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THE EFFECT OF MANIPULATED UNDERSTANDING OF THE TASK END POINT ON PACING DURING SIMULATED RUGBY LEAGUE MATCH-PLAY

by

Thomas Mullen

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A Research Project submitted in partial fulfilment of the requirements of the University of Chester for the degree of M.Sc. Sports Sciences (Exercise Physiology)

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Abstract

The aim of this study was to examine the effect of manipulated understanding of the task end point on pacing profiles during simulated rugby league match-play. Thirteen male rugby players performed three trails of the rugby league match simulation protocol (RLMSP-i). In one trial, subjects were informed they would perform 2×23 min bouts (20 min rest between; control trial, CON). In a second trial, subjects were told to perform 1×23 min bout, following this they were asked to perform an additional 23 min bout (deception trial, DEC). In a third trial, subjects were not told the duration of the RLMSP-i they would perform (up to 80 minutes), but stopped after 2×23 minute bouts (unknown trial, UN). Movement demands, heart rate and blood lactate were measured during all trials, with muscle force/soreness and session rating of perceived exertion (RPE) recorded immediately after the protocol. Maximum sprint speeds were significantly different between trials, significantly rising at the end of bout two in CON (23.5 ± 1.5 km.h-1, *P <0.05*), and at the end of bout one for DEC (23.8 ± 1.6 km.h-1, *P <0.05*), whilst remaining significantly lower in the UN trial (*P <0.05*). Session RPE for DEC (7 ± 1.6) was significantly higher than CON (5.6 ± 1.7) and UN (4.8 ± 2.6; *P <0.05*). Results suggest pacing occurs during simulated match-play, but when an individual’s understanding of the end-point of exercise is manipulated (DEC or UN) their pacing schema significantly differentiates to when knowledge of the end-point is known (CON).
Declaration

No portion of the work referred to in this Research Project has been submitted in support of an application for another degree or qualification of this, or any other University or institute of learning.

The project was supervised by a member of academic staff, but is essentially the work of the author.

Copyright in text of this Research Project rests with the author. The ownership of any intellectual property rights which may be described in this thesis is vested in the University of Chester and may not be made available to any third parties without the written permission of the University.

Thomas Mullen:

Signed ........................................................................................................

Date ........................................................................................................
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1. Introduction

Pacing, or a pacing strategy, refers to the selected distribution of work or energy expenditure over the duration of an exercise task in an attempt to limit homeostatic disturbance and maximise performance (Abbiss & Laursen, 2008; de Konig et al., 2011). It has been proposed that a pacing strategy is generated prior to exercise, and is determined by pre-exercise expectations and experience of task intensity and duration, as well as various psychological (e.g., presence of competitors, motivation level) and physiological inputs (e.g., arterial oxygen saturation, muscle glycogen levels; Noakes, St Clair Gibson & Lambert, 2005; Tucker et al., 2006). Not surprisingly, pacing has been well documented during several ‘closed-loop’ activities, such as 100 km cycling (St Clair Gibson, Schabot & Noakes, 2001) and marathon running (Nicol, Komi & Marconnet, 1991). Early research examining these pacing patterns reported that pacing strategies largely determine performance, through the conscious or subconscious distribution of available resources, during continuous exercise lasting longer than 30 s (Ansley, Noakes, Robson, St Clair Gibson, 2004; Foster et al., 1993; Mauger, Jones & Williams, 2009; Garland, 2005). In such events, whereby winning is achieved by completing a set distance within the quickest time, changes in velocity and power (i.e. pacing profiles) have been well described (van Schenau, de Koning & de Groot, 1994; Garland, 2005; Wilberg & Pratt, 1988), with ‘optimal’ pacing strategies suggested for events spanning various distances (Abbiss & Laursen, 2008). During middle distance events (1.5 – 2 minutes), athletes commonly adopt a positive pacing strategy, whereby speeds progressively decrease following the attainment of peak speed early in the exercise bout (de Konig et al., 2011). During prolonged events (>2 minutes) performance times can be improved from distributing pace more evenly
(Wilberg & Pratt, 1988; de Konig et al., 2011). However research suggests that a ‘parabolic shaped’ pacing strategy is more common during both middle (2000m rowing; Garland, 2005) and long distance events (20 km cycling; Tucker, Rauch & Harley, 2004), where athletes progressively reduce speed from the on-set of exercise but tend to increase speed during latter stages of the event (Abbiss & Laursen, 2008).

Not all exercise endeavours are continuous, as many sport disciplines such as team sports (e.g., soccer, rugby, hockey) require very short (≤ 6 s) bouts of all-out intensity (> 300% of the intensity at which VO$_{2\text{max}}$ is attained) interspersed with incomplete (≤ 30 s) recovery over an extended period (<90 min; Spencer, Bishop, Dawson & Goodman, 2005). During such intermittent exercise, athletes attempt to maintain the highest exercise capacity they can whilst avoiding catastrophic failure of any physiological system (i.e. the cardiovascular system), but in a more complex manner than reported in closed-loop exercise (Billaut, Bishop, Schaerz & Noakes, 2011).

The precise mechanism underpinning pacing in team sports is not-well understood. Edwards and Noakes (2009) proposed a multi-level model of pacing to explain some common features of variable exercise intensity during soccer matches. This model consists of three broad pacing patterns reported during team sports. The first level of this model refers to the overall pacing plan for the match (macro-pacing), whereby running speeds are adopted in order to conclude the match having worked at a vigorous intensity whilst avoiding catastrophic disturbances of homeostasis. This pacing plan explains the frequently reported ‘positive pacing’ profile with reductions in intensity towards the end of match-play (Abbis & Laursen, 2008; Waldron, Highton, Daniels & Twist, 2013) as players are attempting to avoid said catastrophe. The inter-half
pacing plan (meso-pacing), refers to the opportunity to change the pacing profile between match halves in order to maintain the macro-pacing schema. This level of the model is thought to explain the commonly observed reduction in exercise intensity from the first to the second half in several team sports (Black & Gabbett, 2014; Sampson, Fullagar & Gabbett, 2014; Bradley & Noakes, 2013; Waldron et al., 2013a). Finally, the dynamic pacing plan (micro-pacing), allows the constant manipulation of match intensity within the meso- and macro-pacing schema. Substantiation for this level of the model includes the well documented transient reduction in running speeds in the five minutes following acute periods of intense match-play (Mohr, Krstrup & Bangsbo, 2003; Bradley et al., 2009).

The determinants of these commonly reported pacing strategies during team games is unclear, but may include the previously reported efferent feedback from physiological inputs including arterial oxygen saturation, muscle glycogen levels and heart rate as well as external psychological factors such as the presence of competitors, playing home or away and match score (Sullivan et al., 2014; Noakes et al., 2005; Tucker, 2009; Bradley & Noakes, 2013). These determinants then feedback to inform performers’ perceptions of exertion (RPE; Edwards & Noakes, 2009). De Konig et al. (2011), suggests the athlete then continuously compare how they feel at any instant during a match with how they expected to feel at that moment. Subsequently if their RPE is larger than expected at that point in the match then subsequent mechanical work (i.e. running speed) will decrease, in order to prevent further homeostatic disruptions. The process of altering mechanical power output (muscle recruitment) according to the perception of effort is proposed to occur continuously throughout a match and almost certainly takes into account both
the duration remaining till the end of the match and the momentary RPE (de Konig et al., 2011).

Interestingly, analysis of movement intensities throughout match-play has provided evidence that the duration players spend on the pitch appears to have a marked impact on their pacing profile. Players performing the whole-match demonstrate a steady decrease in high intensity running towards the end of match-play (Waldron et al., 2013a), in accordance with other studies in rugby league (Black & Gabbett, 2014; Sampson et al., 2014) and across team sports (Soccer: Mohr et al., 2003; Bradley & Noakes, 2013; Australian Football: Coutts et al., 2010). Conversely, athletes playing as ‘interchanges’ (substituted on and off for a minimum of two bouts) started play at a higher initial exercise intensity (compared to those completing the whole match) which led to a rapid decrease to the end of the first bout, termed the ‘one bout, all out’ pacing strategy. Subsequently, players commenced their second bout at a lower initial exercise intensity (two bout reserve), followed by a sharp increase in running speeds in the final quarter of play, characteristically known as an ‘end-spurt’ (Waldron et al., 2013a). These reported changes in pacing patterns associated with varying playing duration are potentially explained by the knowledge of the endpoint of exercise (St Clair Gibson et al., 2006). This knowledge of the endpoint has been widely reported as a fundamental determinant of an optimised pacing strategy, as without this information an accurate pacing strategy cannot be set (Tucker, 2009). In addition, various studies have revealed that if the expected duration of exercise is altered, such as in deception studies (exercise duration is manipulated or unknown; Ansley, Noakes, Robson-Ansley & St Clair Gibson, 2004; Baden, Mclean, Tucker, Noakes, & St Clair Gibson, 2005; Faulkner, Arnold, & Eston, 2011), perceived exertion and pacing patterns are disrupted.
These studies provide further supporting evidence that the physiological cost of exercise is directly moderated by the knowledge of task duration (Tucker, 2009).

Similarly, when knowledge of the endpoint of exercise is lacking, or there is uncertainty about the exercise duration, lower heart rate responses and exercise intensities have been reported (Eston, Stansfield, Westoby & Parfitt, 2012). Eston et al. (2012) suggested that the altered mechanical work reported from manipulated understanding of the exercise endpoint gives evidence of a process whereby athletes essentially reserve physiological resources due to the limited knowledge of how long the exercise bout will last. Such research has focussed primarily on continuous closed loop exercise activities, whereby exercise distance is known by the athlete (e.g. 10 km race), with constant feedback from a watch showing elapsed time and visual anchors highlighting distance covered (Tucker, 2009).

However, an area of sport competition whereby the duration of performance is often unknown is during team game match-play, due to the interchange of players being an unpredictable yet common occurrence (Waldron et al., 2013a). To date, no studies have examined the influence of task endpoint knowledge on the pacing profiles of team sports players. Accordingly, the current study was designed to examine the effect of manipulated understanding of the task end point on pacing profiles during simulated rugby league match-play.
2. Methods:

2.1. Participants and Design

Thirteen male University level rugby players (league and union; age = 22 ± 3 years, stature = 1.77 ± 0.02 m, body mass = 82.7 ± 8 kg, predicted \( \dot{VO}_{2max} = 54 \text{ ml.kg.min}^{-1} \)) were recruited for the study. The study was conducted with the ethical approval of the Faculty Research Ethics Committee of the University of Chester (appendix 4). All participants received a written and oral explanation of the investigation (appendix 5), after which they provided written informed consent (appendix 6) and completed a pre-test health questionnaire (appendix 7) prior to participating.

The study utilised a repeated measures randomised crossover design. Following baseline measurements of participants' characteristics, participants completed three trials of the rugby league movement simulation protocol (RLMSP-i) with a manipulated understanding of the protocol duration. These trials were considered a control (CON), deception (DEC) and an unknown (UN) trial of the RLMSP-i. Individual testing took place over 23 days, consisting of three days' rest after baseline and 7-10 days' rest between each of the three testing protocols (Figure 1). For methods of pre-exercise standardisation of physical state see, Appendix three.
2.2. Procedures

2.3. Baseline measurements

During the baseline visit participants performed the 20 m multi-stage shuttle run test (MSSRT; Ramsbottom, Brewer, & Williams, 1988) in an indoor sports hall to obtain an estimate of aerobic capacity ($\dot{V}O_{2\text{max}}$). Participants were required to obtain an estimated maximal aerobic power > 45 ml·kg$^{-1}$·min$^{-1}$ (Level 9 - MSSRT) as to replicate an aerobic capacity similar to that of elite rugby league players (Gabbett, 2005). During this baseline visit participants were familiarised with the procedures for isokinetic dynamometry at two different angular velocities (Biodex 3, Biodex Medical Systems, Shirley, NY, USA). Stature, using a wall mounted stadiometer (Harpenden Stadiometer, Wall mounted, Holtain, Crymych, Dyfed, UK) and body mass, using manual balance beam scales (Seca 712, Seca, Hamburg, Germany) were also recorded during the baseline visit (Figure 1). Finally, participants performed two full cycles of the
RLMSP-i (ball in play × 4, ball out of play × 2) to familiarise themselves with the order of events, and reduce any learning effects between trials.

2.4. Rugby League Movement Simulation Protocol

The study used a simulation protocol for rugby league match play designed to replicate the average speeds, distances and playing times of interchanged players (RLMSP-i; Waldron et al., 2013b). Prior to commencing the protocol, participants performed a standardised 10 minute warm-up consisting of varied running intensities and dynamic stretches (self-paced jog, high knees, heel flicks and near maximal running). Participants then performed the RLMSP-i on an artificial 3rd generation playing surface. During each performance of the RLMSP-i environmental temperature and humidity were recorded (THG810, Oregon Scientific Ltd., Berkshire, UK) at the start, middle and end of the protocol.
**Figure 2.** Running course for the RLMSP-i. Y, yellow cone; R, red cone; B, blue cone; W, white cone.

During the protocol participants ran alone, following the instruction of an audio signal (CD player) which dictates the speed of movement between various coloured cones (Figure 2). These movement speeds were established by previously reported locomotive speeds and activities of senior elite rugby league interchanged players during match-play (Waldron et al., 2013a). The RLMSP-i lasts 46 min, comprising two 23 min bouts, replicating the average time that an interchanged forward spends on the pitch during a match (Sykes, Twist, Nicholas & Lamb, 2011; Waldron et al., 2013a). For the full description outlining the order of events (Figure 3) see Appendix 1.
Figure 3. Schematic of the RLMSP-i including measurements taken pre during and post. Note. GPS and heart rate were recorded throughout the entire protocol.

Participants completed three trials of the RLMSP-i (Figure 1) with different feedback conditions. These trials consisted of: a) 2 x 23 min bouts (separated by 20 min) of the RLMSP-i where participants were informed that is what they were going to complete (control; CON trial); b) 2 x 23 min bouts of the RLMSP-i but participants were informed they would complete 1 x 23 min bout (deception; DEC trial); and c) 2 x 23 min bouts of the RLMSP-i where the participants were unaware of the exercise time, but were informed they could be required to perform 2 x 40 min bouts (unknown; UN trial). Prior to commencing each trial, clear instructions to the duration of the RLMSP-i to be completed
were given (Appendix 2). Throughout all performances of the RLMSP-i a clock was made visible, allowing participants to gauge the duration of each exercise bout.

2.5. Blood lactate concentration

Concentration of plasma blood lactate was measured by a capillary blood sample taken from the fingertip. A portable lactate analyser (Lactate Pro, Arkray, Kyoto, Japan) was used to immediately analyse whole blood samples. Blood sample collection took place five minutes before starting each protocol, immediately after the first bout and immediately after termination of the protocol for each trial (Figure 3).

2.6. Perceptual measures

Participants’ RPE (6-20 scale; Borg, 1985) were recorded at the walking intervals after every quartile (5.36 min) of the first and second bout. Session RPE (0-10 scale; Foster et al., 2001), whereby individuals rate their perceived exertion for the entire protocol, was reported within 20 minutes of completing each trial of the RLMSP-i (Figure 3; Herman et al., 2006).

At similar times before and after the protocol (~30 min), perceived muscle soreness was recorded using a visual analogue scale (VAS; Twist & Eston, 2005). This required participants to hold a squat position (90° at the knee) and rate their muscle soreness according to visual anchor points on the scale; between 0 (no muscle soreness), 5 (muscle sore on movement) and 10 (muscle too sore to move; Waldron et al., 2013b).

2.7. Muscle function

Using an isokinetic dynamometer (Biodex 3, Biodex Medical Sytems, Shirley, NY, USA), participants’ knee extensor and flexor peak torques at 60 and 240 deg s⁻¹ were measured in their dominant leg. Prior to testing
participants performed a standardized five minute warm-up, cycling at 50 W on a cycle ergometer (Lode Corival, Groningen, The Netherlands). The participant was then fitted to the dynamometer according to the manufacturer's criteria of knee torque assessment. Visual feedback, displaying real-time force, was provided to encourage maximal efforts. Measurements of knee torque assessment were taken 30 minutes before and within 30 minutes of finishing the protocol for all trials of the RLMSP-i.

2.8. Movement demands and heart rate

Participants wore an appropriately sized vest housing the GPS unit (MinimaxX S5, firmware 6.75, Catapult Innovations, Melbourne, Australia) between the scapulae. The GPS device samples at a rate of 10 Hz. Participants heart rate (HR) was collected using a HR monitor (Polar Electro Oy, Kempele, Finland). Both movement and HR data was downloaded and analysed using Catapult Sprint software (Catapult Optimeye S5, Catapult Innovations, Melbourne, Australia). The number of satellites available during performances of the RLMSP-i ranged from 12-19. The start and end of the RLMSP-i as signalled by the CD player, was documented using the “talking clock”. These times were later used to truncate raw GPS and HR data into quartiles of the first and second playing bouts. Movement data was then analysed per playing quartile (5.36 min) of each bout, including; peak sprint speed (km·h\(^{-1}\)), total distance covered (m), relative distance covered (m·min\(^{-1}\)), and both total and relative distance within high-intensity running (>14.0 km·h\(^{-1}\)).

2.9. Statistical analysis

All hypothesis testing was conducted using the statistical packages for social sciences (SPSS; v.19; SPSS Inc., Chicago, IL), with the alpha level set at \( P < 0.05 \). Once assumptions of parametric tests were met, a three-way
repeated measures analyses of variance (ANOVAs) was performed to assess the variability of the dependent variables taken throughout the protocol (heart rate, RPE and movement variables; total m·min⁻¹, high intensity m·min⁻¹ and peak speeds) due to the trial (UN, CON, and DEC), bout and quartile of the bout (trial × bout × bout quartile). A two-way repeated measures ANOVA was used to assess blood lactate concentrations, muscle soreness scores (VAS) and isokinetic muscle function (trial; time; trial × time). The use of a one way repeated measures ANOVA was performed to assess the variability in the session rating of perceived exertion. Sphericity was assessed using Mauchly’s test, with any violations accounted for via the Greenhouse-Geisser statistic. Where appropriate F statistics were highlighted, post-hoc paired samples t-tests, with Bonferroni adjustment, were conducted to assess pair-wise differences between the trials.

Due to the well-established limitations of null-hypothesis testing, further calculations of magnitude based inferences and effect sizes were utilised, as advocated by Batterham and Hopkins (2006). This established both the magnitude and likelihood of there being a change in the suggested key performance variables (RPE and GPS variables; high intensity running). Previously established thresholds for the probabilities of reporting a substantial effect based on the 90% confidence limits were utilised, they were: < 0.5% most unlikely, 0.5–5% very unlikely, 5–25% unlikely, 25–75% possibly, 75–95% likely, 95–99.5% very likely, > 99.5% most likely (Hopkins, 2006). As suggested by Hopkins, Marshall, Batterham and Hannin (2009), the magnitude of the observed change in these key performance variables were calculated as; the within-participant standard deviation (sd) x 0.3, 0.9 and 1.6 for a small,
moderate and large effect, respectively. The above calculations were completed using a predesigned spreadsheet (Hopkins, 2006).

3. Results

3.1. Movement Demands

For total and high intensity (>14 km·h⁻¹) absolute distances covered, see Appendix nine. Total and high intensity (>14 km·h⁻¹) distance covered relative to playing time and peak speeds obtained in each quartile of the RLMSP-i are displayed below (Figure 4). Analysis of the relative distance covered per minute of playing time highlighted a significant main effect for bout and quartile, with an interaction effect for bout x quartile during the RLMSP-i (F = 89.7; F = 14.8 and F = 18.5, respectively; P < 0.05). Further analysis revealed a significant difference for all quartiles between bouts (P < 0.05; Figure 4). Statistical hypothesis testing failed to show a significant difference between trials for total and relative distance covered per minute of playing time (m·min⁻¹) across all quartiles (Figure 4). However, utilising probabilistic inferences of effect size, deception of the playing duration was likely to elicit a very likely moderate decline in relative distance covered (m·min⁻¹) during the first quartile of the second bout (98 ± 4 m·min⁻¹) when compared to the control trial (102.2 ± 4.4 m·min⁻¹; table 1). During the final quartile of bout two the UN trial (98.3 ± 3.6 m·min⁻¹) is suggested to likely decrease when compared with the CON trial (101 ± 4.9 m·min⁻¹) for relative distance covered.

Relative distance covered in high intensity running (≥ 14 km·h⁻¹), reported a significant main effect for bout (F = 71.8, P < 0.05), quartile (F = 26.5, P <0.05), and an interaction effect for bout x quartile (F = 17.5, P <0.05). In the initial bout there is a stepwise decline in relative distance covered (all trials
compounded) as each quartile decreases significantly from the previous quartile ($P < 0.0125$; Figure 4). The UN and DEC trials were interpreted to result in a likely moderate and likely small decrease when compared to the CON trial in quartile four of bout two respectively (Table 2). Furthermore, the DEC trial is suggested to result in a likely small increase in high intensity running relative to playing time ($\text{m min}^{-1}$) when compared to the UN trial (Table 2).

Analysis of peak speeds obtained for each playing quartiles highlighted a main effect for bout ($F = 22.55$, $P < 0.05$) and quartile ($F = 24.11$, $P < 0.05$) and interaction effects for trial x quartile ($F = 4.4$, $P < 0.05$) and bout x quartile ($F = 5.3$, $P < 0.05$). Post-hoc analysis revealed that deceiving an individual (DEC) to the exercise duration was very likely to result in a significant moderate increase in their peak speed in the second ($t = -4.12$, $P < 0.0125$) and fourth quartile ($t = -5.93$, $P < 0.0125$; Figure 4) of bout one compared to the UN trial. Peak speeds during the final quartile of the second bout for UN and DEC trials were significantly lower than the CON trial ($= 2.79$, $P < 0.0125$; $t = 2.25$, $P < 0.0125$, respectively; Figure 4), interpreted as a very likely moderate or a likely small decrease, respectively (Table 3). For CON and UN trials quartile one was found to be significantly different between bouts for peak speeds ($t = 4.66$, $P < 0.0125$; $t = 2.5$, $P < 0.0125$, respectively). However, during the DEC trial all quartiles were significantly lower in bout two compared to bout one ($P < 0.0125$).
Figure 4. Relative distance covered per minute of playing time (m·min⁻¹) for total and high intensity running (>14 km·h⁻¹) and peak speeds (km·h⁻¹) obtained in each bout and quartile throughout CON, UN and DEC trials of the RLMSP-i; C = control trial; U = unknown trial; D = deception trial; a = significantly different ($P < 0.0125$) to quartile two of the same bout; b = significantly different ($P < 0.0125$) to quartile three of the same bout; c = significantly different ($P < 0.0125$) to quartile four of the same bout; * = significantly different ($P < 0.0125$) to bout one; 2 = significantly different ($P < 0.0125$) to unknown trial; 3 = significantly different ($P < 0.0125$) to deception trial.
Table 1. Statistical summary of the changes in absolute and relative distance covered between trials of the RLMSP-i (control, unknown and deception) during each bout quartile.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Trial</th>
<th>Deception</th>
<th>Control - Unknown</th>
<th>Control - Deception</th>
<th>Unknown - Deception</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Difference</td>
<td>Interpretation</td>
<td>Difference</td>
<td>Interpretation</td>
<td>Difference</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(90% confidence limits)</td>
<td></td>
<td>(90% confidence limits)</td>
<td></td>
<td>(90% confidence limits)</td>
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<tr>
<td>Distance covered (m)</td>
<td>4813 ± 167</td>
<td>4744 ± 131</td>
<td>4764.8 ± 112</td>
<td>-69.8 (± 121)</td>
<td>Unclear</td>
<td>-49.1 (± 56.9)</td>
</tr>
<tr>
<td>m min⁻¹</td>
<td>105.9 ± 3.1</td>
<td>105 ± 2.7</td>
<td>105.5 ± 2.2</td>
<td>-0.9 (± 2.6)</td>
<td>Unclear</td>
<td>-0.4 (± 1.6)</td>
</tr>
<tr>
<td>Bout 1</td>
<td>106.1 ± 4.14</td>
<td>105.5 ± 3</td>
<td>105.6 ± 3.76</td>
<td>-0.6 (± 2.3)</td>
<td>Unclear</td>
<td>0.6 (± 1.8)</td>
</tr>
<tr>
<td>Q1</td>
<td>105.32 ± 3.1</td>
<td>104.18 ± 4</td>
<td>104.29 ± 2.3</td>
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<td>Unclear</td>
<td>0.1 (± 2.5)</td>
</tr>
<tr>
<td>Q2</td>
<td>103.5 ± 2.8</td>
<td>101.8 ± 2.2</td>
<td>102.5 ± 2.9</td>
<td>-1.6 (± 2.5)</td>
<td>Possibly Small ↓</td>
<td>0.1 (± 2.1)</td>
</tr>
<tr>
<td>Q3</td>
<td>101.5 ± 4.5</td>
<td>101.2 ± 3</td>
<td>102.5 ± 2.3</td>
<td>-0.3 (± 3.2)</td>
<td>Unclear</td>
<td>1.3 (± 2.1)</td>
</tr>
<tr>
<td>Q4</td>
<td>103.3 ± 4.2</td>
<td>101.2 ± 3.4</td>
<td>101.5 ± 3.2</td>
<td>-2.1 (± 3)</td>
<td>Possibly Small ↓</td>
<td>-1.8 (± 1.5)</td>
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<tr>
<td>Bout 2</td>
<td>102.2 ± 4.4</td>
<td>100 ± 3.3</td>
<td>98 ± 4</td>
<td>-1.7 (± 3)</td>
<td>Unclear</td>
<td>-3.4 (± 2.2)</td>
</tr>
<tr>
<td>Q1</td>
<td>101.6 ± 3.9</td>
<td>99.5 ± 4.2</td>
<td>99.8 ± 3.7</td>
<td>-2.2 (± 3.1)</td>
<td>Possibly Small ↑</td>
<td>-1.8 (± 2.1)</td>
</tr>
<tr>
<td>Q2</td>
<td>100.9 ± 4</td>
<td>99.6 ± 3.6</td>
<td>100 ± 4.2</td>
<td>-1.3 (± 2.8)</td>
<td>Unclear</td>
<td>-0.8 (± 1.4)</td>
</tr>
<tr>
<td>Q3</td>
<td>101 ± 4.9</td>
<td>98.3 ± 3.6</td>
<td>100 ± 3.5</td>
<td>-3.1 (± 3.2)</td>
<td>Likely Small ↓</td>
<td>-0.8 (± 2.2)</td>
</tr>
<tr>
<td>Q4</td>
<td>101 ± 4.9</td>
<td>98.3 ± 3.6</td>
<td>100 ± 3.5</td>
<td>-3.1 (± 3.2)</td>
<td>Likely Small ↓</td>
<td>2.3 (± 2.6)</td>
</tr>
</tbody>
</table>
Note: Threshold probabilities for a substantial effect were: <0.5% most unlikely, 0.5-5% very unlikely, 25-75% possibly, 75-95% likely, 95-99.5% very likely, >99.5% most likely. Thresholds for the magnitude of the observed change in each dependant variable were determined as the within-participant SD × 0.3, 0.9 and 1.6 for a small, moderate or large effect respectively.

Table 2. Statistical summary of changes in relative distance covered at high intensity (> 14 km·h⁻¹) between trials of the RLMSP-i

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Trial</th>
<th>Unknown</th>
<th>Deception</th>
<th>Control - Unknown</th>
<th>Interpretation</th>
<th>Control - Deception</th>
<th>Interpretation</th>
<th>Unknown - Deception</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bout 1</td>
<td>27.2</td>
<td>25.7</td>
<td>26.8</td>
<td>2.5</td>
<td>-1.5 (± 2)</td>
<td>Possibly Small↓</td>
<td>-0.4 (± 1.4)</td>
<td>Unclear</td>
<td>1.1 (± 0.9)</td>
<td>Possibly Small↑</td>
</tr>
<tr>
<td>Q1</td>
<td>29.6</td>
<td>27.7</td>
<td>28.7</td>
<td>3.3</td>
<td>-2 (± 2.6)</td>
<td>Possibly Small↓</td>
<td>-0.9 (± 2.3)</td>
<td>Unclear</td>
<td>1.1 (± 1.3)</td>
<td>Possibly Small↑</td>
</tr>
<tr>
<td>Q2</td>
<td>27.2</td>
<td>26.3</td>
<td>27.7</td>
<td>3.9</td>
<td>-0.9 (± 2.3)</td>
<td>Unclear</td>
<td>-0.1 (± 1.8)</td>
<td>Unclear</td>
<td>0.8 (± 1.2)</td>
<td>Possibly Small↑</td>
</tr>
<tr>
<td>Q3</td>
<td>26.1</td>
<td>24</td>
<td>25.3</td>
<td>3.4</td>
<td>-2.1 (± 2.3)</td>
<td>Likely Small↓</td>
<td>-1.1 (± 1.9)</td>
<td>Possibly Small↓</td>
<td>0.9 (± 1.5)</td>
<td>Possibly Small↑</td>
</tr>
<tr>
<td>Q4</td>
<td>24</td>
<td>23.1</td>
<td>24.6</td>
<td>2.9</td>
<td>-1.0 (± 1.6)</td>
<td>Possibly Small↓</td>
<td>0.5 (± 1.2)</td>
<td>Possibly Small↑</td>
<td>1.6 (± 1)</td>
<td>Likely Small↑</td>
</tr>
<tr>
<td>Bout 2</td>
<td>24.7</td>
<td>23</td>
<td>23.4</td>
<td>3.1</td>
<td>-1.7 (± 2.2)</td>
<td>Possibly Small↓</td>
<td>-1.3 (± 1.3)</td>
<td>Possibly Small↓</td>
<td>0.4 (± 1.8)</td>
<td>Unclear</td>
</tr>
<tr>
<td>Q1</td>
<td>24.6</td>
<td>23.8</td>
<td>23.3</td>
<td>2.5</td>
<td>-0.9 (± 2.2)</td>
<td>Unclear</td>
<td>-1.3 (± 1.3)</td>
<td>Possibly Small↓</td>
<td>-0.4 (± 1.9)</td>
<td>Unclear</td>
</tr>
<tr>
<td>Q2</td>
<td>24.5</td>
<td>22.8</td>
<td>23</td>
<td>3.4</td>
<td>-1.8 (± 2.1)</td>
<td>Possibly Small↓</td>
<td>-1.5 (± 1.9)</td>
<td>Possibly Small↓</td>
<td>0.3 (± 1.8)</td>
<td>Unclear</td>
</tr>
<tr>
<td>Q3</td>
<td>23.3</td>
<td>22.6</td>
<td>22.8</td>
<td>3.7</td>
<td>-0.7 (± 2.9)</td>
<td>Unclear</td>
<td>-0.5 (± 1.9)</td>
<td>Unclear</td>
<td>0.3 (± 2.1)</td>
<td>Unclear</td>
</tr>
<tr>
<td>Q4</td>
<td>24.6</td>
<td>21.5</td>
<td>23</td>
<td>3.2</td>
<td>-3.2 (± 2.6)</td>
<td>Likely Moderate↓</td>
<td>-1.7 (± 1.2)</td>
<td>Likely Small↓</td>
<td>1.5 (± 2)</td>
<td>Possibly Small↑</td>
</tr>
</tbody>
</table>
(control, unknown and deception) during each bout quartile.

*Note:* For thresholds see Table 1.

**Table 3.** Statistical summary of the changes in peak speeds obtained in each quartile between trials of the RLMSP-i (control, unknown and deception) during each bout quartile.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unknown</th>
<th>Deception</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bout 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>24.4 ± 2</td>
<td>24.1 ± 1.3</td>
<td>25.2 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>-0.3 (± 0.8)</td>
<td>Possibly Small ↓</td>
<td>0.8 (± 1.3)</td>
</tr>
<tr>
<td>Q2</td>
<td>23.7 ± 2.3</td>
<td>23.7 ± 1.7</td>
<td>24.1 ± 1.2</td>
</tr>
<tr>
<td></td>
<td>-0.6 (± 1)</td>
<td>Possibly Small ↓</td>
<td>0.4 (± 1.1)</td>
</tr>
<tr>
<td>Q3</td>
<td>23 ± 2.2</td>
<td>22.7 ± 2</td>
<td>23.4 ± 1.4</td>
</tr>
<tr>
<td></td>
<td>-0.3 (± 1.2)</td>
<td>Unclear</td>
<td>0.4 (± 0.9)</td>
</tr>
<tr>
<td>Q4</td>
<td>22.5 ± 2.4</td>
<td>22 ± 1.6</td>
<td>23.8 ± 1.6</td>
</tr>
<tr>
<td></td>
<td>-0.6 (± 1)</td>
<td>Possibly Small ↓</td>
<td>1.2 (± 0.8)</td>
</tr>
<tr>
<td><strong>Bout 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>23 ± 2</td>
<td>22.7 ± 2</td>
<td>22.6 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>-0.2 (± 1.3)</td>
<td>Unclear</td>
<td>-0.3 (± 0.9)</td>
</tr>
<tr>
<td>Q2</td>
<td>23.1 ± 2.3</td>
<td>22.1 ± 2.4</td>
<td>22.5 ± 2</td>
</tr>
<tr>
<td></td>
<td>-1 (± 1.6)</td>
<td>Possibly Small ↓</td>
<td>-0.6 (± 1.1)</td>
</tr>
<tr>
<td>Q3</td>
<td>22.1 ± 2.4</td>
<td>22 ± 2.7</td>
<td>22 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>-0.1 (± 1.3)</td>
<td>Unclear</td>
<td>-0.2 (± 1)</td>
</tr>
<tr>
<td>Q4</td>
<td>23.5 ± 1.5</td>
<td>21.7 ± 2.6</td>
<td>22.2 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>-1.8 (± 1.1)</td>
<td>Very Likely Moderate ↓</td>
<td>-1.3 (± 1)</td>
</tr>
</tbody>
</table>
Note: For thresholds see Table 1.
3.2. Physiological and perceptual measures

There was a main effect for bout (F = 5.9, \( P < 0.05 \)), quartile (F = 46.5, \( P < 0.05 \)) and an interaction effect for trial x quartile (F = 5.5, \( P < 0.05 \)) for average HR. Between both UN and DEC trials the HR was found to be significantly different in the fourth quartile (\( t = -3.3, P < 0.0125 \)). However, as can be seen from table 4 there is a possibly small and likely small decrease in HR form DEC to UN trials for all quartiles of bout one. For all trials, HR increased significantly from the initial quartile throughout the first bout (\( P < 0.017 \)). Between bouts, only quartile two of the UN (\( t = 2.1 \)) and quartiles two to four of the DEC trial (\( t = 4.9; t = 3.4; t = 3.8 \), respectively) were significantly different between playing bouts (\( P <0.017 \)).

![Figure 5](image.png)

**Figure 5.** Mean heart rate (b·min⁻¹) obtained in each bout and quartile throughout CON, UN and DEC trials of the RLMSP-i; C = control trial; U= unknown trial; D = deception trial; a = significantly different (\( P <0.0125 \)) to quartile two of the same bout; b = significantly different (\( P <0.0125 \)) to quartile three of the same bout; c = significantly different (\( P <0.0125 \)) to quartile four of the same bout; * = significantly different (\( P <0.0125 \)) to bout one; 2 = significantly different (\( P <0.0125 \)) to unknown trial.
Table 4. Statistical summary of the changes in heart rate between trials of the RLMSP-i (control, unknown and deception) during each bout quartile.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unknown</th>
<th>Deception</th>
<th>Control - Unknown</th>
<th>Difference (90% confidence limits)</th>
<th>Interpretation</th>
<th>Control - Deception</th>
<th>Difference (90% confidence limits)</th>
<th>Interpretation</th>
<th>Unknown - Deception</th>
<th>Difference (90% confidence limits)</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Average Heart Rate (b·min⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bout 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>159 ± 24</td>
<td>158 ± 11</td>
<td>162 ± 15</td>
<td>0.3 (± 6.9)</td>
<td>Unclear</td>
<td>2.9 (± 10.2)</td>
<td>Unclear</td>
<td>2.7 (± 4.9)</td>
<td>Possibly Small ↑</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q2</td>
<td>167 ± 23</td>
<td>166 ± 11</td>
<td>171 ± 12</td>
<td>1 (± 6.7)</td>
<td>Unclear</td>
<td>3.1 (± 7.8)</td>
<td>Unclear</td>
<td>3.2 (± 2.6)</td>
<td>Possibly Small ↑</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>169 ± 21</td>
<td>166 ± 11</td>
<td>171 ± 12</td>
<td>-0.9 (± 5.3)</td>
<td>Unclear</td>
<td>1.8 (± 6.9)</td>
<td>Unclear</td>
<td>3.8 (± 3.3)</td>
<td>Possibly Small ↑</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>167 ± 22</td>
<td>165 ± 12</td>
<td>172 ± 12</td>
<td>-1.4 (± 5.2)</td>
<td>Likely Trivial</td>
<td>3.8 (± 5.8)</td>
<td>Possibly Small ↑</td>
<td>5.9 (± 3.2)</td>
<td>Likely Small ↑</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bout 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>160 ± 23</td>
<td>159 ± 10</td>
<td>160 ± 11</td>
<td>0.3 (± 6.8)</td>
<td>Unclear</td>
<td>-1.1 (± 8.9)</td>
<td>Unclear</td>
<td>-0.3 (± 4.4)</td>
<td>Unclear</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q2</td>
<td>166 ± 21</td>
<td>164 ± 12</td>
<td>166 ± 12</td>
<td>-1 (± 5.4)</td>
<td>Unclear</td>
<td>-1.8 (± 7.5)</td>
<td>Unclear</td>
<td>0.5 (± 3.9)</td>
<td>Likely Trivial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>166 ± 21</td>
<td>163 ± 12</td>
<td>167 ± 13</td>
<td>-1.7 (± 6.9)</td>
<td>Unclear</td>
<td>-1.3 (± 7.8)</td>
<td>Unclear</td>
<td>1.5 (± 4.2)</td>
<td>Likely Trivial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>167 ± 22</td>
<td>162 ± 13</td>
<td>167 ± 12</td>
<td>-4.1 (± 7.4)</td>
<td>Possibly Small ↓</td>
<td>-1.5 (± 7.5)</td>
<td>Unclear</td>
<td>3.3 (± 4.5)</td>
<td>Possibly Small ↑</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: For thresholds see Table 1.*
For blood lactate concentrations there was a main effect for trial (F = 5.04, $P < 0.05$), time (F = 8.1, $P < 0.05$) and an interaction effect for trial x time (F = 6.08, $P < 0.05$). As can be seen from figure five, significant differences in blood lactate for the CON and UN trials fall between mid, with a likely small decrease ($t = 2.2$, $P < 0.017$) and post, with a likely moderate decrease ($t = 2.3$, $P < 0.017$). However the DEC trial was only found to be significantly higher compared to UN at the mid time point ($t = -4.6$, $P < 0.017$), described through probability as a most likely moderate increase (table 5).

**Figure 6.** Mean blood lactate (mmol/L) obtained in each bout and quartile throughout CON, UN and DEC trials of the RLMSP-i; C = control trial; U= unknown trial; D = deception trial; y = significantly different ($P < 0.0125$) to mid; z = significantly different ($P < 0.0125$) to post; 2 = significantly different ($P < 0.0125$) to unknown trial.
Table 5. Statistical summary of the changes in blood lactate concentrations between trials of the RLMSP-i (control, unknown and deception) during each bout quartile.

<table>
<thead>
<tr>
<th></th>
<th>Blood lactate (mmol/L)</th>
<th>Control - Unknown</th>
<th>Control - Deception</th>
<th>Unknown - Deception</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Unknown</td>
<td>Deception</td>
<td>Difference (90% confidence limits)</td>
</tr>
<tr>
<td>Pre</td>
<td>3 ± 1.6</td>
<td>2.6 ± 0.8</td>
<td>2.7 ± 1</td>
<td>-0.5 (± 0.9)</td>
</tr>
<tr>
<td>Mid</td>
<td>4.7 ± 3.2</td>
<td>2.9 ± 1.3</td>
<td>5.7 ± 2.6</td>
<td>-1.8 (± 1.4)</td>
</tr>
<tr>
<td>Post</td>
<td>5.6 ± 3.9</td>
<td>2.8 ± 1.7</td>
<td>3 ± 1.8</td>
<td>-2.8 (± 2.1)</td>
</tr>
</tbody>
</table>

Note: For thresholds see Table 1.
There was a main effect on perceived exertion, for trial ($F = 7.5, P < 0.05$) and quartile ($F = 66.8, P < 0.05$) and an interaction effect for bout x quartile ($F = 6.25, P < 0.05$). The UN trial showed a non-significant likely small decrease from the CON trial in all quartiles, excluding possibly small decreases reported in quartiles one and two of the second bout (Table 6). Calculations of magnitude based inferences also inferred a likely small and most likely moderate increase in RPE for quartiles two and three of the first bout between the UN and DEC trials (Table 6). Paired $t$-test revealed that session RPE was significantly different from CON ($5.6 \pm 1.7$) and UN ($4.8 \pm 2.6$; $t = 4.8, P < 0.01$), CON and DEC ($7 \pm 1.6$; $t = -4.5, P < 0.01$), and UN and DEC ($t = -6.06, P < 0.01$).

**Figure 7.** Mean rating of perceived exertion (RPE) obtained in each bout and quartile throughout CON, UN and DEC trials of the RLMSP-i; a = significantly different ($P < 0.0125$) to quartile two of the same bout; b = significantly different ($P < 0.0125$) to quartile three of the same bout; c = significantly different ($P < 0.0125$) to quartile four of the same bout.
Table 6. Statistical summary of the changes in rating of perceived exertion between trials of the RLMSP-i (control, unknown and deception) during each bout quartile.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unknown</th>
<th>Deception</th>
<th>Control - Unknown</th>
<th>Interpretation</th>
<th>Control</th>
<th>Deception</th>
<th>Interpretation</th>
<th>Unknown - Deception</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RPE</strong></td>
<td></td>
<td></td>
<td></td>
<td>Difference</td>
<td>(90% confidence limits)</td>
<td>Interpretation</td>
<td>Difference</td>
<td>(90% confidence limits)</td>
<td>Interpretation</td>
<td>Difference</td>
</tr>
<tr>
<td><strong>Bout 1</strong></td>
<td></td>
<td></td>
<td></td>
<td>Control - Unknown</td>
<td>Interpretation</td>
<td>Control</td>
<td>Deception</td>
<td>Interpretation</td>
<td>Unknown - Deception</td>
<td>Interpretation</td>
</tr>
<tr>
<td>Q1</td>
<td>12.5 ± 1.2</td>
<td>11.8 ± 1.6</td>
<td>12.6 ± 1.6</td>
<td>-0.7 (± 0.5)</td>
<td>Likely Small ↓</td>
<td>0.1 (± 0.6)</td>
<td>Unclear</td>
<td>0.8 (± 0.5)</td>
<td>Likely Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q2</td>
<td>14 ± 1.8</td>
<td>13 ± 1.7</td>
<td>14 ± 1.2</td>
<td>-1 (± 0.6)</td>
<td>Likely Small ↓</td>
<td>-0.1 (± 0.6)</td>
<td>Unclear</td>
<td>0.9 (± 0.4)</td>
<td>Very Likely Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>14.7 ± 1.5</td>
<td>13.8 ± 1.8</td>
<td>15.1 ± 1.6</td>
<td>-0.9 (± 0.7)</td>
<td>Likely Small ↓</td>
<td>0.4 (± 0.6)</td>
<td>Possibly Small ↑</td>
<td>1.3 (± 0.8)</td>
<td>Likely Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>15.4 ± 1.6</td>
<td>14.1 ± 1.5</td>
<td>16.2 ± 1.6</td>
<td>-1.3 (± 0.8)</td>
<td>Likely Small ↓</td>
<td>0.8 (± 0.7)</td>
<td>Possibly Small ↑</td>
<td>2.1 (± 0.8)</td>
<td>Most Likely Moderate ↑</td>
<td></td>
</tr>
<tr>
<td><strong>Bout 2</strong></td>
<td></td>
<td></td>
<td></td>
<td>Control - Unknown</td>
<td>Interpretation</td>
<td>Control</td>
<td>Deception</td>
<td>Interpretation</td>
<td>Unknown - Deception</td>
<td>Interpretation</td>
</tr>
<tr>
<td>Q1</td>
<td>12.9 ± 1.4</td>
<td>12.3 ± 1.2</td>
<td>12.8 ± 2.1</td>
<td>-0.6 (± 0.5)</td>
<td>Possibly Small ↓</td>
<td>-0.1 (± 0.8)</td>
<td>Unclear</td>
<td>0.5 (± 0.7)</td>
<td>Possibly Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q2</td>
<td>13.8 ± 1.5</td>
<td>13.2 ± 1.7</td>
<td>14.1 ± 2</td>
<td>-0.6 (± 0.5)</td>
<td>Possibly Small ↓</td>
<td>0.3 (± 0.7)</td>
<td>Possibly Small ↑</td>
<td>0.9 (± 0.8)</td>
<td>Likely Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q3</td>
<td>14.6 ± 1.8</td>
<td>13.7 ± 1.8</td>
<td>14.7 ± 1.6</td>
<td>-0.9 (± 0.7)</td>
<td>Likely Small ↓</td>
<td>0.1 (± 0.7)</td>
<td>Unclear</td>
<td>1 (± 0.9)</td>
<td>Likely Small ↑</td>
<td></td>
</tr>
<tr>
<td>Q4</td>
<td>15.3 ± 1.6</td>
<td>14 ± 1.5</td>
<td>14.7 ± 1.8</td>
<td>-1.3 (± 0.9)</td>
<td>Likely Small ↓</td>
<td>-0.6 (± 0.8)</td>
<td>Possibly Small ↓</td>
<td>0.7 (± 1)</td>
<td>Possibly Small ↑</td>
<td></td>
</tr>
</tbody>
</table>

Note: For thresholds see Table 1.
3.3. Neuromuscular function

Muscle forces in the knee flexor at 60 and 240 deg s\(^{-1}\) presented a main effect for time (\(F = 28.6, P < 0.001\)) with no interactions. However muscle soreness resulted in a main effect for time (\(F = 35.8, P < 0.001\)) and an interaction effect for trial × time (\(F = 9.7, P < 0.001\)). Perceived muscle soreness was significantly higher immediately following the RLMSP-i in all trials (\(P < 0.05\); Table 7). Reported muscle soreness was significantly higher after the CON compared to the UN trial after completion of the RLMSP-i (Table 7).

**Table 7.** Mean (± standard deviation) muscle force and muscle soreness

<table>
<thead>
<tr>
<th></th>
<th>Trial</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee Flexor (60 deg.s(^{-1}))</td>
<td>Control</td>
<td>143 ± 37</td>
<td>116 ± 16</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>134 ± 22</td>
<td>117 ± 15</td>
</tr>
<tr>
<td></td>
<td>Deception</td>
<td>144 ± 28</td>
<td>114 ± 24</td>
</tr>
<tr>
<td>Knee Flexor (240 deg·s(^{-1}))</td>
<td>Control</td>
<td>90 ± 23</td>
<td>88 ± 21</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>93 ± 15</td>
<td>93 ± 20</td>
</tr>
<tr>
<td></td>
<td>Deception</td>
<td>95 ± 21</td>
<td>88 ± 22</td>
</tr>
<tr>
<td>Muscle Soreness</td>
<td>Control</td>
<td>2.6 ± 2.6</td>
<td>5.1 ± 3 (^#)</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>2.6 ± 2.5</td>
<td>3.4 ± 2.3 (^#)</td>
</tr>
<tr>
<td></td>
<td>Deception</td>
<td>1.8 ± 1.9</td>
<td>4.3 ± 2.6 (^#)</td>
</tr>
</tbody>
</table>
Note: # = significantly different from Pre (P>0.05); ¹ = significant difference from Unknown trial.

There were no significant difference (P>0.05) between all trials for temperature and humidity recorded pre (CON = 27°C, 32%; UN = 24.9°C, 30.3%; DEC = 22.3°C, 33.8%), during (CON = 26.1°C, 30.9%; UN = 23.7°C, 29.6%; DEC = 21.8°C, 35.1%) and post (CON = 25.6°C, 29.6%; UN = 24°C, 31.8%; DEC = 22.2°C, 28.2%) RLMSP-i.

4. Discussion:

4.1. Movement demands

The findings of the current study suggest that pacing not only occurs, but can be manipulated during prolonged intermittent exercise (RLMSP-i). Results indicate that when an individual’s understanding of the end-point of exercise is manipulated (DEC or UN) their macro-pacing schema significantly differentiates compared to when the knowledge of the end-point is known (CON). These reported movement profiles (m·min⁻¹ and HIT m·min⁻¹; Figure 4) imitate movement profiles of interchange players during rugby league match-play (Waldron et al., 2013a). High intensity running in the first bout adopts a ‘one bout, all out’ pacing strategy, characterised by a stepwise decline in total and high intensity running towards the end of the first bout (Waldron & Highton, 2014). The commencement of the second playing bout presented significantly lower running intensities throughout all quartiles, representative of a ‘second bout reserve’ pacing strategy (Waldron & Highton, 2014). Arguably the peak speeds achieved during the CON trial over the course of the RLMSP-i better represent the pacing profiles outlined above (Figure 4). Although the pacing
profile is similar throughout the initial bout of the CON trial (one bout, all out; Figure 4), the second bout culminates in significantly higher peak speeds ($P<0.05$) compared to UN and DEC trails. Representative of the ‘end-spurt’ phenomenon, previously reported during team game match-play for interchanged players (Waldron et al., 2013a). This variation in high intensity running speeds provides evidence supporting the existence of several pacing strategies (Tucker & Noakes, 2009; de Konig et al., 2011).

Running intensities are significantly lower in the second bout compared to the first, which is indicative of a meso-pacing strategy (Black & Gabbett, 2014). The reported end-spurt (CON) suggests that due to previous periods of ‘holding back’ energy resources, the participants are able to increase their running intensity at later stages of the protocol (de Konig et al., 2011). This conscious or subconscious physiological reservation is less likely to be required in the closing stages of the exercise bout, and participants are able to increase running speeds without harmful risk of homeostatic disturbances. In addition, a ‘hazard score’ developed by de Konig et al., (2011) has demonstrated that athletes tend to change pace during competitive simulations in accordance with how they feel at that moment (RPE) and how much of the event remains. This theory highlights the importance of perception of effort and knowledge of exercise duration in developing an optimal pacing strategy. Similarly, evidence suggests an ‘end-spurt’ occurs during the DEC trial (peak sprint speeds; Figure 4). However this occurs towards the end of the first bout due to the deception of the exercise duration. Conversely, due to the misinformed participant’s knowledge of the end-point during the UN trial, significantly lower peak speeds were reported throughout both bouts of the protocol. Comparable findings have been reported when athletes were misinformed as to the duration of the end-
point of exercise during continuous endurance activities (Eston et al., 2012). It can be speculated that the macro-pacing strategy required the reservation of energy sources in anticipation of exercising for up to 80 minutes. This pacing could allow glycogen sparing due to a potential reduction of anaerobic glycolysis rates, with subsequent reductions in associated metabolite accumulation (lactate and hydrogen ions).

4.2. Perceptual response

A plethora of research suggest that these variations in movement variables (Figure 4), are directly regulated by an individual’s perception of exertion (RPE; st Clair Gibson et al., 2006; de Konig et al., 2011; Noakes et al., 2005; Billaut et al., 2011). Interestingly, in the current study no significant differences were highlighted between the trials of the RLMSP-i for RPE ($P >0.05$; Figure 6). A potential justification is that RPE is reported to not reach maximal values during team game match-play, as individuals pace efforts to facilitate a physiological reserve for; i) the end of the exercise bout; and, ii) anticipation of any large fluctuations in high intensity activities dictated by the variability of match-play (i.e. having to sprint to make a tackle; Waldron et al., 2014). As a result of this ‘physiological reserve’, RPE in the current study may be subconsciously ‘capped’ at sub-maximal levels. In which case even between the different trials it would prove difficult to find differences in RPE, as they would all be near to this theoretical ‘ceiling’ during such high intensity prolonged intermittent exercise. Although the movement demands during the RLMSP-i are prescribed, hence the random variation that occurs in match-play is removed. The team games players used in this study may inherently pace themselves in the described manner, with a physiological reserve, due to years of competitive
team games experience. As Abbiss and Laursen (2008) suggest pacing strategies are developed according to the previous experience of the player. Interestingly, session RPE was significantly higher in DEC compared to CON and UN, and CON was significantly higher than UN ($P < 0.01$). The deception could have increased session RPE due to feelings of anger. In the UN trial however, the down-regulation of high intensity efforts throughout (Figure 4) potentially caused minimal homeostatic disturbances, hence feeding back to lower the RPE.

4.3. Physiological response

RPE is commonly regarded a ‘central regulator’ within the central nervous system (CNS) which is influenced by various afferent feedback from peripheral physiological systems (HR, metabolite accumulation, core temperature). Therefore, the physiological efferent feedback which construct the momentary RPE indirectly informs changes in physiological exertion. During DEC the profile of average HR across all quartiles can be compared with the peak speeds obtained (Figure 4 and 5). The significant very likely moderate increase in peak speeds during the final quartile of bout one is in conjunction with the likely small significant increase in HR during the same playing quartile. Conversely, following the active recovery (DEC) average HR was significantly lower in quartiles two to four of the second bout, similar to the significantly lower peak speeds achieved in the second bout for all quartiles following the deception of the initial bout.

Although non-significant, there is a definite trend for the average HR in the UN trial to be lower than both the CON and DEC trials throughout the protocol (Figure 5). This coincides with the reduced sprint speeds throughout
the RLMSP-i during the UN trial. Speculated to avoid catastrophic failure of any physiological system associated with sustained higher running intensities (e.g. glycogen depletion). Therefore the amount of high intensity activity is significantly down-regulated throughout all playing quartiles (Figure 4), thus reducing the rate of anaerobic glycolysis, sparing glycogen stores for later surges in high intensity work.

Blood lactate concentrations during the DEC trial peak immediately following the first bout (~6 mmol·L\(^{-1}\)) followed by a significant decrease to baseline values by the end of the second bout (~2.5 mmol·L\(^{-1}\); Figure 6). The limited research available on deception studies would suggest that this pattern (reduced speeds in bout two) informs the new pacing schema. Following deception, high intensity efforts were down-regulated to potentially generate a physiological reserve in anticipation of an unexpected change in exercise duration (further deception). Possibly further exaggerated due to feelings of anger and a lack of trust for the information given on the exercise duration of the subsequent bout (Billaut et al., 2011). Whereas in the CON trial blood lactate concentrations peak immediately post RLMSP-i, suggesting that as the correct information on duration to the exercise end-point was provided, a more ‘optimal’ pacing strategy was employed. As the closer to the end-point of exercise team players are, the more willing they are to exert themselves to bearable discomfort related to their ‘hazard score’, under the condition that the exercise end point is accurately known (de Konig et al., 2011). Providing further evidence that match performance (high intensity running) is regulated by a subconsciously functioning central governor (Edwards & Noakes, 2009).
This is not to say that either increased HR or the associated accumulation of hydrogen ions with elevated blood lactate concentrations solely cause an individual to down-regulate efforts. Rather they operate as part of a complex multifactorial process incorporating peripheral responses to exercise which in turn feedback to the CNS, bringing about a reduced central motor ‘drive’ in order to prevent catastrophic failure of any one physiological system (Edwards and Noakes, 2009). This constant regulation of exercise intensity through complex afferent feedback and efferent feed-forward systems constantly operate (micro-pacing strategy) in order to fulfil the previously developed macro-pacing schema to ensure that at the point of cessation of exercise, the individual has worked at a vigorous intensity whilst avoiding any catastrophic failure (Edwards & Noakes, 2009), as evidenced in the CON trial.

5. Conclusion

5.1. Summary and applied implications

The current study conveys two main findings, these are; i) that pacing does occur during a team game specific simulation of rugby league match-play and is comparable to pacing profiles during elite rugby league match-play; and ii) that manipulating the knowledge of the exercise end-point can significantly alter pacing throughout the RLMSP-i. Future research may look to assess how altered perceived exertion (nutritional supplementation e.g. caffeine) can manipulate pacing during a simulation of match-play.
6. References


the brain and peripheral physiological systems in pacing and perception of effort. *Sports Medicine, 36*(8), 705-722.


7. Appendices

**Additional Methodology**

**Appendix 1.** Order of events in the RLMSP-I

**Appendix 2.** Standardised instructions for deception

**Appendix 3.** Pre-exercise standardisation of physical state

**Additional Results**

**Appendix 8.** Means and SD for absolute distance covered

**Additional Materials**

**Appendix 4.** Ethical Approval Form

**Appendix 5.** Participant Information Sheet

**Appendix 6.** Informed consent

**Appendix 7.** Health Screening Form
Appendix 1: Order of events during the RLMSP-i

Each cycle of the RLMSP-i consists of two parts; the first (ball in play) lasting 60.32 s is performed twice and the second (ball out of play) lasting 48.25 s. The order of activity is as follows: 13.5 m sprint, 15 m jog (deceleration), 8 m sprint to contact, 7.0 s simulated contact (tackling a soft, cylindrical tackle bag; Gilbert Rugby, East Sussex, England; mass = 23 kg; dimensions = 138 x 45 cm). The contact involves tackling the bag with the shoulder at approximately hip height. Once landed in a prone position, the participant was instructed to roll 360° laterally whilst holding the bag, touching it on the floor, before rolling laterally 360° back to the original position. This contact was alternated with a ‘flapjack’ movement (once per cycle), requiring the participant to sprint in an 8 m line (as previous; Figure 2), then go from a standing to a prone position, followed by a 360° lateral roll, before rolling back 360° to the original position and standing up. Following the contact the following order of events ensue; 20.5 m jog (ball in play), and 13.5 m walk x 2, 13.5 m jog, 13.5 m walk (ball out of play). This cycle is repeated 24 times (2 × 12 cycles) with a 20 min passive recovery half way through to simulate both half time and substitution time.
Appendix 2: Standardised instructions for RLMSP-i (DEC, UN, CON)

The aim of this study is to examine the influence of game duration on exercise performance during simulated rugby league match play. You will complete 3 different rugby protocols where the game duration is modified. Details of these conditions are as follows:

**Condition 1**
A rugby league match consists of 2 x 40 min halves of exercise with an approximate 15 minute break in-between. During a match, interchanged players typically exercise for 23 minutes in two bouts separated by 20 minutes (a total of 46 minutes of exercise). You are being asked to complete **TWO** x 23 minutes of simulated rugby league match play, separated by 20 minutes of rest, to replicate this typical exercise pattern of an interchanged rugby league player. A clock will be provided from which you can gauge the duration of your exercise bout.

**Condition 2**
A rugby league match consists of 2 x 40 min halves of exercise with an approximate 15 minute break in-between. During a match, interchanged players typically exercise for 23 minutes in two bouts separated by 20 minutes (a total of 46 minutes of exercise). You are being asked to complete **ONE** x 23 minutes of simulated rugby league match play to replicate an aspect of the typical exercise pattern of an interchanged rugby league player. A clock will be provided from which you can gauge the duration of your exercise bout.

**Condition 3**
A rugby league match consists of 2 x 40 min halves of exercise with an approximate 15 minute break in-between. During a match, interchanged players typically exercise for 23 minutes in two bouts separated by 20 minutes (a total of 46 minutes of exercise). In this trial, you will not be informed of your exercise duration, but you may be asked to exercise for up to 2 x 40 minutes. A clock will be provided from which you can gauge the duration of your exercise bout.
Appendix 3: Pre-exercise standardisation of physical state

All testing was conducted at similar times of day (i.e. consistently between either 9 am – 11 am, 12 pm – 2 pm or 4 pm – 6 pm), to control for circadian alterations in exercise capacity. Participants were instructed to refrain from strenuous exercise for 24 hours before each testing session. Food diaries were recorded in the 48 hours immediately before trial one (Figure 1) and participants were asked to repeat this diet in the 48 hours before the remaining two trials to control for effects of pre exercise dietary intake on performance (Waldron, Highton & Twist, 2013).
Appendix 4. Ethical Approval Form

Thomas Mullen

9th July 2014

Dear Thomas,

Study title: Influence of knowledge of playing duration on pacing during a simulated rugby league protocol.
FREC reference: 919/14/TM/S6
Version number: 2

Thank you for sending your application to the Faculty of Life Sciences Research Ethics Committee for review.

I am pleased to confirm ethical approval for the above research, provided that you comply with the conditions set out in the attached document, and adhere to the processes described in your application form and supporting documentation.
Appendix 5. Participant Information Sheet

Influence Of Playing Duration On Exercise Performance During A Simulation Of Rugby League Match Play

You are being invited to take part in this MSc dissertation research study. Before you decide, it is important for you to understand why the research is being conducted and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask me if there is anything that is not clear or if you would like more information before you decide whether or not you wish to take part.

Thank you for reading this.

What is the purpose of the study?
The purpose of this study is to document the physiological and perceptual responses to varying durations of a forward-specific simulated rugby league match protocol.

Why have I been chosen?
You have been selected to participate in this study as you are a moderately trained male, and you are currently playing rugby (league or union) competitively at the University of Chester.

Do I have to take part?
It is up to you to decide whether or not to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive in any way.

What will happen to me if I take part?
You will be required to visit the laboratory on four occasions. On the initial (baseline) visit, you will complete the multi-stage running test to estimate your maximal aerobic capacity. Thereafter, you will complete three trials of the interchange Rugby League Movement Simulation Protocol (RLMSP-i), which involves intermittent movement (standing walking, jogging, sprinting and tackling a cylindrical tackle bag) dictated by audio cues from a CD. The movement will be between colored cones on a 3G artificial playing field, and is made to replicate movement speeds found in competitive rugby league match play. Each trial will consist of a duration of the RLMSP (up to 80 minutes) and will be performed with 7-10 days between each trial. During each trial blood lactate, heart rate, muscle function (isokinetic dynamometer), rating of
perceived exertion and movement intensity (with a 5 Hz global positioning system) will be recorded. You will be required to complete a food diary in the 48 hours before the first trial, and will be asked to repeat this dietary intake in the 48 hours before the following two trials.

**What are the possible disadvantages and risks of taking part?**
The slippery surfaces on the 3G could incur slips and trips leading to cuts and grazes. The study will also push you to physical exhaustion (multi-stage fitness test) and may involve some muscle soreness and weakness which may last for 72 hours after taking part. You will also be required to avoid any demanding physical exercise in the 24 hours prior to testing, so this may disrupt your daily routines slightly. The blood lactate analysis will require fingertip blood samples and may cause slight pain and bruising but will be alleviated 48 hours post exercise.

**What are the possible benefits of taking part?**
You will gain experience of taking part in a post graduate research project in exercise physiology. An evaluation of your aerobic fitness will also be provided from which recommendations for improving your performance will be provided, plus the invaluable data from GPS analysis will be made accessible to you, reporting your movement demands performed during the testing (e.g. sprint speed, distance covered).

**What if something goes wrong?**
If you wish to complain or have any concerns about any aspect of the way you have been approached or treated during the course of this study, please contact Professor Ken Green, Head of Department, Department of Sport and Exercise Sciences, University of Chester, Parkgate Road, Chester, CH1 4BJ, 01244 513426.

If you are harmed by taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone’s negligence (but not otherwise), then you may have grounds for legal action, but you may have to pay for this.

**Will my taking part in the study be kept confidential?**
All information that is collected about you during the course of the research will be kept strictly confidential so that only the researcher carrying out the research (Thomas Mullen) and his research supervisor (Dr Craig Twist) will have access to the information, furthermore all data will be coded to ensure anonymity.

**What will happen to the results of the research study?**
The results will be written up for a Level 7 (MSc) dissertation. In addition, the material might be published in an academic peer-reviewed journal. Individuals who participate will not be identified in any subsequent report or publication. You are most welcome to request a copy of the results of said dissertation should you wish.

**Who is organising and funding the research?**
An MSc student (Thomas Mullen) from the Department of Sport and Exercise Sciences at the University of Chester will be involved in organising and carrying out the study, and no external funding will be provided to conduct this research.

**Who may I contact for further information?**
If you would like more information about the research before you make your decision to participate (or not), please do not hesitate to contact:

Thomas Mullen

E-mail: @chesterac.uk

Thank you for your interest in this research.
Appendix 6. Informed consent

Title of Project: Influence of knowledge of playing duration on pacing during a simulated rugby league protocol

Name of Researcher: Thomas Mullen

Please initial box

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions.

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason.

3. I agree to take part in the above study.

___________________                  _________________                  _______________
Name of Participant    Date   Signature

Thomas Mullen

Researcher    Date   Signature
Appendix 7. Health Screening Form

PRE-TEST HEALTH QUESTIONNAIRE

(PLEASE NOTE THAT THIS INFORMATION WILL BE CONFIDENTIAL)

Name:…………………………….  DOB:…………….. Age:…………

Study Title: Influence of knowledge of playing duration on pacing during a simulated rugby league protocol

Researcher: Thomas Mullen (MSc Student)

Please answer these questions truthfully and completely. The purpose of this questionnaire is to ensure that you are fit and healthy enough to participate in this laboratory practical/research project.

1. Have you in the past suffered from a serious illness or accident. □   □
   If Yes, please provide details

2. Have you consulted your doctor the last 6 months □   □
   If Yes, please provide details

3. Do you suffer, or have you suffered from:

   Yes   No
   Asthma   □   □
   Diabetes   □   □
   Bronchitis   □   □
   Epilepsy   □   □
   High blood pressure   □   □

4. Is there any history of heart disease in your family □   □

5.
6. Are you suffering from any infectious skin diseases, sores, blood wounds, or infections i.e., Hepatitis B, HIV, etc.?  
If Yes, please provide brief details 
..........................................................................................................................................................  
..........................................................................................................................................................

7. Are you currently taking any medication  
If Yes, please provide details 
..........................................................................................................................................................

8. Are you suffering from a disease that inhibits the sweating process  

9. Is there anything to your knowledge that may prevent you from participating in the testing that has been outlined to you?  
If Yes, please provide details 
..........................................................................................................................................................

Your Recent Condition  

- Have you eaten in the last 2 hours?  
  If Yes, please provide details 
  ..........................................................................................................................................................

- Have you consumed alcohol in the last 24hr 
  □  □

- Evaluate your diet over the last two days.  **Poor Average Good Excellent**

- Have you had any kind of illness or infection in the last 2 weeks  
  □  □

- Have you exercised in the last 2 days?  
  □  □

  If Yes, please describe below

Persons will not be permitted to take part in any experimental testing if they:
- have a known history of medical disorders (i.e. hypertension, heart or lung disease)
- have a fever, suffer from fainting or dizzy spells
- are currently unable to train because of a joint or muscle injury
- have had any thermoregulatory disorder
- have a history of infectious diseases (i.e. HIV or Hepatitis B)

My responses to the above questions are true to the best of my knowledge and I am assured that they will be held in the strictest confidence.

Name: (Participant)……………………………………..  Date:…………………………

Signed (Participant): ………………………………………

Name:  **Thomas Mullen**  Date:…………………………
Signed (Researcher): ...........................................
Appendix 8: Total and high intensity distance covered over the course of the RLMSP-i.

<table>
<thead>
<tr>
<th></th>
<th>Bout 1</th>
<th>Bout 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Q1</td>
<td>Q2</td>
</tr>
<tr>
<td><strong>Distance covered (m)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>621 ± 24</td>
<td>616 ± 18</td>
</tr>
<tr>
<td>Unknown</td>
<td>617 ± 18</td>
<td>609 ± 23</td>
</tr>
<tr>
<td>Deception</td>
<td>618 ± 22</td>
<td>610 ± 13</td>
</tr>
<tr>
<td><strong>HIT Distance covered (m)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>173 ± 25</td>
<td>159 ± 19</td>
</tr>
<tr>
<td>Unknown</td>
<td>161 ± 22</td>
<td>153 ± 11</td>
</tr>
<tr>
<td>Deception</td>
<td>168 ± 17</td>
<td>158 ± 15</td>
</tr>
</tbody>
</table>

*Note. a = significantly different (P <0.0125) to quartile two of the same bout; b = significantly different (P <0.0125) to quartile three of the same bout; c = significantly different (P <0.0125) to quartile four of the same bout; * = significantly different (P <0.0125) to bout one; 2 = significantly different (P <0.0125) to unknown trial; 3 = significantly different (P <0.0125) to deception trial.*