered to have reached $\dot{V}O_{2peak}$ if they achieved one of the following conditions: respiratory exchange ratio > 1.1 or heart rate ≤ 10 $b \cdot min^{-1}$ of their age-predicted maximum (Tanaka, Monahan, & Seals, 2001). All participants achieved at least one of these conditions during their graded exercise test.

Table 1. Reliability and smallest worthwhile change of the performance variables.

Performance Measures	Mean	±SD	Change in Mean (%)	Lower CI	Upper CI	TE CV (%)	Lower CI	Upper CI	ICC	SWC
TD (m)	4344.1	343.1	2.0	-2.3	6.6	4.3	3.0	8.4	0.86	1.9
Walk (m)	813.7	69.0	0.0	-5.4	5.8	5.5	3.7	10.8	0.73	2.0
Jog (m)	1499.4	103.6	2.6	-3.5	9.1	6.0	4.1	12.0	0.69	1.9
Run (m)	1258.0	180.9	1.1	-4.5	7.0	5.7	3.9	11.1	0.93	3.3
Sprint (m)	704.5	36.3	3.2	-1.1	7.7	4.2	2.9	8.2	0.65	1.4
1^{st} sprint ($m \cdot s^{-1}$)	5.6	0.4	4.9	1.5	8.4	3.2	2.2	6.2	0.83	1.5
RSC ($m \cdot s^{-1}$)	5.6	0.4	3.1	-0.5	7.0	3.6	2.5	7.0	0.81	1.4
Relative Contribution (%)										
Walk	18.8	1.0	-1.3	-6.1	3.7	4.9	3.4	9.6	0.34	1.2
Jog	34.6	1.2	0.9	-1.2	3.0	2.0	1.4	3.9	0.76	0.7
Run	28.8	2.2	-0.8	-3.3	1.8	2.5	1.7	4.9	0.94	1.6
Sprint	16.3	1.2	0.9	-1.2	3.1	2.1	1.4	4.0	0.96	1.5

CI, confidence limit; TE CV, typical error as a co-efficient of variation; ICC, intraclass correlation; SWC, smallest worthwhile change; TD, total distance; RSC, repeated-sprint clusters.

$\dot{V}O_{2peak}$ was calculated as the average of the three highest consecutive 10 s $\dot{V}O_2$ values before the participant reached volitional fatigue. Ventilatory threshold was calculated using the V-slope method (Beaver, Wasserman, & Whipp, 1986). Two researchers independently determined the ventilatory threshold of each participant using a customised excel spreadsheet. When a discrepancy occurred, the conflict was resolved through discussion between the two researchers. Ventilatory threshold and $\dot{V}O_{2peak}$ were subsequently used to prescribe the relative intensity of the experimental warm-up conditions.

3.4 Experimental Trials

Participants arrived at the laboratory 2 h before testing to ingest a telemetric temperature pill (CorTemp, Palmetto, Florida). At this time, participants consumed 600 mL of a commercially available sport drink (Gatorade G Series 02 Perform, Victoria, Australia) supplemented with 1 $g \cdot kg^{-1}$ of body weight of glucose powder (Lotus Foods, San Francisco, California) (Jeacocke & Burke, 2010). A carbohydrate supplement was provided to the participants before the trials to limit the impact of carbohydrate availability on metabolite accumulation and exercise performance. A general guideline of carbohydrate consumption before commencing exercise within 1-4 hr is 1-4 $g \cdot kg^{-1}$ (Jeacocke & Burke, 2010).

Upon returning to the laboratory, a venous blood sample was taken to determine hydration status using plasma osmolarity. After voiding their bladder, the participant's pre-trial body weight and T_c were determined before commencing the warm-up. After completing the warm-up, participants sat on a chair, placed on the non-motorised treadmill, for 20 min before starting the TSS. Participants were asked to remain seated during the 20 min recovery to control for activity levels between participants. When the participant

completed the TSS, they sat for another 10 min to enable determination of the overall trial rating of perceived exertion (RPE) (Ribeiro, Alves, da Silva, & Fontes, 2013).

3.4.1 Warm-Up Conditions

Participants performed three experimental warm-up conditions in a block randomised manner: CON, HVY and SEV. All warm-up conditions were performed on a motorized treadmill (HP Cosmos Pulsar, Nussdorf-Traunstein, Germany) set at a 4% slope to enable running velocity to be prescribed based on results from the graded exercise test. The CON involved the participants walking for 6 min at 4 $km \cdot h^{-1}$. The HVY and SEV involved running for 6 min at a speed equivalent to 30% and 70% (Δ) of the difference between ventilatory threshold and $\dot{V}O_{2peak}$, respectively. The duration and intensity of the warm-up conditions was selected to replicate warm-up protocols previously demonstrated to improve performance during constant-speed cycling exercise (Bailey et al., 2009).

A facemask (7450 V2, Hans Rudolph, Shawnee, Kansas) and two way respiratory valve (Hans Rudolph, Shawnee, Kansas), were fitted to the participants and $\dot{V}O_2$ and $\dot{V}CO_2$ determined in 10 s increments using a metabolic gas analyser (Parvomedics, Sandy, Utah). The gas analyser and flowmeter were calibrated immediately before each warm-up following the manufacturer's guidelines. Metabolic blood parameters and T_c were measured pre and post warm-up. RPE was recorded post warm-up (Foster's scale 0-10) (Foster et al., 2001).

3.4.2 Team-Sport Running Simulation

Participants performed a 30 min TSS on a non-motorized treadmill. Before testing, the friction force and inertia resistance of the treadmill belt was set at 37.3 N and 34.0 kg,

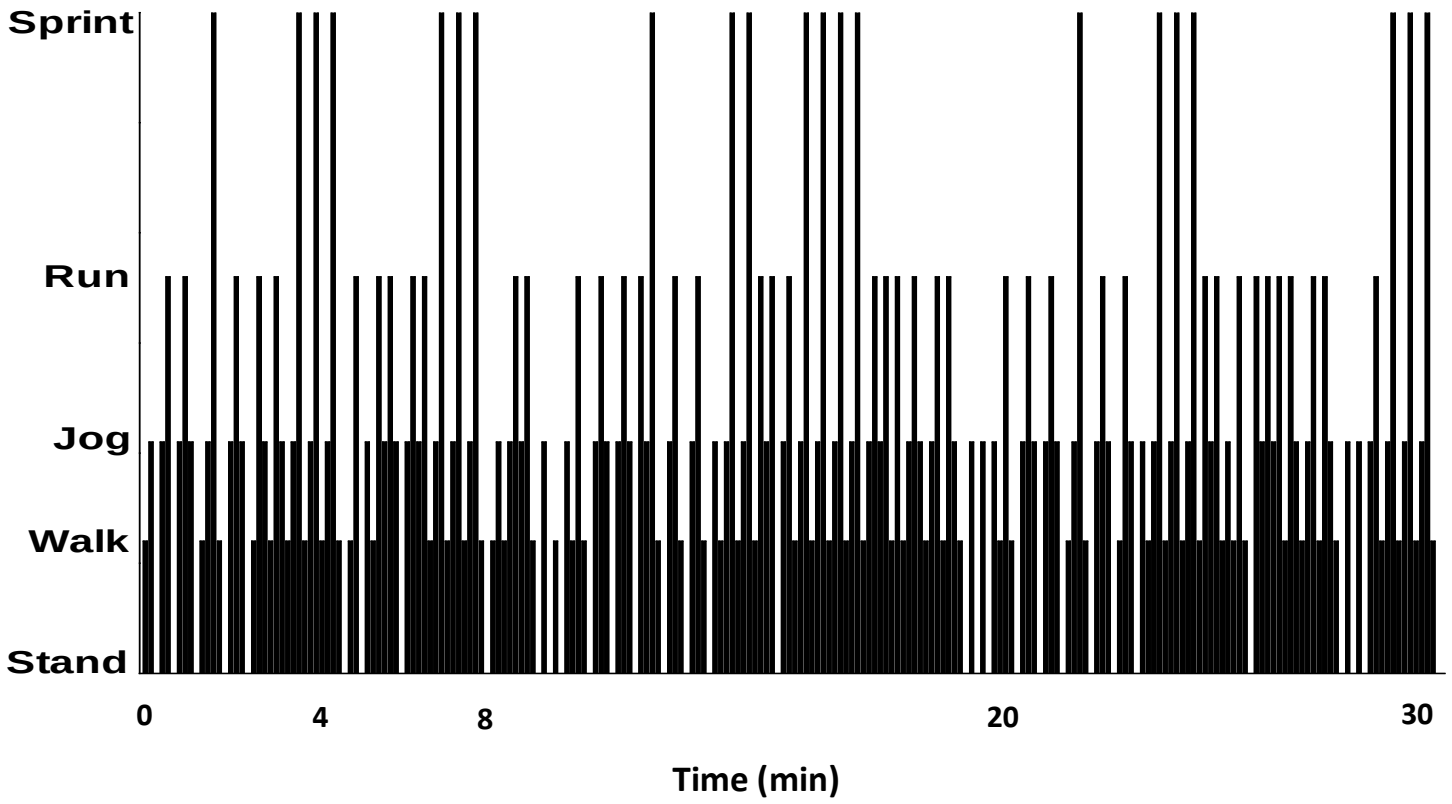
respectively. Data from the non-motorized treadmill was sampled at 200 Hz, and the velocity and acceleration filters were set at 20 and 16 Hz respectively (Pacer Performance System, Perth, Australia).

The same facemask and attachments used in the warm-up were re-fitted to the participants, and $\dot{V}O_2$ and $\dot{V}CO_2$ determined in 10 s increments. The five speed activity profiles performed during the TSS were standing, walking, jogging, running and sprinting (Figure 1). The participants were instructed on each activity profile to be performed via automated, synchronised visual and audio cues from a projector and speakers (UL7400U, Mitsubishi Electric, Rydalmere, Australia), whilst not receiving any feedback on their performance. Although the category of speed was provided as an instruction, the actual locomotive speed was self-selected. This procedure permitted any differences in self-selected running to be analysed between experimental conditions.

One researcher provided verbal instruction to the participants when a sprint was about to occur during the TSS. Furthermore, the same researcher provided verbal motivation to the participants during the sprints to help maintain a maximum effort throughout the sprint. The terminology and tone used during the verbal instruction and motivation was carried out by the same researcher for all trials, and was consistent for all participants.

The TSS was designed to closely match the time-motion running demands of team-sport athletes. The percentage time spent performing the speed activities of the TSS was similar to the prolonged, high-intensity, intermittent running protocol created by Sirotic and Coutts (2007) to mimic the running work profile of team-sports. The mean heart rate and blood lactate profiles determined by Sirotic and Coutts during the prolonged, high- intensity,

Figure 1. The simulated team-sport running protocol with the 5 speed activities and repeated-sprint clusters.



The simulated team-sport protocol was designed to closely match the time-motion demands of team-sport athletes. The percentage of time spent performing each speed activity was standing 17%, walking 24%, jogging 33%, running 19% and sprinting 7%. The vertical bars represent the occurrence of an activity but the intensity of these efforts was self-selected by the participants.

intermittent running protocol are similar to reported values from the Loughborough Intermittent Shuttle Test (Sirotic & Coutts, 2007). The Loughborough Intermittent Shuttle Test has been shown to replicate the physiological and performance demands of real-world soccer athletes (Nicholas, Nuttall, & Williams, 2000). These associations may provide confidence that the present TSS closely matched the physical and physiological demands of team-sport.

The specific variables measured were the total distance covered (m) of the TSS, distance covered (m) and average speed ($m \cdot s^{-1}$) performed in each speed activity category (walking, jogging, running and sprinting), distance covered and average speed within specific time-periods of the simulation (0-4 min, 4-8 min, 8-20 min, 20-30 min), distance covered and average speed performed in each speed activity within the specific time-periods, and distance covered and average speed sprinting during the 1^{st}, 2^{nd} and final repeated-sprint clusters. Metabolic blood parameters, T_c and RPE were measured at 0, 4, 8 and 30 min, and T_c and RPE were also measured at 20 min into the TSS (Figure 2).

3.4.3 Blood Collection

Metabolic blood parameters using arterialised, fingertip blood were determined using a handheld lactate monitor (Lactate Pro 2, Arkray Inc, Kyoto, Japan) and i-STAT device (Abbott Point of Care, Princeton, New Jersey). The i-STAT $CG4^{+}$ cartridges were used to determine pH, partial pressure of carbon dioxide (PCO_2), partial pressure of oxygen (PO_2), base excess (BE), bicarbonate (HCO_3), total carbon dioxide (TCO_2), saturation of oxygen (sO_2) and lactate, and were calibrated before testing according to the manufacturer's guidelines.

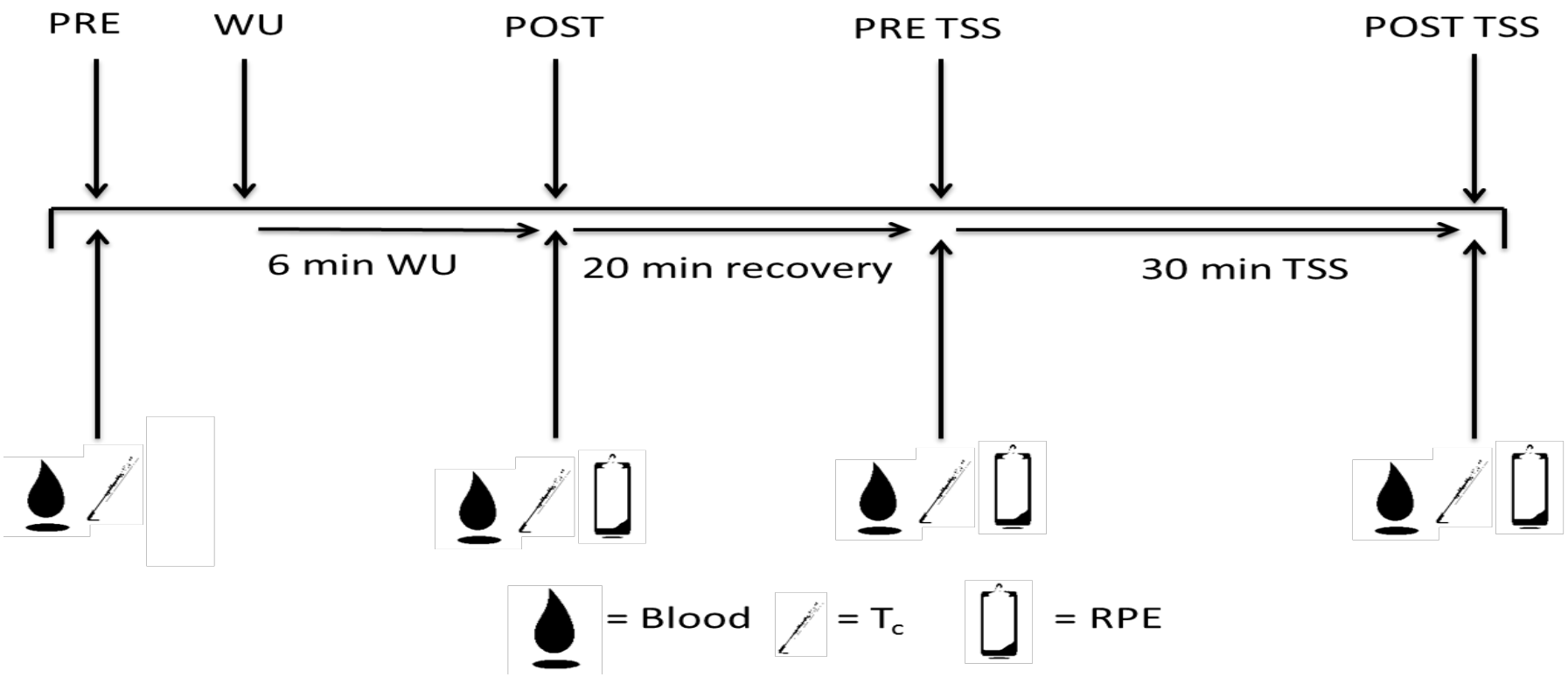
Approximately 100 µL of arterialised, fingertip blood was collected by placing participant's right hand in a bucket of warm water for 5-30 s. This hand was subsequently dried, cleansed with an alcohol swab, and a puncture created with a disposable lancet. Two heparinised capillary tubes (~95 µL total) were used to collect blood for analysis with the i-STAT handheld device. Subsequently, the surface blood was wiped away, and a 0.3 µL blood sample collected with the lactate monitor. Originally, the Lactate Pro 2 analyser was used as a back-up if the required blood sample for the i-STAT was inadequate.

A 5 mL venous blood sample was collected after the participants had been lying supine for 15 min. This procedure was enforced as plasma volume and concentrations can fluctuate due to postural changes (Hagan, Diaz, & Horvath, 1978). A 21 gauge needle was inserted into an antecubital vein and blood collected into a lithium-heparinised tube (Greiner Bio-One, Frickenhausen, Germany). Plasma was separated by centrifuging the whole blood at 4000 rpm (Thermoline Scientific, Wetherill Park, NSW, Australia), and plasma osmolarity assessed in triplicate (Advanced Instruments, Norwood, Massachusetts). The osmometer was calibrated using a 290 $mOsmol \cdot kg^{-1}$ standard sample.

3.4.4 Rating of Perceived Exertion

At the beginning of the first experimental trial, participants were advised on the meaning and purpose of the RPE scale (Borg, 1982). Foster's 0-10 RPE scale with verbal anchors was shown to the participants, and they were instructed to select a number representing their immediate level of exertion at specific time-points throughout experimental trials (Foster et al., 2001). These time-points were immediately post warm-up,

Figure 2. The experimental protocol.



WU, warm-up; TSS, team-sport simulation; T_c, core temperature; RPE, rating of perceived exertion. Blood markers also taken at 4 min and 8 min of the TSS. T_c and RPE were taken at 4 min, 8 min and 20 min of the TSS.

pre and post TSS, and 4, 8 and 20 min into the TSS (Figure 2). Furthermore, the participants were instructed to provide an RPE score 10 min after the completion of the TSS to establish their perception of effort of the entire trial (Ribeiro et al., 2013).

3.4.5 Core Temperature

Participants ingested the telemetric temperature pill 2 h prior to the warm-up protocol. It has been determined that 2 h is enough time for the pill to travel through the stomach, and be a valid and reliable measure of T_c (Kolka, Levine, & Stephenson, 1997). Furthermore, the participants were advised to abstain from eating during this 2 h period and drinking water 1 h before testing to maintain the reliability of the temperature measures (Byrne & Lim, 2007). The participants T_c was determined immediately pre and post warm-up, pre and post TSS, and 4, 8 and 20 min into the TSS (Figure 2).

3.5 Statistical Analysis

All variables were log transformed to reduce bias due to non-uniformity of error. Differences between conditions were assessed using percentage statistic with 90% confidence intervals on a customized spreadsheet (Hopkins, 2006). Differences were assessed relative to the CV% determined for each variable in the familiarisation trials or from previous work (Crouter, Antczak, Hudak, Dellavalle, & Haas, 2006; Dascombe, Reaburn, Sirotic, & Coutts, 2007; Gant, Atkinson, & Williams, 2006; Hill-Haas, Coutts, Rowsell, & Dawson, 2008). The magnitude of difference (increased or decreased) were classified as 'important' when there was a ≥75% likelihood the true value of the statistic was practically or mechanistically important, and when there was <5% chance that statistic would occur in the opposite direction (Batterham & Hopkins, 2006; Hopkins, 2007). Effects with less

certainty were categorised as trivial, and when the likelihood that the true value of the statistic could occur in both directions was >5%, the effect was reported as unclear.

Chapter 4: Results

4.1 Participants

The average plasma osmolarity level determined across the experimental trials was 291.5 ± 2.5 mOsmol·kg^{-1}. Body weight loss during the HVY, SEV and CON trials were 0.97 ± 0.30, 0.93 ± 0.23, and 0.76 ± 0.24 kg respectively.

4.2 Warm-Up

4.2.1 $\dot{V}O_2$ responses

The $\dot{V}O_2$ profiles of the HVY, SEV, and CON are shown as relative $\dot{V}O_2$ (Figure 3), percentage of estimated critical velocity (Figure 4A), and percentage of $\dot{V}O_{2peak}$ (Figure 4B). There was an important difference in the $\dot{V}O_2$ response in the HVY and SEV compared to the CON from 30 s to 360 s (HVY & SEV > CON). An important difference in the $\dot{V}O_2$ response was also identified for the SEV compared to the HVY from the 120 s to 360 s (SEV > HVY). However, differences in the rate of $\dot{V}O_2$ change from the 120 s to 360 s for the HVY and SEV were only trivial.

4.2.2 Blood markers

There were only trivial differences for the blood markers before the start of the warm-up conditions (Table 2). However, an important increase in blood lactate and partial pressure of O_2 was detected pre-post HVY and pre-post SEV compared to pre-post CON. Furthermore, an important decrease in pH and HCO_3 was also observed pre-post HVY and pre-post SEV compared to pre-post CON. Partial pressure of CO_2 was importantly decreased but only for pre-post SEV compared to pre-post CON. There were only trivial differences between all warm-up conditions for sO_2 levels.

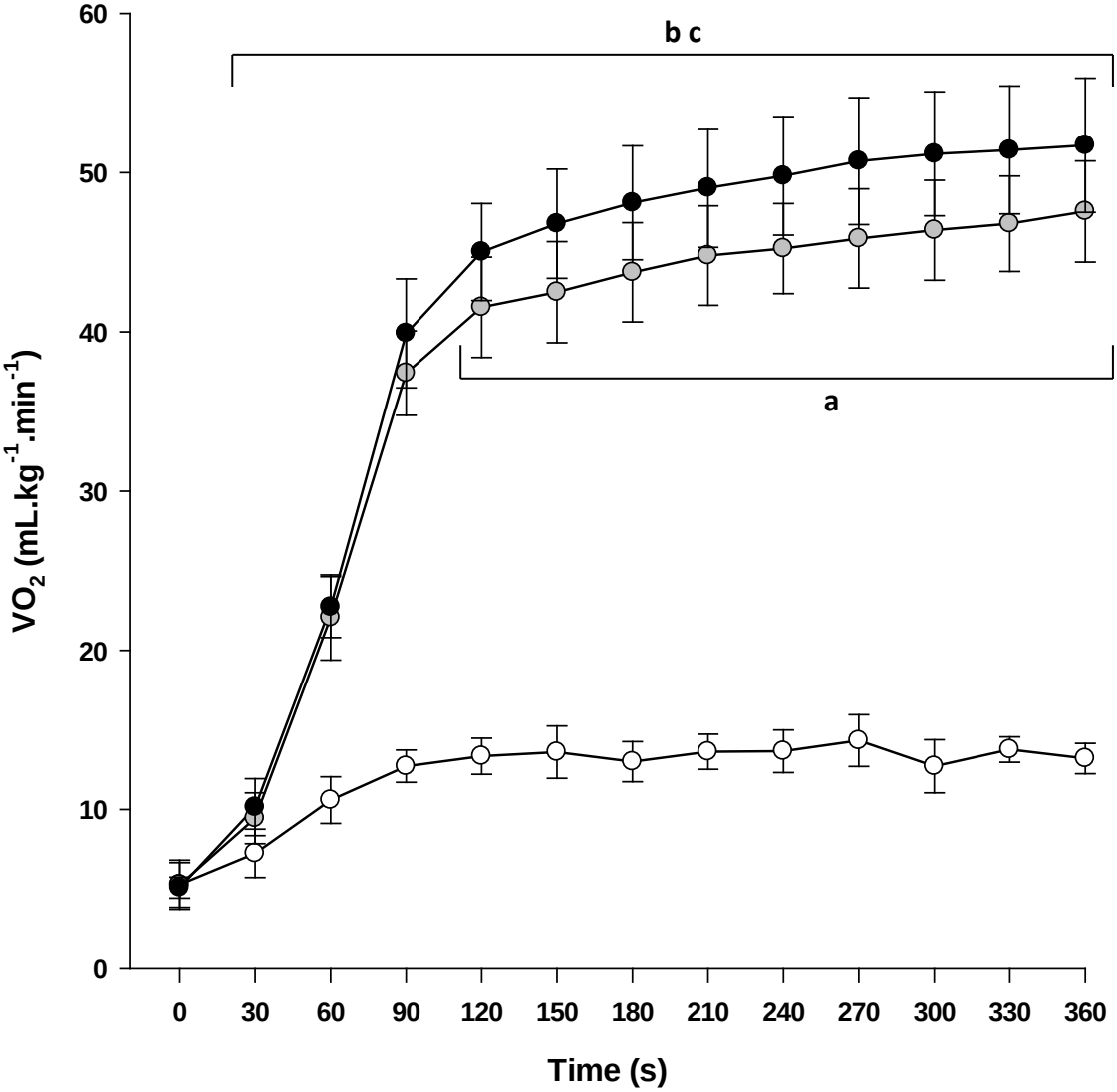


Figure 3. Relative $\dot{V}O_2$ profiles during the warm-up conditions. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between the SEV and HVY. 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

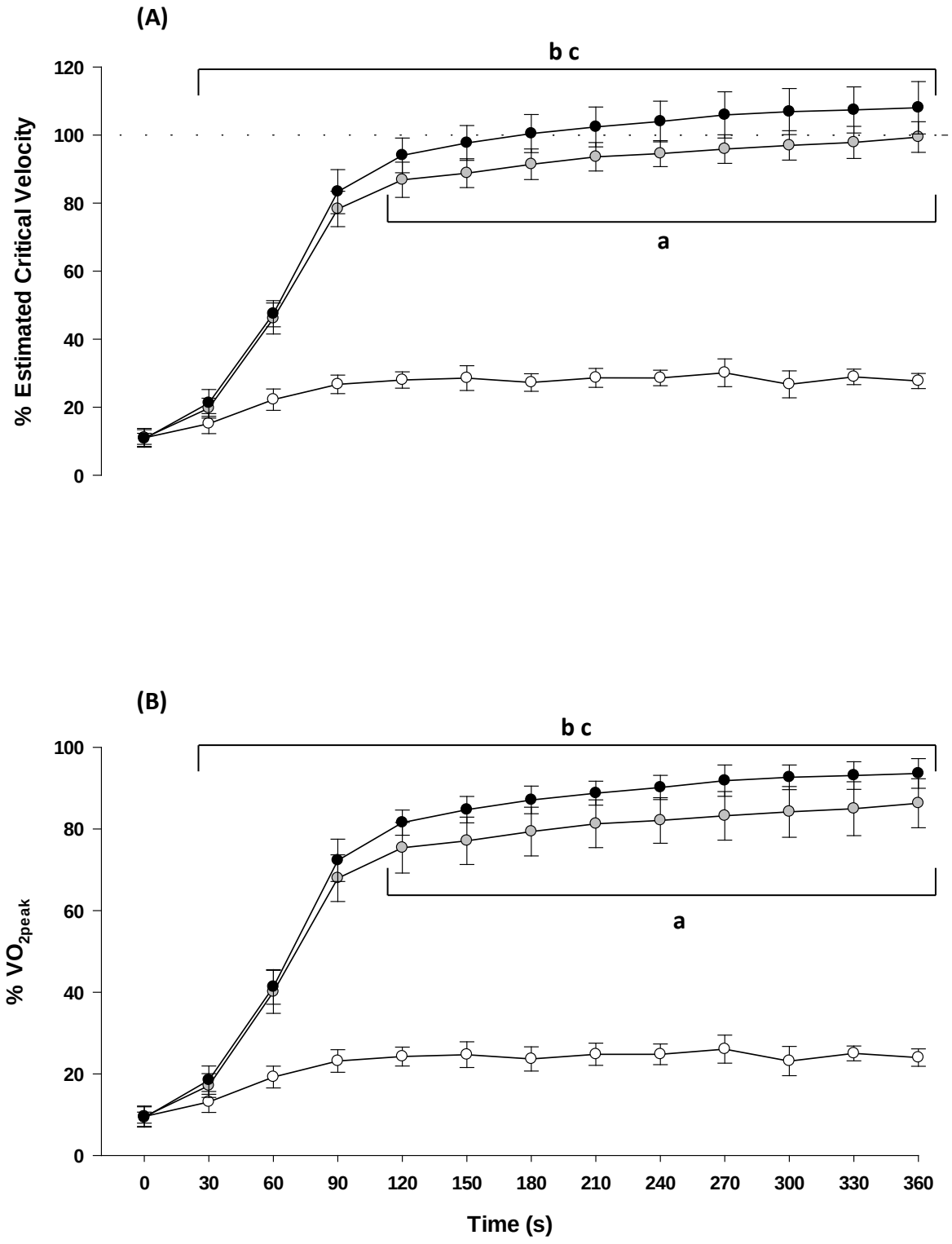


Figure 4. $\dot{V}O_2$ profiles as a percentage of estimated critical velocity (A) and $\dot{V}O_{2peak}$ (B) during the warm-up conditions. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between the SEV and HVY. 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

Table 2. The blood marker profiles pre-post warm-up conditions.

Blood Markers	Warm-Up Conditions	PRE-WU	POST-WU	(Δ)
pH	CON	7.42 ± 0.01	7.43 ± 0.01	0.00 ± 0.01
	HVY	7.42 ± 0.03	7.32 ± 0.04	-0.09 ± 0.05 b
	SEV	7.42 ± 0.02	7.18 ± 0.07	-0.24 ± 0.07 a c
Lactate ($mmol \cdot L^{-1}$)	CON	1.36 ± 0.42	0.90 ± 0.32	-0.46 ± 0.29
	HVY	1.31 ± 0.30	7.02 ± 2.86	5.71 ± 2.79 b
	SEV	1.31 ± 0.57	14.17 ± 2.80	12.87 ± 2.87 a c
HCO_3 ($mmol \cdot L^{-1}$)	CON	26.29 ± 2.05	26.25 ± 2.22	-0.05 ± 0.95
	HVY	25.84 ± 1.47	19.93 ± 3.19	-5.92 ± 2.93 b
	SEV	25.28 ± 1.64	13.57 ± 2.77	-11.72 ± 2.35 a c
PCO_2 (mmHg)	CON	40.31 ± 3.03	39.94 ± 3.45	-0.37 ± 1.21
	HVY	40.22 ± 3.28	38.03 ± 4.16	-2.18 ± 4.21
	SEV	39.44 ± 2.94	36.21 ± 3.55	-3.23 ± 2.55 c
PO_2 (mmHg)	CON	80.82 ± 9.47	83.36 ± 7.17	2.55 ± 10.22
	HVY	78.58 ± 9.57	98.00 ± 7.17	19.42 ± 7.76 b
	SEV	79.67 ± 12.60	101.67 ± 5.07	22.00 ± 11.81 c
sO_2 (%)	CON	95.91 ± 1.38	96.27 ± 1.35	0.36 ± 1.69
	HVY	95.33 ± 2.19	97.00 ± 0.60	1.67 ± 2.23
	SEV	95.17 ± 2.25	95.83 ± 1.11	0.67 ± 2.50

(Δ), rate of change; HCO_3, bicarbonate; PCO_2, partial pressure of carbon dioxide; PO_2, partial pressure of oxygen; sO_2, percentage saturation of oxygen in the blood. CON, control warm-up; HVY, heavy-intensity warm-up; SEV, severe-intensity warm-up. 'a' denotes important difference between SEV and HVY; 'b' denotes important difference between HVY and CON; 'c' denotes important difference between SEV and CON. Data mean ± SD.

4.2.3 Core Temperature

There were only trivial differences before the start of the warm-up conditions for T_c (Figure 5). An important increase in T_c (0.44 ± 0.20 v 0.41 ± 0.29^{o}C) was detected pre-post HVY and pre-post SEV compared to pre-post CON (0.19 ± 0.17^{o}C). However, the differences between T_c between the HVY and SEV were unclear.

4.2.4 Rating of Perceived Exertion

An important difference in RPE (4.5 ± 1.5 v 7.5 ± 1.8) was detected post-HVY and post-SEV compared to post-CON (1.0 ± 0.4) (SEV & HVY > CON). In addition, an important difference was also observed post-SEV compared to post-HVY (SEV > HVY) (Figure 6).

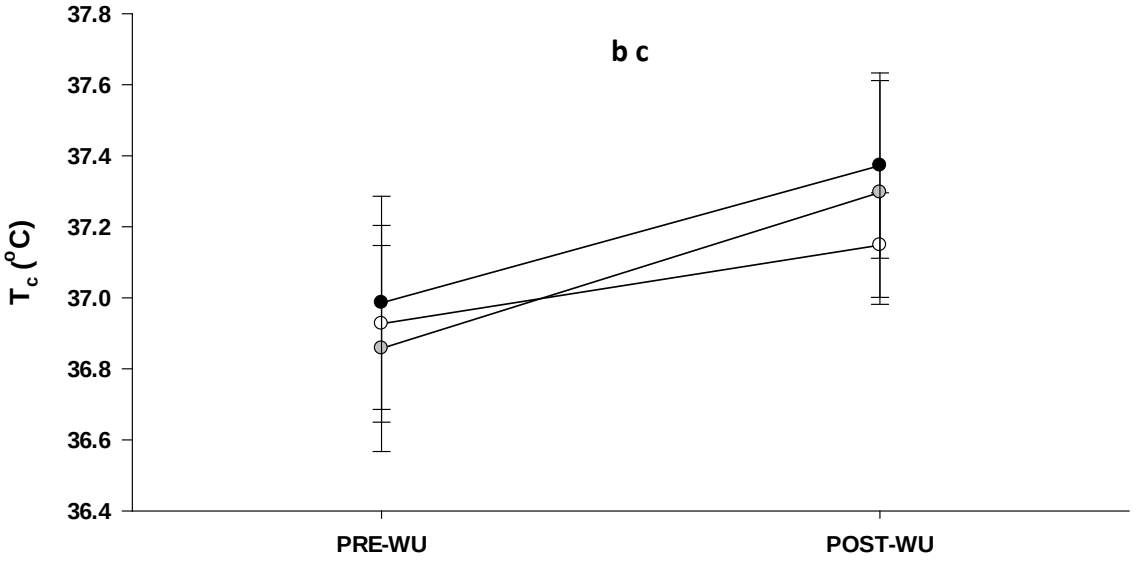


Figure 5. Changes in core temperature (T_c) pre-post warm-up conditions. white = CON, grey = HVY, and black = SEV. 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

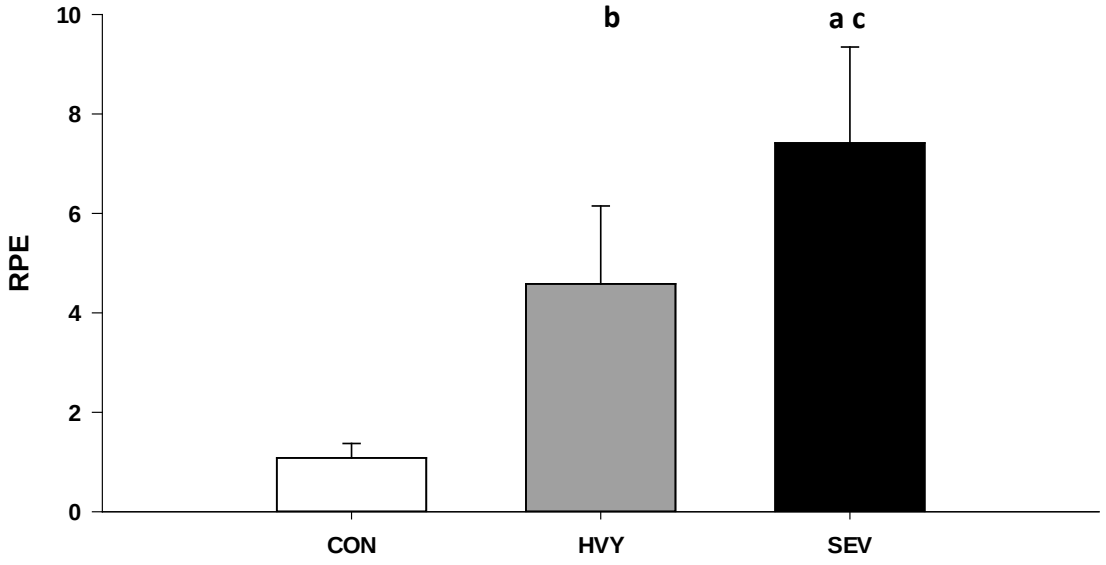


Figure 6. Rating of perceived exertion (RPE) post warm-up conditions. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between the SEV and HVY; 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

4.3 Team-Sport Simulation

4.3.1 Performance

The total distance covered of the TSS between the HVY, SEV and CON were trivially difference (Figure 7). The distances covered performing the speed activities of the TSS were also only trivially different between the warm-up conditions (Figure 8).

Trivial differences were found between the HVY, SEV and CON for average speed generated within the 0-4, 4-8, and 20-30 min time periods. However, there was an important decrease in average speed within the 8-20 min time period (2.25 ± 0.24 v 2.38 ± 0.23 $m \cdot s^{-1}$) for the SEV compared to the CON (SEV < CON) (Figure 9).

There were only trivial differences between the warm-up conditions for average speed walking, running and sprinting within all time periods of the TSS. Though, there was an important decrease in average speed jogging during the 8-20 min time period (2.43 ± 0.28 v 2.60 ± 0.27 $m \cdot s^{-1}$) in the SEV compared to the CON (SEV < CON) (Figure 10B).

The relative contribution of walking, jogging and running to the total distance covered throughout the TSS, and within all time-periods, was only trivial between the warm-up conditions. However, the relative contribution of sprinting to the total distance covered throughout the TSS (17.4 ± 1.4 v 16.9 ± 1.1%), and within time-periods 0-4 min (23.3 ± 2.3 v 22.4 ± 2.0%) and 8-20 min (14.5 ± 1.3 v 14.1 ± 1.0%) was importantly increased in the SEV compared to the CON (SEV > CON) (Table 3).

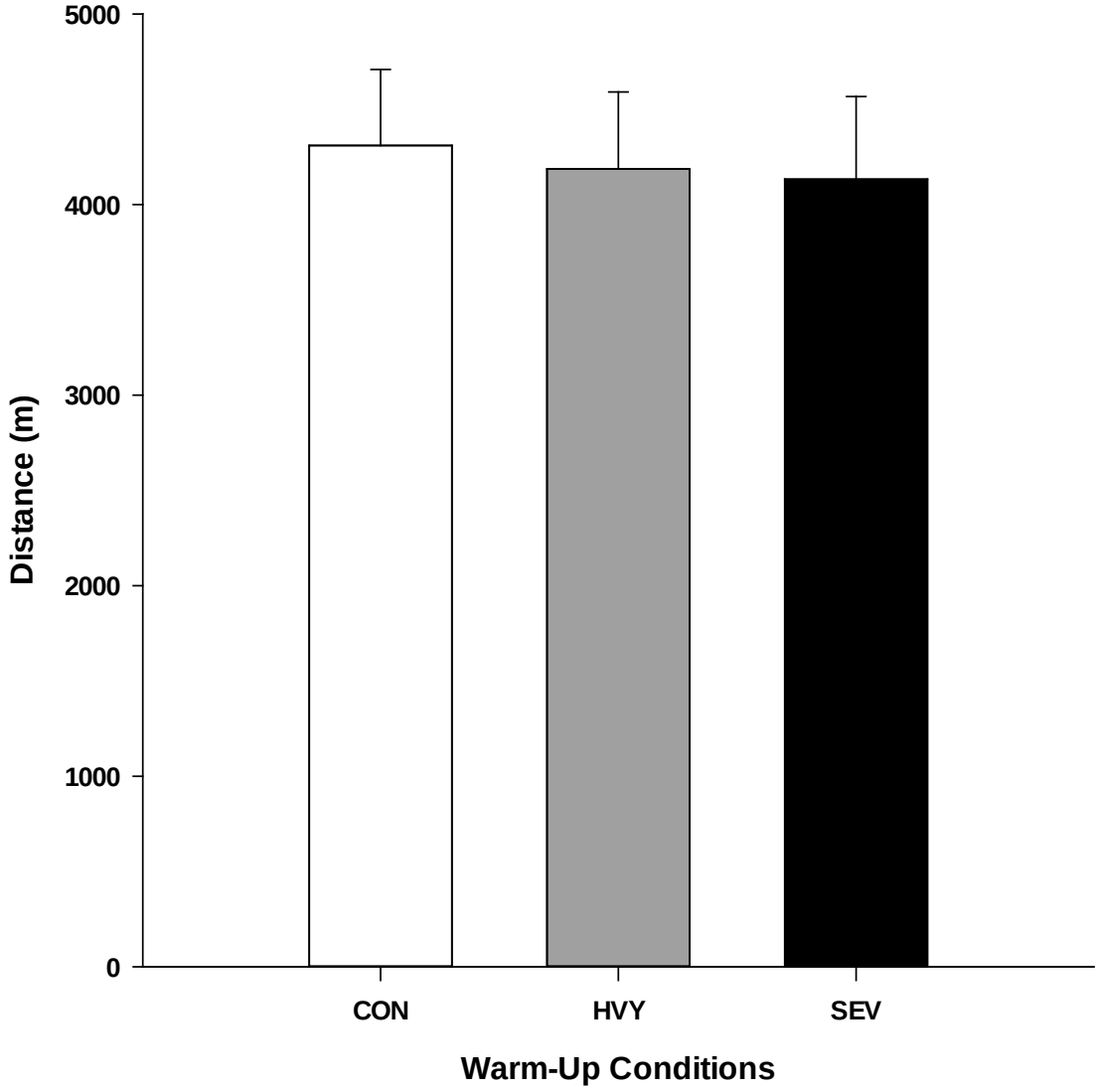


Figure 7. Total distance covered during the team-sport simulation. white = CON, grey = HVY, and black = SEV. Data mean ± SD.

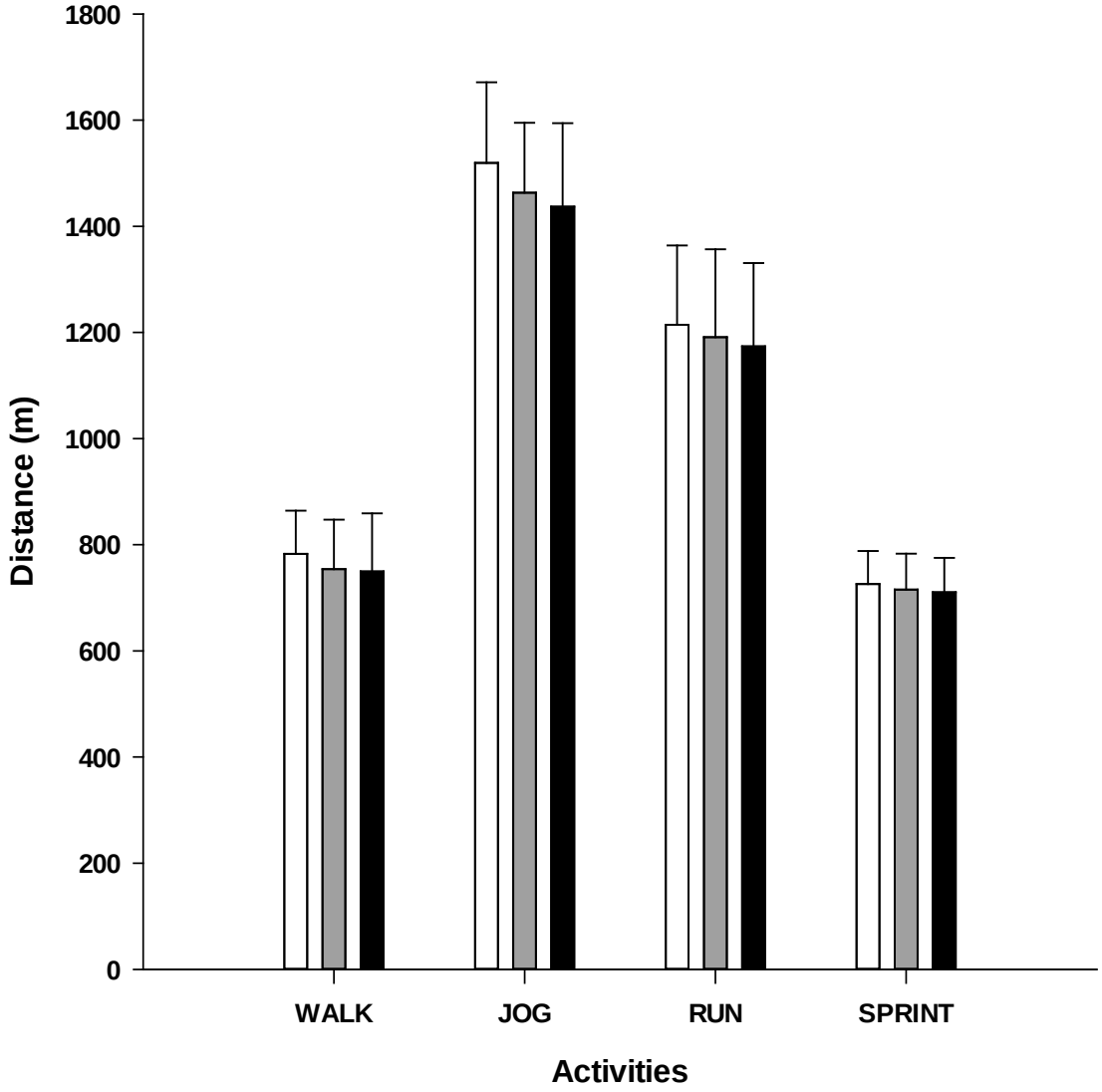


Figure 8. Distance covered performing the speed activities of the team-sport simulation. white = CON, grey = HVY, and black = SEV. Data mean ± SD.

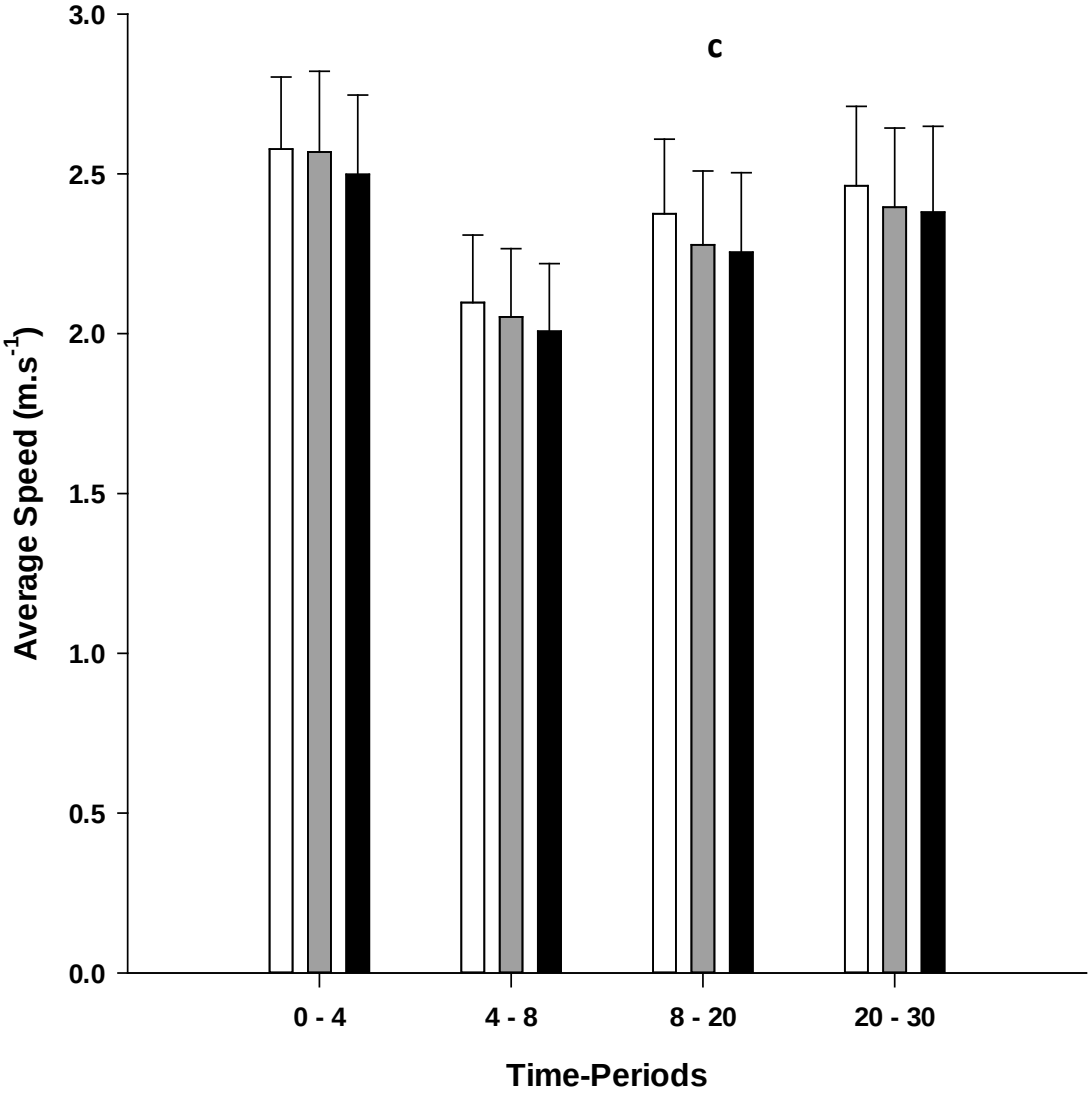


Figure 9. Average speed within the time-periods of the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'c' denotes important difference between the SEV and CON. Data mean ± SD.

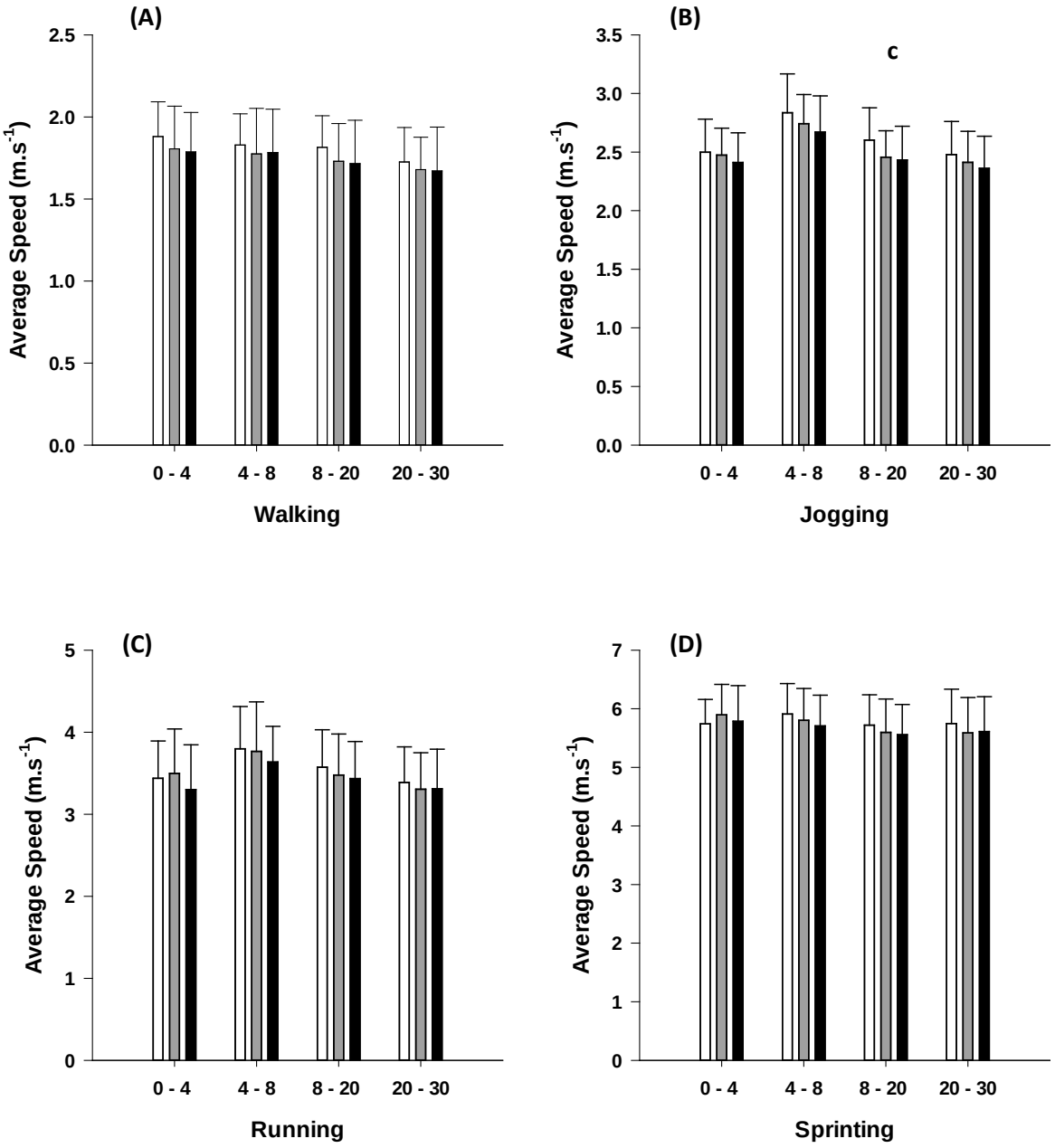


Figure 10. Average speed of walking (A), jogging (B), running (C) and sprinting (D) within the time-periods of the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'c' denotes important difference between the SEV and CON. Data mean ± SD.

Table 3. The relative contribution (%) of walking, jogging, running and sprinting to the total distance covered during the team-sport simulation.

Activities	Warm-Up Conditions	Team-Sport Simulation				
		0 - 4 min	4 - 8 min	8 - 20 min	20 - 30 min	Full Trial
Walking	CON	20.68 ± 1.67	20.37 ± 1.22	16.83 ± 0.83	17.91 ± 1.04	18.17 ± 0.92
	HVY	19.87 ± 1.58	20.13 ± 1.73	16.69 ± 1.16	17.94 ± 1.18	18.06 ± 1.20
	SEV	20.25 ± 1.75	20.67 ± 1.46	16.72 ± 1.40	17.89 ± 1.17	18.13 ± 1.33
Jogging	CON	33.91 ± 1.78	32.65 ± 1.47	36.63 ± 1.54	35.10 ± 1.51	35.25 ± 1.35
	HVY	33.75 ± 1.21	32.36 ± 1.23	36.11 ± 1.30	35.14 ± 1.26	34.92 ± 1.14
	SEV	33.79 ± 1.62	32.14 ± 1.31	36.06 ± 1.42	34.64 ± 1.83	34.63 ± 1.32
Running	CON	21.07 ± 1.63	24.08 ± 1.24	30.96 ± 1.95	29.23 ± 2.16	28.14 ± 1.77
	HVY	21.52 ± 2.11	24.42 ± 2.54	31.40 ± 2.56	29.29 ± 2.24	28.35 ± 2.47
	SEV	20.85 ± 2.13	24.17 ± 1.90	31.39 ± 2.29	29.53 ± 2.46	28.37 ± 2.28
Sprinting	CON	22.38 ± 1.99	21.21 ± 1.64	14.14 ± 1.03	16.31 ± 1.04	16.89 ± 1.18
	HVY	23.03 ± 1.60	21.28 ± 1.52	14.42 ± 1.12	16.29 ± 1.07	17.17 ± 1.17
	SEV	23.25 ± 2.32 c	21.41 ± 1.67	14.50 ± 1.27 c	16.51 ± 1.38	17.40 ± 1.47 c

CON, control warm-up; HVY, heavy-intensity warm-up; SEV, severe-intensity warm-up. ‘c’ denotes important difference between SEV and CON. Data mean ± SD.

There were only trivial differences between the SEV and CON, and the HVY and SEV for average speed of 1st sprint. However, an important increase in average speed of 1st sprint (6.09 ± 0.47 v 5.76 ± 0.45 $m.s^{-1}$) was determined in the HVY compared to the CON (HVY > CON) (Figure 11A). Trivial differences were found between the warm-up conditions for average speed of the 1st, 2nd and final repeated-sprint clusters (Figure 11B).

For the percentage difference between average speed of the 1st sprint and average speed within the 1st repeated-sprint cluster, trivial differences were determined between the HVY, SEV and CON. However, important decreases in performances were found within the 2nd (96.1 ± 10.2 v 95.0 ± 8.2%) and final (90.7 ± 12.3 v 92.6 ± 9.5%) repeated-sprint clusters for the HVY and SEV compared to the CON (102.9 ± 9.2%, 99.4 ± 14.0%) (HVY & SEV < CON) (Figure 12).

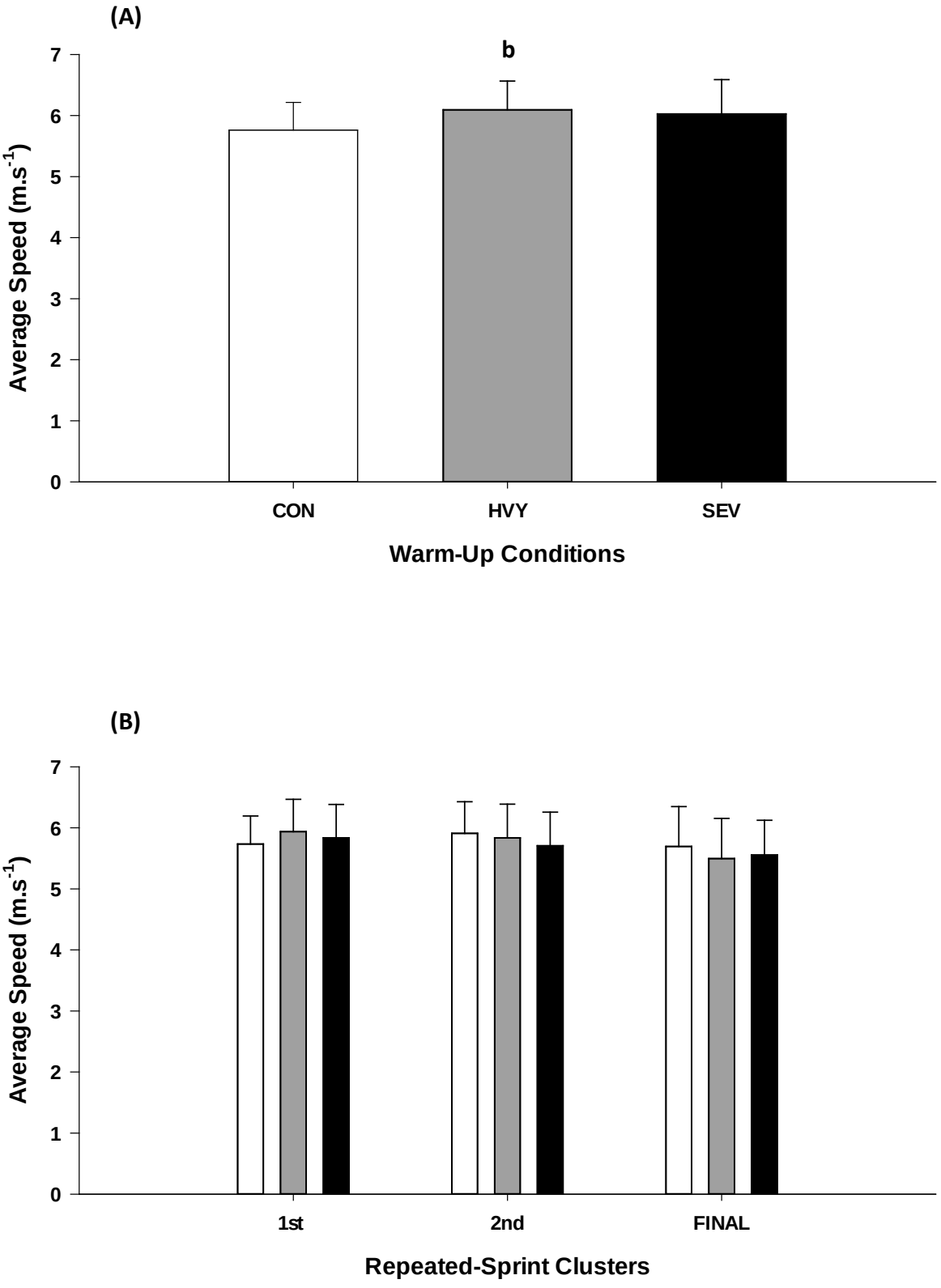


Figure 11. Average speed of 1st sprint (A) and average speed of the 1st, 2nd and final repeated-sprint clusters (B) during the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'b' denotes important difference between the HVY and CON. Data mean ± SD.

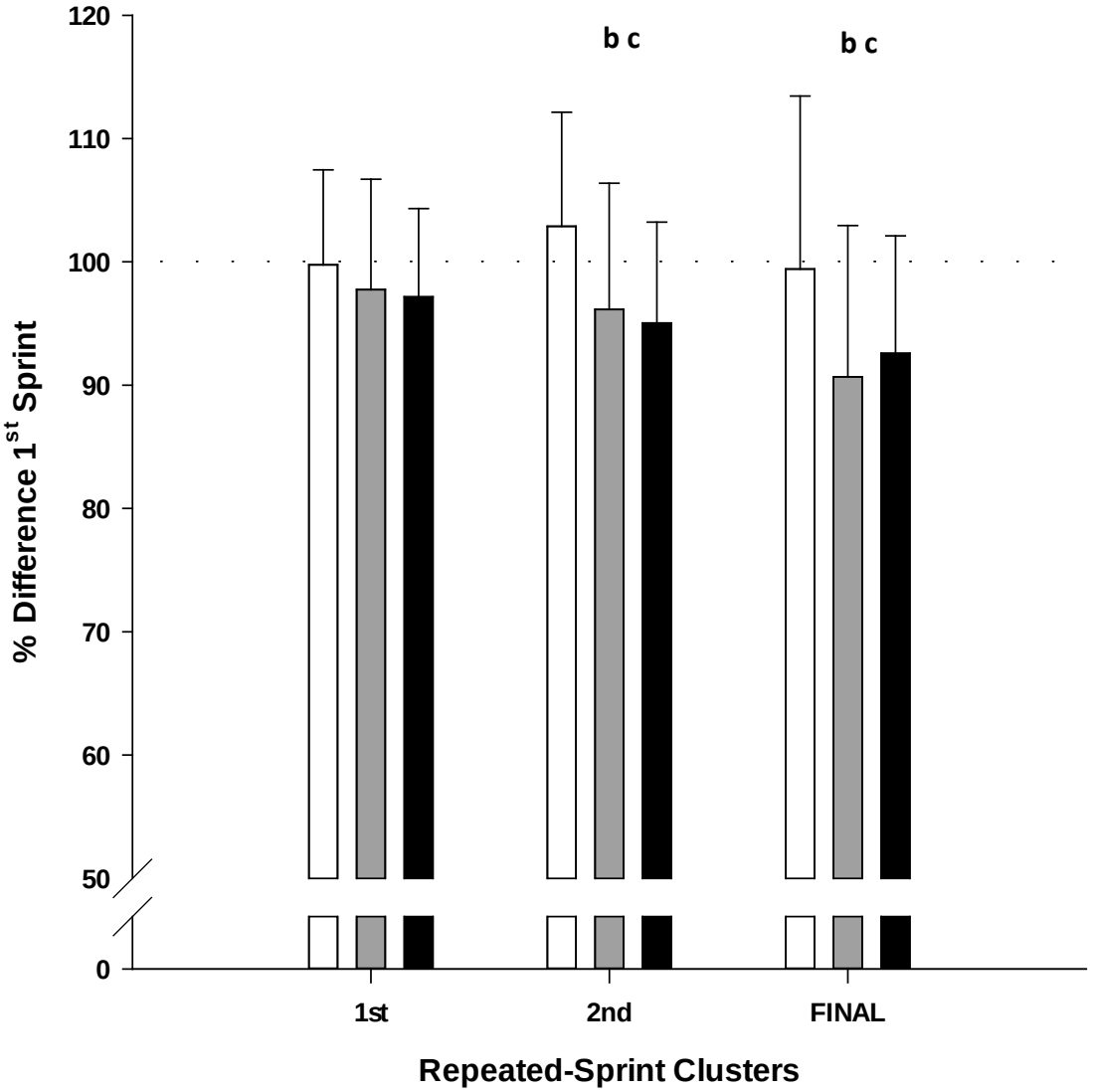


Figure 12. Average speed of repeated-sprint clusters compared to average speed of 1st sprint during the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

4.3.2 Blood Markers

There were important increases for lactate (3.3 ± 2.0 v 9.1 ± 3.6 mmol·L^{-1}) and decreases for HCO_3 (23.4 ± 2.4 v 17.1 ± 3.9 mmol·L^{-1}) at the start of the TSS in the HVY and SEV compared to the CON (0.8 ± 0.3 mmol·L^{-1}, 26.0 ± 2.1 mmol·L^{-1}) (Figure 13). There was also important differences in pH (7.3 ± 0.0 v 7.4 ± 0.0), PCO_2 (34.6 ± 3.6 v 39.8 ± 4.4 mmHg) (SEV < CON) and PO_2 (88.4 ± 7.8 v 81.0 ± 7.1 mmHg) (SEV > CON) in the SEV compared to the CON (Figure 13 & 14). Lactate (SEV > HVY), HCO_3, PCO_2 and pH (SEV < HVY) in the SEV compared to the HVY (PCO_2 – 38.6 ± 3.8 mmHg, pH – 7.4 ± 0.0) were also importantly different at the beginning of the TSS.

An important reduction in pH within the 0-4 min time period was detected in the HVY and SEV compared to the CON, with the SEV reduced more than the HVY. Furthermore, an important reduction in pH was determined between the SEV and CON during the 4-8 min time-period. Trivial differences were found between warm-up conditions for PCO_2 throughout the TSS, except for an important increase observed within the 8-30 min time-period for the SEV compared to the HVY and CON. There were only trivial differences between the warm-up conditions for sO_2 and PO_2 throughout the TSS (Figure 13 & 14).

An important attenuation of elevated lactate accumulation, and a reduced decrease in HCO_3, within the 0-4 min and 4-8 min time-periods, was detected in the HVY and SEV compared to the CON, with the SEV affected more than the HVY. An important attenuated elevation in lactate and a reduced decrease in HCO_3 were determined in the SEV and CON within the 8-30 min time-period (Table 4).

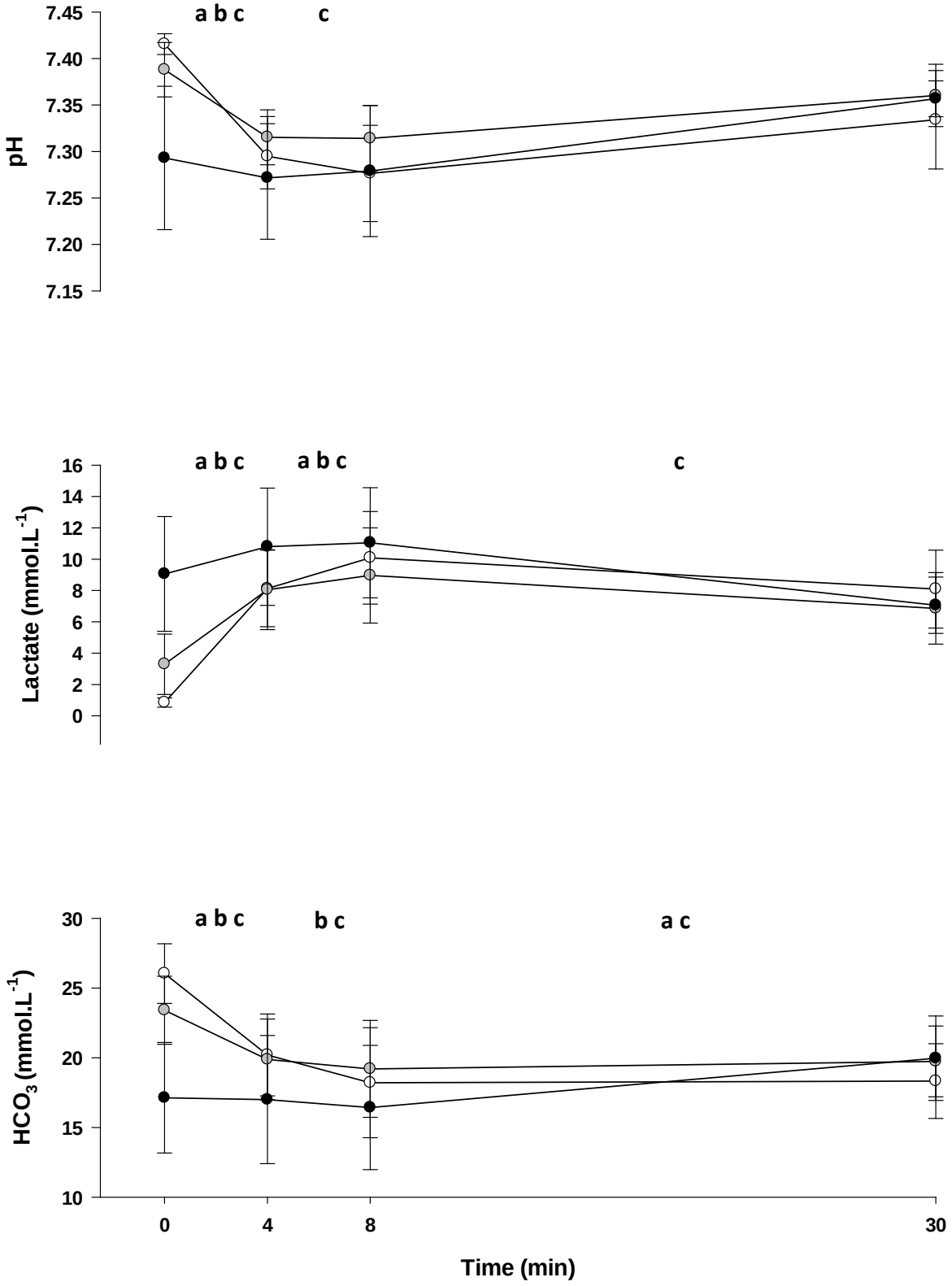


Figure 13. Blood marker profiles of pH, lactate and HCO_3 throughout the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between the SEV and HVY. 'b' denotes important difference between the HVY and CON; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

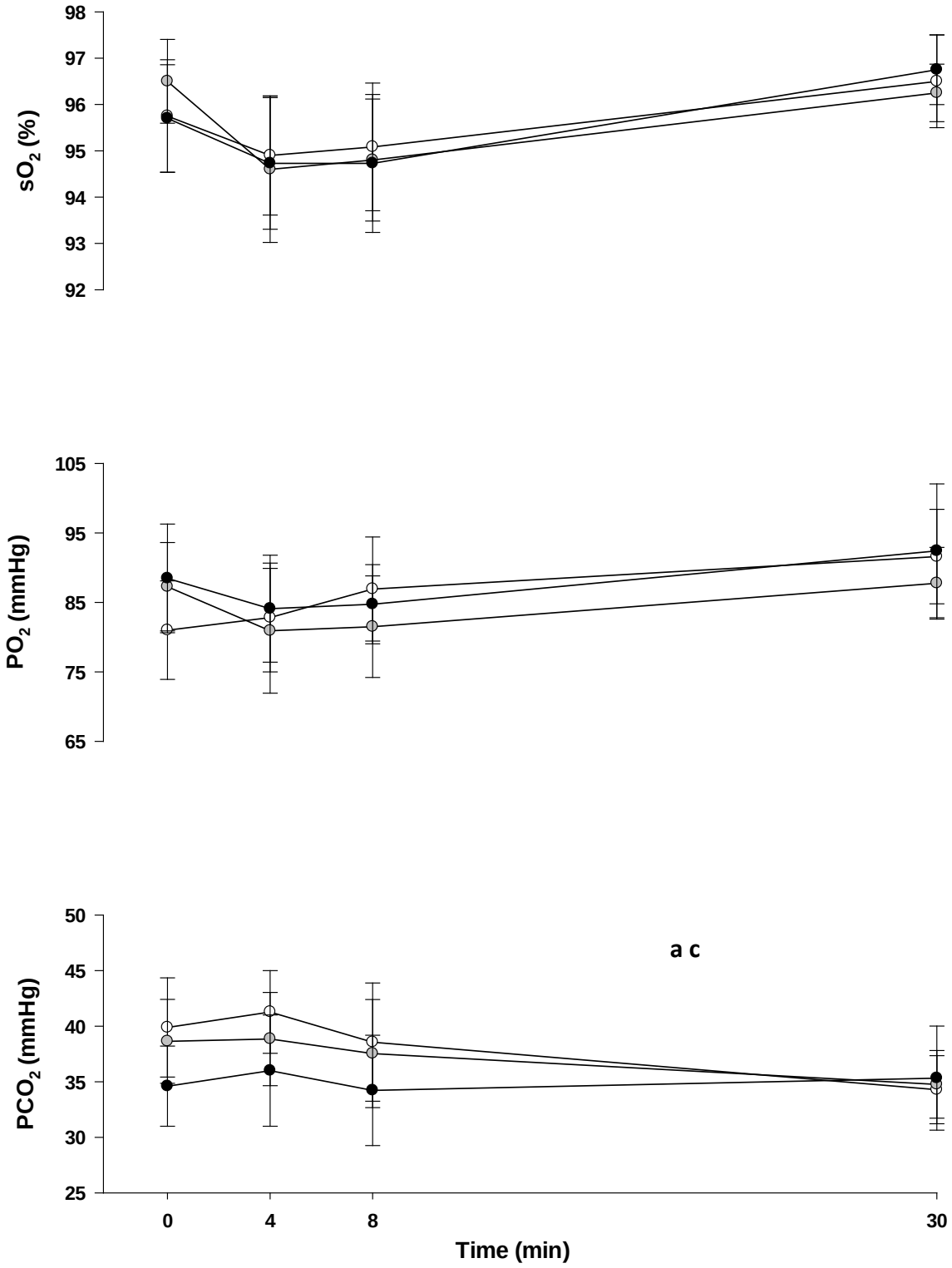


Figure 14. Blood marker profiles of sO_2, PO_2 and PCO_2 throughout the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between the SEV and HVY; 'c' denotes important difference between the SEV and CON. Data mean ± SD.

Table 4. Differences in the rate of change of blood markers throughout the team-sport simulation.

Blood Markers	Warm-Up Conditions	Team-Sport Simulation		
		0 - 4 min (Δ)	4 - 8 min (Δ)	8 - 30 min (Δ)
pH	CON	-0.12 ± 0.04	-0.02 ± 0.02	0.06 ± 0.05
	HVY	-0.07 ± 0.04 b	0.00 ± 0.01	0.05 ± 0.04
	SEV	-0.02 ± 0.07 a c	0.02 ± 0.03 c	0.08 ± 0.06
Lactate ($mmol \cdot L^{-1}$)	CON	7.28 ± 2.46	1.92 ± 0.82	-2.00 ± 2.68
	HVY	4.62 ± 2.09 b	0.68 ± 0.90 b	-2.35 ± 2.52
	SEV	1.59 ± 3.05 a c	-0.40 ± 1.38 a c	-3.68 ± 2.71 c
HCO_3 ($mmol \cdot L^{-1}$)	CON	-5.82 ± 1.83	-2.31 ± 1.61	0.12 ± 2.68
	HVY	-3.51 ± 2.14 b	-0.56 ± 1.65 b	0.67 ± 2.43
	SEV	0.20 ± 3.19 a c	-0.22 ± 1.10 c	3.29 ± 2.93 a c
PCO_2 (mmHg)	CON	1.45 ± 2.38	-3.02 ± 3.68	-4.28 ± 3.61
	HVY	0.27 ± 3.45	-1.08 ± 2.82	-2.73 ± 2.67
	SEV	1.70 ± 3.03	-1.94 ± 1.98	0.84 ± 3.76 a c
PO_2 (mmHg)	CON	1.09 ± 8.08	4.18 ± 8.40	4.67 ± 6.91
	HVY	-5.27 ± 7.25	1.70 ± 4.11	7.40 ± 6.92
	SEV	-5.20 ± 10.05	-0.10 ± 3.41	7.91 ± 10.25
sO_2 (%)	CON	-0.80 ± 0.79	0.20 ± 1.55	1.42 ± 1.44
	HVY	-1.90 ± 1.60	0.44 ± 1.13	1.60 ± 1.07
	SEV	-1.00 ± 1.41	0.00 ± 1.15	2.00 ± 1.34

(Δ), rate of change; CON, control warm-up; HVY, heavy-intensity warm-up; SEV, severe-intensity warm-up. ‘a’ denotes important difference between SEV and HVY, ‘b’ denotes important difference between HVY and CON, ‘c’ denotes important difference between SEV and CON.

4.3.3 Core Temperature

There were important differences in T_c (37.2 ± 0.2 v 37.4 ± 0.2 v 37.7 ± 0.3^{o}C) at the start of the TSS for the CON, HVY and SEV (SEV > HVY > CON). Trivial differences between the rates of change of T_c occurred during the 0-4, 4-8, and 20-30 min time-periods. However, an important attenuation in T_c (0.8 ± 0.2 v 0.6 ± 0.1 ^{o}C) was detected in the HVY and SEV compared to the CON (0.9 ± 0.2 ^{o}C) (HVY & SEV < CON) during the 8-20min time-period, with SEV importantly less increasing than HVY (HVY > SEV) (Figure 15A).

4.3.4 Rating of Perceived Exertion

Only trivial differences were determined between the warm-up conditions within the 4-8 and 20-30 min time-periods for RPE. In contrast, an important decrease in perception of effort (3.2 ± 2.1 v 3.7 ± 0.8 v 3.9 ± 1.6) was found in the SEV compared to the CON and HVY during the 0-4 min time period (SEV < HVY & CON). An important decrease in perceived effort (0.7 ± 0.8 v 1.7 ± 1.0) was also found in the SEV compared to the CON within the 8-20 min time-period (SEV < CON). There were only trivial differences between the warm-up conditions for overall trial RPE (Figure 15B).

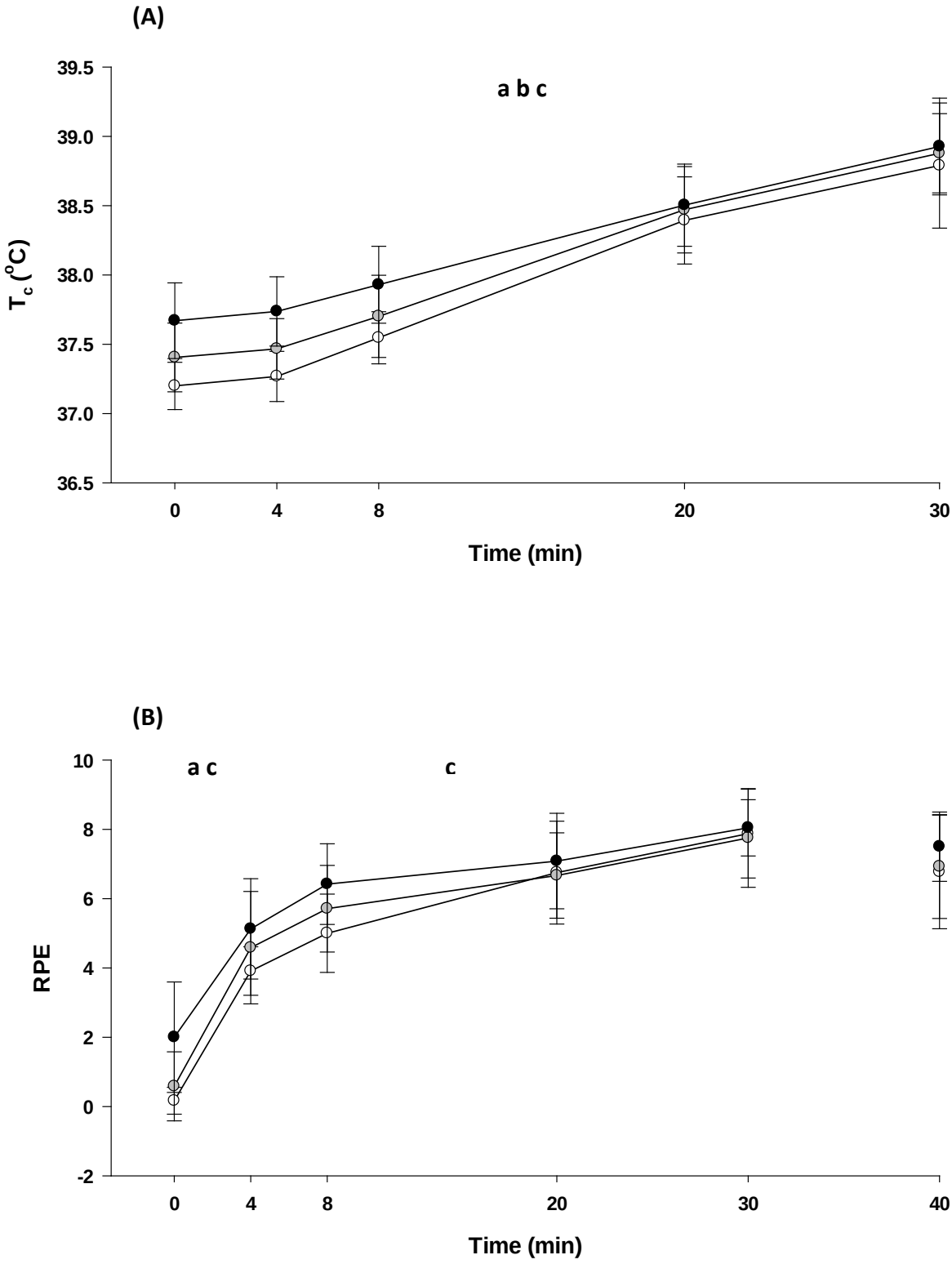


Figure 15. Changes in core temperature (T_c) (A) and rating of perceived exertion (RPE) (B) throughout the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'a' denotes important difference between SEV and HVY. 'b' denotes important difference between the HVY and CON. 'c' denotes important difference between the SEV and CON. Data mean ± SD.

4.3.5 Ventilation

There was an important difference in baseline $\dot{V}O_2$ (0.44 ± 0.05 v 0.40 ± 0.07 L.min^{-1}) and $\dot{V}_E$ (18.4 ± 4.0 v 15.1 ± 2.5 L.min^{-1}) before beginning the TSS for the SEV compared to the CON (SEV > CON). In addition, there was an important attenuation in $\dot{V}_E$ (285.3 ± 40.1 v 264.6 ± 36.9 L.min^{-1}) in the SEV compared to the CON during the 0-4 min time-period of the TSS (SEV < CON) (0 = baseline ventilation). There were only trivial differences between conditions for $\dot{V}O_2$ during the TSS. This was also observed for $\dot{V}_E$ after the 0-4 min time period (Figure 16).

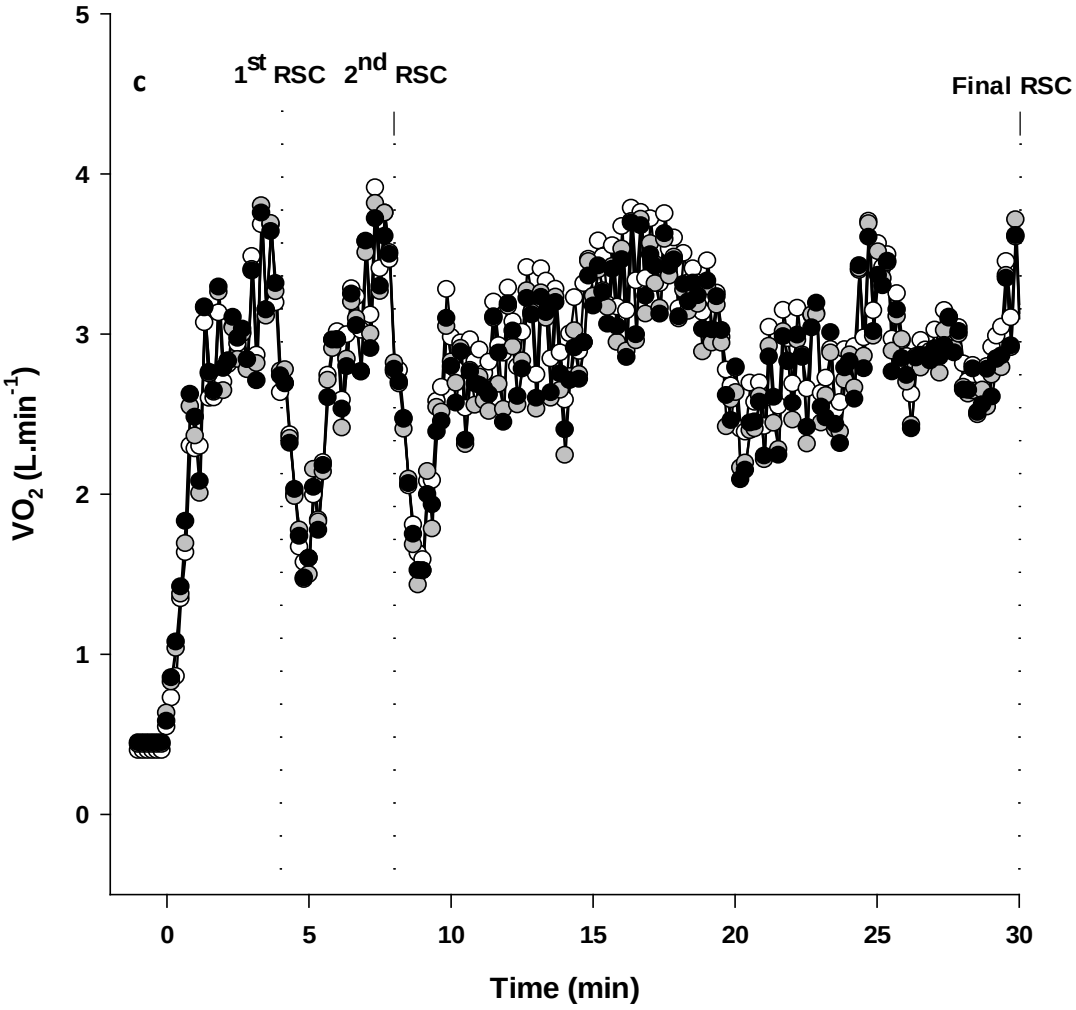


Figure 16. Changes in $\dot{V}O_2$ during the team-sport simulation. white = CON, grey = HVY, and black = SEV. 'c' denotes important difference between the SEV and CON. Data mean ± SD.

Chapter 5: Discussion

The primary finding of this research was that both a heavy-intensity (HVY) and severe-intensity (SEV) warm-up provided little performance benefit compared to the CON during the team-sport running simulation (TSS). At the beginning of the TSS, *important* differences were observed in T_c, pH, lactate, HCO_3, PO_2, PCO_2 and baseline $\dot{V}O_2$ and $\dot{V}_E$ following the SEV and CON. However, these differences were dissipated across the TSS and by the end all physiological parameters were similar between conditions.

The expected outcomes of the study were that the total distance covered during the TSS would be greater following the SEV compared to the HVY and CON. Based on previous reports, it was expected that the SEV would create a 'priming effect' that would enhance the aerobic metabolism contribution during the TSS (Gerbino et al., 1996). The upregulation of aerobic metabolism was expected to improve jogging and running activity profiles, and improve repeated-sprint performance because of enhanced phosphocreatine kinetics and reduced accumulation of fatigue-related metabolites (Forbes et al., 2008). However, the differences in total distance covered between warm-up conditions were determined to be *trivial*. Furthermore, the differences in the distance covered performing the speed activities of the TSS, including jogging and running, were also found to be *trivial* between the warm-up conditions.

A possible explanation for the trivial findings is the task-dependent nature of the priming effect. A number of studies have determined that a supralactate warm-up can alter subsequent $\dot{V}O_2$ kinetics while cycling. The major modification to $\dot{V}O_2$ kinetics after performing a supralactate warm-up is a reduced slow component amplitude (Burnley et al., 2000). However, modifications to the $\dot{V}O_2$ kinetic profile have not been observed while running, even though the same relative intensities have been prescribed between modalities (Jones, DiMenna, et al., 2008).

Previously, the priming effect on $\dot{V}O_2$ kinetics had been shown to be intensity dependent (Gerbino et al., 1996), making the warm-up intensity the presumed primary modifiable factor which affects subsequent metabolism. Instead, the priming of subsequent $\dot{V}O_2$ kinetics following a warm-up may be exclusive to cycling. For example, differences in the $\dot{V}O_2$ kinetic profile are observed between cycling and running in a 'un-primed' condition (before intervention). The primary component time-constant tends to be shorter and the slow component is smaller during running compared to cycling (Billat et al., 1998; Carter et al., 2000; Jones & McConnell, 1999). It is speculated that differences in active muscle mass, muscle contraction regimen, duty cycle, blood flow, fatigue resistance, and fibre recruitment between the exercise modes may influence the varying 'un-primed' $\dot{V}O_2$ profiles (Jones, DiMenna, et al., 2008). With running exercise having a fast 'un-primed' $\dot{V}O_2$ kinetic profile, any improvements expected by the SEV were most likely superfluous.

The priming effect on $\dot{V}O_2$ kinetics following a supralactate warm-up had also been shown to occur during subsequent supralactate exercise. Conversely, the same supralactate warm-up provided no effect on priming $\dot{V}O_2$ kinetics during subsequent sublactate exercise (Gerbino et al., 1996). The $\dot{V}O_2$ profile during sublactate exercise can reach a steady-state in ~3 min, and can be described without a slow component. The attenuated slow component during sublactate exercise offers little benefit to perform a warm-up designed to improve $\dot{V}O_2$ kinetics. Because the priming effect mainly reduces the slow component amplitude, expectations of improvements in submaximal activity profiles (walking, jogging and running) may have been unfounded. With these activities likely occurring at work-rates near or below lactate threshold, the priming effect may not have modified metabolism at these intensities.

It is also suggested that warm-up protocols designed to influence $\dot{V}O_2$ kinetics may be more effective when prescribed to participants who are not conditioned in the chosen exercise modality (Jones, DiMenna, et al., 2008). In the 'un-primed' condition, participants who present with longer

primary component time-constant and larger slow component $\dot{V}O_2$ kinetic profiles, may be more responsive to the effects of a supralactate warm-up (Carter et al., 2000; Jones, DiMenna, et al., 2008). The participants in this research had similar running capacities to other studies, and were accustomed to the demands of team-sport running. It is likely the participants may have possessed fast 'un-primed' $\dot{V}O_2$ kinetics because of their current fitness levels. It is also speculated that the priming effect may vary from person to person, making some individuals more responsive to the warm-up protocols than others (Jones, DiMenna, et al., 2008).

Numerous studies have shown that supralactate warm-ups reduce the appearance of lactate and hydrogen ions, increase O_2 utilisation and acetylcarnitine concentration (Bailey et al., 2009; Forbes et al., 2008) and blunt motor unit recruitment (Burnley et al., 2002b) during supine lower-body and cycling exercise. These priming effects are associated with the changes in the $\dot{V}O_2$ kinetics response. It has been speculated that these effects are still generated during subsequent running exercise, but they may not measurably impact $\dot{V}O_2$ kinetics. For example, a reduced rate of blood lactate accumulation has been observed during subsequent running exercise, even though the $\dot{V}O_2$ kinetic profile was unaltered (Jones, DiMenna, et al., 2008).

In this study, a reduced rate of blood lactate accumulation was observed during the 0-4 min and 4-8 min time-periods for the SEV compared to the CON. Concurrently, a reduced drop in blood pH and HCO_3 concentration was seen during the same time-periods. The changes in lactate, pH and HCO_3 possibly demonstrate a reduced reliance on anaerobic metabolism at the start of the TSS for the SEV.

On the other hand, increased baseline acidification at exercise onset can also cause changes in the mobilisation and utilisation of anaerobic and aerobic substrates (Taylor, 1975). The increased baseline acidification can also alter the efflux of lactate from the muscle (Juel, 2001), making it difficult to interpret the reasons for the modified blood acid-base profiles. More research is required

to ascertain if priming effects generated by a supralactate warm-up can be observed during subsequent running exercise.

There were *important* underlying performance differences between the SEV and CON during the TSS. The average speed and average speed jogging during the 8-20 min time-period was greater in the CON compared to the SEV. This result was contradictory to the expected outcomes. It was expected the SEV would enhance the total distance covered by improving submaximal activity profiles. However, the consequences of performing a SEV may have overshadowed the possible benefits.

The expected detrimental effects of performing a SEV include reductions in phosphocreatine and pH, and increased accumulation of inorganic phosphates and blood lactate (Jones, Wilkerson, et al., 2008). The commencement of subsequent exercise with these effects present has been shown to negatively affect performance (Bishop, Lawrence, & Spencer, 2003; Jones, Wilkerson, et al., 2008).

Conversely, intramuscular phosphocreatine stores and hydrogen ion concentrations are restored back to baseline levels within 15 min following 6 min of heavy-intensity plantar-flexion exercise (Forbes et al., 2008). Furthermore, anaerobic work capacity, which reflects how anaerobic energy stores and fatigue-related metabolites may influence performance, has been shown to approximate baseline levels after severe-intensity exercise within 15 min (Ferguson et al., 2010). The negative effects of performing a SEV should be nullified, and should not impact subsequent exercise when a 20 min rest period is prescribed. However, in this study, blood acid-base profiles had not returned to expected levels before the beginning of the TSS.

A previous investigation reported blood lactate levels of 8.0 ± 1.6 $mmol \cdot L^{-1}$ post-SEV and had decreased to 3.0 ± 0.8 $mmol \cdot L^{-1}$ after a 20 min passive recovery (Bailey et al., 2009). In the current study, blood lactate reached 14.2 ± 2.8 $mmol \cdot L^{-1}$ post-SEV and only decreased to 9.1 ± 3.6 $mmol \cdot L^{-1}$

after 20 min passive recovery. Consequently, the anaerobic work capacity was not likely restored before the start of the TSS (Bailey et al., 2009), which may explain the different performance results during the 8-20 min time period.

In opposition of the suggestion that the varied baseline blood markers caused the performance decrements during the 8-20 min time-period is the absence of a performance change at the start of the TSS. It would be expected, if the different blood marker concentrations affected performance, that performance would be impaired at the beginning of the TSS. The blood lactate concentration was well above the considered optimal pre-trial blood lactate range. In fact, starting severe-intensity exercise with a baseline blood lactate of ≥6 mmol·L^{-1} has been shown to significantly decrease performance (Burnley, Doust, & Jones, 2005a).

At the 8 min mark of the TSS, blood pH, lactate, HCO_3 and $\dot{V}O_2$ were *trivially* different between the SEV and CON. Furthermore, the rate of change of blood pH and $\dot{V}O_2$ during the 8-30 min time period was also *trivially* different. This would suggest that the participants were exercising under the same physiological constraints at the 8 min mark, and during the middle and final stages of the TSS. However, performance differences were still observed. It is likely other mechanisms may have caused the performance decrements.

Another explanation for the negative performance result during the 8-20 min time-period is temperature regulation. At the beginning of the TSS, there was an *important* difference found for T_c between the SEV and CON (SEV > CON). Previously, the commencement of constant-rate exercise with an elevated T_c had been shown to be inversely related to time-to-exhaustion performance (González-Alonso et al., 1999). Reaching an earlier critical hyperthermic T_c because of the elevated baseline T_c was acknowledged as the cause of the performance decrement. This theory has been referred to as the critical limiting temperature hypothesis (González-Alonso et al., 1999).

However, during self-paced exercise, cerebral thermic centres alter exercise behaviour by regulating exercise intensity (Marino, 2004; Tucker et al., 2006; Tucker, Rauch, Harley, & Noakes, 2004). Exercise intensity is determined based on the rate heat is being stored, and the actual T_c, to anticipate the effort required to execute the desired performance (Marino, 2004; Tucker et al., 2006). The reduction in exercise intensity occurs well before body temperature reaches hyperthermic critical levels (Schlader, Z. J. et al., 2011; Tucker et al., 2004) to prevent the attainment of a critical T_c and avoid the catastrophic effects of hyperthermia.

At the 8 min mark of the TSS, T_c was still found to be *importantly* different between the SEV and CON (SEV > CON); however, at the 20 min mark, there was only a *trivial* difference between the warm-up conditions, which was maintained until the end of the TSS. Furthermore, the rate of change of T_c was either *unclear* or *trivial* from the 0-8 min time-period between the warm-up conditions. During the 8-20 min time-period, the rate of change in T_c was *importantly* less in the SEV compared to the CON conditions (SEV < CON).

Aligning the performance results and T_c changes during the 8-20 min time period between the SEV and CON, it can be speculated that some form of pacing strategy may have been adopted. It may be possible that the execution of a SEV compared to the CON may negatively impact performance during the middle stages of a TSS because of the generation of an elevated baseline T_c, which is maintained up to 8 min.

The regulation of low and moderate-intensity activities during team-sport matches has previously been determined (Coutts et al., 2010; Spencer et al., 2004). It has been speculated that players reduce the intensity of nonessential activities as a match progresses to preserve high-intensity performance (Duffield, Coutts, & Quinn, 2009). Notably, the decrease in low and moderate-intensity activities during team-sport matches has also been strongly correlated with changes in T_c (Duffield et al., 2009). It has been concluded that the regulation of T_c during an Australian football

match was achieved because the athletes adopted a pacing strategy within moderate-intensity activities to ensure the control of high internal temperature loads. The application of pre-cooling before and during warm-up procedures had been shown to improve submaximal bouts of exercise during an intermittent-sprint protocol (Duffield & Marino, 2007). These findings would suggest that elevating T_c by performing a high-intensity warm-up at the beginning of team-sport matches may cause a reduction in performance to occur earlier. Adopting a subconscious pacing strategy may imply that athletes find themselves out-of-position to create, or defend, scoring opportunities during team-sport matches.

The blood acid-base levels before beginning the TSS were unexpected. Similar procedures had been undertaken in other studies, including prescribing the same relative intensity for the SEV (70% Δ between gas exchange threshold and $\dot{V}O_{2peak}$), but the observed blood acid-base responses didn't match (Bailey et al., 2009). A potential explanation for the varying results is the different exercise modalities, cycling and running, recruiting different amounts of muscle mass. In support of this theory, another investigation determined that blood lactate post 6 min of severe-intensity cycling reached 10.1 ± 1.0 mmol·L^{-1} and had been restored after 15 min to 6.4 ± 1.5 mmol·L^{-1} (Ferguson et al., 2010). This finding resembles the blood lactate profile observed in the Bailey et al. (2009) study. However, comparisons made between cycling and running while exercising at the same relative intensities have shown that blood lactate profiles are not significantly different (Billat et al., 1998; Carter et al., 2000).

Another possible explanation for the varied blood marker results between studies was the method used to determine ventilation threshold. Bailey et al. (2009) used a cluster of measurements to subjectively determine ventilation threshold, while this study used the V-slope method. The V-slope method objectively determines the deflection point between $\dot{V}CO_2$ and $\dot{V}O_2$. It was thought that implementing an objective technique to determine ventilation threshold, instead of relying on

visual detection, would provide a more reliable determination of ventilation threshold. The varying methods used to determine ventilation threshold could explain the large blood marker variances between studies. However, as both methods have been shown to agree reliably with lactate threshold (Beaver et al., 1986; Gaskill et al., 2001) this possibility is just speculative.

Another explanation is the different lactate analyser used to determine the blood lactate changes between studies. In this thesis, the i-STAT device and (as a back-up) Lactate Pro 2 analyser were used to determine blood lactate changes, while Bailey et al. (2009) used the YSI 1500 lactate analyser. The lactate concentrations determined by the i-STAT and Lactate Pro 2 analysers had poor agreement (Whyte et al. In Press), possibly demonstrating that this could happen when comparing results between the i-STAT and YSI 1500 analysers.

The varied blood marker results may have also been caused by the assumed location of the critical velocity. Investigations have shown that critical velocity exists ~50% Δ between lactate threshold and $\dot{V}O_{2peak}$ (Smith & Jones, 2001; Wilkerson, Koppo, Barstow, & Jones, 2004b). Then again, these findings have been determined based on cycling and not running. Given that lactate threshold, as a percentage of $\dot{V}O_{2max}$, can occur at a different percentage within the same individual during cycling and running (Carter et al., 2000), it is likely that critical velocity does the same. Critical velocity (power) has been shown to exist with running and cycling at ~85% and ~75% of VO_{2max} respectively (Jones & Burnley, 2005). The assumption that critical velocity existed at 50% Δ between ventilation threshold and $\dot{V}O_{2peak}$ may have been incorrect in this study.

The use of critical velocity to separate the heavy and severe-intensity domains has also been questioned. Critical velocity is a theoretical concept expected to provide an accurate estimation of the maximal work-rate in a state of physiological aerobic balance (Dekerle, Baron, Dupont, Vanvelcenaher, & Pelayo, 2003; Moritani, Nagata, Devries, & Muro, 1981). Heavy-intensity exercise

is seen as the highest work intensity at which blood lactate and $\dot{V}O_2$ can still plateau, while blood lactate and $\dot{V}O_2$ fail to stabilise during severe-intensity exercise.

Based on these characteristics, a common association made with critical velocity is the maximal lactate steady-state. However, the association between critical velocity and maximal lactate steady-state is only moderate (Dekerle et al., 2003). Critical velocity has been found to be a higher value than maximal lactate steady-state, meaning critical velocity may overestimate metabolic rate. Others have suggested that maximal lactate steady-state provides a better indication of the boundary between the heavy and severe-intensity domains (Pringle & Jones, 2002).

To ensure the participants in this study were exercising in the HVY and SEV (without calculating critical velocity) an intensity buffer was created at 20% above and below the estimated critical velocity. Other studies had used similar procedures with apparent success. Unfortunately, there were some participants whose $\dot{V}O_2$ profiles did not represent the characteristics typically expected during exercise in the heavy and severe-intensity domains. These discrepancies may also be the reason for the varied blood lactate profiles post-SEV.

Furthermore, the uncharacteristic $\dot{V}O_2$ profiles during the warm-up conditions may have been caused by the maximal lactate steady-state, which has been shown to range from 33-83% Δ between lactate threshold and $\dot{V}O_{2max}$ (Smith & Jones, 2001). Even if critical velocity and maximal lactate steady-state were the same phenomena, this range is slightly larger than this study's intensity buffer. It is likely that some participants exercised at a higher intensity than desired.

Whatever the reason for the varied baseline blood markers or the uncharacteristic $\dot{V}O_2$ profiles, the only way to resolve this limitation is for subsequent studies to calculate critical velocity or maximal lactate steady-state. One of the main issues with determining critical velocity is the numerous trials required to calculate the hyperbolic relationship between power and time.

However, the determination of critical velocity or maximal lactate steady-state would provide confidence the results are reflective of the appropriate warm-up intensity. This may also ensure blood markers are consistently generated between studies.

Another major finding of the study was that average speed of 1st sprint was *importantly* increased in the HVY compared to the CON. The improvement in 1st sprint performance following a warm-up during a prolonged intermittent-sprint protocol has previously been determined (Yaicharoen, Wallman, Bishop, et al., 2012). With T_c found to be *importantly* increased at the start of the TSS between the HVY and CON, it is possible that T_m was also elevated in the lower-body (Asmussen & BØJe, 1945; Mohr, Krustrup, Nybo, Nielsen, & Bangsbo, 2004). Maximal sprint performance can be improved by ~10% per 1°C increase in T_m (Sargeant, 1987). The potential mechanisms which may have improved 1st sprint performance include the changes in the force-velocity relationship of the muscle (Davies & Young, 1983) and the increased degradation of high-energy phosphates (Febbraio et al., 1996).

However, there was only a *trivial* difference between the SEV and CON for 1st sprint performance. It would be expected the same mechanisms impacting 1st sprint following the HVY would be active following the SEV. It is possible the elevated baseline blood acid-base levels before beginning the TSS may have offset the generated benefits of the SEV. Based on this result, a HVY may provide acute performance benefits which could be useful for player interchange and substitutions during critical stages of match-play.

It was determined that there were *trivial* differences between the warm-up conditions for repeated-sprint performance. This finding goes against the expected outcomes that repeated-sprint performance would be improved in the SEV compared to the CON. It was predicted the priming effect generated by the supralactate warm-ups would enhance phosphocreatine kinetics and reduce fatigue-related metabolite accumulation during the repeated-sprints and recovery between sprints

(Forbes et al., 2008). Consequently, this may have enabled sprinting performance during multiple sprints to be improved.

When average speed within the repeated-sprint clusters was compared to the average speed of the 1st sprint, it was found that the 2nd and final repeated-sprint clusters were *importantly* decreased between the HVY and SEV compared to the CON. A time-motion study on Australian football determined that the performance of higher-intensity activities at the start of a match or at half-time can significantly impair total distance, high-intensity, and low-intensity activities during later stages of matches (Coutts et al., 2010). Even though *trivial* differences were found for performance between the warm-up conditions at the start of the TSS, the execution of a HVY and SEV may have caused fatigue similar to performing high-intensity activities at the start of a match. This explanation is supported by the elevated blood marker levels observed, especially in the SEV, before beginning the TSS.

A possible shortcoming of this study's findings is the lack of performance compensation observed in other measures. Within the 8-20 min time-period, average speed and average speed jogging were *importantly* reduced in the SEV compared to the CON. However, at the end of the TSS, there were only *trivial* differences between the warm-up conditions for total distance covered and distance covered performing each of the speed activities. It would be expected if a change in performance was observed at the 8-20 min time-period, and subsequently all warm-up conditions were *trivially* different at the end of the TSS, that some form of performance compensation must have happened i.e., increase in running or sprinting performance within another time-period. There was an *important* increase in the relative contribution of sprinting to the total distance covered in the TSS during the SEV compared to CON. However, other relative contribution measures were calculated to be *trivially* different. It is possible that the *trivial* differences in multiple other speeds (i.e. the change is spread across the other activities) and therefore no single speed activity changed

by enough to be *important*. The allocated statistical threshold dividing *trivial* and *important* differences in this thesis may have left some performance changes undetected.

5.1 Summary and Conclusions

This study indicates that a HVY and SEV provided little performance benefit during a subsequent TSS. It was expected the SEV would create a 'priming effect' which may have enabled a greater total distance to be covered, including favourably altering submaximal activity profiles. Although the accumulation of lactate was attenuated, and the drop in pH and HCO_3 was reduced, potentially demonstrating an improved aerobic metabolism response, this had no bearing on subsequent performance. In contrast, the elevation of baseline T_c for the SEV compared to CON, which was maintained for 8 min into the TSS, may have negatively affected the participants pacing strategy during the middle stages of the TSS. However, the uncharacteristic $\dot{V}O_2$ and blood marker profiles pre and post the HVY and SEV may demonstrate that some participants were exercising in the same intensity domain for both warm-up conditions. The apparent disassociation between expected and observed outcomes further emphasises the need for additional research to investigate the most effective warm-up strategy for team-sport athletes.

5.2 Practical Applications

The findings from this thesis provide evidence-based knowledge on the impact of warm-up intensity on team-sport running performance. It is suggested that team-sport athletes expecting to compete for prolonged periods during matches should not perform high-intensity warm-up routines. It is possible that athletes who execute a high-intensity warm-up may reduce jogging speed during the middle stages of the 1st quarter, and possibly through later stages of a match. The elevated baseline T_c may contribute to the change in exercise behaviour to attenuate the accumulation of heat production. The high-intensity warm-up efforts may also affect repeated-sprint ability as the

quarter, half, and match progress. Team-sport coaches and athletes are advised that *'less is more'*, meaning the extra exercise load prescribed in high-intensity or prolonged warm-up protocols may negatively affect subsequent performance. In contrast, a HVY may provide an acute performance benefit which could be pertinent with respect to player interchanges or substitutions during critical periods of match-play.

5.3 Directions for Future Research

An area for future research to investigate is if a moderate-intensity warm-up can improve team-sport running performance. Also, investigations into the impact of warm-up on a different TSS, one with more 'worst case scenarios', may enable more subtle differences in performances to be detected. Lastly, future research should investigate if a warm-up can improve other aspects of running mechanics, such as changes in stride length and stride frequency, during a TSS.

Appendices

Appendix A

Letter to Participant

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PARTICIPANT INFORMATION LETTER

PROJECT TITLE: Impact of warm-up intensity on simulated team-sport running performance.

INVESTIGATOR 1: Dr Doug Whyte
INVESTIGATOR 2: Dr Stuart Cormack
STUDENT RESEARCHER: Mr Grant Rowe
STUDENT'S DEGREE: MExSc(Res) – Exercise Science

Dear Participant,

You are invited to participate in the research project 'Impact of warm-up intensity on simulated team-sport running performance'.

What is the project about?
The project aims to determine the impact of three different warm-up intensities on a simulated team-sport match using a Non-Motorised Treadmill (NMT).

Who is undertaking the project?
This project will be conducted by Mr Grant Rowe as part of his Master's thesis, under the supervision of Drs Doug Whyte and Stu Cormack.

How much time will the project take?
If you agree to participate in the study you will be required to attend the Exercise Physiology Laboratory at Australian Catholic University for six sessions over 4-6 weeks. The time commitment for sessions 1-3 will be ~45-90 min; and sessions 4-6 ~3 hours.

What will I be asked to do?
The first three visits will allow you to become familiarised with running on the NMT. In each of these sessions you will perform the 30 min team sport match simulation that will be used in the later experiments. During the session you will be instructed to change your running speed to mimic the different movement patterns that you would expect if you were playing a match. These will include standing, walking, jogging, running and sprinting.

In addition, during your third visit you will perform a $\dot{V}O_{2peak}$ test which will assess the maximal amount of oxygen your body can use. The test requires you to run at a series of increasing speeds while we measure the amount of oxygen you are consuming. A computer screen directly in front of you will display your current

speed and we will instruct you to maintain a specific pace. Throughout the test you will wear a face mask and a heart rate monitor so we can monitor your response. After 20 min passive recovery you will then complete the final familiarization trial. It is important that you arrive at this session completely hydrated. When you arrive at the laboratory we will take a blood sample from a forearm vein and weigh you in order to determine your hydration status.

Visits 4-6 will involve three experimental warm-ups, each 6 min long, that will be composed of either walking or running at a moderate or hard intensity on the NMT. Following a 20 min rest you will then complete the 30 min simulated team sport protocol. Two hours prior to testing you will be required to arrive at the university laboratory to ingest a temperature pill (the size of a jellybean). Two hours is required for the pill to transit past your stomach and be a reliable measure of body temperature. This pill will help us measure body temperature changes during the testing sessions. The pills are covered with a protective coating to allow impermeable and unobstructed passage through your body. The following day the pill will pass and exit your body when you next go to the toilet. The temperature pills are approved by the U.S. Food and Drug Administration for consumption. After ingesting the pill, you will be given a wrist band to wear for 48 hours, identifying that you have ingested a temperature pill and that no MRI scans are to be performed on you without checking that the device has been passed (e.g. x-ray). You will also receive a carbohydrate supplement and Gatorade drink to consume during this time. Furthermore, 6 fingerprick blood samples will be collected from you throughout each of these sessions. As with the tests described above, oxygen consumption and heart rate will be collected during the exercise protocols.

Are there any risks associated with participating in this project?

As with any bout of intense exercise there are a number of small risks associated with participating in this study, including the potential for injury during maximal exercise testing. However, we will minimise these risks by familiarising you with running on the treadmill, requiring you to meet certain fitness standards to be eligible to participate and by including sufficient warm up prior to each trial. There is also a slight risk of infection and /or bruising as a result of blood sampling. These risks will be minimised by having only trained staff take your blood using strict sterile techniques. Lastly, with any physical activity there is inherent risk of experiencing a cardiac event. However, as you have identified that your age is between 18 – 30 years, you have above average fitness levels, and that you don't have any pre-existing cardiopulmonary conditions this risk is minimised.

What are the benefits of the research project?

Your participation in this study will help with understanding the impact warm-up intensity has on team-sport running performance. We anticipate these findings will help coaches prescribe warm-up protocols that optimise training and competition performance for team-sport athletes. There are no direct benefits in participating in this experiment; however, you will gain insight into the research process using laboratory-based exercise equipment. You will also learn what your physiological fitness capacities are during the research testing.

Can I withdraw from the study?

Participation in the study is voluntary and if you do choose to participate but later change your mind for any reason, you may withdraw without any consequences.

Will anyone else know the results of the project?

Your personal information and any data collected during this study will be kept completely confidential. The only people who will have access to this information are the researchers (Doug Whyte, Stuart Cormack, and Grant Rowe). After all data have been collected and analysed, average scores for the group as a whole will also

be used in sports science presentations and publications; however, no individually identifiable data will be apparent in such presentations/publications.

Who do I contact if I have questions about the project?
Any questions regarding this project should be directed to investigators and the student researcher:

Dr Doug Whyte
Ph: 03 9953 3557

Dr Stuart Cormack
Ph: 03 9953 3133

Mr Grant Rowe
Ph: 0409 420 152

School of Exercise Science
Australian Catholic University
115 Victoria Pde
Fitzroy, Victoria, 3065

Will I be able to find out the results of the project?
Upon the completion of the project all of your results will be available to you, and also given to you in a printed summary.

What if I have a complaint or any concerns?
This study has been approved by the Human Research Ethics Committee at Australian Catholic University. In the event that you have any complaint or concern, or if you have any query that the Supervisor or Student Researcher have not been able to satisfy, you may write to the Chair of the Human Research Ethics Committee care of the nearest branch of the Research Services Office:

Chair, HREC
C/- Research Services
Australian Catholic University
Melbourne Campus
Locked Bag 4115
FITZROY VIC 3065
Tel: 03 9953 3158
Fax: 03 9953 3315

Any complaint or concern will be treated in confidence and fully investigated. The participant will be informed of the outcome.

I want to participate! How do I sign up?
If you agree to participate in this project, you should sign both copies of the Consent Form, retain one copy for your records and return the other copy to the Investigator or Student Researcher.

Appendix B

Consent Form

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CONSENT FORM

Copy for Participant

TITLE OF PROJECT: Impact of warm-up intensity on simulated team-sport running performance.

INVESTIGATOR 1: Dr Doug Whyte

STUDENT RESEARCHER: Mr Grant Rowe

I ..... have read and understood the information provided in the 'Letter to Participants'. Any questions I have asked have been answered to my satisfaction. I agree to participate in this study and understand it will involve:

- Six (6) laboratory visits to the Exercise Physiology Laboratory at ACU
- A graded exercise test
- Six (6) team-sport running simulations
- Three (3) blood sample taken from a forearm vein and up to eighteen (18) finger prick blood samples
- Ingesting a telemeter pill two (2) hours prior to each of the experimental sessions.

I understand that I can withdraw my consent at any time without any adverse consequences. I agree that research data collected for the study may be published or may be provided to other researchers in a form that does not identify me in any way.

I consent for my photo to be used by the investigator in presentations and/or publications associated with this research. If any photographs are used the investigators will obscure my face either by the angle of the photo taken or by covering my face using computer software.

YES / NO

NAME OF PARTICIPANT:

SIGNATURE ..... DATE .....

SIGNATURE OF INVESTIGATOR 1: DATE:.....

SIGNATURE OF STUDENT RESEARCHER: DATE:.....

Appendix C

Pre-Exercise Questionnaire

Pre-Exercise Questionnaire

Name: Mr/Mrs/Ms/Miss			
Phone:	H:	W:	M:
Email:		DOB:	
Address:			
Suburb:			
Emergency Contact:		**Phone:**	

HOW WOULD YOU RATE YOUR OVERALL STATE OF HEALTH?

Poor_____ Good_____ Fair_____ Excellent_____

PLEASE GIVE A BRIEF DESCRIPTION OF YOUR CURRENT WEEKLY PHYSICAL ACTIVITY:

HEALTH CLEARANCE

Do you suffer/have you ever suffered from:

1. Diabetes	_____YES _____No	**6.** Chest Pain	_____YES _____No
2. Epilepsy	_____YES _____No	**7.** High Blood Pressure	_____YES _____No
3. Stroke	_____YES _____No	**8.** Currently Pregnant	_____YES _____No
4. Heart Disease	_____YES _____No	**9.** Any infections or infectious diseases	_____YES _____No
5. Heart Condition	_____YES _____No	**10.** A bleeding disorder	_____YES _____No

If you answered 'Yes' to any of the above you must obtain a medical clearance

MEDICAL INFORMATION

In the past **12 months** have you suffered from the following:

11. Illness	_____YES _____No	**16.** Muscular pain or cramps	_____YES _____No
12. Arthritis or joint pain	_____YES _____No	**17.** Back pain	_____YES _____No
13. Asthma (or other lung disease)	_____YES _____No	**18.** Chronic Cough	_____YES _____No
14. Hernia	_____YES _____No	**19.** High Cholestorol	_____YES _____No
15. Any major injuries	_____YES _____No	**20.** Liver/Kidney conditions	_____YES _____No

21. Does your family have a history of premature (<60 years) cardiovascular disease? _____YES _____No

22. Are you currently using any medication, homeopathic or alternative therapies or taking any dietary supplements? (please list in 'details') _____YES _____No

23. Are you currently receiving any physio/osteo/chiropractic treatment? (please list in 'details') _____YES _____No

DETAILS

Appendix D

Food Diary

AUSTRALIAN CATHOLIC UNIVERSITY

24 Hour Pre-test Diet

Please write down the food and drink you consumed during the 24 hours prior to, and the morning of your test. Write down what you ate or drank within the appropriate box as well as the time you had it. You do not need to write something in each box (e.g., if you did not have a morning snack leave this box blank). Please remember to include any fluids you drank during this period as well.

It is very important that you are hydrated at the start of testing, as you will not be able to drink during the test itself. Therefore, please try to drink a sufficient volume of fluid (~500 – 1000 mL) the night before the test.

A sample pre-test diet is provided below, to illustrate the level of detail required to complete this form.

Please refrain from any structured exercise during this time period.

Please submit this form at the beginning of your next scheduled testing session.

SAMPLE PRE-DIET

	Time of meal	
Breakfast	7:00AM	Cereal (2c) + skim milk (1c), Strawberries (1/2c), Toast (1 slice) + jam/honey, 1 fruit juice
Morning Snack	10:30AM	1 banana, water (2g)
Lunch	12:15PM	1 roll + ham + salad (no butter), 1 carton low-fat fruit yogurt, (1g) water
Afternoon Tea	3:45PM	English muffin (1) + honey (2 tsp), water (1g)
Dinner	6:30PM	Chicken (breast, no skin) + (2c) rice + 2 stir-fry vegetables, fruit salad, water (1g)
Evening Snack	10:00 PM	Hot chocolate (1c)
Breakfast	6:30AM	Cereal (2c) + skim milk (1c), Peachers (1/2c), Toast (1 slice) + jam/honey, 1 fruit juice

Abbreviations: c = cup(s), tsp = teaspoon, tbsp = tablespoon, g = glass(es).

PRE-TEST DIET

	Time of meal			
Breakfast				
Morning Snack				
Lunch				
Afternoon Tea				
Dinner				
Evening Snack				
Breakfast				
Exercise	**Time of exercise**	**Mode of exercise**	**Duration**	**Exertion (1-10)**

Appendix E

Team-Sport Simulation Protocol

Phase	Start	Speed	Load	Instruction
a1	0	50	0	Walk
a2	8	10.5	0	Jog
a3	16	0	0	Stand
a4	20	18	0	Run
a5	26	10.5	0	Jog
a6	36	0	0	Stand
a7	40	10.5	0	Jog
a8	48	18	0	Run
a9	58	10.5	0	Jog
a10	68	0	0	Stand
a11	74	6	0	Walk
a12	80	10.5	0	Jog
a13	84	30	0	Sprint
a14	90	6	0	Walk
a15	96	0	0	Stand
a16	102	10.5	0	Jog
a17	108	18	0	Run
a18	114	10.5	0	Jog
a19	122	0	0	Stand
a20	128	6	0	Walk
a21	132	18	0	Run
a22	142	10.5	0	Jog
a23	152	6	0	Walk
a24	156	18	0	Run
a25	162	10.5	0	Jog
a26	170	6	0	Walk
a27	176	10.5	0	Jog
a28	180	30	0	Sprint
a29	186	6	0	Walk
a30	198	10.5	0	Jog
a31	202	30	0	Sprint
a32	208	6	0	Walk
a33	220	10.5	0	Jog
a34	224	30	0	Sprint
a35	230	6	0	walk
a36	240	0	0	Blood
a37	300	6	0	Walk
a38	308	10.5	0	Jog
a39	318	0	0	Stand
a40	324	18	0	Run
a41	330	6	0	Walk
a42	336	18	0	Run
a43	342	10.5	0	Jog
a44	352	18	0	Run
a45	360	10.5	0	Jog
a46	368	0	0	Stand
a47	378	10.5	0	Jog
a48	388	18	0	Run
a49	394	10.5	0	Jog
a50	402	18	0	Run
a51	408	6	0	Walk
a52	416	10.5	0	Jog
a53	420	30	0	Sprint
a54	426	6	0	Walk
a55	438	10.5	0	Jog
a56	442	30	0	Sprint
a57	448	6	0	Walk
a58	460	10.5	0	Jog
a59	464	30	0	Sprint
a60	470	6	0	Walk
a61	480	0	0	Blood
a62	540	6	0	Walk
a63	548	10.5	0	Jog
a64	558	6	0	Walk
a65	562	10.5	0	Jog
a66	570	18	0	Run
a67	578	10.5	0	Jog
a68	588	18	0	Run
a69	600	6	0	Walk
a70	604	0	0	Stand
a71	610	10.5	0	Jog
a72	620	0	0	Stand
a73	626	6	0	Walk
a74	630	0	0	Stand
a75	636	10.5	0	Jog
a76	652	6	0	Walk
a77	658	18	0	Run

Phase	Start	Speed	Load	Instruction
b1	670	6	0	Walk
b2	674	0	0	Stand
b3	678	10.5	0	Jog
b4	686	18	0	Run
b5	692	10.5	0	Jog
b6	704	0	0	Stand
b7	710	10.5	0	Jog
b8	720	18	0	Run
b9	732	10.5	0	Jog
b10	740	0	0	Stand
b11	748	18	0	Run
b12	760	10.5	0	Jog
b13	764	30	0	Sprint
b14	770	6	0	Walk
b15	778	0	0	Stand
b16	784	10.5	0	Jog
b17	794	18	0	Run
b18	800	6	0	Walk
b19	804	0	0	Stand
b20	810	10.5	0	Jog
b21	822	18	0	Run
b22	828	6	0	Walk
b23	832	0	0	Stand
b24	840	10.5	0	Jog
b25	850	6	0	Walk
b26	858	10.5	0	Jog
b27	862	30	0	Sprint
b28	868	6	0	Walk
b29	880	10.5	0	Jog
b30	884	30	0	Sprint
b31	890	6	0	Walk
b32	898	18	0	Run
b33	904	10.5	0	Jog
b34	914	0	0	Stand
b35	920	18	0	Run
b36	926	10.5	0	Jog
b37	938	18	0	Run
b38	944	6	0	Walk
b39	952	10.5	0	Jog
b40	956	30	0	Sprint
b41	962	6	0	Walk
b42	974	10.5	0	Jog
b43	978	30	0	Sprint
b44	984	6	0	Walk
b45	996	10.5	0	Jog
b46	1000	30	0	Sprint
b47	1006	6	0	Walk
b48	1018	10.5	0	Jog
b49	1022	30	0	Sprint
b50	1028	6	0	Walk
b51	1040	10.5	0	Jog
b52	1050	18	0	Run
b53	1058	10.5	0	Jog
b54	1066	18	0	Run
b55	1078	6	0	Walk
b56	1086	18	0	Run
b57	1098	6	0	Walk
b58	1102	10.5	0	Jog
b59	1110	18	0	Run
b60	1116	10.5	0	Jog
b61	1126	6	0	Walk
b62	1130	10.5	0	Jog
b63	1140	18	0	Run
b64	1146	6	0	Walk
b65	1150	18	0	Run
b66	1162	10.5	0	Jog
b67	1172	6	0	Walk
b68	1180	0	0	Stand
b69	1188	10.5	0	Jog
b70	1198	0	0	Stand
b71	1206	10.5	0	Jog
b72	1214	0	0	Stand
b73	1220	10.5	0	Jog
b74	1236	6	0	Walk
b75	1240	18	0	Run

Phase	Start	Speed	Load	Instruction
c1	1252	6	0	Walk
c2	1256	0	0	Stand
c3	1260	10.5	0	Jog
c4	1270	18	0	Run
c5	1276	10.5	0	Walk
c6	1280	0	0	Stand
c7	1288	10.5	0	Jog
c8	1298	18	0	Run
c9	1310	10.5	0	Jog
c10	1318	0	0	Stand
c11	1322	6	0	Walk
c12	1326	10.5	0	Jog
c13	1330	30	0	Sprint
c14	1336	6	0	Walk
c15	1344	0	0	Stand
c16	1352	10.5	0	Jog
c17	1362	18	0	Run
c18	1368	10.5	0	Jog
c19	1376	0	0	Stand
c20	1382	6	0	Walk
c21	1390	18	0	Run
c22	1396	10.5	0	Jog
c23	1406	0	0	Stand
c24	1412	10.5	0	Jog
c25	1420	6	0	Walk
c26	1428	10.5	0	Jog
c27	1432	30	0	Sprint
c28	1438	6	0	Walk
c29	1450	10.5	0	Jog
c30	1454	30	0	Sprint
c31	1460	6	0	walk
c32	1472	10.5	0	jog
c33	1476	30	0	Sprint
c34	1482	6	0	Walk
c35	1492	18	0	Run
c36	1504	10.5	0	Jog
c37	1512	18	0	Run
c38	1524	6	0	Walk
c39	1532	10.5	0	Jog
c40	1542	6	0	Walk
c41	1550	18	0	Run
c42	1556	6	0	Walk
c43	1564	0	0	Stand
c44	1570	18	0	Run
c45	1576	10.5	0	Jog
c46	1584	18	0	Run
c47	1590	10.5	0	Jog
c48	1600	18	0	Run
c49	1608	6	0	Walk
c50	1612	18	0	Run
c51	1618	10.5	0	Jog
c52	1628	6	0	Walk
c53	1632	10.5	0	Jog
c54	1640	18	0	Run
c55	1652	6	0	Walk
c56	1656	18	0	Run
c57	1668	10.5	0	Jog
c58	1676	6	0	Walk
c59	1680	0	0	Stand
c60	1684	10.5	0	Jog
c61	1692	0	0	Stand
c62	1696	10.5	0	Jog
c63	1704	0	0	Stand
c64	1708	10.5	0	Jog
c65	1724	18	0	Run
c66	1730	6	0	Walk
c67	1736	10.5	0	Jog
c68	1740	30	0	Sprint
c69	1746	6	0	Walk
c70	1758	10.5	0	Jog
c71	1762	30	0	Sprint
c72	1768	6	0	Walk
c73	1780	10.5	0	Jog
c74	1784	30	0	Sprint
c75	1790	6	0	Walk
c76	1800	0	0	End

Appendix F

Blood Marker Data Sheet

Experimental Trial Worksheet

Subject No:	**Date:**	**Session:**	
Warm-up Speed:	**Food Diary (Y/N)**	**Pill Taken Time:**	
Ambient Temp:	**Barometric Pressure:**	**RH:**	**Plasma Osmolality:**

	PRE WU	POST WU	PRE TSS	4 MIN	8 MIN	20 MIN	POST TSS	10 MIN POST
BW								
Tcore								
HR								
RPE								
pH								
PCO2								
PO2								
Lactate								
TCO2								
HCO3								
Be								
SO2								
Specific Comments:								

Comments:

Appendix G

Rating of Perceived Exertion Scale

Rating	Descriptor
0	Rest
1	Very, very easy
2	Easy
3	Moderate
4	Somewhat Hard
5	Hard
6	-
7	Very Hard
8	-
9	-
10	Maximal

Appendix H

Approval of Ethics Certificate

Human Research Ethics Committee
Committee Approval Form

Principal Investigator/Supervisor: Dr Doug Whyte

Co-Investigators: Dr Stuart Cormack

Student Researcher: : Mr Grant Rowe

Ethics approval has been granted for the following project:
The impact of warm-up intensity on team-sport running performance.

for the period: 27/06/2014 - 28/02/2014

Human Research Ethics Committee (HREC) Register Number: 2013 130V

Special Condition/s of Approval
Prior to commencement of your research, the following permissions are required to be submitted to the ACU HREC:
N/A

The following standard conditions as stipulated in the *National Statement on Ethical Conduct in Research Involving Humans* (2007) apply:

(i) that Principal Investigators / Supervisors provide, on the form supplied by the Human Research Ethics Committee, annual reports on matters such as:
- security of records
- compliance with approved consent procedures and documentation
- compliance with special conditions, and

(ii) that researchers report to the HREC immediately any matter that might affect the ethical acceptability of the protocol, such as:
- proposed changes to the protocol
- unforeseen circumstances or events
- adverse effects on participants

The HREC will conduct an audit each year of all projects deemed to be of more than low risk. There will also be random audits of a sample of projects considered to be of negligible risk and low risk on all campuses each year.

Within one month of the conclusion of the project, researchers are required to complete a *Final Report Form* and submit it to the local Research Services Officer.

If the project continues for more than one year, researchers are required to complete an *Annual Progress Report Form* and submit it to the local Research Services Officer within one month of the anniversary date of the ethics approval.

K. Pashley.

Signed: .... Date: .... 07/03/2014.....
(Research Services Officer, McAuley Campus)

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