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The recovery of muscle function and glycogen levels following game-play in young elite male ice hockey players

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Abstract

Despite the frequent occurrence of congested game fixtures in elite ice hockey, the postgame recovery pattern has not previously been investigated. The purpose of the present study was therefore to evaluate the acute decrements and subsequent recovery of skeletal muscle glycogen levels, muscle function and repeated-sprint ability following ice hockey game-play. Sixteen male players from the Danish U20 national team completed a training game with muscle biopsies obtained before, postgame and following ~38 h of recovery (day 2). On-ice repeated-sprint ability and muscle function (maximal voluntary isometric [MVIC] and electrically induced low- (20 Hz) and high-frequency (50 Hz) knee-extensor contractions) were assessed at the same time points, as well as ~20 h into recovery (day 1). Muscle glycogen decreased 31% ($p < 0.001$) postgame and had returned to pregame levels on day 2. MVIC dropped 11%, whereas 50 and 20 Hz torque dropped 21% and 29% postgame, respectively, inducing a 10% reduction in the 20/50 Hz torque ratio indicative of low-frequency force depression (all $p < 0.001$). While MVIC torque returned to baseline on day 1, 20 and 50 Hz torque remained depressed

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by 9%–11% ($p = 0.010$–0.040), hence restoring the pre-exercise 20/50 Hz ratio. Repeated-sprint ability was only marginally reduced by 1% postgame ($p = 0.041$) and fully recovered on day 1. In conclusion, an elite youth ice hockey game induces substantial reductions in muscle glycogen content and muscle function, but only minor reductions in repeated-sprint ability and with complete recovery of all parameters within 1–2 days postgame.

KEYWORDS

fatigue, intermittent exercise, performance, team sport

1 | INTRODUCTION

Ice hockey is a highly physically demanding team sport characterized by numerous repeated high-intense actions such as accelerations, decelerations, changes of direction, and body impacts.[1,2] This activity pattern has been shown to provoke reductions in the number of high-intense actions performed towards the end of the game,[3-6] accompanied by impairments in repeated-sprint ability,[7] suggestive of cumulative fatigue development. However, little is known about the time course of recovery after an elite ice hockey game, which is critical to develop optimal strategies for load distribution among players, manage the training process and implement appropriate recovery approaches and to inform stakeholders to optimize tournament planning. This is highly relevant in modern elite ice hockey as players endure tightly congested in-season game schedules and during cup tournaments, where games are often played on consecutive days interspersed with less than 24 h of recovery. In the National Hockey League, it is for example normal routine to compete in four games weekly, spending large amounts of time traveling and leaving limited time to restore physiological homeostasis, which may increase the predisposition to injuries and lead to performance deteriorations.[8]

It has previously been demonstrated that muscle force production in university level ice hockey players is impaired by up to 30% during a weekly cycle of games and training.[9] In particular, the force production obtained by low-frequency (20 Hz) electrical stimulation of the muscle surface was reduced postexercise and during recovery, a common phenomenon determined "prolonged low-frequency force depression".[10] Thus, low-frequency force production was still impaired the day after each practice/game session. This more pronounced and sustained force-loss at low compared to high stimulation frequencies may be attributable to altered Ca^{2+} release or impaired myofibrillar Ca^{2+} sensitivity and could induce performance impairments/increased sensation of fatigue despite no apparent reductions in single maximal efforts.[10] In other high-intensity intermittent team sports, such as soccer, the time course of recovery postgame has been extensively explored, demonstrating sustained reductions in sprint ability, vertical jump height and measures of muscle strength for up to 72 h.[11] The exact mechanism behind this long-lasting suppression of muscle function following soccer games awaits to be clearly understood, but has been linked with signs of cellular muscle damage and cytokine accumulation suggesting low-level inflammation, and elevated oxidative stress responses.[12–14] The postgame recovery pattern in ice hockey, however, is likely different due to the shorter game duration and possible differences in the muscular demands of skating compared to running.[2] Accordingly, plasma creatine kinase concentrations have been found to be up to two-fold higher when sampled 24 h after a soccer game compared to an ice hockey game suggestive of substantial differences in the degree of muscle damage,[3] although the precision of blood creatine kinase as a marker of muscle damage can be questioned.[15]

The resynthesis of muscle glycogen following soccer game-play has similarly been shown to be delayed for up to 72 h.[13] This has been attributed to the large eccentric component of the running-based soccer movements potentially causing a disruption in postexercise muscle glucose uptake.[16] In ice hockey, the high-intensity activity pattern provokes a comparable reduction in muscle glycogen as that observed in soccer (~50% depletion), despite the shorter game duration, including full depletion in a majority of individual fibers, conceivably involved in the fatiguing process.[1,7,17] Hence, if the time-course of muscle glycogen resynthesis is similarly delayed following ice hockey game-play, this would result in incomplete pre-game levels during subsequent game exposure. Nevertheless, due to the possible differences in the degree of muscle damage following ice hockey game-play, the disruption of postexercise muscle glucose uptake could be less severe, resulting in a potential faster rate of resynthesis.[3]

The aim of the present study was therefore to examine the reduction and subsequent recovery of muscle glycogen stores, muscle function, and repeated-sprint ability in response to ice hockey game-play in elite male players.

We hypothesized that an ice hockey game would lead to substantial reductions in muscle glycogen accompanied by impaired muscle function and repeated-sprint ability, but that these measures would be recovered within 2 days postgame. Moreover, we hypothesized that the reduction in low-frequency force would be exacerbated and possibly more prolonged (≥2 days).

2 | MATERIALS AND METHODS

2.1 | Participants

Sixteen young adult male ice hockey players from the Danish U20 national team participated in the study. Subject characteristics are presented in Table 1. Sample size was estimated based on previous power calculations using the present primary outcome variables.[7,18] For the muscle glycogen measurements five subjects were deemed necessary to yield a power of 0.80 with an α-level of 0.05,[7] while 12–16 subjects were found to be required for the MVIC assessments.[18] The present study included nine forwards and seven defensemen. The participants were informed of potential risks, discomforts and benefits associated with the study, before signing an informed consent. The study procedures adhered to the Declaration of Helsinki and were approved by the Regional Committees on Health Research Ethics for Southern Denmark (Project-ID: S-20210063).

2.2 | Study design

The participants took part in a training game that was completed in compliance with official ice hockey game rules. The game consisted of three periods lasting 20 min each, separated by 18 min of recovery. Each team consisted of three lines of defensemen and four lines of forwards whom rotated freely as determined by the coaches, reflecting a natural game scenario and inevitably leading to more time on ice for defensemen compared to forwards. The teams were evenly matched by the Danish national team head coach to make the game as competitive as possible. To further simulate a realistic competitive scenario, official referees officiated the game and spectators were allowed to attend. The game was played in the middle of the regular season and few weeks prior to the final roster selection for the U20 World Championships 2021 held in Denmark. Accordingly, players were in top physical form and highly motivated to perform at their best abilities. All players refrained from strenuous activity and caffeine 48 and 12 h before the game, respectively. Players were gathered for a training camp on the days of the study, where all participants received three standardized meals per day. While the participants were instructed to follow their habitual dietary intake during the time course of the study, no carbohydrate intake was allowed during the game. On the day after the game (~14 h after) the players participated in a light recovery session on the ice lasting 30 min. Otherwise, players rested between measurements. A local positioning system and heart rate recorders were used to continuously measure in-game activity patterns and physiological loading. In addition, a battery of performance tests was completed at four different time-points: prior to the game (baseline), immediately after the game (postgame), ~19–23 h postgame (day 1), and 37–39 h postgame (day 2) as outlined in detail below (see Figure 1 for complete study overview). In addition, muscle biopsies were sampled at the same time points, except that no biopsy was obtained on day 1 after the game.

2.3 | Pretesting

1–2 weeks prior to the study, most players ($n = 13$) performed a Yo-Yo Intermittent Recovery Level 1 Ice Hockey Test (Yo-Yo IR1-IH) as described in detail previously.[19] The remaining players ($n = 3$) were unable to participate in this test due to a busy game schedule and traveling constraints. Moreover, all participants were familiarized with the test procedures by undergoing one complete testing session of each type (repeated-sprint testing and assessments of muscle function).

TABLE 1 Subject characteristics. Values are presented as mean and range.

	All ($n = 16$)	Forwards ($n = 9$)	Defensemen ($n = 7$)
Age (y)	19.4 (18.2–19.9)	19.2 (18.2–19.9)	19.5 (19.1–19.9)
Height (cm)	184 (174–194)	183 (174–192)	186 (181–194)
Body mass (kg)	85.2 (76.9–102.5)	83.9 (76.9–100.8)	87.0 (77.8–102.5)
Body fat (%)	14.4 (9.6–20.9)	15.0 (10.1–20.3)	13.6 (9.6–20.9)
Muscle mass (kg)	41.8 (36.6–53.3)	40.8 (37.6–49.3)	43.1 (36.6–53.3)
Yo-Yo IR1-IH (m)	2329 (1800–2720)	2340 (1800–2720)	2312 (2080–2480)

Abbreviation: Yo-Yo IR1-IH, Yo-Yo intermittent recovery level 1 ice hockey test.

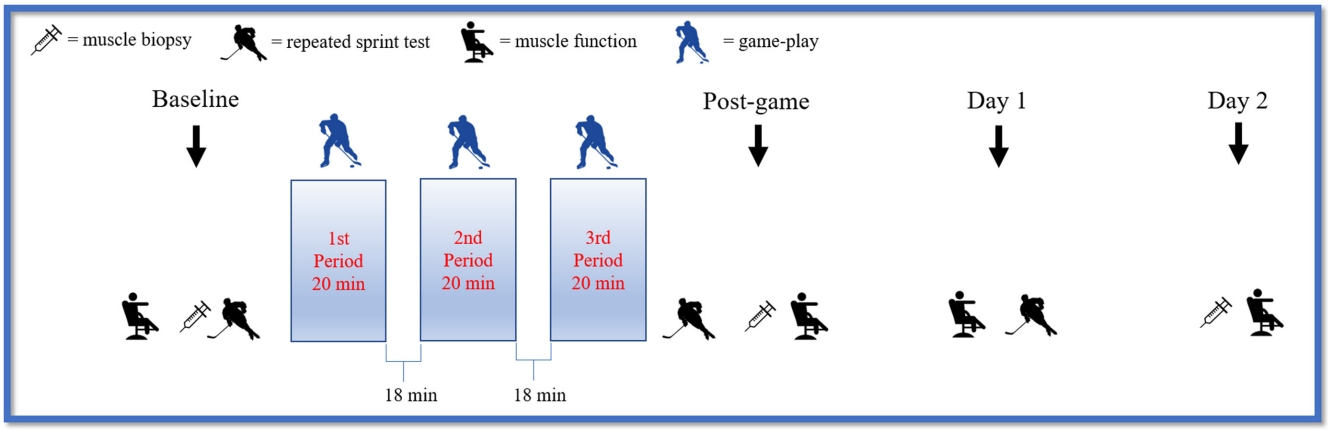

FIGURE 1 Illustration of the study overview. Arrows show time points for muscle sampling, repeated-sprint test and/or maximal voluntary contraction test with low and high frequency stimulation. Baseline, postgame, day 1 and day 2 is prior to, immediately after, 19–23 h and 37–39 h after the game, respectively.

2.4 | Repeated-sprint testing

A repeated-sprint test was performed on-ice at baseline (as the final part of the pregame warmup), postgame (~5–10 min after the game), and on day 1 during recovery (~23 h postgame) (see Figure 1 for study overview). The test consisted of five 33 m (goal line to second blue line) sprints separated by ~25 seconds of recovery (sprints initiated every 30 s) where the players skated slowly back to the starting line, as previously described.[7] The test was performed in full game gear including hockey stick. The sprints were timed using photocell instrumented timing gates (Witty Gate Wireless Training Timer; Microgate). The players initiated the sprints from a static starting position 1 m behind the starting line to avoid premature triggering of the timing gates. Fastest sprint time, mean sprint time and fatigue decrement score (%) calculated as the total sprint performance relative to the ideal sprint performance (fastest sprint time x 5 divided by actual total sprint time x 100) were obtained as results.

2.5 | Muscle function

Muscle function was evaluated with a maximal voluntary isometric knee-extensor contraction test (MVIC) supplemented with low (20 Hz) and high (50 Hz) frequency electrical stimulation tests as previously described.[20] The rationale behind the use of 20 Hz and 50 Hz stimulations was to investigate both low and high frequency fatigue in the knee-extensors as highlighted previously[9] and to include assessments of muscle function without involvement of the central nervous system. The tests were performed prior to the game (~2–4 h pregame), immediately after (~30–160 min postgame), and on day 1 (~19–21 h postgame) and day 2 (~37–39 h postgame) into recovery (see Figure 1). After a 5-min warm-up on a bicycle ergometer (100 W; Monark Ergomedic 874E, Monark), players were seated in a mobile isometric dynamometer (MID-Chair) (Science to Practice, S2P), mounted with model 1-Z6FC3/200 kg torque transducers (HBM GmbH) and fastened by straps around the waist and around the proximal thigh. The chair seat and backrest were individually adjusted to ensure alignment of the rotational axes of the knee joint and torque arm of the dynamometer. Individual adjustments were noted for replication in subsequent tests and the mechanical knee angle set at 70°. Torque signals were continuously recorded at 1000 Hz on an external computer using a build-in analog-to-digital 16-bit (A/D) converter (ARS, Science to Practice, S2P). Prior to placing stimulation electrodes (Valutrode, 5 × 10 cm, Axelgaard), a razor was used to remove hair and prepare the specific stimulation areas on the thigh. To allow for swift measurements without having to change the chair setup between each individual, all players were assessed using their right leg at all time points. The electrodes were placed on the distal and proximal part of the m. quadriceps femoris and placements marked for subsequent trials. Specific warm-up following the 5-min of cycling consisted of a series of voluntary submaximal isometric contractions at increasing knee extensor torque. After the warm-up, the players performed two MVICs separated by 60-s of recovery. Participants were instructed to contract as fast and forcefully as possible for ~3 s accompanied by standardized verbal encouragement. After MVIC testing, muscle surface electrical stimulation of the knee extensors was applied using stimulation frequencies of 20 and 50 Hz provided by an external stimulator box (model DS7 Digitimer Electronic).[20] Following 1–2 stimulations at submaximal electrical current for habituation, two 20 Hz trains (stimulation duration: 600 ms) were delivered with a rest period of 30 s between successive trains. Subsequently, two 50 Hz trains were delivered (600 ms duration) interspaced with

30 s rest. All stimulations were delivered with a current of 125 mA as this current was tolerated by all players and enabled the achievement of >50% MVIC during the 50 Hz stimulations for all players. Players were instructed to relax as much as possible prior to the stimulations to avoid pre-contractions. The recorded data were subsequently analyzed in Matlab (Math Works, MATLAB R2021b). Smoothing of the raw torque signals was performed using a digital fourth-order zero-lag Butterworth low-pass filter, using a cut-off frequency of 15 Hz.[21] Measurements were excluded if there was a countermovement resulting in a negative torque exceeding 1% of peak torque. The trials with the highest peak torque during each of the MVIC, 20 Hz and 50 Hz stimulation conditions, respectively, were included in the statistical analysis.

2.6 | Muscle sampling

Muscle biopsies (70–100 mg w.w.) were obtained from the m. vastus lateralis using the Bergstrøm needle technique with suction.[22] Biopsies were performed pregame (3–4 h pregame), postgame (within 15–60 min) and on day 2 (37–39 h postgame) (see Figure 1 for study overview). Players rested in the supine position on portable beds placed in a room next to the rink. Local anesthesia (Lidocaine 2%) was administered before making a small incision in the skin and fascia. After the muscle biopsy was obtained, the incision point was covered with sterile bandage and sterile gauze. The muscle biopsies on the game-day were obtained from the right leg, while the biopsy on day 2 was taken from the left leg.

2.7 | In-game activity pattern and heart rate recordings

A local positioning system (LPS tracking; KINEXON Precision Technologies, KINEXON ONE, version 1.0) was used to monitor in-game activity patterns. Sixteen antennas were positioned around the rink and each player equipped with a LPS sensor placed at the upper back in a manufacture designed vest. Position data were collected live at 30 Hz and analyzed using the system software. System software and firmware corresponded to the latest releases at the time of the game (November 2021). Speed zones were coded as previously described in ice hockey studies[3,7] and expressed in absolute values, as well as relative to playing time with game interruptions included. We were unable to quantify active playing time due to technical constraints, but recorded total time on ice for each player including stoppages. However, we estimated the players' active playing time by calculating the ratio between the period's length in active playing time (20 min) and actual total period length (including game interruptions), which was ~0.79 in order to introduce these estimates in the discussion. Heart rate was continuously monitored during the game with breaks between periods excluded from the analysis (Team Polar, Polar Electro Oy). HR_{MAX} was defined as the maximal heart rate measured during the Yo-Yo IR1-IH test or during the game. For the three players with no Yo-Yo IR1-IH assessment, the HR_{MAX} obtained during the game was applied as their maximum heart rate since it has previously been shown that players reach ~97% of HR_{MAX} during games, which aligns well with the findings obtained for the remaining players of the present study.

2.8 | Muscle biopsy analysis

Muscle biopsies were immediately frozen in liquid N_2 and stored at −80°C until further analysis. To determine the water content, the muscle samples were weighed before and after freeze-drying. After freeze-drying, the samples were dissected free of visible blood, connective tissue, and fat. Glycogen content was determined spectrophotometrically (Beckmann DU 650) as previously described.[23] In brief, freeze-dried muscle tissue (1–1.5 mg) was boiled in 0.5 mL of HCL (1 mol/L) for 150 min before it was quickly cooled, whirl mixed, and centrifuged at 3500 g for 10 min at 4°C. Forty microliters of boiled muscle sample and 1 mL of reagent solution containing Tris buffer (1 mol/L), distilled water, ATP (100 mmol/L), magnesium chloride (1 mol/L), nicotinamide adenine dinucleotide phosphate (100 mmol/L), and glucose-6-phosphate dehydrogenase were mixed before the process was initiated by adding 10 μL of diluted hexokinase. Absorbance was recorded for 60 min before the glycogen content was calculated. Muscle glycogen was expressed as millimoles per kilogram of dry weight (d.w.).

2.9 | Statistical analysis

Data are presented as group means and 95% confidence interval unless otherwise stated. Normality of studentized residuals and homoscedasticity were assessed with normal probability plots and distribution of residuals. To detect differences in activity pattern and heart rate between periods, and to detect differences in muscle glycogen, muscle function and repeated-sprint ability between the different time points, a linear mixed model was applied with period or time as fixed effect and subject ID as random effect. The linear models were followed by Bonferroni post hoc tests when appropriate. Finally, correlation coefficients were calculated using Pearson Product Moment Correlation

where r values were interpreted as r ≤ 0.1 (trivial), 0.1–0.29 (small) 0.3–0.49 (moderate), 0.5–0.69 (large), 0.7–0.89 (very large), and ≥0.9 (nearly perfect).[24] Significance was set at $p \leq 0.05$ (two-tailed testing). All statistical analysis were performed in Stata/IC16 (StataCorp, College Station) while graphs were created using GraphPad Prism 9 (GraphPad Software).

3 | RESULTS

3.1 | Muscle function

Peak torque during 20 Hz electrically-induced contractions was reduced by 29% [37;21] postgame ($p < 0.001$) and remained 13% [1;21] lower ($p = 0.010$) on day 1 until returning to pregame values on day 2 ($p = 0.533$) (Figure 2A). Evoked 50 Hz peak torque measured was 20% [11;29] reduced ($p < 0.001$) after the game and 9% [1;18] lower on day 1 ($p = 0.040$). No difference was observed between 50 Hz knee extensor torque on day 2 compared to baseline ($p = 1.000$; Figure 2B). Knee extensor peak torque (MVIC) decreased by 11% [3;18] immediately after the game ($p = 0.001$), but returned to baseline on day 1 ($p = 0.644$) and day 2 ($p = 1.000$) (Figure 2C). The 20/50 Hz ratio was reduced by 11% [4;18] postgame ($p < 0.001$), while there was no difference on day 1 and day 2 when compared to baseline ($p = 0.575$ and 0.714, respectively) (Figure 2D). No positional differences were apparent in the reduction in muscle function postgame nor in the recovery period ($p = 0.203$–0.801).

3.2 | Repeated-sprint test

After the game, mean sprint time was 1% [1;2]) slower compared to baseline ($p = 0.041$), while there was no difference between mean sprint times at baseline compared to day 1 ($p = 1.000$) (Figure 3). When evaluating the differences for each sprint, this decline was significant only for sprints 2 and 3 in the repeated sprint test ($p = 0.004$ and $p = 0.042$, respectively), though the fastest single sprint time observed (irrespective of sprint number) did differ significantly ($p = 0.033$) with a 1% slower time postgame. The fatigue index calculated as the actual total sprint performance relative to the ideal sprint performance (fastest sprint x 5) was not different postgame compared to baseline ($p = 0.983$) (~97% of ideal

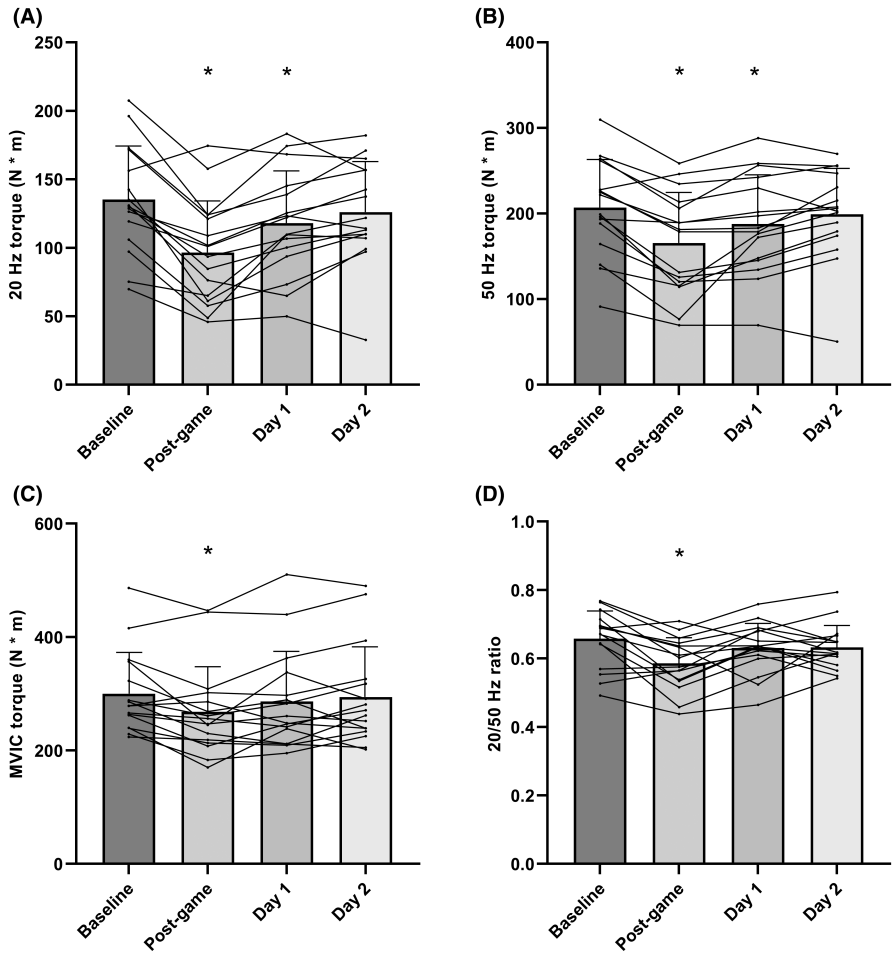

FIGURE 2 Assessment of muscle function. Peak torque during (A) 20 Hz, (B) 50 Hz, (C) MVIC, and (D) the 20/50 Hz ratio prior to the game (Baseline), immediately after (postgame), 19–21 h after (day 1), and 37–39 h after (day 2) the game ($n = 16$). Values are presented as means and individual values. * denotes significant difference from baseline ($p < 0.05$).

sprint performance at both time points). The decline in repeated sprint ability was not different between positions ($p = 0.171$).

3.3 | Muscle glycogen concentration

Muscle glycogen concentrations were 31% [19;43] reduced acutely after game-play ($p < 0.001$) reaching an average concentration of 344 [273;414] mmol·kg^{-1} d.w (Figure 4A). However, for players covering the longest total distance (~4790 m, $n = 8$ i.e., the top-fifty percentile) the magnitude of glycogen depletion postgame was much more pronounced (lowered by 48% [40;57], $p < 0.001$; Figure 4B). Across all players, muscle glycogen stores were fully restored on day 2 ($p = 1.000$). Analyzing players by position revealed no differences between forwards and defensemen at baseline ($p = 0.225$), but a lower postgame glycogen level (228 [164;292] mmol·kg^{-1} d.w vs. 365 [274;456] mmol·kg^{-1} d.w, $p = 0.024$) in defensemen mediated by a trend towards a larger relative decline (50% [35;65] vs. 26% [4.5;47], $p = 0.072$).

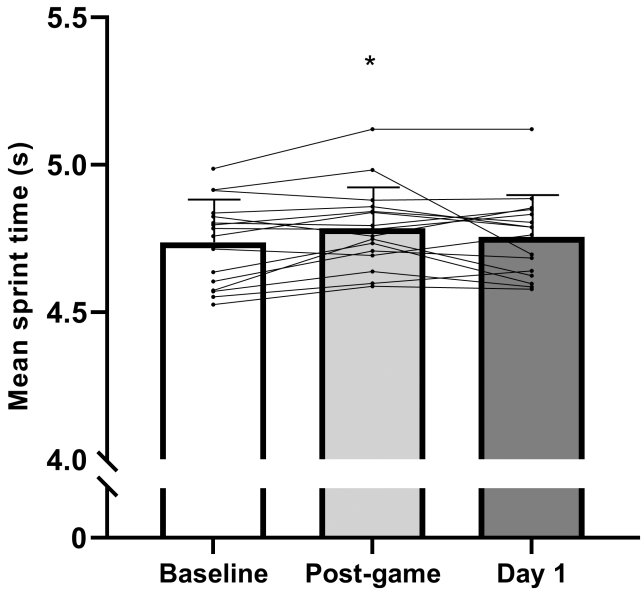

FIGURE 3 Mean sprint times (s) for all 5 sprints in the repeated-sprint test (5 × 33 m with 25 s recovery) before (Baseline), immediately after (postgame) and 23 h (day 1) after the game ($n = 16$). Values are presented as means and individual values. * denotes significant difference from baseline ($p < 0.05$).

3.3.1 | Game activity

Total average on-ice time (game interruptions included) during the game was 21.5 [18.7;24.4] min, with 7.1, 6.9 and 7.1 min in period 1, 2 and 3, respectively. Players skated 4421 [4059;4785] m during the game of which 20, 13 and 18% was covered by very slow speed skating (1.0–10.9 km/h), slow speed skating (11.0–13.9 km/h) and moderate speed skating (14.0–16.9 km/h), respectively. In addition, 27, 14, and 9% was covered by fast speed skating (17.0–20.9 km/h), very fast speed skating (21.0–23.9 km/h), and sprint skating (>24.0 km/h), respectively. There were no differences ($p = 0.948$) in the total distance covered during the first, second and third periods (1466 [1365;1566], 1479 [1318;1639], and 1467 [1321;1614] m, respectively). A longer distance per min was covered in the second period (219 [204;234] m/min), compared to the first (207 [196;218] m/min, $p < 0.001$) and third periods (210 [196;223] m/min, $p = 0.003$). The number of accelerations per minute were reduced in the third period (1.5 [1.1;1.8]) compared to the first period (1.8 [1.5;2.0]) ($p = 0.020$), while there was no difference in the number of decelerations between periods ($p > 0.292$). A trend towards a lower number of changes in pace per minute was observed in the third period (2.7 [2.2;3.3]) compared to the first period (3.4 [3.0;3.9]) ($p = 0.051$). In addition, the number of turns per minute was lower in the second (2.0 [1.6;2.5]) and third (1.8 [1.3;2.2]) period compared to the first period (2.8 [2.4;3.3]) ($p < 0.001$). In addition, the number of wide turns per minute were lower in the second

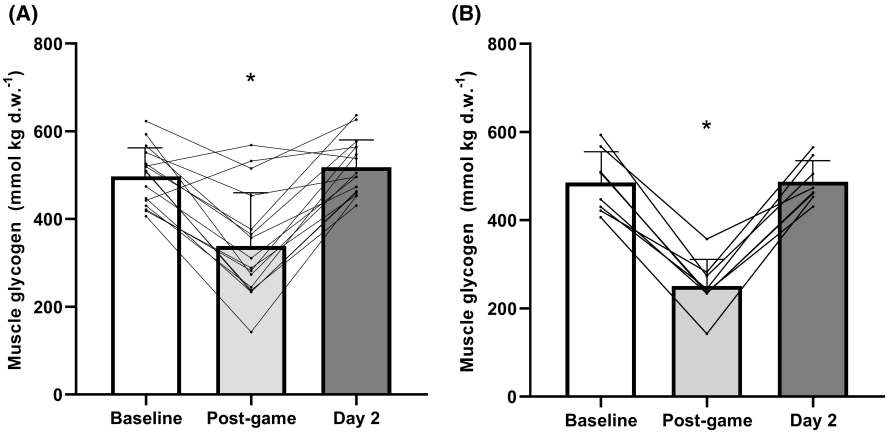

FIGURE 4 Muscle glycogen concentration for (A) all players and (B) for the half of the players covering the longest total distance, before (Baseline), immediately after (postgame), and 37–39 h after (day 2) an ice hockey game. Values are presented as means and individual values. * denotes significant difference from baseline ($p < 0.05$).

(1.8 [1.4;2.2]) and third (1.6 [1.1;2.0]) periods compared to the first period (2.4 [2.1;2.9]) ($p < 0.001$).

Finally, positional differences were captured with defensemen accumulating more time on ice than forwards (26.1 [22.2;30.1] min vs. 18.6 [16;21] min, $p = 0.001$) and more total distance (4881 [4332;5431] m vs. 4134 [3706;4561] m, $p = 0.021$) as a result of more very slow speed skating (1217 [814;1620] m vs. 692 [617;768] m, $p = 0.001$), slow speed skating (809 [561;1056] m vs. 471 [409;533] m, $p = 0.001$) and moderate speed skating (1001 [833;1169] m vs. 656 [571;740] m, $p < 0.001$) but on the contrary less very fast speed skating (416 [213;620] m vs. 693 [630;757] m, $p = 0.002$) and sprint skating (237 [−16;491] m vs. 451 [338;563] m, $p = 0.04$). Moreover, the total number of accelerations tended to be higher for defensemen (41 [33;48] vs. 31 [23;38], $p = 0.054$), whereas the number of decelerations was significantly higher (60 [50;71] vs. 47 [41;54], $p = 0.017$). When game activities were expressed relative to time on ice (m/min or counts/min) forwards accumulated 18% more total distance ($p = 0.005$), ~20% less very slow- and slow speed skating ($p = 0.02$–0.054) but 35% more fast speed skating ($p = 0.011$), 126% more very fast speed skating ($p < 0.001$) and 145% more sprint skating ($p = 0.007$) per min compared to defensemen. No differences were captured in the number of accelerations or decelerations per min ($p = 0.46$–0.77).

3.4 | Heart rate profile

The players exhibited a mean heart rate of 77.2% [75.2;79.3] and a peak heart rate of 99.6% [99.1;100.0] HR_{MAX} throughout the game. Mean heart rate was higher in the second and third periods (78.0% [76.0;79.9] and 78.1% [75.8;80.5] HR_{MAX}, respectively) compared to the first period (75.6% [73.1;78.0] HR_{MAX}), $p = 0.013$ and 0.005, respectively. No positional differences in mean or maximum heart rate were captured ($p = 0.177$–0.766).

3.5 | Correlation analyses

A strong correlation was observed between total covered distance and absolute reduction in muscle glycogen concentration during the game ($r = 0.79$, $p = 0.001$) (Figure 5A). In accordance with this, on-ice time and absolute reductions in muscle glycogen concentration were strongly correlated ($r = 0.68$, $p = 0.011$) (Figure 5B). Likewise, strong correlations were observed between total distance covered and relative reductions in knee-extensor peak torque during both 20 Hz and 50 Hz stimulations ($r = 0.59$, $p = 0.032$) and ($r = 0.63$, $p = 0.021$) (Figure 6A,B), respectively. In contrast, relative reductions in voluntary peak torque (MVIC) were unrelated to total distance covered (r = 0.47, $p = 0.108$). Finally, time on ice tended to correlate with relative reductions in peak torque during both 20 Hz ($r = 0.55$, $p = 0.050$) and 50 Hz stimulations ($r = 0.52$, $p = 0.069$) (Figure 6C,D), but not relative reductions in peak torque during MVIC ($r = 0.34$, $p = 0.254$). Neither time on ice nor total distance covered were found to correlate with relative reductions in MVIC peak torque, or 50 Hz and 20 Hz evoked torque ~24 h postgame. Absolute postgame glycogen levels did not correlate with relative reductions in peak torque during 20 Hz, 50 Hz and MVIC, nor with increases in the mean sprint time during the repeated-sprint test after the game.

4 | DISCUSSION

The present study is the first to examine the time course of changes in muscle glycogen, repeated-sprint ability and muscle function after and in the recovery period

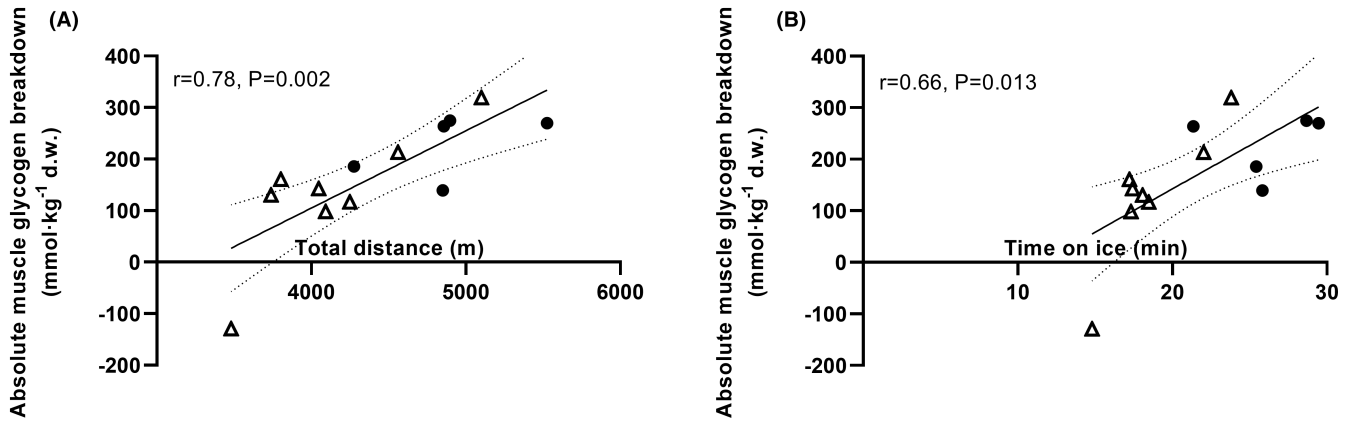

FIGURE 5 Game-induced decrease in muscle glycogen against total distance covered (A) and time on ice (including stoppages) (B) ($n = 13$) for forwards (triangles) and defensemen (circles).

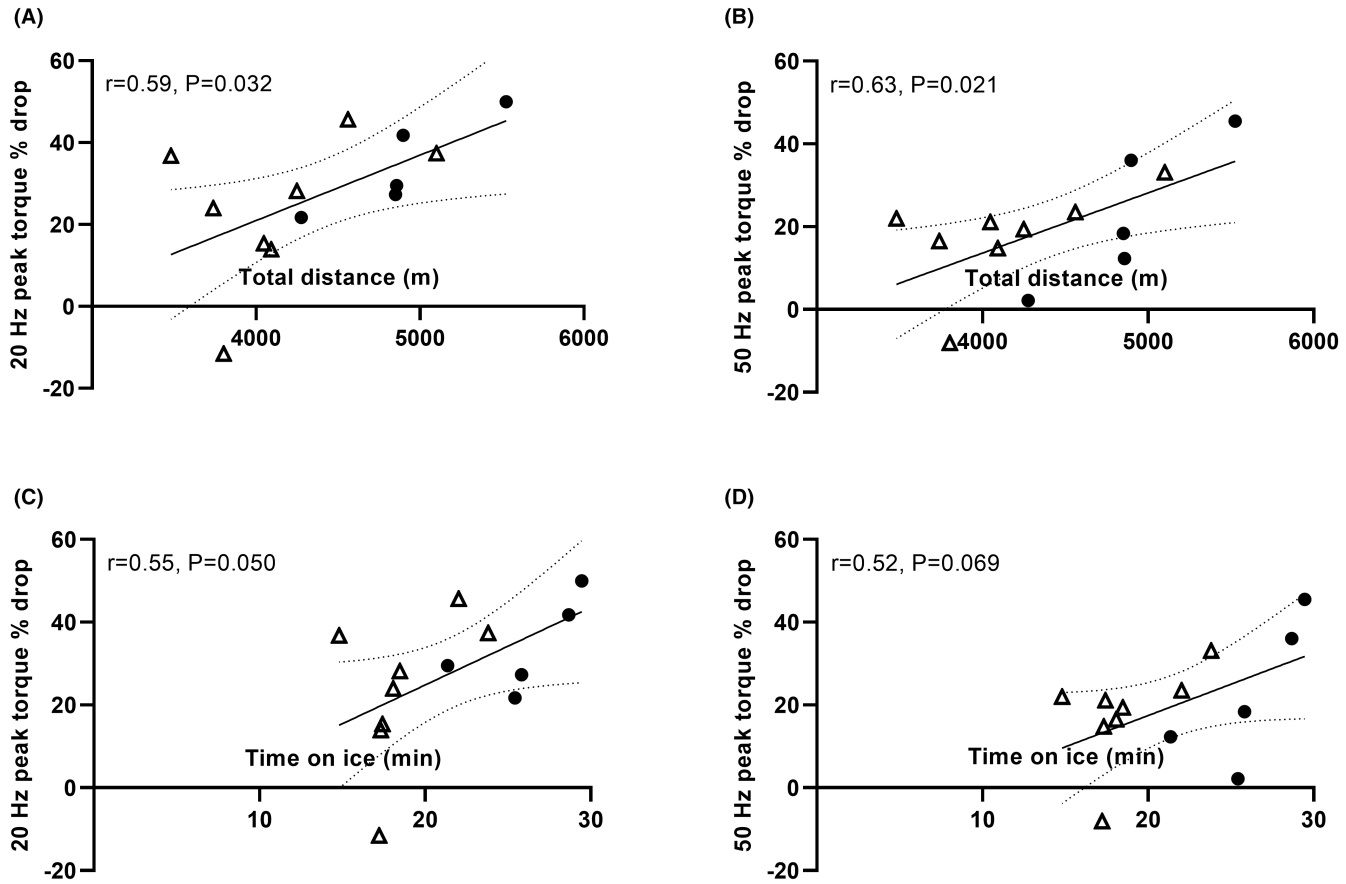

FIGURE 6 Game-induced decrease in peak torque in % during 20 Hz stimulations (A) and (C), as well as peak torque during 50 Hz stimulations (B) and (D) against total distance covered and time on ice (including stoppages) ($n = 13$) for forwards (triangles) and defensemen (circles).

following an ice hockey game in youth elite male players. The major findings were that (1) despite substantial reductions in muscle glycogen concentrations, dependent on individual playing time and distance covered, restorations to pregame levels were obtained rapidly within ~38 h postgame, whereas (2) maximum and repeated-sprint ability was only marginally impaired postgame and fully restored within ~23 h. Finally, (3) muscle function was substantially impaired postgame, including exacerbated low-frequency force depression, and only partially recovered on the subsequent day where the degree of low- and high-frequency force depression was similar.

The total distance covered during the game and distance covered at high skating speeds are in line with previous time-motion studies in competitive ice hockey[3,4] and with a more pronounced high-intensity activity pattern in forwards compared to defensemen confirming previous results.[3,5,25] In addition, the average heart rate during the game was ~77% of HR_{MAX} (between-shifts included) with peak values approaching maximal levels, as previously reported.[7] In contrast, the 31% reduction in muscle glycogen postgame is less than previously reported,[1,7,17] however, the starting level was higher in the present study, meaning that in absolute levels, the glycogen degradation was comparable with previous observations. Consequently, the present study seems to reflect a competitive game setting. Importantly, the load imposed on each player is highly dependent on specific on-ice playing time and the present study therefore reflects the challenge when averaging an on-ice time of 22 min (range: 15–29 min) whereas in elite ice hockey, some players may accumulate substantially more (or less) playing time than this. Accordingly, the muscle glycogen degradation was largely correlated with time on ice ($r = 0.68$) and very largely correlated with total distance covered ($r = 0.79$) and with position-specific on-ice times leading to lower postgame levels in defensemen. Notably, the time on ice in the present study was recorded including game interruptions meaning that the active ice time was of shorter duration (estimated average: 17 min; range: 12–23 min).

In the present study, substantial reductions were demonstrated in volitional and electrically induced muscle torque postgame as MVIC, 50 Hz and 20 Hz evoked torques were reduced by 11, 21 and 29%, respectively. Importantly, these measurements were obtained at least 30 min postexercise and therefore underestimates the immediate postgame

reductions due to transitory perturbations of the metabolic and ionic intracellular environment.[26] Moreover, the reductions in muscle function assessed with electrical stimulation were moderately-to-largely dependent on total distance covered ($r = 0.059–0.63$) but with no significant positional differences present. In addition, all these measures were independent of the absolute postexercise muscle glycogen concentration, which aligns with previous studies demonstrating that single maximal voluntary or electrically-induced contractions are unrelated to low muscle glycogen levels.[20,27] The reduction in MVIC torque was comparable to that observed after a soccer game assessed with similar delays,[12,28] despite the playing time in ice hockey being 4–5 times shorter and with less than half the distance covered. In addition, Thorlund et al.[29] reported a comparable 11% acute decrease in knee-extensor MVIC after a team handball game in male elite players. Thus, the degree of acute postgame muscle fatigue seems comparable to that reported in other team sports irrespective of the differences in game durations.

In addition, the ability to perform high-intense dynamic actions appeared to be impaired towards the end of the game as reflected by a lower number of accelerations per minute in the third period compared to the first period and reductions in the number of turns and changes in pace, complementing previous ice hockey studies, and suggestive of accumulative fatigue development.[3,4,7] In partial agreement, repeated-sprint ability was impaired postgame, however, only marginally (~1%). This represents a less pronounced reduction than previously reported following acute ice hockey game-play[7] and following other intermittent team sports such as soccer[30,31] suggesting that the ability to perform repeated-sprint activities is relatively well preserved. These small declines in repeated-sprint ability were unrelated to postgame muscle glycogen levels and playing position. This was despite degradations of muscle glycogen for the most severely used players to values of around ~250 $mmol \cdot kg^{-1}$ d.w. comparable to that observed after soccer games.[13,30,31] This level is identical with a proposed critical glycogen threshold level where muscle excitation-contraction coupling has been suggested to become compromised, leading to impaired exercise performance.[20,32,33] Although muscle glycogen was not fully depleted at a global level in the present study, Vigh-Larsen et al.[7] previously showed that glycogen in several individual fibers was substantially depleted after a simulated ice hockey game with a similar degree of glycogen degradation at the whole-muscle level as in the present study. Moreover, glycogen levels below 250 $mmol/kg^{-1}$ d.w. were associated with impaired repeated-sprint performance measured in a laboratory setting concomitant with single-fiber depletion and depletion of glycogen in certain microenvironments in the muscle cell.[20,34,35] However, in the present study the lack of correlation between postexercise glycogen levels and repeated-sprint ability (and negligible decline herein) may be related to the fact that glycogen was not depleted substantially below this proposed critical glycogen level with the playing times employed in the present study.

Moreover, the repeated-sprint ability test in the present study was performed after a 5–10 min rest period following the game and included brief sprints (~4–5 s duration) interspersed by ~25–26 s of recovery. These rest periods are longer than what players commonly endure during repeated-sprint scenarios in the game and provoked only a small reduction in performance from the first to the last sprint (~3%) at each time point. In addition, no intense braking or change of direction actions were included in the test, which could be speculated to be more prone to alterations in muscle function than concentric-based sprint skating strides. Thus, it is likely that we may underestimate the degree of acute fatigue occurring during ice hockey-specific actions in the most demanding passages of play. Finally, we did not control for potential pacing during the tests, although we did observe a linear reduction in sprint ability from the first to the last sprint suggestive of true maximal efforts.

Immediately postgame we observed exacerbated low frequency force depression (reduced 20/50 Hz ratio), which is defined as a relatively larger reduction in evoked muscle torque production during low compared to high-frequency stimulations.[10] Low frequency force depression has been linked with impaired Ca^{2+} handling,[10,20] entailing either impaired Ca^{2+}-release from the sarcoplasmic reticulum and/or a reduction in myofibrillar Ca^{2+}-sensitivity, which is specifically present during low-frequency stimulations due to the sigmoidal nature of the force-Ca^{2+} relationship.[10] Thus, even if the ability to maximally engage the muscles when needed during the repeated-sprint test was only marginally impaired, exacerbated low-frequency force depression could increase the sensation of fatigue and demand increased firing frequencies from the central nervous system to sustain the in-game activity pattern. Although speculative, this could potentially lead to alterations in the pacing strategies that have previously been reported in ice hockey in relation to playing time.[36]

During the recovery period, repeated-sprint ability and maximal voluntary knee-extensor torque were rapidly restored within 1 day. In contrast, low and high-frequency electrically-induced torque production remained impaired (~10%) at day 1 before reaching full recovery on day 2. Thus, contrasting the initial hypothesis, the depression of low-frequency torque did not persist for longer after the game compared to that of high-frequency stimulations. Nevertheless, although no impairments were observed in repeated-sprint ability at this time point, the persistent

reductions in electrically-induced muscle function indicates that the players may not be completely recovered and possibly prone to accelerated impairments in muscle function when games are separated by less than 24 h of recovery. Nonetheless, this recovery pattern of muscle function is markedly faster than previously reported following soccer games where reductions persisting for up to 72 h have been reported.[12,13]

Similarly, the muscle glycogen levels had returned to pregame values already ~38 h postgame. This was evident in all players, despite large reductions in muscle glycogen in individual players after the game. Thus, not only the recovery of muscle function but also the glycogen resynthesis rate in ice hockey is apparently fast compared to that observed in soccer where similar reductions in muscle glycogen levels have been shown to persist for more than 48 h postgame.[12,13] This delayed recovery has been attributed to the degree of muscle damage induced by soccer matchplay, impairing the muscle glucose uptake, resulting in a prolonged restoration of the muscle glycogen content.[16,37] Thus, one explanation for the faster glycogen resynthesis rate and recovery of muscle function in ice hockey could be that differences in the movement pattern (skating compared to running) or that the lower total work performed induces a lower degree of muscle damage postgame, at least in the quadriceps muscle assessed. In support of this, plasma creatine kinase levels have been shown to be 1-2-fold lower 24 h after an ice hockey game compared to a soccer game.[3] Thus, as a unique feature of ice hockey, the high-intensity activity pattern results in a markedly higher muscle glycogen breakdown per unit of work performed due to the inefficient ATP production through glycolysis at high exercise intensities (~10 times less ATP per glucose molecule) resulting in large glycogen reductions relative to the actual total work performed.[38] However, since no muscle biopsies were obtained on the first day after the game it remains uncertain whether muscle glycogen stores are adequately refilled if games are interspaced by very short recovery time (<24 h), which could potentially result in subsequent premature glycogen depletion.

4.1 | Limitations

Due to the time needed to test each player, there was a delay (~30–160 min) in the testing of voluntary MVIC and evoked 20 Hz and 50 Hz muscle torque production postgame, which means that the results do not capture the acute transitory decrements immediately postgame, but rather reflect relatively stable decrements with a prolonged recovery time (hours/days). Despite this, we investigated if there were any associations between the test order between players and the degree of impairment in muscle function. No correlations were found for peak torque during MVIC, 20 Hz and 50 Hz contractions, nor in the 20/50 Hz ratio, suggesting that the effect of player testing order was negligible. Furthermore, there may have been a small resynthesis of muscle glycogen during the time delay before sampling the muscle biopsies. However, this resynthesis should predominantly take place within the first ~20 min postexercise due to the transiently increased availability of muscle lactate for gluconeogenesis and glycogen synthesis postexercise and thus should not differ significantly between the first and last participants sampled.[39,40] Accordingly, since the increase in muscle lactate at the end of an ice hockey game is not very large (~20 mmol·kg^{-1} d.w.) the potential for glycogen resynthesis postexercise is limited. In addition, the players in the present study were juniors (~19 years of age) with a different body composition and training status as compared to that of older professional players and therefore a potentially different degree of fatigue development and subsequent time needed to recover from a game.[41,42] Finally, the players were not allowed to ingest carbohydrates during the game in contrast to during competitive games where some players may ingest carbohydrate, which could potentially alter the muscle glycogen depletion pattern and/or performance, although no major effects on glycogen metabolism has been reported previously.[43]

5 | CONCLUSION

In conclusion, the recovery of muscle glycogen stores, repeated-sprint ability and muscle function following an ice hockey game is rapid, taking place within 1–2 days postexercise despite substantial reductions immediately postgame. However, since electrically stimulated muscle function is still partially depressed the day after a game this could impose a problem for players exposed to congested game schedules with <24 h of recovery. In addition, the degradation of muscle glycogen content and muscle function are closely related to individual time on ice and game activities highlighting the heterogeneity between players in relation to the actual game exposure.

5.1 | Perspectives

Our findings demonstrate that the recovery of performance and muscle glycogen stores is relatively fast after an ice hockey game and can be achieved within 1–2 days postgame. However, since the in-season ice hockey game schedule in an elite setting entails multiple congested fixtures it could pose a problem for performance and injury risk mitigation that muscle function is only partially

restored the day after a game. Moreover, our findings demonstrate the heterogeneity in playing exposure in ice hockey and the association to fatigue development (reductions in muscle function) and the degree of glycogen breakdown, which may be useful for coaches. As such, it is possible on the basis of the present data to estimate the degree of fatigue and glycogen use following a certain playing exposure/activity pattern. Moreover, the maximal individual active on-ice time in the present study was ~24 min but can be as high as >30 min in the most challenging scenarios for key players, which may pose a different exacerbated fatigue and recovery scenario. Therefore, coaches should be careful when rationing playing time between players at the crucial stages of the season to ensure sustained performance and player health.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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