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Rehabilitation and nutrition protocols for optimising return to play from traditional ACL reconstruction in elite rugby union players: A case study

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ABSTRACT

Current nutrition and exercise focus during rehabilitation periods has been on reducing muscle atrophy associated with immobilisation. This case report outlines a best practice anterior cruciate ligament (ACL) rehabilitation programme undertaken by two professional rugby athletes, with the addition of an evidence-based supplementation (gelatine and vitamin C) and exercise protocol focused on collagenous tissue. Both players ruptured their left ACL and were repaired with a traditional hamstring graft. Players undertook a structured rehabilitation programme for 34 weeks before being clinically assessed ready to play. Players saw minimal changes in body composition in the early rehabilitation period (P1 – 0.8 kg; P2 – 0.4 kg). Leg lean mass reduced in both legs of Player 1 (Injured – 0.8 kg, Non-injured – 0.6 kg) at 17 weeks, with Player 2 only experiencing a loss of 0.3 kg of lean tissue in the injured leg. Both players returned to baseline body compositions after 24 weeks. Leg strength returned to a maximum at 24 and 15 weeks, respectively, with knee function returning to baseline by 30 weeks. This case report provides evidence that nutrition and rehabilitation programmes targeted at minimising the effects of disuse in both muscle and connective tissue may assist return to play after ACL injury.

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KEYWORDS
ACL; collagen; rehabilitation; nutrition

Introduction

Rugby union is a high impact sport with frequent severe injuries resulting in long periods (>10 weeks) of recovery and rehabilitation away from the game (Williams, Trewartha, Kemp, & Stokes, 2013). Typically, rehabilitation follows a pattern of acute injury with surgical repair, short to moderate periods of immobilisation, longer retraining programmes before returning to competitive sport. Immobilisation is associated with muscle atrophy and has been shown to prolong return to play (Wall, Morton, & van Loon, 2015).

During periods of immobilisation best practice interventions focus on minimising muscle atrophy through the maintenance of mechanical stimulus via neuromuscular electrical stimulation (NMES) (Dirks et al., 2014; Gibson, Smith, & Rennie, 1988) or low load strength training (Burd et al., 2010). To support and amplify the mechanical stimulus additional nutrition interventions have been suggested. These include feeding of high leucine (>3 g) protein sources (Wall et al., 2015), omega-3 fish oils (Smith et al., 2011), and creatine (Johnston, Burke, MacNeil, & Candow, 2009). Recently, Milsom and colleagues outlined the combination of these interventions in a well-planned nutrition and exercise rehabilitation programme in a professional soccer player returning from Anterior Cruciate Ligament (ACL) injury (Milsom, Barreira, Burgess, Iqbal, & Morton, 2014). Atrophy was observed within the initial eight-week immobilisation phase (5.8 kg whole body lean mass, 1.35 kg immobilised leg lean mass). This while undertaking a low carbohydrate-high protein diet, supplemented with Vitamin C, HMB, creatine and fish oil in combination with twice daily physiotherapy and NMES exercise.

A subsequent 14-week rehabilitation phase of region-specific resistance training and moderate carbohydrate-high protein diet led to increases in lean mass above baseline by 2.5 kg, with a subsequent reduction in fat mass of 0.6 kg. Immobilised leg lean mass increased by 1.2 kg between 12 and 24 weeks. This provides evidence that protocols to minimise atrophy can enhance return to train in elite athletes after injury.

To date, little attention has been given to supporting the repair and regeneration of damaged connective tissue. Immobilisation for even short periods (10 days) has been shown to reduce both myofibrillar and collagen synthesis by up to 50% (de Boer et al., 2007). In this work, de Boer et al. (2007) demonstrated myofibrillar protein synthesis reductions stabilised after 10 days of immobilisation; however, declines in collagen synthesis continued to be seen at 21 days. Although declines in collagen synthesis did not result in decreased cross-sectional area of the patellar tendon, reductions in tendon stiffness were observed (de Boer et al., 2007). Therefore, decreased collagen turnover is potentially associated with impaired muscle-tendon function. In support of this hypothesis, reductions in muscle volume (~350 g or 3.1%) associated with disuse, accounts for only a small percentage of the reduction in muscle function (~25%) (Wall et al., 2013).

The collagen-rich extracellular matrix (ECM) of muscle, tendon and ligaments plays an essential role in the function of these tissues. A major stimulus for ECM adaptation is strain,

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with fibroblasts of the ECM responding accordingly (Humphrey, Dufresne, & Schwartz, 2014). One adaptive response to mechanical loading is the synthesis of new collagen. Short bouts of mechanical loading appear sufficient to optimise collagen production within in vitro ligament models (Paxton, Hagerty, Andrick, & Baar, 2012). Additionally, the availability of key nutrients (proline and ascorbic acid) in combination with this strain stimulus have demonstrated further superior responses to typical or sub-optimal availability (Hagerty et al., 2012). These works together suggest that optimising the availability of key nutrients, at times when collagen synthesis is upregulated via mechanical stimulation may support connective tissue repair and regeneration after significant injury. Research to date has been equivocal at demonstrating the benefit of nutrition support for collagen synthesis, with both positive (Babraj et al., 2005; Farup et al., 2014; Miller et al., 2005) or no effect (Dideriksen et al., 2011; Moore, Phillips, Babraj, Smith, & Rennie, 2005). Additionally, the transient nature of blood flow to tendon and ligaments associated with exercise may influence the availability of key amino acids following protein consumption.

We have hypothesised that feeding a food source high in glycine, proline and hydroxyproline peptides (e.g., gelatine) with ascorbic acid prior to repeat short, low-intensity exercise bouts, separated by 6 h of rest, could potentially improve the repair of the ACL donor site and ligamentisation of the ACL graft. Recently, we have demonstrated that feeding gelatine and vitamin C prior to skipping, separated by 6 h of rest, could double markers of collagen synthesis in vivo (Shaw, Lee-Barthel, Ross, Wang, & Baar, 2017).

This paper presents a case report of two professional rugby players where gelatine/vitamin C and intermittent loading were added to traditional best practice protocols for rehabilitation following ACL rupture and repair. Of note, this case report was started prior to recent data regarding the dose-response of gelatine being published (Shaw et al., 2017). Therefore, the amount of gelatine is lower than what the current evidence would suggest may be optimal.

Methodology

Player injury and surgical repair

Both players were international representative, male, professional rugby union players (Table 1). Both had represented their country for extended periods and had spent over 10 years in structured training programmes. Both athletes ruptured their left ACL during a rugby game and were surgically repaired using an 8.5 mm and 9 mm right hamstring graft, respectively. Players were in full competition training at the time of injury (1–2 gym sessions, 2–3 rugby sessions, 1 position-specific session per week). Players were weight stable and body composition had been maintained for >3 months. Player 1 had ruptured the same left ACL during a game, 12 months prior. The ACL was repaired with a hamstring graft and had been rehabilitated and clinically assessed as ready to play. Players provided their written consent to share their unidentified data in a case study format.

Interventions

Training interventions (*Tables 2 and 3*)

Players undertook a structured rehabilitation programme overseen by a rehabilitation specialist, physiotherapist, and sport medicine doctor. In short, the rehabilitation programme started immediately post-surgery with range of motion (ROM) exercises. Within 24 h of surgery, ROM exercises were combined with NMES protocols (Compex SP 6.0, Compex Sport, France). These exercise sessions were repeated 2–3 times per day with at least 6 h of rest between sessions. This programme was designed to replicate the optimal growth stimulus for both muscle and connective tissue. Post-rehabilitation, players were progressed through a structured hypertrophy focused retraining programme as outlined in Tables 2 and 3. Key ROM and functional milestones were achieved before progressing through each stage of the programmw (Figure 2). Due to Player 1's previous injury history, rehabilitation for the first 16 weeks was undertaken, under supervision, outside of the clubs training environment, to enable maintenance of mental health over the return to play period. Additionally, through consultation with performance support staff and the consulting orthopaedic surgeon, Player 1 was advised to reduce lean mass in the upper body over the rehabilitation period. Therefore, upper body hypertrophy or strength training was a minimal focus throughout their rehabilitation.

Nutrition interventions (*Table 4*)

Players were instructed to follow best practice nutrition guidelines for injury rehabilitation as outlined by Tipton (2015) and Wall et al. (2015) with the addition of nutrients proposed to support sinew repair. Briefly, players were instructed to consume six servings of high leucine protein (>0.3 g protein per kg BM and >3 g leucine) per day separated by 2–3 h with the daily addition of creatine monohydrate (loading: 20 g x 5 days immediately post-surgery, then 3 g/day) and a high dose omega-3 fish oil (EPA 2000 mg/day) throughout the rehabilitation programme. To aid in the repair of grafted hamstring tendon and reduce atrophy in connective tissue, players were instructed to consume 10 g of gelatine with 250 mg vitamin C, 30 min prior to all exercise sessions throughout the first 24 weeks of the rehabilitation period, including ROM exercises early in the rehabilitation period.

Table 1. Player information.

Player	Age (y)	Wt (kg)	Ht (cm)	∑7 Skinfold (mm)	DXA lean mass (kg)	DXA fat mass (kg)	Injury specifics
1	26	103	184	47	92	11	Left knee ACL rupture occurred while changing direction 61 minutes into a match
2	31	113	186	75	86	22	Left knee ACL rupture with a grade 3 medial ligament tear occurred tackling an opposition player 20 minutes into a match

Table 2. Strength and conditioning ACL rehabilitation plan overview.

	Rehabilitation phase			Retraining phase				Return to play phase	
	Weeks 1–4	Weeks 4–10	Weeks 10–12	Weeks 13–16	Weeks 16–18	Weeks 18–20	Weeks 20–23	Weeks 24–32	Week 33+
STAGE/AIMS	Immobilisation, in brace and on crutches ROM 2–3 x Daily	Increase ROM + commence weight bearing + strength training. Partial weight bearing week 5, full weight bearing week 6, out of brace week 7	Progress strength + commence running re-education	Progress to water based plyometrics and compliant surface run drills/ plyometrics	Progress running on compliant surfaces + gym based mechanical power	Straight line overground running + compliant surface multidirectional running and elastic and reactive power	Commence overground multidirectional running and contact progressions	Reintroduce Rugby training	Return to play
LOWER BODY STRENGTH & POWER									
Frequency of training	2–3 x Daily			3–4/week				2–3/week	
Main lifts	Out of brace for ROM exercises and NMES	Commence closed kinetic chain (CKC) DBL body weight in brace progressing to loaded SL				CKC DBL or SL loaded exercises + mechanical power (i.e., olymplic lifting variations)			
Set range			2–5			3–5		2–5	
Rep range			6–12			2–6		2–6	
Focus of training of accessory exercises/ muscles		Non-rotational trunk integrity 2–3/ week, electromuscular stimulation daily, ROM 4/week	Rotational trunk integrity 2–3/week, electromuscular stimulation daily, non-impact foot, ankle and lower leg intrinsic muscle development		Rotational Trunk integrity 2–3/week, foot, ankle and lower leg intrinsic muscle development (including low level impact skipping and hopping)		Commence and progress overground straight line and multidirectional plyometrics		
RUNNING RE-EDUCATION									
Frequency of training			2–3/week	2–4/week	2–3/week	2–4/week		3–5/week	4–7/week
Session(s) focus			Straight line, low impact, basic running drills (i.e., A-drill variations)	Pool based straight line and multidirectional plyometrics + sand or sprung gymnastics floor basic running drills	Sand or gymnastics floor multidirectional running drills	Overground running drills and straight line running + compliant surface for multidirectional running drills	Overground progressing from controlled to uncontrolled change of direction	Introduce to non-contact rugby for several weeks and continue with contact progressions, including low tackles (i.e., blows to the knee)	
Surface			Water depth = 1.2 m	Water depth = 1.2 m, gymnastics sprung floor, sand	Gymnastics sprung floor, sand, non-compliant surface for skipping/foot & ankle integrity	Gymnastics sprung floor, sand, non-compliant surface for run drills & leg/ ankle/foot/integrity		Overground	
FITNESS CONDITIONING									
Modality		Upper body cond. (e.g., swimming with pool buoy, grinder)	Pool (e.g., deep water running, flipper kicking, swimming), bike, rower, stair + uphill walking, paddle boarding		Sand conditioning (e.g., wrestling drills), overground skipping		Run conditioning + non-impact and low impact modalities		
Frequency			3–5/week		4–6/week	3–5/week		4–7/week	
Duration			20–60 min		30–60 min			40–80 min	60–90 min

Table 3. Functional milestones throughout the rehabilitation period.

	Weeks 1–4	Weeks 5–10	Weeks 11–12	Weeks 13–16	Weeks 17–18	Weeks 19–20	Weeks 21–23	Weeks 24–32	Week 33+
Physical									
Player 1						Full ROM W 19		Knee Normal	
Player 2				Full ROM W 14					Knee normal
Movement									
Player 1		Out of brace w 6		Gait normal		Commence run drills (e.g., A-drills)		**Max** MRV (9.1 m/s) Acc (4.2 m/s/s)	
Player 2		Out of brace w 6	Gait normal				Commence run drills (e.g., A-drills)	**Max** MRV (7.2 m/s); Acc (3.8 m/s/s)	
Strength – Power									
Player 1							**Max** RDL (120 kg x 3) SV (110 kg x 3) SL BE (80 kg x 3 EL)		
Player 2				**Max** RDL (90 kg x 6) SV (170 kg x 4) SL BE (20 kg x 6EL)					

MRV: Maximum constant running velocity; Acc: Acceleration; RDL: Romanian Deadlift; SV: Squat variation (e.g., trap bar deadlift); SL BE: Single leg back extension.

Table 4. Example meal plan for players during the early rehabilitation phase and additional intake for the return to train (RTT) phase.

Meal	Options	Energy (cal)	Protein (g)	CHO (g)	Fat (g)
Breakfast 6–7am	Poached egg x 2 1 serve of other Breakfast protein option (eg 150 g leg ham, steak) ½ avocado 1 cup porridge oats 2 Fish oil tablets	860	60	40	50
RTT additional	2 slices of mixed grain bread Glass of juice	276	9	50	3
Post morning training/Mid-morning snack	WPI (30 g) shake PLUS 3–4 x crackers with ~100 g cottage cheese or 90 g tin tuna + sliced tomato/cucumber/Avocado OR 1 x High protein yoghurt 170 g tub and 50 g of Almond	525	50	20	30
RTT additional	Large banana	123	2	25	0
Lunch	½ plate of green salad or vegetable option 200g plate lean protein option (e.g., grilled chicken/fish/beef/etc) 2/3 cup filling carbohydrate (e.g., sweet potato, brown rice, couscous salad)	790	65	35	40
RTT additional	1.5 additional cup of grain	385	8	80	2
Post afternoon training/mid afternoon snack	WPI (30g) shake PLUS 3–4 x crackers with ~100g cottage cheese or 90g tin tuna + sliced tomato/cucumber/Avocado OR 1 x High protein yoghurt 170g tub and 50g of Almond	525	50	20	30
Dinner	250g lean protein (e.g., grilled chicken/fish/beef/etc) Large serve of green salad or vegetable option 2 Fish oil tablets	720	60	20	40
RTT additional	¾ cup filling carbohydrate (sweet potato, quinoa)	317	5	35	2
Pre bed	1 x berry protein frappe (WPI (30g), ½ cup frozen berries, ½ cup skim milk, lot of ice) PLUS Bowl diet jelly	215	35	15	<5
	Total	3635 ~40 Kcal/kg FFM	320 ~3g/kg BM	150 ~1.5g/kg BM	190 ~1.9g/kg BM
	RTT additional	4736 ~45 Kcal/kg FFM	344	340 ~3.5g/kg BM	197

The timing of nutrients throughout the day was proximal to exercise to enhance nutrient availability and take advantage of cellular processes activated by specific exercise stimuli.

The major focus of the dietary advice outside of the above interventions was adequate energy availability. Energy intakes were targeted at approximately 40 kcal/kg BM during the rehabilitation phase. Macronutrient targets for carbohydrate and fat were subsequently assigned. Although initial carbohydrate intakes were low, in part driven by individual player nutrition philosophies, to help maintain appropriate calorie intake, players increased carbohydrate intake (as directed by the club sports dietitian GS) as training load increased. As training volume and load increased players were encouraged to maintain energy availability targets through increasing carbohydrate-containing food sources around training sessions with daily intakes reaching on average 2–4 g/kg/day when in full training.

Body composition assessment

Players underwent body composition assessment throughout the rehabilitation period. Dual Energy X-ray Absorptiometry (DXA) assessments were undertaken at key time points throughout the rehabilitation period, subject to player availability. Skinfold and body mass assessments were undertaken at a greater frequency due to the portability of equipment and player familiarity with the technique. Assessments, where possible, were undertaken within 24 h of each other with body mass measured at the same time as DXA scan, upon waking, after an overnight fast. Skinfolds were assessed at a convenient time over the day, away from exercise, as per ISAK standards (Stewart, Marfell-Jones, Olds, & de Ridder, 2011). DXA was assessed via a GE Lunar Prodigy (GE Healthcare, Madison, WI), using subject standardisation and scan assessment protocols to ascertain whole body and segmental composition (Nana et al., 2016). Upper leg region of interest (ROI) composition was delineated and measured as per the protocols described by Haakonssen, Jenkins, Burke, and Martin (2017). Baseline measures prior to injury were obtained from the closest DXA assessments to injury available. For player 2 this was on a Hologic Discovery DXA scanner (Hologic Inc, Bedford, MA). The results of this scan are translated into equivalent values using the regression equations outlined by Malouf et al. (Malouf et al., 2013) for comparison purposes. Scans were undertaken at time points that aligned with player availability in the first 4 months and then at regular intervals once players returned to the club training environment (Aug 2014 – April 2015).

Clinical results

*Body composition (**Figure 1**)*

Players saw minor body mass loss over the first 2 weeks post injury (0.8 kg, 0.8% and 0.4 kg, 0.35%, respectively). This corresponded with minor increases in adiposity for player 1 (Σ7SF 2.7 mm, 6.7%) and larger increases for player 2 (Σ7SF 7.9 mm, 11%). Variability in body mass over the course of 38 weeks was larger in player 1 (–3.5 kg), compared to player 2 (–1.2 kg). Whole body lean mass was reduced by 2.9 kg (–3.2%) and 2.0 kg (–2.3%) in player 1 and 2, respectively, at 17 weeks. Player 1 saw significant mass loss over the initial rehabilitation and return to train phases, attributed to the loss of upper body lean mass (–1.9 kg 17 w and –2.5 kg 24 w), matching the rehabilitation plan of the player. Conversely, player 2 saw an incremental increase in lean mass back to baseline over a period of 31 weeks.

Player 1 had no difference between injured and non-injured leg at baselines and saw small drops in lean mass of both legs after 17 weeks (0.8 kg, 5% Left, 0.6 kg, 4% Right) which was mostly apparent in the upper leg (0.6 kg, 5% Left, 0.5 kg, 5% Right). This drop was consistent between both immobilised and non-immobilised legs, returning to baseline measures at 31 weeks. Player 2 had a 0.7 kg difference in leg lean mass at baseline with lean mass measures stable in the non-injured leg over the rehabilitation period. Swelling at 2 weeks, likely increased lean mass measures in the injured leg masking the leg mass differences. At 13 weeks the injured leg was 0.4 kg of lean mass lower than baseline, a difference that was returned to within measurement error by 24 weeks.

*Functional progression (**Tables 2** and **3** and **Figure 2**)*

As outlined in Tables 2 and 3 and illustrated in Figure 2, players progressed through rehabilitation at a consistent pace. Both players were out of the ROM brace at 6 weeks and had achieved full ROM by 19 and 12 weeks, respectively. Run training commenced at 20 weeks with both players returning to previously measured maximum speed and acceleration by 27 weeks (15 Hz SPI-HPU, GPSports, Canberra, Australia). Maximal leg strength was achieved by player 2 at 15 weeks and player 1 at 24 weeks. Both players, therefore, returned to maximal strength before pre-injury lean mass returned. Player 1 suffered unrelated upper respiratory tract infections during rehabilitation and had associated surgery at 24 weeks. Both players returned to full rugby specific training at 24 weeks. Player 1's knee function was assessed via a Power-cut hop test (Serpell et al., 2016) at 30 weeks with no significant differences observed between legs. Both players were medically cleared to play at 34 weeks; however, due to game availability, players 1 and 2 played their first competitive rugby games post injury at 49 and 36 weeks, respectively.

Discussion

The goal of this case report is to outline the progression of two elite rugby players through a purposeful exercise and nutrition ACL rehabilitation programme. The programme for both players was targeted at minimising the deleterious effects of immobilisation, specifically minimising the functional differences between legs at the end of the 8-week rehabilitation phase and enhancing progression through a traditional ACL return to play programme. Consensus suggests that postoperative ACL rehabilitation should continue for up to 9–12 months, with 35% of ACL reconstructions not returning to pre-injury levels of sport within 2 years (van Melick et al., 2016). One key limiter of enhanced rehabilitation progression appears to be muscle atrophy associated with injury and repair. In fact, key metrics of successful and accelerated rehabilitation programmes are return of strength and

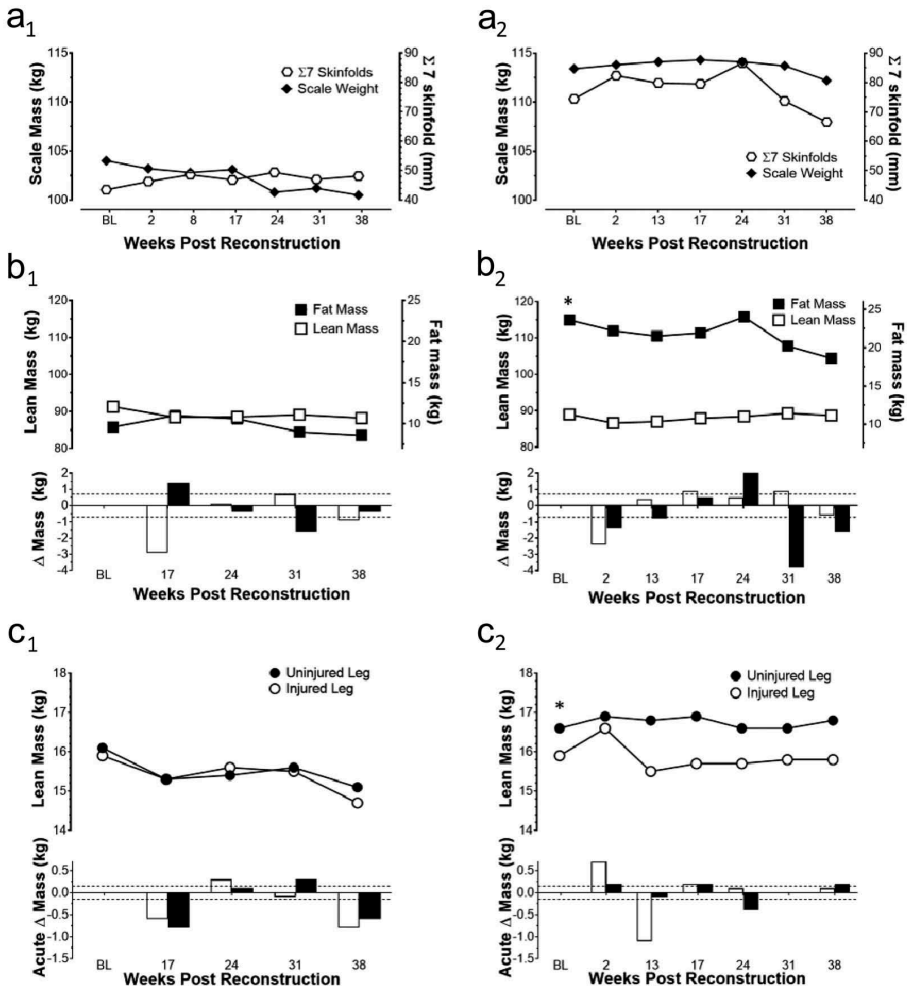

Figure 1. Whole body composition changes over the 38-week rehabilitation period. Skinfold (⎔) and Scale mass (◆) for (A_1) player 1 and (A_2) player 2. Total DXA Lean Mass (□), and Fat Mass (■), as well as an acute change in lean (open bars) and fat (closed bars) mass in (B_1) player 1 and (B_2) player 2. Leg mass changes over the 38-week rehabilitation period. Changes in lean mass of the injured (○) and non-injured (●) leg of (C_1) player 1 and (C_2) player 2. Acute changes in leg lean mass from the prior test for the injured (open bars) and uninjured (black bars) are displayed below. The dotted line (B_1, B_2, C_1, C_2) indicates the smallest worthwhile effect for DXA Lean mass (Nana, Slater, Stewart, & Burke, 2015). *indicates that the DXA scan was performed on a Hologic Discovery machine and converted as per Malouf et al. (2013).

function of injured limbs (Wilk & Arrigo, 2017). Although accelerated rehabilitation via atrophy minimisation has been described in ACL reconstructions of soccer players (Milsom et al., 2014), this is a report of players being ready to play competitive contact sport 34 weeks post hamstring graft.

Body mass and skinfolds assessments within the first 13 weeks showed minimal changes in physique. At 13 weeks in player two whole body lean mass losses were restricted to 2 kg with lean mass losses of 0.4 kg within the injured leg and no changes within the non-injured leg. Milsom et al. (2014) reported losses of 3.5 kg of the whole body lean mass at 12 weeks with losses in the injured and non-injured legs of 1.4 kg and 1.1 kg, respectively. Both interventions were based on previously hypothesised, evidence-based guidelines for minimising the "anabolic resistance" associated with disuse atrophy (Wall et al., 2015). The current case report provides further objective support for the effectiveness of exercise and nutrition protocols aimed at minimising lean mass losses associated with disuse in injured limbs.

This report outlines the author's first attempts at designing and implementing a novel exercise and nutrition intervention with the purpose of aiding in the repair of the ACL graft and site of harvest. Exercise and nutrient availability have been shown to optimise collagen synthesis in engineered ligaments (Hagerty et al., 2012; Paxton et al., 2012). Armed with this knowledge the authors designed a programme to replicate these key objectives within the rehabilitation phase of the ACL return to play programme. Through the supplementation of gelatine and vitamin C as outlined within this case study, we attempted to provide key amino acids (glycine, proline) in combination with ascorbic acid (necessary for proline hydroxylation prior to integration into collagen) at a time when

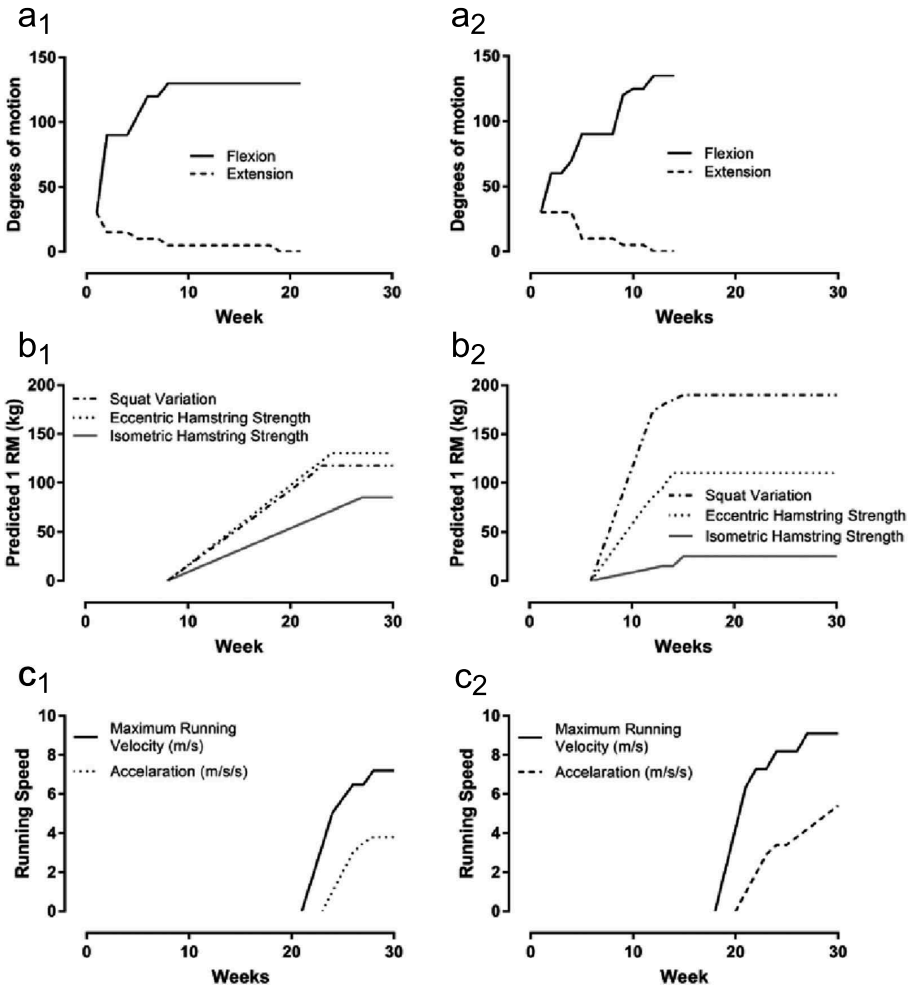

Figure 2. Functional progression through rehabilitation and return to train period of 30 weeks. The range of motion progression reported as degrees of motion for (A_1) Player 1 and (A_2) Player 2. Lower Body Strength progression reported as predicted 1RM by equations of Baker (2004) for (B_1) Player 1 and (B_2) Player 2. Running progression reported for (C_1) Player 1 and (C_2) Player 2.

availability may be limited. The timing of ingestion was optimised to ensure the peak availability of nutrients as blood flow to the injury site was enhanced due to exercise stimulus. In this, we attempted to maintain an adequate nutrient availability to support a strain stimulated collagen synthesis and attenuate drops in collagen synthesis previously reported during immobilisation.

Figure 2 demonstrates a consistent progression through the return to play programme that was not hampered by muscle atrophy during the immobilisation phase. Of interest was the early introduction of resistance exercise in the lower body at 6 weeks. This, in combination with the use of NMES from 48 h postsurgery could help explain the minimal atrophy observed in the injured legs throughout the rehabilitation phase. Consequently, allowing both players to return to maximal lower body strength prior to the return of lean mass to baseline levels. In non-athletes, isometric hamstring strength has been shown to increase rapidly between the fourth and eighth week of rehabilitation after hamstring graft ACL repair. This strength progression then slows such that by 12 weeks hamstring strength is still only 76% of the uninjured leg (Harput, Kilinc, Ozer, Baltaci, & Mattacola, 2015). Player 2 demonstrated no difference in isometric hamstring strength (single leg back extension) of injured and non-injured legs at week 14. Although this demonstrates return of neuromuscular recruitment potential, it is also important to acknowledge that without significant healing of the graft site, return of full muscle function would not be possible. Player 1 saw a less rapid increase in strength but saw maximum hamstring strength returning at 24 weeks. This is less of a surprise when the absolute strength of the players is considered. Others have suggested that hamstring strength is a key marker of progression through accelerated rehabilitation programmes (Wilk & Arrigo, 2017) and this hypothesis is supported by the current case report. Of particular note is the fact that player 1 saw no significant differences between injured and non-injured knee performance in a power-cut hop test undertaken at week 30.

This test was designed to simulate the change of direction manoeuvre typically seen in non-contact ACL injuries (Serpell et al., 2016), suggesting a complete return to baseline knee function at 30 weeks post injury.

Since these case reports were completed, we have demonstrated that nutrition can enhance markers of collagen synthesis in vivo. Through the consumption of food sources high in nutrients shown to enhance in vitro ligament growth (Paxton, Grover, & Baar, 2010), together with short periods of exercise, we were able to demonstrate enhanced collagen synthesis both in vitro and in vivo (Shaw et al., 2017). Additionally, this combination of purposeful connective tissue focused exercise and amino acid availability has been shown to aid in tendinopathy rehabilitation (Baar, 2018).

Conclusion

Significant work is still required to directly demonstrate enhanced healing of connective tissue through the nutrition and exercise strategies outlined above. However, this report outlines our first attempts at translating in vitro work into an effective return to play protocol. Additionally, it demonstrates that well-planned purposeful exercise and nutrition interventions can minimise lean tissue loss during periods of immobilisation. The associated attenuated loss of function may support a more rapid progression through a return to play programme. Finally, it should be acknowledged that the players undertaking this return to play programme were extremely diligent and compliant with the interventions outlined. With the small, meaningful benefits these interventions may provide, highly structured rehabilitation plans are required, completed by compliant athletes, in an individualised training environment.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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