Metabolic demands and replenishment of muscle glycogen after a rugby league match simulation protocol.

Abstract

Objectives: The metabolic requirements of a rugby league match simulation protocol and the timing of carbohydrate provision on glycogen re-synthesis in damaged muscle were examined. **Design:** Fifteen (mean ± SD: age 20.9 ± 2.9 y, body-mass 87.3 ± 14.1 kg, height 177.4 ± 6.0 cm) rugby league (RL) players consumed a 6 g·kg·day-1 CHO diet for 7-days, completed a time to exhaustion test (TTE) and a glycogen depletion protocol on day-3, a RL simulated-match protocol (RLMSP) on day-5 and a TTE on day-7. Players were prescribed an immediate or delayed (2-h-post) re-feed post-simulation. **Methods:** Muscle biopsies and blood samples were obtained post-depletion, before and after simulated match-play, and 48-h after match-play with PlayerLoad and heart-rate collected throughout the simulation. Data were analysed using effects sizes ± 90% CI and magnitude-based inferences. **Results:** PlayerLoad (8.0 ± 0.7 AU·min-1) and %HRpeak (83 ± 4.9%) during the simulation were similar to values reported for RL match-play. Muscle glycogen *very likely* increased from immediately after to 48-h post-simulation (272 ± 97 cf. 416 ± 162 mmol·kg-1d.w.; ES ± 90%CI) after immediate re-feed, but changes were *unclear* (283 ± 68 cf. 361 ± 144 mmol·kg-1d.w.; ES ± 90%CI) after delayed re-feed. CK *almost certainly* increased by 77.9 ± 25.4% (0.75 ± 0.19) post-simulation for all players. **Conclusions:** The RLMSP presents a replication of the internal loads associated with professional RL match-play, although difficulties in replicating the collision reduced the metabolic demands and glycogen utilisation. Further, it is possible to replete muscle glycogen in damaged muscle employing an immediate re-feed strategy.

Keywords: Physiology, Nutrition, Team-sport, Contact, Metabolism, Carbohydrate

Word Count: 3000

Introduction

Rugby league (RL) is a high-intensity, intermittent team sport played over 80 minutes during which players perform repeated high-intensity efforts such as sprints, high-impact collisions, and rapid changes of direction.[1] These efforts are interspersed with periods of low-intensity activity such as jogging, walking, and standing.[2, 3] The prevalence of the actions performed during a match are specific to playing position,[2] and are likely to influence the metabolic requirements for an individual player.

While muscle glycogen has recently been found to be depleted by ~40% after a professional RL match,[4] further studies to examine the metabolic requirements imposed on players are challenged by the large inter-match variations in movement characteristics observed in RL match play.[5] Accordingly, a case for utilising a simulated RL match protocol replicating the physiological demands and movement patterns of real matches might be appropriate to permit greater control whilst conducting dietary intervention studies. Observations of a validated RL simulation protcol[6] reported similar total distance covered and $\%HR_{peak}$ to elite RL match-play[7] suggesting an accurate reflection of real match movement demands. However, the metabolic demands and glycogen utilisation of a simulated RL match remain unknown and warrant investigation.

Although the match characterictics,[3, 8] and nutritional requirements to fuel profesional players during a RL match have previously been described,[4] nutritional requirements for optimal recovery are poorly understood. Despite major differences in the match-day demands between soccer and rugby, similar changes in pre- to post-match muscle glycogen concentrations have been reported,[4, 9] with many players finishing a match with less than 200 $mmol \cdot kg^{-1}$ dry weight (d.w.) of muscle glycogen in both rugby[4] and soccer.[9] The extent to which muscle glycogen is depleted heavily influences the magnitude of re-synthesis,[10] with current nutritional guidelines suggesting ingestion of 1.2 $g \cdot kg^{-1}$ $bw \cdot h^{1}$ of carbohydrate (CHO) for three hours post-match.[11] However, recommended nutritional guidelines for athlete recovery[12] are based on data from soccer[13] and translated for use in RL. Given differences in match-play activities between soccer and rugby, the suitability of using such studies to inform nutritional practices of rugby are therefore questionable.[14] Accordingly, a study to examine the magnitude of glycogen repletion of RL players after simulated match play and the role of different nutritional strategies in

facilitating this is necessary.

Glucose transport in to the muscle cell via GLUT-4 to facilitate glycogenesis can be regulated by insulin and exercise, and is characterised by two phases; the insulin dependent phase (in the presence of glucose), and the non-insulin dependent phase (exercise induced increase in GLUT-4 translocation), which can last between 0.5 to 2-h after exercise in undamaged muscle.[15] A damaged muscle however might reduce the efficacy of glucose uptake resulting in decreased glycogen synthesis.[16] Furthermore, inflammatory cells present within damaged muscle have an affinity for glucose oxidation, competing with glycogen depleted muscle cells for blood glucose,[17] resulting in a reduction in glycogen synthesis. Despite high volumes of eccentric muscular contraction throughout real or simulated RL match-play from repeated bouts of acceleration, deceleration, rapid changes in direction, and impacts,[18] it is currently unknown how the resulting muscle membrane damage[19] affects muscle glycogen repletion in the days after. Therefore, the aims of the present study were: 1) to examine the metabolic demands of a simulated RL match and compare with previously published data from professional RL match play and 2), assess the efficacy of an immediate or delayed carbohydrate re-feed on muscle glycogen re-synthesis in damaged muscle after a simulated match. Accordingly, we assessed university RL players who performed a validated RL match simulation protocol (RLMSP),[6] after which they consumed either an immediate or delayed re-feed. We hypothesised that: 1) the simulated match would result in similar muscle glycogen utilisation to that previously reported in a professional RL match;[4] 2) the immediate re-feed would result in better muscle glycogen re-synthesis; and 3) with a correct re-feeding strategy it will be possible to replenish a damaged muscle after a simulated rugby match.

Methods

In a matched pairs design (body mass used to match pairs) and using a random number generator, players were randomly allocated into an immediate re-feed (n = 8) or a delayed re-feed (n = 7) group. Testing took place over two separate weeks in an indoor training facility to ensure an identical playing surface and weather conditions. Over a 7-day period, players consumed a standardised diet and completed a time to exhaustion test (TTE) as a non-damaging marker of performance followed by a glycogen depleting protocol on Day-3, a validated RLMSP[6] to deplete muscle glycogen on Day-5, followed by a final TTE on Day-7 (Figure 1). Due to technical issues, blood samples were not collected on 5 players therefore all performance, HR and muscle biopsy data are reported as n=15, whereas bloods data are presented on *n*=10.

(Insert Figure 1 here)

Fifteen male university RL players (mean ± SD: age 20.9 ± 2.9 y, body-mass 87.3 ± 14.1 kg, height 177.4 ± 6.0 cm) playing in the British Universities and College Sports (BUCS) Northern 1A league volunteered to take part in this study. Ethics approval for the study was granted by the ethics committee of Liverpool John Moores University and all participants provided written informed consent.

Players performed a maximal incremental cycling test to volitional fatigue on a Lode ergometer (Daum Electronic Premium 8i, Furth, Germany) for determination of peak power output (PPO) and time to exhaustion (TTE). Tests were started every 5 minutes with players in different areas of the laboratory with partitioning screens to ensure that there was no competition bias. The maximal incremental protocol commenced at 150 W for 2-min, with work rate increased by 30 W every minute thereafter until exhaustion.[20] TTE was recorded as a measure of performance, and the highest power output (W) attained used to inform exercise intensity during the glycogen depletion protocol. After the TTE test, players were given 15-minutes to rest before beginning an intermittent glycogen-depleting cycling protocol. Previous studies from our group have shown that without this pre-trial depletion, pre-exercise muscle glycogen concentrations are extremely variable between participants.[4] Therefore, employing this procedure, followed by 48-h of controlled carbohydrate intake, ensured that all participants commenced

the RLMSP with similar muscle glycogen concentrations. An adapted version of the protocol used by Pederson et al, (2008)[21] was used for the glycogen depleting exercise protocol. After a 5-min warm-up at a self-selected intensity, participants commenced cycling at 90% of PPO for 2-min followed immediately by 1-min of an active recovery at a self-selected intensity. This work-recovery protocol was maintained until the participants were unable to complete 2-min at 90% PPO, determined as an inability to maintain a cadence of 60 rpm for 15 s. When players could not maintain 2-min at 90%, the work bouts were reduced to 1.5 min, while maintaining the 1-min recovery bouts at a self-selected intensity. When players were unable to maintain the 1.5-min at 90%, the work period was reduced to 1-min. Once the participants could not maintain 90% of PPO for 1-min, the intensity was lowered to 80%, 70% and finally 60% of PPO following the same work to rest pattern. Exercise was terminated when players could not complete 1-min of cycling at 60% of PPO at a cadence of 60 rpm. This protocol was chosen so as to maximally deplete muscle glycogen in both Type I and Type II muscle fibers and to standardize muscle glycogen concentration between participants. Verbal encouragement was given and water was consumed *ad libitum* throughout exercise.

Individualised 7-day diets were designed and provided to all participants. Diets consisted of 6 $g \cdot kg \cdot day^{-1}$ CHO, 2 $g \cdot kg \cdot day^{-1}$ protein and 1 $g \cdot kg \cdot day^{-1}$ fat, with habitual diet ingested before the intervention. All meals were designed and distributed to the players by a Sport and Exercise Registration (SENr) accredited practitioner and prepared by a recognised catering food and drink supplier (Soulmate Food, UK) complete with meal plans. All players were given strict instructions to follow the diet explicitly, not to consume any foods or liquids (apart from water) other than what was provided, and to finish all meals provided. Players self-reported that they had strictly adhered to the diets prescribed to them. All supplements prescribed were Informed Sport, a quality assurance program that certifies all nutritional supplements/ingredients have been tested for banned substances. Players were randomly allocated to one of two dietary re-feed conditions; immediate or delayed ingestion of CHO after the RLMSP. The immediate dietary group consumed a beverage containing 90 g CHO (Maltodextrin, My Protein, UK) and 30 g PRO (True Whey, My Protein, UK), followed by a further 90 g CHO (My Protein maltodextrin) beverage 1-hour later. The delayed dietary group consumed visually identical beverages containing 30

g PRO (True Whey, My Protein, UK), followed by a zero calorie hypotonic sports drink 1-hour later. All participants continued with prescribed diets 2-h after the simulation. Post-exercise drinks were delivered as an absolute, rather than relative dose of carbohydrate and protein given that there is currently no consensus as to the optimal amount of protein to be consumed after exercise, and such practice is common in elite rugby.

An adapted version of the RLMSP[6] was used to avoid large between player variation in match activities associated with real matches.[5] The movements for the protocol are based on the mean locomotive speeds and activities of whole-match players established during competitive RL matches.[6] Briefly, the protocol lasted for 89 min 20 s (2 x 44 min 40 s separated by 10-min to simulate half-time), replicating the mean time that a whole-match player spends on a pitch including stoppages. The protocol comprises 40 identical cycles lasting 2-min 14 s. Each cycle comprised Part A (ball in play) lasting 48.4 s, which was performed twice, and Part B (ball out of play) lasting 49.3 s and is performed once. Part A involved: 10.5 m jog and turn, 10.5 m walk and turn, 20.5 m sprint, 8 m deceleration, contact with 30 kg tackle bag and 4 s 'wrestle' (first time) or down and up of the ground (second time), 13 m jog and 15.5 m walk. Part B involved: 10.5 m walk and turn, 10.5 m walk and turn, 6 s passive rest, 15.5 m jog and turn, 15.5 m walk and turn, 4.75 s passive rest. Players wore a custom designed vest fitted with a micro-technology device (Optimeye S5, Catapult Innovations, Melbourne, Australia) positioned between the scapulae to measure accumulated PlayerLoad™ (AU). Accumulated PlayerLoad™ is derived from the micro-technology device's embedded tri-axial accelerometer and is presented as an arbitrary value based on the combined rate of change of acceleration in three planes of movement; forward, lateral and vertical. The metric provides an accepted method to quantify match and training loads of collision sports,[22] particularly in this case where the study was conducted indoors and GPS was not available. Heart rate was also monitored using a coded transmitter unit (Polar, Oy, Finland) strapped to the chest with data transmitted and recorded to GPS units for later download and analysis. Perceived exertion (RPE) was collected using the category ratio (e.g., 0-10) RPE scale of Borg from which session RPE (sRPE; AU) was calculated by multiplying RPE by total time of exercise bout.

Muscle samples were obtained using a biopsy gun (Monopty 12g, BARD, Brighton, UK) from the middle of the *vastus lateralis* muscle as previously described.[23] Given the increase in inflammatory markers after a muscle biopsy have been well documented, it was decided that biopsies would be taken in a randomised order from alternate legs at each time point, with the second incisions on each leg made close to the original site (~2 cm proximal). All muscle samples were immediately snap frozen in liquid nitrogen and stored at -80°C for later analysis. Venous blood samples (5 ml) were drawn from a superficial vein in the anticubital fossa of the forearm using standard venipuncture techniques (Vacutainer Systems, Becton, Dickinson). Samples were collected into three vacutainers (Serum Separating Tube, EDTA and Lithium Heparin tubes, Nu-Care Products, UK), and were stored on ice (apart from serum) until centrifugation at 1500 RCF for 15-min at 4°C. Biopsy and blood samples were taken within a 15-min time-period which was achieved by having three qualified researchers performing the biopsies simultaneously.

Muscle glycogen concentration was determined as previously described.[23] Briefly, approximately 2-3 mg of freeze-dried sample was dissected free of all visible non-muscle tissue and subsequently hydrolyzed by incubation in HCI for 3-h at 100°C. After cooling to room temperature, samples were neutralized by the addition of Tris/KOH saturated with KC1. After centrifugation at 10,000 RCF for 10 min at 4° C, 200 µl of the supernatant was analysed by spectrophotometry in duplicate using Randox Daytona (Randox Laboratories, Antrim, UK) for glucose concentration according to the hexokinase method at 340 nM using commercially available kit (GLUC-HK, Randox Laboratories, Antrim, UK). Intra-assay coefficients of variation was <5%. Glycogen concentrations are expressed as nM and need to be converted to mmol·kg $^{-1}$ d·w^{-1}. Blood was analysed spectrophotometrically for glycerol, non-esterified fatty acid (NEFA) and CK concentrations using commercially available kits (Randox, Laboratories, Antrim, UK) with intra-assay CV of <5% for all assays.

Magnitude-based inferential statistics were employed to provide information on the size of the differences allowing a more practical and meaningful explanation of the data. Differences in i) muscle glycogen concentrations and blood metabolites before and after rugby match-play and 48-hours after rugby match-play and ii), TTE scores between the pre- and re-test between immediate and delayed

dietary groups using Cohen's effect size (ES) statistic ± 90% confidence limits (CL) and magnitude-based inferences.[24] Threshold probabilities for a meaningful effect based on the 90% confidence limits (CL) were: <1%, *almost certainly not*; 1-5%, *very unlikely*; 5-25%, *unlikely*; 25-75%, *possibly*; 75-97.5%, *likely*; 97.5-99% *very likely*; >99%, *almost certainly*. Effects with confidence limits across a likely small positive or negative change were classified as unclear.[26] All analyses were completed using a predesigned spreadsheet.[26]

Results

Differences in PlayerLoad™ during the RLMSP (2 ± 9%, 0.02 ± 0.01) were *unclear* between the immediate (7.3 ± 0.43 AU·min^{-1}) and delayed re-feed (8.0 ± 0.33 AU·min^{-1}) groups. Similarly, %HR_{peak} (84 ± 4 cf. 82 ± 6%; 2 ± 6 %, 0.03 ± 0.08) and sRPE (mean ± SD cf. mean ± SD; 7 ± 11 %, 0.54 ± 0.85,) were *unclear* for immediate and delayed re-feed groups, respectively.

Muscle glycogen data are reported in Figure 2. All players sufficiently depleted muscle glycogen after the depletion protocol (<50 mmol·kg^{-1}d.w.) and mean muscle glycogen concentrations were relatively high before commencing the simulation after following the standardized diet (337 *cf.* 376 mmol·kg^{-1}d.w. in immediate and delayed re-feed groups, respectively). Muscle glycogen concentrations were decreased immediately after the simulation by 25 ± 14% (ES ± 90% CI: -1.38 ± 0.90, *very likely*) and 24 ± 12% (-1.6 ± 0.92, *very likely*) in the immediate and delayed re-feed groups, respectively. Muscle glycogen concentrations were increased 48-h after the simulation by 51 ± 47% (0.88 ± 0.66, *very likely*) in the immediate re-feed but only 24 ± 49% (0.7 ± 1.23, *unclear*) in the delayed re-feed group. Between group analysis revealed that muscle glycogen concentrations were slightly lower before the simulation in the immediate compared with delayed re-feed group (-4 ± 58%, 0.66 ± 0.74, *likely*) but were *unclear* after depletion (12 ± 18%, -0.06 ± 0.85), immediately (11 ± 38 %, 0.17 ± 0.7) and 48-h after the simulation (-10 ± 35%, -0.32 ± 0.79).

Changes in blood metabolites (plasma glycerol, NEFA and CK) are reported in Figure 3. Glycerol concentrations were increased after the simulation by 60 ± 60% (0.78 ± 0.52, *likely*) and 103 ± 23% (4.13 ± 0.76, *almost certainly*) in the immediate and delayed re-feed group, respectively. Glycerol concentrations were *unclear* between groups before (0.42 ± 1.26) and after the simulation (-0.32 ± 1.47), although the delayed group presented higher concentrations than the immediate re-feed group 48-h after (1.36 ± 0.87, *very likely*). NEFA concentrations were increased after the simulation by 1049 ± 581% (2.01 ± 0.40, *almost certainly*) and 998 ± 1060% (2.4 ± 0.86, *almost certainly*) in the immediate and delayed re-feed group, respectively. Differences between groups in NEFA concentrations were *unclear* before (0.29 ± 0.76), immediately (0.10 ± 0.87), and 48-hr after the simulation (0.58 ± 0.92). CK

concentrations were increased after the simulation by 128 ± 43% (0.60 ± 0.18, *very likely*) and 84 ± 41% (0.63 ± 0.23, *very likely*) in the immediate and delayed re-feed group, respectively. CK concentrations remained higher but decreased at 48-h after by 29 ± 38% (-0.42 ± 0.61, *likely*) and 30 ± 20% (-0.55 ± 0.43, *likely*) in the immediate and delayed re-feed groups, respectively. Differences between groups in CK concentrations were *unclear* before (-0.4 ± 0.9), after (-0.38 ± 0.88), and 48-h after the simulation (-0.24 ± 0.6).

TTE decreased from 671 ± 127 s to 586 ± 202 s (-14 ± 11%; -0.66 ± 0.54, *likely*) in the immediate re-feed group and 611 ± 171 s to 564 ± 202 s (-10 ± 7% (-0.35 ± 0.28, *likely*) in the delayed re-feed group. Differences in TTE between groups were *unclear* for the initial (-10 ± 19 %; -0.47 ± 0.92) and follow up assessment (-11 ± 25 %; -0.34 ± 0.85).

Discussion

The aim of the present study was to assess the metabolic demands and glycogen utilisation of a simulated RL match and assess glycogen re-synthesis up to 48-h afterwards. We provide novel data demonstrating that 1) simulated match-play utilises ~50% less muscle glycogen than previously reported for professional RL match-play. This is likely because of the difficulties in replicating physical collisions and exertions during the simulation; 2) a *very likely* increase in muscle glycogen repletion was observed when CHO was ingested immediately post-exercise compared with an *unclear* difference following the delayed re-feed; 3) this occurred despite increases in CK suggesting muscle damage has resulted from the simulated activity therefore implying that it is possible to acutely replete muscle glycogen in a damaged muscle following a simulated rugby performance.

Muscle glycogen was depleted by ~21% during the simulation, which is less than previously reported in RL match-play (~40%).[4] A lower glycogen depletion occurred despite heart rate (~83 $\%HR_{peak}$) and PlayerLoad (~7.7 $AU \cdot min^{-1}$) during the simulation being consistent with the loads reported in competitive matches.[7, 27] Furthermore, analysis of blood metabolites revealed comparable rates of lipid mobilization to those reported in elite RL match-play,[4] suggesting that the RLMSP successfully reflected the intermittent nature of the sport. That PlayerLoad was similar to actual match-play, despite the lack of true physical collisions in the simulation, is probably explained by the higher movement speeds in the simulation protocol.[6] The lower glycogen depletion in the simulation is likely a reflection of an inability to replicate the physicality of collisions in matches, with tackles and wrestling movements performed on a passive 30 kg tackle bag rather than an opposing player. The smaller magnitude of increase in CK activity when compared to match play[28] also suggests the simulation was unable to replicate the collision. Our findings reaffirm the difficulties in simulating physical contact[29] and, more importantly, highlight the large metabolic cost of the tackle in rugby league. Future study should now attempt to increase the physicality of the RLMSP to more accurately reflect the physical demands of rugby match-play.

The immediate re-feed elicited a *very likely* increase (~53%) in muscle glycogen concentration 48-h after the simulated match, whereas the difference was *unclear* (~27%) in players who consumed the delayed re-feed. This large discrepancy probably highlights the importance of the short-lived non-insulin dependent phase for rapidly resynthesizing muscle glycogen after exercise in the presence of CHO.[30,31] Furthermore, while not reported in the literature, anecdotal evidence demonstrates that players struggle to consume food in the immediate period post-match and often do not compensate for this in the following days. The accumulative effect of this might lead to improper recovery and players dropping body mass throughout the competitive season, accentuating the importance of an appropriate nutrition strategy for recovery. Differences in glycogen repletion might also be because the delayed re-feed group consumed 180 g less CHO than the immediate re-feed group. Given that all players continued with the same high CHO diet for a further 48-h this is however unlikely, and highlights the importance of immediately re-feeding post-exercise for optimal muscle glycogen re-synthesis. Between group analysis revealed that there was a small, yet likely, lower muscle glycogen concentration in the immediate re-feed group compared with the delayed re-feed group before the match simulation (~4% difference), which may have contributed to the discrepancy in glycogen re-synthesis between groups. This difference was surprising given that both groups performed the same glycogen depleting protocol and then followed identical diets in an attempt to standardise pre-simulation muscle glycogen concentrations. However, given that the simulation elicited a ~21% muscle glycogen depletion, this minor discrepancy is unlikely to have had any meaningful effects. The difference is likely attributable to variability in muscle glycogen concentrations within individual muscle fibres[9] and represents known limitations in the muscle biopsy technique rather than any physiological difference in muscle glycogen concentration. Similarly, the unclear differences in muscle glycogen concentration between the 2 groups 48-h after the simulation is likely attributable to the large between-participant variability in muscle glycogen repletion and the aforementioned limitations of the biopsy technique.[9]

Despite raised CK concentrations indicating muscle damage did occur, meaningful muscle glycogen re-synthesis was possible in the immediate dietary re-feed group, suggesting that with appropriate feeding strategies it is possible to replenish a damaged muscle. However, the *vastus lateralis* is composed of

283 both fast and slow twitch muscle fibres permitting the extraction of individual muscle fibres from either
284 fibre type. Future studies should therefore look to employ histochemical analysis of individual skeletal
285 muscle fibers to assess muscle damage, and the efficacy of nutritional intervention on glycogen
286 utilisation and repletion around RL match-play.
287
288 The TTE performance test was used to observe assess the physiological consequences of the two dietary
289 strategies 48-h after simulated match-play, and to mimic the schedule of professional rugby players.[14]
290 A lower TTE 48-h after match-play for both groups is likely attributed to a reduction in force generating
291 capacity from exercise-induced muscle damage[32] caused by the simulation protocol.[6] This decrease did
292 not appear to be affected by. That the effect of manipulating CHO immediately after exercise on
293 subsequent TTE performance was *unclear*, suggests that a larger sample is required or a more sensitive
294 measure of performance was needed to resolve the uncertainty. Despite the lack of specificity to rugby,
295 the use of the TTE performance measure was necessary to avoid tissue damage that would be associated
296 with load-bearing exercise. Moreover, it would be unlikely for players to perform prolonged rugby
297 specific exercise 48-h post match. This notwithstanding, further work is required to detect the effect of
298 different feeding strategies on light exercise performed in the days after a match.
299
300 This study is not without limitations, most of which are due to the controlled nature in which data were
301 collected. It was necessary to standardise the exercise time and distance covered for all participants,
302 which means the data do not reflect the match demands of interchange players. Furthermore, replicating
303 contacts with a tackle bag was necessary to control the number and intensity of the collisions, but means
304 the blunt force trauma induced to participants was much lower compared to match-play.[28] Finally, CK
305 activity revealed evidence of tissue damage after the simulation protocol, but demonstrated large
306 between-participant variability in resting values and was not measured at 24-h where peak values would
307 have been expected.[28] These data reaffirm the limitations of using blood-borne parameters to confirm
308 the magnitude of tissue damage in rugby league players.[33] Future studies might consider measures such
309 as muscle soreness and function to support the occurrence of exercise-induced muscle damage after such

exercise.

Conclusion

We have for the first time demonstrated that simulated RL match-play elicits lower muscle glycogen utilisation (21 *cf.* 40 %) despite similar player load and metabolic demands to a professional RL match. We attribute this to the difficulties of replicating extensive structural damage and physical exertion from collisions during a simulation. We also show substantial muscle glycogen re-synthesis was possible in the immediate dietary re-feed group despite evidence of muscle damage via increase blood proteins suggesting that with appropriate feeding strategies it is possible to replenish a damaged muscle.

Practical Implications

- The present study has reaffirmed that the RLMSP elicits a similar internal load, but the metabolic demands are lower than during professional RL match-play.
- Collision events in rugby league present a large metabolic cost to the player and are difficult to simulate in training and research settings
- The RLMSP might be used as a specific conditioning tool to condition or evaluate a player's readiness for match-play.
- Despite high carbohydrate consumption in the 48-h after RL match-play, the immediate re-feed seems to be a major contributor to muscle glycogen re-synthesis in damaged muscle.

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Figure legends:

Figure 1 – Schematic representation of the study time line expressed as days away from simulation (Sim): Sim-4, Sim-3, Sim-2, Sim-1, Sim, Sim+1, Sim+2.

Figure 2 - Muscle glycogen concentrations at four time points: Depletion, Pre- and Post-match-play, and 48-h Post-match-play presented in $mmol \cdot kg^{-1}$ d.w. taken from 15 university rugby league players. * = difference in immediate group from previous time point. # = difference in delayed group from previous time point.

Figure 3 - Plasma glycerol, non-esterified fatty acid (NEFA), and creatine kinase (CK) concentrations at four time points: Depletion, Pre- and Post-Simulation, and 48-h Post-simulation presented in $umol \cdot L^{-1}$, $mmol \cdot L^{-1}$ and $U \cdot L^{-1}$ respectively. * = difference in immediate group from previous time point. # = difference in delayed group fro previous time point.