Impact of Lower Limb Muscle Morphology on Sprint Performance

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Abstract

The ability to rapidly accelerate and reach maximal sprinting velocity is paramount to success in running-based sports. However, there is a lack of scientific literature examining the muscular determinants of overground sprint acceleration and maximal velocity in athletes. The purpose of this study was to 1) identify the spatial patterns of lower limb muscle volumes and peak cross-sectional areas in sub-elite rugby league athletes who were affiliated with a National Rugby League (NRL) club; and 2) identify if spatial differences in normalised muscle volumes and peak cross-sectionals area were associated with 10-m sprint times, 30-m sprint times and peak sprint velocity. Nineteen male under-20 rugby league athletes (mean ± SD, age, 19.2 ± 0.7 years; height, 180.7 ± 5.6 cm; mass, 89.9 ± 10.0 kg) from a single NRL club, performed a series of 30-m sprints in order to derive 10-m sprint times, 30-m sprint times and peak sprint velocity. Magnetic Resonance (MR) imaging was used to calculate the normalised muscle volumes and peak cross-sectional areas of the gluteus maximus (GMAX), medius (GMED) and minimus (GMIN), tensor fascia latae (TFL), sartorius (SART), iliopsoas (ILIOP), gracilis (GR), adductor magnus, longus and brevis (ADD), rectus femoris (RF), vastus lateralis (VL), vastus intermedius (VI), vastus medialis (VM), semimembranosus (SM), semitendinosus (ST), biceps femoris long head (BFLH), biceps femoris short head (BFSH), soleus (SOL), gastrocnemius medialis (GM) and gastrocnemius lateralis (GL). Stepwise linear regression models were used to determine the extent to which sprint performance was explained by muscle morphology. The results revealed that variation in 10-m sprint times was best explained by ADD peak CSA ($r^2 = 0.582$, $p = 0.005$) and TFL and VI normalised volume ($r^2 = 0.570$, $p = 0.026$). Variation in 30-m sprint times were best explained by ADD, VI and VM peak CSA ($r^2 = 0.751$, $p = 0.030$) and ILIOP and TFL normalised muscle volumes ($r^2 = 0.672$, $p = 0.021$). Variation in peak sprint velocity was best

explained by GMIN and GL peak CSA ($r^2 = 0.445$, $p = 0.029$) and ILIOP, GL, RECF and ADD normalised muscle volume ($r^2 = 0.820$, $p = 0.035$). The findings of this study demonstrate that sub-elite male rugby league athletes display non-uniform patterns of lower limb muscle size and suggest the possibility that preferential hypertrophy of the proximal hip- and knee-spanning muscles may have important implications for improving sprint acceleration and maximal velocity performance.

Statement of Originality

This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself.

(Signed)_  _
Christopher Martin Cooper

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List of Abbreviations

- ADD – Adductors
- BFLH – Biceps femoris long head
- BFSH – Biceps femoris short head
- CSA – Cross-sectional area
- CT - Computer tomography
- DXA - Dual energy x-ray absorptiometry
- F_H – Horizontal ground reaction force
- F_V – Vertical ground reaction force
- GCT – Ground contact time
- GL – Gastrocnemius lateral head
- GM – Gastrocnemius medial head
- GMAX – Gluteus maximus
- GMED – Gluteus medius
- GMIN – Gluteus minimus
- GPS – Global positioning systems
- GR – Gracilis
- GRF – Ground reaction force
- ILIOP – Iliopsoas
- $m \bullet s^{-1}$ – Metres per second
- MRI – Magnetic resonance imaging
- NRL – National rugby league
- PCSA – Physiological cross-sectional area
- RECF – Rectus femoris
- RFD – Rate of force development
- SART – Sartorius
- SM – Semimembranosus
- SOL – Soleus
- ST – Semitendinosus
- TFL – Tensor fascia latae
- VI – Vastus intermedius
- VL – Vastus lateralis
- VM – Vastus medialis

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1.1 Lay Description

In running-based team sports such as rugby league, the ability to accelerate and attain top sprinting velocity is an important determinant of individual and team performance (Till et al., 2017; Gabbett 2006; De Lacey et al., 2014). Sprint performance can vary considerably between athletes, and this can influence their position both on the field and on the team (De Lacey et al., 2014). For example, faster athletes may be better able to manipulate the speed of play, cover field position and provide more point scoring opportunities than slower athletes (Samozino et al., 2015; Haugen et al., 2019; Korhonen et al., 2009). Despite a significant amount of research, the muscular determinants of sprint performance are still poorly understood. Theoretically, larger muscles are more powerful than smaller muscles (Lieber and Friden, 2000) and might be expected to confer an advantage for sprinting performance. However, the addition of muscle size also increases the mass of the limb, which may confer a speed disadvantage. Interestingly, the distribution of lower limb muscle size in elite sprinters is significantly different to non-sprinters, which suggests the possibility that certain muscles may be more important than others for maximising sprint performance (Handsfield et al., 2017). The purpose of this study was to explore the relationship between the size of the hip-, knee- and ankle-crossing muscles with 10-m and 30-m sprint times and maximal sprint velocity in a group of team-sport athletes. An improved understanding of these factors may improve talent identification practices in elite sport and may inform the design of training studies targeting at improving sprint performance.

1.2 Aims

1) Map the spatial patterns of lower limb muscle volumes and peak cross-sectional areas (CSA) in a group of male under-20 rugby league athletes affiliated with a National Rugby League (NRL) team; and
2) Determine if regional differences in normalised muscle volumes and peak cross-sectional areas are associated with 10-m sprint times, 30-m sprint times, and peak sprint velocity.

1.3 Objectives

1) Employ magnetic resonance imaging (MRI) to determine the peak anatomical cross-sectional areas and volumes of the hamstrings, hip adductors, gluteals, hip flexors, quadriceps and triceps surae muscles, in male under-20 rugby league athletes;
2) Assess 10-m and 30-m sprint times, and peak sprint velocity via electronic timing gates during maximal over-ground sprints in male under-20 rugby league athletes;
3) Employ regression analyses to determine the association between the spatial patterns of lower-limb muscle size and acceleration and peak sprint velocity.

1.4 Hypotheses

1) Male under-20 rugby league athletes will display heterogenous patterns of lower-limb muscle size; and
2) Rugby league athletes who display greater normalised muscle volume and greater peak cross-sectional area of the semitendinosus, biceps femoris long head, adductors and gluteus maximus will display faster 10-m and 30-m sprint times and greater peak sprint velocity.

1.5 Significance

Recently, Handsfield and colleagues (2017) reported that sprint trained athletes display heterogenous patterns of lower limb muscle size when compared to non-sprinters. In this study, elite sprinters displayed significantly larger hip and knee-crossing muscles, normalised to the height-mass product, when compared to non-sprinters. Although muscle volume is proportional to its power generating capacity (Lieber & Friden 2000), a uniform increase in hypertrophy along the lower limb may result in an increase in the moment of inertia which would presumably have a negative effect on the ability to accelerate and reach maximal sprint velocity (Handsfield et al., 2017; Sugisaki et al., 2018). It has been proposed that an increase in muscle mass of more proximally located muscles may be beneficial for enhanced sprint performance (Handsfield et al., 2017; Sugisaki et al., 2018; Sugisaki et al., 2011; Hoshikawa et al., 2006). For example, Miller et al. (2020) reported that the relative muscle volumes of the hip extensor muscle group and the individual muscle volume of gluteus maximus (GMAX), explained 31% and 34%, respectively, of the variance in season best 100-m sprint times. Further, Sugisaki et al. (2018) demonstrated that the GMAX to quadriceps femoris ratio could explain 23% of the variance in 100-m sprint times. However, limited work has examined the muscular determinants of sprint performance in team-sport athletes (Chelly et al., 2010; Edouard et al., 2018; Morin et al., 2015[A]), and no study has explored these characteristics in rugby league players. Elite sprinters (i.e., 100-m sprinters) do not typically reach peak sprint velocity until the 50-m to 60-m mark (Haugen et al., 2019), whereas most of the sprints that occur in a rugby-league match are of distances ≤20-m and frequently involve collisions with opponent players (Gabbett 2012). Given the unique physical demands of rugby league (Haugen et al., 2019; Gabbett 2012), it is reasonable to assume that these

athletes would display a different distribution of lower limb muscle size to other running-based athletes. An improved understanding of the distribution (i.e., spatial patterns) of lower limb muscle size and its association with sprint performance may have important implications for the design of interventions targeted at improving this parameter in rugby league athletes (Maffiuletti et al., 2016; Waldron et al., 2014; Akagi et al., 2009; De Lacey et al., 2014).

2.0 Background Review

2.1 Introduction

In running-based team sports such as rugby league, acceleration and peak sprint velocity are important determinants of individual and team performance (Till et al., 2017; Gabbett 2006; De Lacey et al., 2014).Technology used to determine player locomotive movement patterns, such as Global Positioning Systems (GPS) and time-motion analyses (McLellan et al., 2011; Gabbett, 2012) have revealed that, on average, rugby league athletes perform up to 34 sprints per game and can accumulate distances of 150-300 metres at high-speed, depending on their playing position. Increasing the number of tries being scored, enhanced ball carrying to gain line, increased tolerance to larger impact forces, and greater ball playing speed are examples of the positive outcomes that can be attained through optimising and improving sprint performance (Till et al., 2017: Gabbett et al., 2011). With most sprints in rugby league occurring over distances ≤20m (67.5%) (Gabbett 2012), the need to further understand what factors may improve the ability to accelerate and reach peak sprint velocity are warranted.

Performing explosive movements such as accelerating or sprinting, requires a substantial amount of muscular power (Miller et al., 2020; Andersen & Aagaard 2006). Whilst neural

factors such as the rate and magnitude of electromyographical activity (EMG) play a pivotal role in how much force or power a muscle can generate (Andersen & Aagaard 2006), a muscle's force producing capacity is also proportional to its physiological cross-sectional area (PCSA). In a more recent investigation, Trezise et al. (2016) demonstrated that muscle size as measured by cross-sectional area (CSA), was a major determinant in knee extensor torque, with proximal muscle belly CSA showing a greater association compared to mid or distal measures of CSA. Therefore, training interventions targeted at increasing lower limb muscle CSA would theoretically result in improvements in force production and thus power production (Cormie et al., 2011). However, uniform hypertrophy in all lower limb muscles may increase the moment of inertia during sprinting which would presumably have a negative effect on the ability to optimally accelerate and reach maximal sprint velocity. At present, little to no research has examined whether the distribution (i.e., spatial patterns) of lower limb muscle size is related to sprint performance in team-sport athletes. Handsfield et al. (2017) previously identified significant differences in the magnitude and distribution of lower limb muscle volumes when comparing elite-sprinters to non-sprinters. In this study (Handsfield et al, 2017), the semitendinosus (ST) (54%), gracilis (GR) (42%), tensor fascia latae (TFL) (41%), rectus femoris (RECF) (40%), sartorius (SART) (37%) and GMAX (31%) were 5 of 19 muscles that were significantly larger in elite sprinters compared to non-sprinters. More recently, Miller et al. (2020) reported that elite sprinters displayed larger relative hip extensor muscle volumes than sub-elite sprinters, and that the relative volume of GMAX was associated with 100-m season best performance. Further, Sugisaki et al. (2011) demonstrated that in a group of short and middle-distance college runners, measures of anatomical CSA in the adductors (ADD) were able to best explain 30-m sprint times. While the aforementioned studies highlight the importance of proximal hip-

and knee-spanning muscle size for sprint performance, it should be recognised that each of these studies have used track and field and/or sprint specialists as their participants. Further, sprint performance is often based on retrospective season-best times (Miller et al., 2020) over relatively long distances (i.e., 60-m to 100-m), which limits the interpretation and generalisability of the findings. The spatial patterns of lower limb muscle size and their association with 10-m sprint times, 30-m sprint times and peak sprinting velocity in rugby league athletes has yet to be explored.

2.2 Kinematic and Kinetic Determinants of Acceleration and Maximal Sprint Velocity

At the simplest level, sprint performance is determined by the interaction of stride length and stride rate (Weyand et al., 2000). Despite the optimal interaction for stride length and stride rate being inconclusive, the influence that each factor has during various phases of acceleration and peak sprint velocity have been heavily documented (Hunter et al., 2004). Studies that have previously investigated the interaction between stride length and stride rate, have provided interesting insights into the changes that occur when progressing from the acceleration phase to the maximal velocity phase of sprinting (Hunter al., 2004; Dorn et. al., 2012). Dorn et al. (2012) previously suggested that during the acceleration phase of sprinting (3.5 to 7.0 $m \cdot s^{-1}$), the ability to exert a large amount of ground reaction force (GRF) had been associated with increases in sprint speed as well as increases in stride length. Further, when speeds greater than 7.0 $m \cdot s^{-1}$ were attained, a shift from increasing stride length to increasing stride rate were seen when approaching maximal sprint velocity. However, when investigating the changes that occur during the acceleration phase of a 60-m sprint protocol, Nagahara et al. (2014) demonstrated that in a group of highly trained sprinters, stride rate was the first kinematic variable to reach its maximum

value and not stride length. As depicted in Figure 1, the maximum rate for stride rate had occurred by the fourth step of acceleration, while stride length increased in a similar fashion to sprint velocity, with sprint velocity reaching its maximum value by the twenty-third step and stride length by the twenty-fourth step (Nagahara et al., 2014). It should be recognised that the differences in findings between Dorn et al. (2012) and Nagahara et al. (2014) were possibly due to the differences in data collection. Dorn et al. (2012) had used the resultant GRF, which is the sum of both horizontal force (F_H) and vertical force (F_V), for their analysis on maximal sprint velocity, while Nagahara et al. (2014) assessed both F_H and F_V as separate components of GRF. Further to, the faster velocities that were attained during the early phase of acceleration in the Nagahara et al. (2014) study were noted to occur via a greater pushing-based action, with foot contact being behind the centre of gravity owing to the increase in stride rate (Nagahara et al., 2014). The increases that were witnessed for both stride length and sprint velocity were noted to occur in combination with changes in foot placement. It was identified that as sprint velocity increased, foot placement had begun to change with the foot strike occurring in front of the centre of gravity rather than behind the centre of gravity (Nagahara et al., 2014). When further investigating ground reaction forces during acceleration, Nagahara et al. (2018[B]) discovered that larger propulsive impulses and smaller braking forces contributed to greater acceleration at 55-95% and 75-95%, respectively, of maximal sprint velocity. The greater propulsion and smaller braking forces which aid the early acceleration phase of sprinting are in line with previous findings (Nagahara et al., 2014). Despite an increase in braking force being present, a subsequent decrease in ground contact time (GCT), an increase in stride length and an increase in vertical force are witnessed when increases in maximal sprint velocity occur (Nagahara et al., 2018[B]; Paradisis et al., 2019). Further, it has previously been identified that reductions in maximal sprinting velocity and increases

in GCT have been associated with reductions in stride length and stride rate, respectively (Korhonen et. al., 2009).

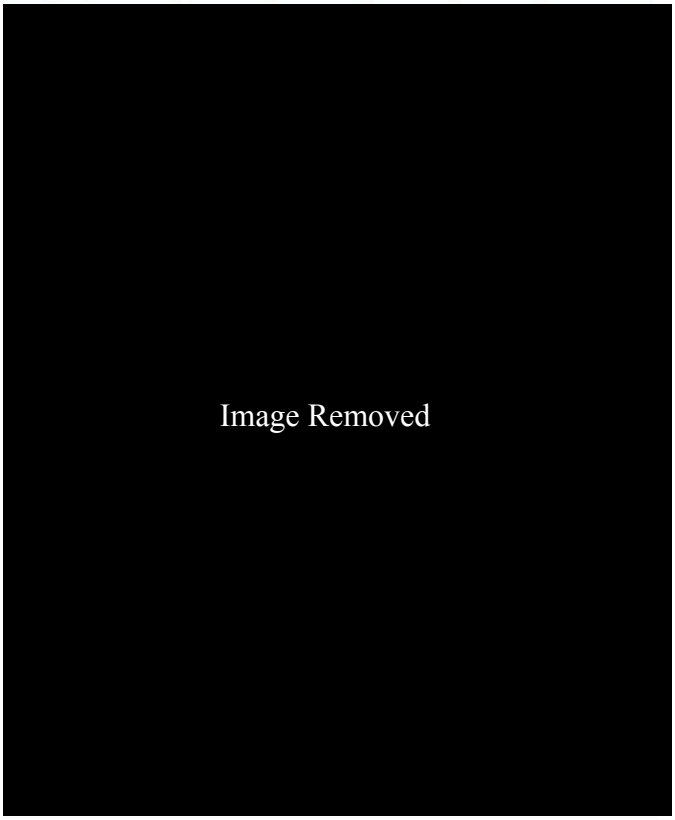


Figure 1. Illustration extracted from Nagahara et al. (2014) representing the relationship between D) stride length and time; and G) stride frequency and time during a six second sprint. Vertical solid lines with numbers indicate the step number.

It is heavily agreed upon that the defining kinematic variable separating faster sprinters from slower sprinters is stride length due to its strong relationship with maximal sprinting velocity (Paradisis et al., 2019; Korhonen et. al., 2009). Although several studies have demonstrated the importance of longer stride lengths on maximal sprint velocity, the importance of higher stride rates should not be undervalued. When athletes are able produce high maximal running speeds, it is typically seen that different combinations of

stride rates and stride lengths are produced, despite the similarities that may be seen in running speed (Hunter et al., 2004). When disparities are seen in factors such as stride rate and stride length, but similar results are attained in sprint speed, determining the factors that influence these kinematic variables may be an important step to further identify the characteristics which enhance acceleration and maximal sprinting tasks. During a 25-m maximal effort sprint task, it was revealed by Hunter et al. (2004) that when vertical velocity increased by 0.20m•s^{-1} (42%) for speed of foot take-off, there was an increase in stride length but a decrease in stride rate by 0.26-m (14%) and 0.52Hz (12%) respectively, with no changes occurring in sprint velocity. In contrast to this, when horizontal velocity increased by 0.20m•s^{-1} (2%) for speed of foot take-off, stride length had increased by 0.02-m (1%), stride rate remained unchanged, while overall the sprint velocity was increased by 0.10m•s^{-1} (1%) (Hunter et al., 2004). Interestingly, stride length was better related to sprint velocity than stride rate, however, in the trials where athletes produced their fastest time, a higher stride rate was used compared to slower trials (Hunter et al., 2004). Further, when decreases in stride rate have previously been reported, an associated increase in GCT has accompanied, indicating that a longer period of time is required in order to generate the needed force to propel the foot forward which is disadvantageous for maximal velocity sprinting (Paradisis et. al., 2019; Korhonen et. al., 2009).

The magnitude and orientation of ground reaction force has been noted to be a determining factor during maximal speed running with suggestions that an increase in the ability to produce force, via increases in muscular strength, are accompanied with improvements in sprint performance (McBride et al., 2009; Comfort et al., 2012). Comfort et al. (2012) previously demonstrated that maximum back squat strength had

been correlated with 5-m (r = -0.61), 10-m (r = -0.62) and 20-m (r = -0.60) sprint times. It was further suggested that faster 10-m and 20-m sprint times in the well-trained sprinters had been attributable to larger forces being produced at higher velocities (Comfort et al., 2012). Early research by Weyand et al. (2000) suggested that when analysing maximal sprinting speed through the use of a motorised treadmill, the noted increase in stride length had been achieved through increases in F_V which led to the increases in maximal sprinting velocity. In support of Weyand et al. (2000), during a 10-m sprint protocol it was demonstrated that reductions in GCT were correlated with F_V owing to the faster 10-m sprint times achieved by team-sport athletes (Lockie et al., 2013). Nagahara et al. (2018[A]) also reported that increases in F_V were associated with reduced GCTs in a 60-m sprint protocol. Interestingly, when a group of semi-professional AFL athletes were required to progress their running speed from 40% to 60% of maximal sprinting speed, F_V significantly increased with the changes in sprint speed (Brughelli et al., 2011). However, when the same group of AFL athletes progressed from 60% to 100% of maximal sprinting speed, there were no significant changes in F_V. Brughelli et al. (2011) proposed that the changes that had occurred for 60%-100% of maximal sprinting speed were due to athletes producing more F_H as opposed to more F_V. It was further postulated that due to the vertically orientated position that the participants started in, the influence of F_V on sprint speed was altered (Brughelli et al., 2011). Producing force over a specific period of time is a measure of impulse and has previously been utilised by researchers to assess both horizontal and vertical components in sprinting (Nagahara et al., 2018[A]; Hunter et al., 2005). Nagahara et al. (2018[A]) previously highlighted that during the acceleration phase of sprinting, vertical impulse rather than F_V was correlated with both stride length and stride rate. It was suggested that due to longer GCT occurring during the acceleration phase of sprinting, greater forces are able to be produced reflecting

the positive influence in which vertical impulse had in the Nagahara et al. (2018[A]) study. It is, however, commonly suggested that F_H is the determining factor for the acceleration phase of sprinting, while F_V may have a greater influence on maintaining maximal sprint speed once attained (Weyand et al., 2000; Nagahara et. al., 2018[B]). It should be acknowledged that differences may be present between team-sport athletes and elite sprinters when sprinting, given the differences in the physical requirements of their sport (Haugen et al., 2019; Lockie et al., 2013).

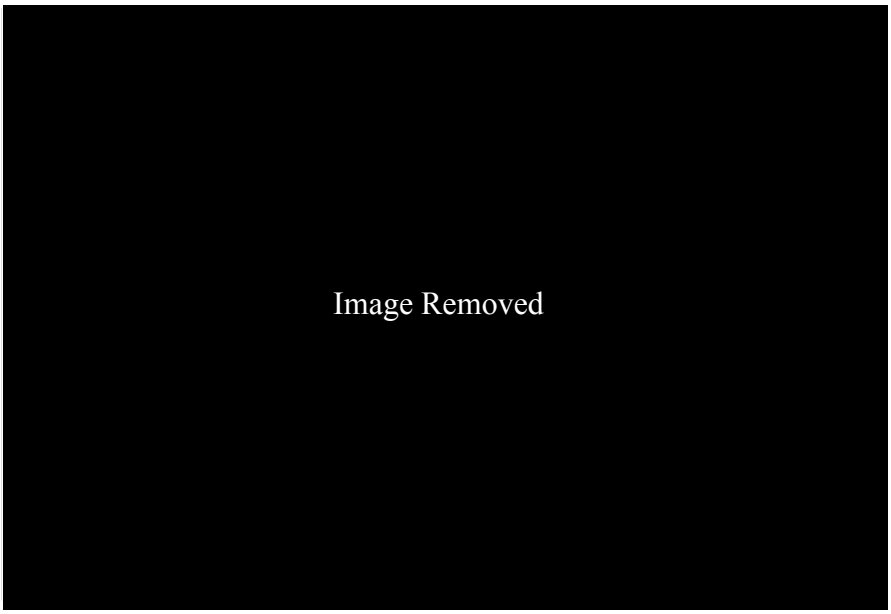


Figure 2. Illustration extracted from Morin et al. (2012). The figure depicts differences in the ratio of force % (RF%), representing the ratio of horizontal force [FH] relative to the total ground reaction force [GRF], between faster and slower 100-m sprint times. Black circles represent the RF% and running velocity of the faster 100-m sprint times, and white circles represent the RF% and running velocity of slower 100-m sprint times.

While F_V has shown to influence stride length, contact time and maximal sprint speed (Weyand et. al., 2000; Lockie et al., 2013; Nagahara et al., 2018[A]), greater F_H has been

prominent in differentiating faster sprinters from slower sprinters (Rabita et al, 2015). Previously, Morin et al. (2012) demonstrated that as sprinting velocity increases, the ability to apply and maintain a greater ratio of force in the horizontal direction (RF%) separated faster 100-m sprinters from slower 100-m sprinters (Figure 2). In a similar finding, Rabita et al. (2015) detailed that while analysing the orientation of force between elite sprinters and sub-elite sprinters during various sprint distances (10-m to 40-m), it was recognised that the elite sprinters had an enhanced ability to generate a greater RF% in the horizontal direction (F_H) rather than the vertical direction. Although previous studies have suggested greater resultant force is a determining factor for sprint speed (Weyand et al., 2000; Dorn et al., 2012), more recent investigations seem to suggest otherwise (Rabita et al., 2015). Further, sub-elite sprinters have previously been shown to produce a greater amount of resultant force when compared to elite sprinters, however, slower speeds were still attained by sub-elite sprinters due to an inability to effectively apply GRF (Rabita et al., 2015). The elite sprinters in the above study were noted to have applied greater amounts of F_H as opposed to greater resultant forces, leading to faster sprinting speeds being attained (Rabita et al., 2015). Greater propulsion forces, smaller braking forces and smaller vertical forces that occur in the early acceleration phase of sprinting have shown to be an advantage when applying forward leaning GRF i.e., F_H (Nagahara et al., 2018[B]). The ability to apply large amounts of F_H during the early acceleration phase have shown to further enhance the capability of producing higher speeds post-acceleration phase (Nagahara et al., 2018[B]). To further highlight this point, in a 40-m sprint protocol analysing GRF impulse, the athletes who produced the highest amounts of horizontal impulse in the acceleration phase were the athletes who reached the highest sprinting speeds (Morin et al., 2015[B]). When horizontal impulse was broken down into the propulsive phase (positive impulse) and the braking phase (negative

impulse), it was the ability to perform a greater pushing-based action in the acceleration phase (first 6-7 steps) which allowed a greater amount of positive horizontal impulse to be generated (Morin et al., 2015[B]). This finding by Morin et al. (2015[B]) emphasises the need for higher amounts of horizontal net impulse per unit of body mass to accelerate optimally. Similar findings were identified by Hunter et al. (2005) who found that the strongest predictor for sprint velocity was the application of relative horizontal impulse in the acceleration phase of a sprint. It was speculated that greater amounts of vertical impulse would be disadvantageous in the acceleration phase with suggestions that only the minimum amount of vertical impulse should be generated to allow effective repositioning of limbs, with all other efforts directed towards producing greater horizontal impulses (Hunter et al., 2005). Given that the initial acceleration phase of sprinting involves accelerating a static body, reducing the amount of F_V will allow an increase in GCT which may allow for greater F_H to be developed (i.e., because slower rates of shortening allow for the development of greater forces) (Hunter et al., 2005).

The horizontal and vertical forces needed to accelerate at high rates and reach maximal sprinting speeds, which both ultimately affect stride rate and stride length, are predominantly generated by the muscles of the lower limb (Handsfield et al., 2017; Morin et al., 2015[B]; Nuell et al., 2020). While muscle size is strongly related to its force and power production (Akagi et al., 2000; Blazevich et al., 2009; Trezise et al., 2018), increasing muscle size uniformly along the lower limb may be disadvantageous to sprint performance as it would also increase the limb's moment of inertia (Miller et al., 2020; Sugisaki et al., 2011; Hoshikawa et al., 2006). To effectively apply ground reaction force and produce the kinematics required to sprint optimally, the development of muscular force and power for specific muscles/muscle groups should be further understood.

2.3 Influence of Maximal Force Production on Acceleration and Maximal Sprint Velocity

The relationship between muscular strength and sprint performance has been the focus of significant research efforts, with suggestions that an increase in muscular strength will be accompanied by an enhanced ability to apply ground reaction force (McBride et al., 2009). As demonstrated in Figure 3, training interventions in rugby league athletes involving the back squat exercise have shown improvements of 7.6%, 7.3% and 5.9% with 5-m, 10-m and 20-m sprint times, respectively, suggesting that increases in lower body strength result in a positive effect for sprinting ability (Comfort et al., 2012). Styles et al. (2016) found similar results when introducing an in-season strength program in a cohort of professional soccer athletes. It was determined that improvements in back squat strength had a positive effect on sprint performance with improvements of 5%, 3% and 1% observed for 5-m, 10-m and 20-m sprint times (Styles et al., 2016). The association between improvements in back squat strength and sprint performance are suggested to reflect the longer GCT present during the initial phase of sprinting, whereby a greater amount of force is able to be generated in order to accelerate the body (Comfort et al., 2012). With team sport athletes performing a large portion of their sprints across distances ≤30-m (Wisloff et al., 2004; Gabbett 2012), the ability to produce a significant amount of force via increases in lower body strength may further allow athletes to meet the physical demands of their sport (Comfort et al., 2012; Wisloff et al., 2004; Gabbett 2012). Additionally, the inclusion of a 10-week Nordic hamstring exercise (NHE) protocol demonstrated improvements in 10-m sprint times in a group of male soccer players (Ishoi et al., 2018). It had been suggested by Ishoi et al. (2018) that the improvements seen in 10-m sprint times with the inclusion of the NHE, resulted from an improved ability to

produce more F_H. Interestingly, Baker & Newton (2008) demonstrated that despite elite rugby league athletes being significantly stronger than sub-elite athletes, no differences were witnessed in acceleration and sprint speed. However, the significant differences in maximal strength and the heavier body masses (8.9%) in the elite playing group resulted in greater amounts of momentum (7.0%) being generated. As body mass increases, the 17% and 12% differences in maximal leg strength and maximal power, respectively, may facilitate improvements in acceleration and sprint speed, further resulting in changes in momentum (Baker & Newton, 2008). As previously outlined, due to the close proximity and the collisions involved in rugby league (Till et al., 2017: Gabbett et al., 2011) optimising momentum may be advantageous for playing performance.

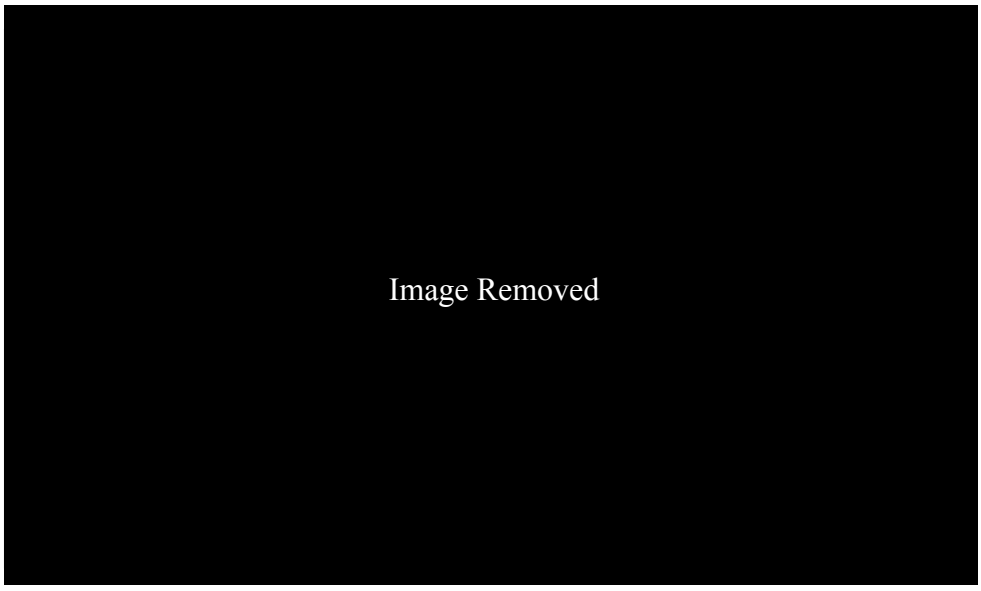


Figure 3. Illustration extracted from Comfort et al. (2012). The figure depicts the changes in 5-m, 10-m and 20-m sprint times in professional rugby league athletes after an 8-week strength/power training intervention.

While absolute and relative strength measures have shown to be favourable for sprint performance, elements such as the rate of force development (RFD) may further

differentiate faster and slower runners (Tillin et al., 2013). When previously assessing the rate of torque development (RTD) in the hamstring muscle group, Ishoi et al. (2019) identified that the ability to produce a greater amount of force in ≤100ms had been associated with 0 to 5-m, 0 to 15-m and 0 to 30-m sprint times in a group of elite football players. These findings are in agreement with Tillin et al. (2013) who demonstrated that the ability to produce a greater proportion of maximal force in ≤150ms separated faster (<1s) from slower rugby union athletes (>1s) in a 5-m sprint protocol. It was detailed that as maximal force production was comparable between fast and slow rugby union athletes, the ability to produce a greater amount of force at 50ms, 100ms and 150ms had been defining factors demonstrating an enhanced ability for RFD (Tillin et al., 2013). Andersen and Aagaard (2006) reported that 80% of the variance in voluntary RFD (150-250 ms) could be explained by maximal muscle strength. Further, Ishoi et al. (2019) and Tillin et al. (2013) suggested that RFD, and not maximal force, was the primary determinant of acceleration performance in faster athletes. Both Akima et al. (2000) and Andersen & Aagaard (2006) previously identified that the extent to which a muscle is voluntarily activated, inherently influences the amount of force it can produce as well as the rate it can be produced. Theoretically, an increase in muscle mass coupled with an increase in the level of muscle activation would lead to an increase in maximal force production and RFD (Andersen & Aagaard 2006; Maffiuletti et al., 2016; Akima et al., 2000).

2.4 Physiological Factors Influencing Acceleration and Maximal Sprint Velocity

Through previous investigations, it can be gathered that athletes who have larger CSAs of type II muscle fibres are able to produce both higher contractile velocities as well as

higher contractile forces (Korhonen et. al., 2006; Maffiuletti et. al., 2016). In addition, training status also influences the ability to use adenosine triphosphate (ATP) through greater contributions of phosphocreatine (PCr) (Korhonen et. al., 2006; Glaister, 2005; Girard et. al., 2011; Cristea et. al., 2008). Sprinting is one of the most dominant actions performed in several sports, and is largely determined by the physical and physiological characteristics of individual athletes (Comfort et al., 2012; De Lacey et al., 2014; Faude et al., 2102). Due to the regular requirement for acceleration and sprinting in rugby league, increasing type II fibre size as well as improving metabolic responses for sprinting using resistance-based training and mixed methods of sprint training is warranted (Bishop et al., 2011). Given that sprinting requires the generation of substantial muscular power, understanding the physiological factors that allow a muscle to produce a substantial amount of force within a limited amount of time, may be of interest for improving sprinting ability.

When repeated bouts of acceleration and sprinting occur as is the case in rugby league (Gabbett, 2012), the overall performance and the ability to repeat sprints reduce as a direct result of metabolic fatigue (Bishop et al., 2011; Greenhaff et al., 1994). Maximal intensity exercise such as sprinting is typically limited by the rate at which ATP can be supplied and resynthesised within the active muscles (Glaister, 2005; Girard et. al., 2011). Peak ATP turnover has previously been detailed to only supply the human body with 1-2s of maximal work, and with maximal sprint efforts lasting 2-10s, PCr becomes significantly important for the resynthesis of ATP as it is the most immediate reserve for rephosphorylation (Glaister, 2005; Girard et. al., 2011). The influence and dominance that type II muscle fibres have on sprinting is supported by the findings of Greenhaff et al. (1994). In a maximal 30s sprint effort, Greenhaff et al. (1994) demonstrated that both

type I and type II fibres had a substantial decline in both PCr and glycogen. However, type II fibres were noted to have incurred a greater depletion in both PCr and glycogen by a further 25% and 64%, respectively, when compared to type I fibres. Strength and RFD have previously been shown to be influential for sprint performance (Comfort et al., 2012; Baker & Newton 2008; Tillin et al., 2013) and due to the ability of type II muscle fibres to produce higher amounts of force and greater RFD than type I fibres (Maffiuletti et al., 2016), it would make reasonable sense to promote positive adaptations in type II fibres.

2.5 Neurological Factors Influencing Acceleration and Maximal Sprint Velocity

Voluntary activation of skeletal muscle is an important determinant of the rate and magnitude of force production (Ross et al., 2001; Bishop et al., 2011). Increases in sprinting speed are paralleled by an increase in surface electromyographical (EMG) activity of most hip-, knee- and ankle-spanning muscles (Howard et al., 2018; Paradisis et al., 2019; Korhonen et. al., 2009). However, changes in EMG activity appear to be non-uniform between different lower limb muscles throughout the sprint gait cycle, suggesting that various muscles may play more important roles during different phases of the sprint cycle (Howard et al., 2018). Specifically, Howard et al. (2018) noted in their systematic review that during the stance phase of the sprint cycle the hamstrings, quadriceps, gastrocnemius and soleus (SOL) were active through the entire phase, while GMAX and tibialis anterior were only active during first contact and early phase of the stance phase. Further, during the swing phase of the sprint gait cycle, the hamstrings in addition to vastus lateralis (VL), GMAX, gastrocnemius and SOL were noted to be active

during the late swing phase, while RECF had two distinct bursts in the early and late phase of swing (Howard et al., 2018). While it's well established that larger muscles can produce more force compared to smaller muscles, previous investigations have identified a strong relationship between torque and muscle recruitment (Akima et al., 2000). Using functional MRI and isokinetic dynamometry, Akima et al. (2000) identified that the extent to which a muscle is activated (quantified via exercise-induced changes in the transverse [T2] relaxation time) inherently influences the amount of force it can produce. Akima et al. (2000) found that whilst having a larger PCSA improves capacity to produce more force, the ability to activate or recruit a larger portion of muscle fibres within a muscle may have a greater effect on force production compared to PCSA alone. The difference in muscle fibre recruitment seen in the Akima et al. (2000) study was witnessed between two participants displaying a PCSA of 314cm^2 and 281cm^2. Due to an increased ability in recruiting a greater number of muscle fibres within the quadriceps femoris muscle group, the participant with the PCSA of 281cm^2 produced more isometric force (291 N.m vs 398 N.m) than the participant with greater PCSA (Akima et al., 2000). In support of this, Trezise et al. (2016) demonstrated that the combination of muscle size (i.e., CSA), fascicle angle and the level of quadriceps activation were predictors of isometric knee joint torque.

The number of motor units (MUs) recruited and the rate at which motor neurons discharge (rate coding) significantly influence force production (Maffiuletti et al., 2016). When high-threshold MUs containing type II muscle fibres are recruited, due to the increased discharge rates of these MUs, greater increases in muscular force and rates of muscular force can be achieved (Maffiuletti et al., 2016). Whilst increased GCT during the acceleration phase may provide an advantage for horizontal force production (Kawamori

et al., 2013; Hunter et al., 2005), the time required to produce maximal force (>300ms) is typically greater than the time allowed during the acceleration phase (<300ms) and the maximal velocity phase (<200ms) (Tillin et al., 2013; Paradisis et al., 2019; Styles et al., 2016; Ishoi et al., 2019). As outlined in section 2.3, Tillin et al. (2013) demonstrated that the ability to produce more force earlier (i.e., 50-150 ms), was a determining factor separating faster and slower rugby union athletes. This is consistent with the findings of Ishoi et al. (2019) who demonstrated that greater RTD in the hamstring muscle group was associated with sprint acceleration performance. Similarly, Paradisis et al. (2019) demonstrated that faster athletes had reduced GCT as well as a shorter propulsive phase than slower athletes. Although the magnitude of propulsive horizontal force is strongly associated with acceleration and maximal sprint velocity (Morin et al., 2015[B]; Hunter et al., 2005; Nagahara et al., 2018[B]), the reduction in GCT observed in faster sprinters (Paradisis et al., 2019; Nagahara et al., 2018[A]) highlights the need for greater RFD. In support of this, Lockie et al. (2011) previously discovered that differences in time to reach peak vertical and horizontal force separated faster and slower field-sport athletes in a 10-m sprint protocol. The reduced GCT was the major step characteristic separating faster athletes from slower athletes, and as peak force was similar between groups, it was recognised that the greater efficiency in force production displayed by faster athletes provided an advantage in faster 10-m sprint times (Lockie et al., 2011). With previous investigations by Handsfield et al. (2017) and Miller et al. (2020) highlighting disproportionate increases lower limb muscle mass in elite sprinters, specifically hip and knee-crossing muscles, preferential recruitment of these muscles may result in improved sprint performance. Given the potential differences in the activation patterns of individual muscles throughout the sprint gate cycle (Howard et al., 2018), the changes in muscular

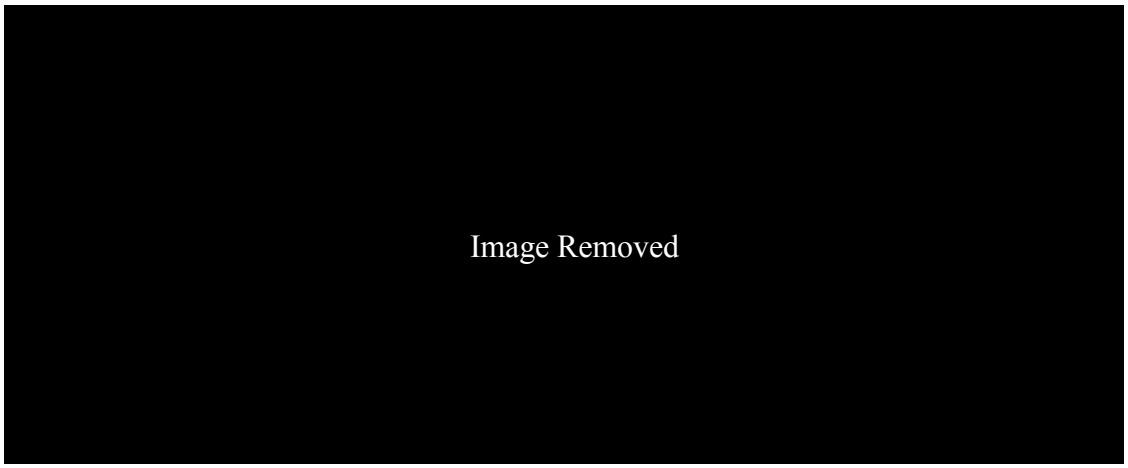
structure seen in elite sprinters and well-trained athletes may reflect the physical demands placed upon each muscle in order to complete an acceleration or sprint-based task.

2.6 Morphological Factors Influencing Acceleration and Maximal Sprint Velocity

Muscle size has a substantial impact on force production (Akima et al., 2000; Trezise et al., 2016) and therefore sprint performance. A muscle's architecture, which includes muscle length, fibre length, pennation angle and physiological cross-sectional area (PCSA) are important determinants of its function (Fukunaga et al., 2001; Lieber & Friden 2000). It is well recognised that the effective application of force in both the horizontal and vertical direction heavily influences sprinting ability (Hunter et al., 2004; Rabita et al., 2015). Further, with previous reports suggesting muscle activity changes throughout the sprint cycle (Howard et al., 2018), it can be postulated that specific muscles or muscle groups may be need to be preferentially targeted to produce the kinetics and kinematics required to accelerate and sprint optimally.

PCSA is the only architectural characteristic that is proportional to a muscle's force generating capacity (Liber and Friden, 2000). PCSA encompasses the cross-sectional area of a muscle perpendicular to the orientations of its fibres and reflects the number of sarcomeres in parallel (Lieber & Friden 2000). In contrast, anatomical cross-sectional area (ACSA) is measured perpendicular to the longitudinal axis of a muscle, so does not account for all fibres contained in pennate muscle (Fukunaga et al., 2001). Blazevich et al. (2009) conducted an experiment to examine the extent to which quadriceps muscle volume, PCSA, ACSA and muscle architecture could predict isokinetic knee extensor moments. The combined measurement of PCSA and fascicle length were identified as

being the best predictors when measuring knee extensor moments at 300°s^{-1}, explaining 59% of the variance that occurred at this speed (Blazevich et al., 2009). As PCSA is a major determinant of force production and given that fascicle length heavily influences contraction velocity, it logical that the combination of these two factors would influence force production at higher movement speeds (Blazevich et al., 2009). This study also revealed that quadriceps muscle volume was the best predictor at of knee extensor moment at 0°s^{-1} and 30°s^{-1}, explaining 60% and 74% of the variance in isometric and slow concentric speeds, respectively (Blazevich et al., 2009). However, in this study no statistical differences were apparent between ACSA, muscle volume and PCSA (Blazevich et al., 2009), which suggests that ACSA may be an appropriate surrogate of muscle size when more robust methods are not available. These findings from Blazevich et al. (2009) support the idea that the active range of a muscle and its force capabilities are heavily influenced by both increases in the sarcomeres in-series, leading to longer muscle fibres, and the increase in muscle volume/size.



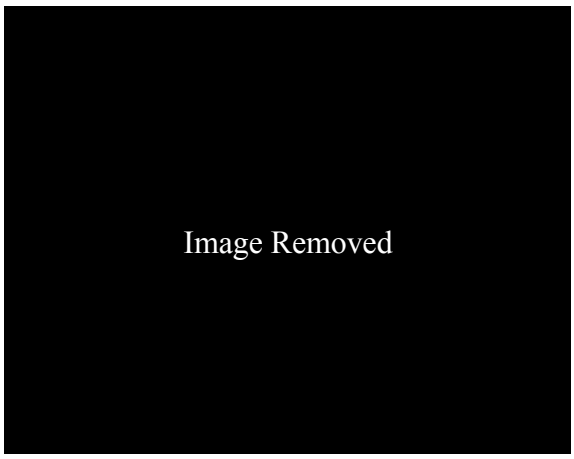


Figure 4. Illustration extracted from Aagaard et al. (2001). Figure depicts changes that occur after a 14-week strength training program in quadriceps CSA (top left), quadriceps volume (top right) and maximal isometric voluntary contraction (bottom).

When investigating morphological changes that occur as a consequence of a 14-week strength training program, Aagaard et al. (2001) reported that VL muscle volume, ACSA and maximal isometric knee extensor strength increased by 10.3%, 10.2% and 16.2%, respectively, which can be seen in Figure 4. It was further identified that there was an increase in type II muscle fibre CSA by 18.4%, demonstrating a disproportionate increase between ACSA, volume and muscle fibre CSA (Aagaard et al., 2001). Aagaard et al. (2001) suggested that the disproportioned increase in muscle fibre size reflected the changes which occurred in the pennation angle of the VL (+35.5%), allowing an increase in contractile muscle tissue to occur, further explaining the increases seen in muscle strength. In a comparison between sprint specialists, middle-distance runners and long-distance runners, Weyand & Davis (2005) reported that sprint specialists displayed the largest muscle masses. It is believed that when approaching faster sprinting speeds, the ability to produce greater support forces is required and is further aided with an increase in muscle mass (Weyand & Davis 2005). Although it was suggested that an increase in

muscle mass would be an advantage for sprinting, the increases seen in muscle mass, or volume, are typically accompanied with an increase in total body mass, which may be a disadvantage when sprinting (Handsfield et al., 2017). Despite an increase in the strength capacity of the limb, a uniform increase in muscle mass may negatively affect sprint performance via increasing the limb's moment of inertia (Handsfield et al., 2017; Sugisaki et al., 2018). This increase in inertia has been hypothesised to reduce the angular acceleration of the limb and requires a greater amount of effort to accelerate and decelerate the limb at fast speeds (Handsfield et al., 2017; Sugisaki et al., 2018; Sugisaki et al., 2011; Hoshikawa et al., 2006).

In a recent investigation, Handsfield et al. (2017) examined differences in lower limb muscle volumes between a group of 15 elite sprinters and 24 non-sprinters as can be seen in Figure 5. The assessment of normalised muscle volume was conducted for 38 lower limb muscles for both cohorts via MRI. When comparing the two cohorts, 19 lower limb muscles were significantly larger in the elite sprinter group compared to the non-sprinting group. Notably, Handsfield et al. (2017) identified that despite significantly larger muscle volumes being evident in the elite sprinter group, there were a number of muscle volumes that were identified as being the same size, and in some cases smaller, in the elite group when compared to the non-sprinting group. As an indication of the differences seen for the muscle volumes; ST, RECF, GMAX, ADD, and BFLH were 54%, 40%, 31%, 26% and 26% larger in the elite sprinting group, respectively. A recent study by Miller et al. (2020) attempted to assess the differences between elite sprinters, sub-elite sprinters and untrained subjects. Miller et al. (2020) examined the association between measures of muscle volume, isometric strength and anthropometry with 100-m sprint performance. While a number of muscles and muscle groups were significantly larger in the elite group

than the untrained group, the relative volumes of the hip extensor group, TFL, SART and GMAX were 15%, 37%, 28% and 25% larger in the elite than sub-elite group, respectively. Though there were differences in muscle volume between the two sprint groups, there were no differences in measures of strength between these two groups. Further, none of the strength measures collected in this study were related to season best 100-m sprint time (Miller et al., 2020). Overall, it was the relative muscle volumes of the hip extensor muscle group and the GMAX that could explain 31.4% and 33.6% of the variance, respectively, in season best 100-m sprint times. In another study exploring associations between lower limb muscularity and 100-m sprint times in Japanese sprinters, Sugisaki et al. (2018) discovered that neither the measurements of absolute nor normalised muscle volume could predict 100-m sprint times. However, the GMAX to quadriceps femoris ratio explained 23.4% of the variance in 100-m sprint times, possibly via contributing to greater propulsive forces. In an ecological study whereby, national sprinters were followed across a 5-month in-door training cycle with no interference by investigators, Nuell et al. (2020) discovered that improvements in sprint performance, across various sprint distances, were accompanied by increases in hamstring, ADD and quadriceps muscle volume. It was recognised that the degree of muscle volume change did not directly transfer to improvements in sprint performance. However, notable improvements of 5.3% and 7.2%, were identified for maximal sprint velocity and F_H production, respectively, after the 5-month training cycle phase (Nuell et al., 2020). Specifically, for 10-m and 40-m sprint times, improvements of 7.0% and 4.9% were identified, respectively, while the muscle volumes of the hamstrings, ADD and quadriceps increased by 10.1%, 12.2% and 6.7%, respectively, after following a 5-month periodised plan designed by the head coaches (Nuell et al., 2020).

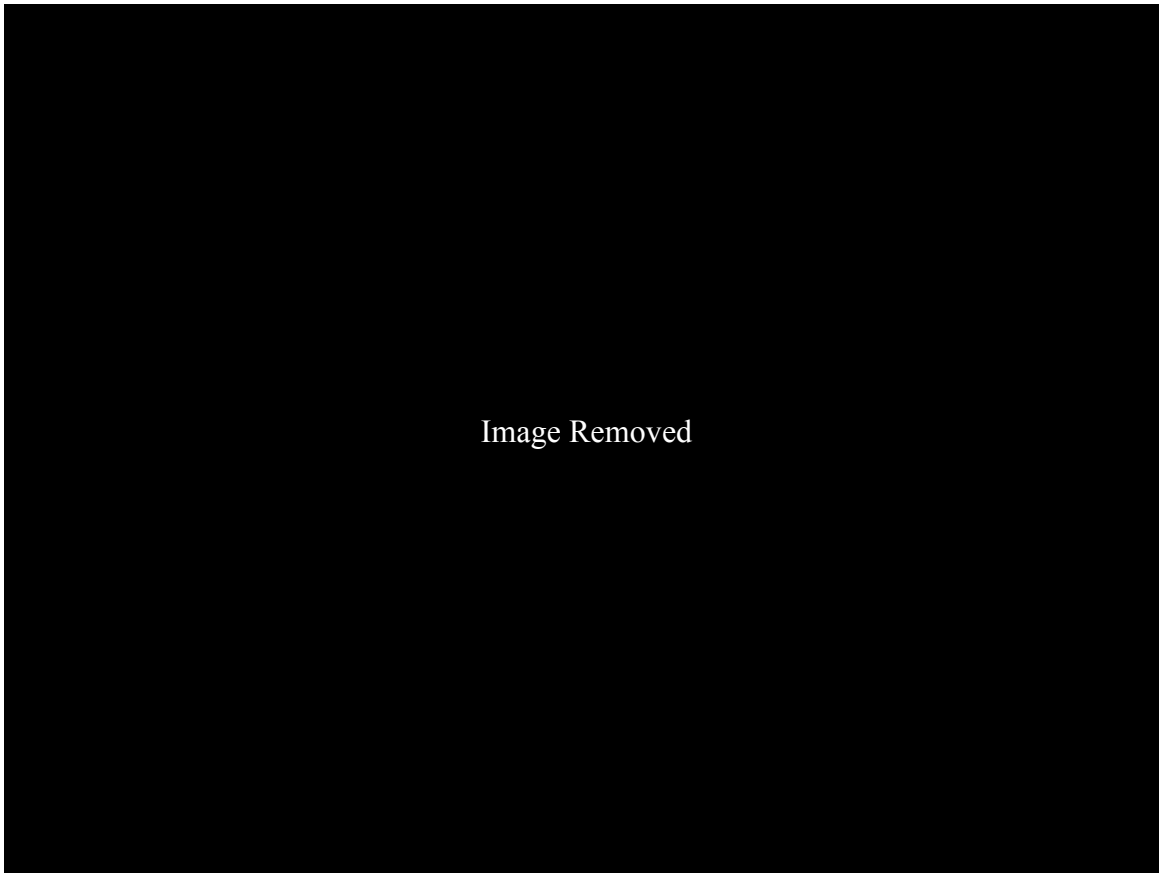


Figure 5. Illustration extracted from Handsfield et al. (2017). Figure depicts muscle volume differences between sprinters and non-sprinters. Filled circles indicate significant differences, non-filled circles indicate non-significant differences. Rectus femoris (RF), vastus medialis (vast med), vastus intermedius (vast int), vastus lateralis (vast lat), adductor magnus (ad mag), adductor longus (ad long), adductor brevis (ad brev), gluteus maximus (glut max), gluteus medius (glut med), TFL, biceps femoris long head (BFlh), semitendinosus (semitend), semimembranosus (semimemb), biceps femoris short head (BFsh), gastrocnemius lateral head (lat gastroc), gastrocnemius medial head (med gastroc), flexor digitorum longus (fdl), flexor hallucis longus (fhl), extensor digitorum longus and extensor hallucis longus (edl & ehl), tibialis anterior (ta).

The idea that increases in lower limb muscle mass, and thus increases in inertial load, would have a negative effect on sprint performance (Sugisaki et al., 2018) are supported

by Hoshikawa et al. (2006) who investigated the association between quadriceps femoris, hamstrings and psoas major CSA with 100-m sprint performance. Hoshikawa et al. (2006) identified that for 100-m sprint time, an increase in the psoas major to quadriceps femoris CSA ratio was able to explain 38% and 33% of the variance for the boys and girls, respectively. It is postulated that while sprinting, having a lower quadriceps femoris CSA and a larger psoas major CSA may potentially aid forward acceleration of the limb, with increases in psoas major CSA influencing the power generated at the hip which is required to decelerate the hip extension created in the prior phase (Hoshikawa et al., 2006). While larger proximal than distal muscles are likely advantageous for sprinting (Hoshikawa et al., 2006; Sugisaki et al., 2018), the potential importance of the ADD muscle group has been highlighted by Sugisaki et al. (2011). One of the major factors differentiating faster and slower athletes is the ability to apply large amounts horizontal force, which has been identified to be a major role of the ADD muscle group as a prime mover of hip extension (Morin et al., 2015[B]; Hunter et al., 2005; Sugisaki et al., 2011). Sugisaki et al. (2011) discovered that the CSA of the ADDs explained 60% of the variation in a 30-m sprint protocol. A possible explanation of Sugisaki et al. (2011) not being able to explain more variance in 30-m sprint times, is the exclusion of GMAX CSA from the study. However, during positions in which there are greater degrees of hip flexion, the moment arm of the ADDs is greater than that of the GMAX, owing to a greater emphasis on the adductors to then extend the hip (Sugisaki et al., 2011). Interestingly, when comparing absolute and normalised muscle volumes for elite female sprinters and female non-sprinters, Yasuda et al. (2019) identified no difference in any of the variables other than that of normalised adductor brevis volume. An r value of -0.652 was described for the correlation between adductor brevis and 100-m sprint time, suggesting that the larger normalised adductor brevis was associated with faster 100-m

sprint time. The adductor brevis has also been noted for its contribution to hip flexion and medial rotation when placed into an extended position, which may potentially support the role of psoas major and further assist in 100-m sprint performance (Yasuda et al., 2019).

While measures of CSA and muscle volume provide valuable insight into the muscular determinants of sprint performance, results are not always consistent. Previous studies analysing CSA or muscle volume have used 1) a limited number of muscles for the analysis, 2) a limited number of sites to determine muscle volume, and 3) different methods to determine CSA site and peak CSA. In relation to the latter, some measures of CSA and peak CSA have been determined relative to limb length and/or muscle length. The drawback from these values is that these measures of CSA may not truly represent the peak CSA of the muscle. Further, when estimating muscle volume from measures of CSA, although Lund et al. (2002) demonstrated a 0.004% difference in muscle volume estimates when using 8 versus 50 CSA slices, large errors (<15%) in volume estimates were observed when using 4 CSA slices. Although valuable information can be attained from single slice CSA measures, when it comes to estimating muscle volume it is clear that a greater number slices are required for more accurate measures. It should also be acknowledged that the majority of existing research examining associations between muscle morphology and sprint performance has been conducted on track and field athletes or specialist sprinters, which may not be applicable to other running-based athletes.

2.7 Methods to Assess Lower Limb Muscle Morphology

Several methods are available for the assessment of whole body and regional muscle mass, but these can vary significantly in their accessibility and cost (Franchi et al., 2018).

Dual Energy X-ray Absorptiometry (DXA) and Computer Tomography (CT) scanning are recognised as cost-effective options to assess both the regional and total lean masses of individuals. However, studies that require a large exposure to DXA and CT raise ethical concerns due to the ionizing radiation that accompanies these methods (Franchi et al., 2018).

Ultrasound imaging has become a popular method for the application of measuring muscle architecture in vivo (Franchi et al., 2018). The use of ultrasound has been highly sort after due to its ability to measure muscle thickness, pennation angle and fascicle length which have been noted to be valuable in estimating the PCSA of muscle (Fukunaga et al., 2001; Aagaard et al., 2001). It was previously demonstrated that following a 12-week training program aiming to increase hypertrophy of the VL, ultrasound imaging at a single site (50% of femur length) was able to detect changes in muscle size (Franchi et al., 2018). However, it was noted that the changes that were detected using ultrasound imaging were different to MRI measures (Franchi et al., 2018). The possible explanation for the difference in percentage change is due to the VL not being perfectly symmetrical along the length of the muscle, meaning that a size change in a single region may not represent changes in other regions (Franchi et al., 2018). Despite showing promise in being able to estimate muscle volume with a low standard error of estimate (SEE) of 6-8% (Fukunaga et al., 2001) and being a reliable tool for measuring fibre angle and fibre length which are fundamental to assessing the PCSA (Blazevich et al., 2009; Franchi et al., 2018), ultrasound imaging may not be a viable tool in inferring whole muscle characteristics (i.e., muscle volume). Trezise et al., (2016) discovered that using a single CSA measurement gathered from ultrasound imaging may present error depending on the site in which it was obtained. It was demonstrated that a CSA slice obtained at 50% of

quadriceps femoris muscle length, which includes the CSA of VL, vastus medialis (VM), RECF and vastus intermedius (VI), had a lower predictability for knee extension torque prediction compared to using a measure that was more proximal on the limb (Trezise et al., 2016). There are suggestions that due to differences seen among the regions of a muscle for size and fibre type, taking a single measure may not be viable for assessing muscular size and force production (Trezise et al., 2016; Blazevich et al., 2009).

MRI is recognised as the gold standard tool for the measurement and assessment of muscle volume. MRI technology provides useful information for the analysis of soft tissue through its detection of magnetic activity of hydrogen nuclei contained in tissue water and fat molecules (Mendiguchia et al., 2013). Although the use of MRI can be difficult to justify in certain circumstances due to its high cost and limited accessibility, this technique allows for a non-invasive, high resolution spatial assessment of muscular characteristics without exposure to potentially harmful ionizing radiation (Franchi et al.,2017). As a consequence, MRI overcomes many of the spatial limitations associated with ultrasound imaging (Trezise et al., 2016; Blazevich et al., 2009); however, it does not allow for an assessment of muscle architecture. Nevertheless, MR-derived assessments of quadriceps muscle volume have consistently been shown to be a reliable indicator of a knee extension torque production capability (Blazevich et al., 2009). Further, several studies have employed MRI to map the distribution of muscle size in athletes and determine its association with performance (Mendiguchia et al., 2013; Akima et al., 2000; Kinugasa et al., 2006).

2.8 Summary

The ability to accelerate optimally and reach high sprinting velocities is an important determinant of individual and team success in rugby league (Till et al., 2017; Gabbett 2006; De Lacey et al., 2014). Faster players are better able to create and tolerate high impact forces, enhance ball carrying to gain line, increase ball playing speed, and may increase the number of try scoring opportunities (Till et al., 2017: Gabbett et al., 2011). Sprint performance is ultimately determined by the interaction of stride rate and stride length with the ability to effectively apply horizontal and vertical force further influencing acceleration and maximal sprint velocity (Hunter et al., 2004; Rabita et al., 2015; Nagahara et al., 2018[A]; Nagahara et al., 2018 [B]). Increases in lower limb muscular strength appear to positively influence sprint performance (Akima et al., 2000; Trezise et al., 2016; Comfort et al., 2012), and a muscle's force generating capacity is directly related to its size. However, uniform lower limb hypertrophy is likely disadvantageous to acceleration and maximal sprint speed due to changes that occur in the moment of inertia, which would increase the work required to accelerate and decelerate the lower limbs (Handsfield et al., 2017; Sugisaki et al., 2018; Hoshikawa et al., 2006). It has been proposed that non-uniform increases in lower limb muscle mass, particularly of proximal hip- and knee-spanning muscles, may be an important determinant of improved sprint performance (Miller et al., 2020, Sugisaki et al., 2011; Nuell et al., 2020). While the relationship between muscle morphology and sprint performance has previously been investigated (Miller et al., 2020; Sugisaki et al., 2018), these investigations have typically been confined to specialist track and field athletes. The spatial patterns of lower limb muscle size and the contributions of peak CSA and muscle volume to 10-m sprint times, 30-m sprint times and peak sprinting velocities have yet to be explored in rugby league athletes.

3.0 Methods

3.1 Participants

Nineteen male rugby-league players (mean ± SD, age: 19.2 ± 0.7 years; height: 180.7 ± 5.6 cm; body mass 89.9 ± 10.0 kg) participated in this study. All participants were from the same elite development (i.e., youth) system within an Australian National Rugby League (NRL) club. All participants were involved in full training and competition, had at least four years of resistance training experience, and had no history of lower-limb injury in the prior twelve months. Prior to inclusion, all participants completed a cardiovascular risk factor questionnaire to ensure that it was safe for them to participate in intense exercise, in addition to a MRI metals checklist with a qualified radiographer to ensure it was safe for them to undergo this type of medical imaging. All participants provided written informed consent prior to participating in this study, which was approved by the Griffith University Human Research Ethics Committee (GU Ref No: 2017/344).

3.2 Experimental Design

This study was conducted after the 2019 National Rugby League preseason. At a single time-point, participants underwent MRI of their lower limbs to enable calculation of muscle volumes and anatomical CSAs (ACSAs) of the major hip, knee and ankle-spanning muscles. In a separate session 48 hours later, participants completed a series of 30-m maximal sprints on a flat grass oval which were timed using dual-beamed timing gates.

3.3 Magnetic Resonance Imaging (MRI)

Axial T1-weighted 3-dimensional (3D) fast field echo (FFE) sequences were acquired using a 3-T MRI scanner (Philips Medical Systems) from the level of the iliac crest to the ankle spanning both legs while the participant lay supine in the scanner. The images were acquired in 5 stations with ~210 slices per station and a 10 mm overlap between stations. Slice thickness was 2.0 mm, inter-slice gap was 0.0 mm, repetition time was 4.1 ms, echo time 1 was 1.44 milliseconds, and echo time 2 was 2.60 ms. The CSAs of the major lower limb muscles for both legs were determined at predefined lengths of 20, 30, 40, 50, 60, 70 and 80% of the total muscle length by manually tracing the margin of the respective muscle in the relevant axial slices using image analysis software (Mimics, version 17; Materialise). The analysed muscles were the GMAX, gluteus medius (GMED) and minimus (GMIN), TFL, SART, iliopsoas (ILIOP), GR, adductor magnus, longus and brevis (ADD), RECF, VL, VI, VM, semimembranosus (SM), ST, biceps femoris long head (BFLH), biceps femoris short head (BFSH), SOL, gastrocnemius medialis (GM) and gastrocnemius lateralis (GL). The identification of the borders between adductor magnus, longus and brevis was difficult; therefore, these muscles were analysed as a muscle group and referred to as adductors (ADD). This method of forming the ADD muscle group is supported by Sugisaki et al. (2018) who had analysed adductor magnus, longus and brevis as a single variable in order to identify a relationship between lower limb muscle volume and 100-m sprint times. All segmentations were performed by two members of the research team (Figure 6 to 12). Muscle volume was calculated under the assumption that the muscle is an ideal cylinder whereby the known distance between slices was multiplied by the sum of the CSA of the total number of traced slices. This methodology (i.e., using 7 slices) has been shown to have high levels of agreement (i.e., differences of <0.005%) with muscle volume calculations using 50 slices (Lund et al., 2002). In order to reduce effects of body size on muscle size differences, we normalised

muscle volume by the height-mass product (Hansfield et al., 2017). Peak CSA for each muscle was determined as the slice with the largest CSA among sites located at 20, 30, 40, 50, 60, 70 and 80% of the length of the muscle. No significant differences in peak CSA or normalised volume were detected between the left and right leg for any muscle; therefore, individual muscle values from both legs were summed for all analyses. The inter-rater reliability of the normalised muscle volume of the ST, SM, BFLH, BFSH, GR, ADD, GMAX, GMED and GMIN was excellent (mean ICC = 0.973, CV = 2.0%).

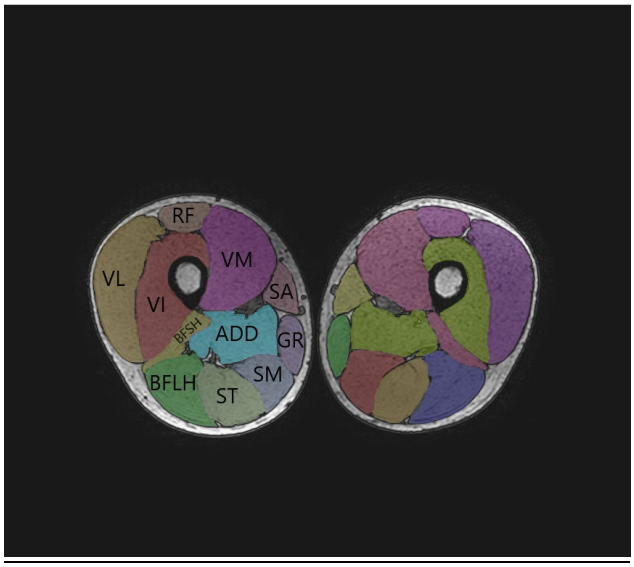


Figure 6. Typical T1-weighted magnetic resonance image (MRI) displaying the cross-sectional areas (CSAs) of the biceps femoris long head (BFLH), biceps femoris short head (BFSH), semitendinosus (ST), semimembranosus (SM), vastus intermedius (VI), vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF), adductors (ADD), gracilis (GR), and sartorius (SA).

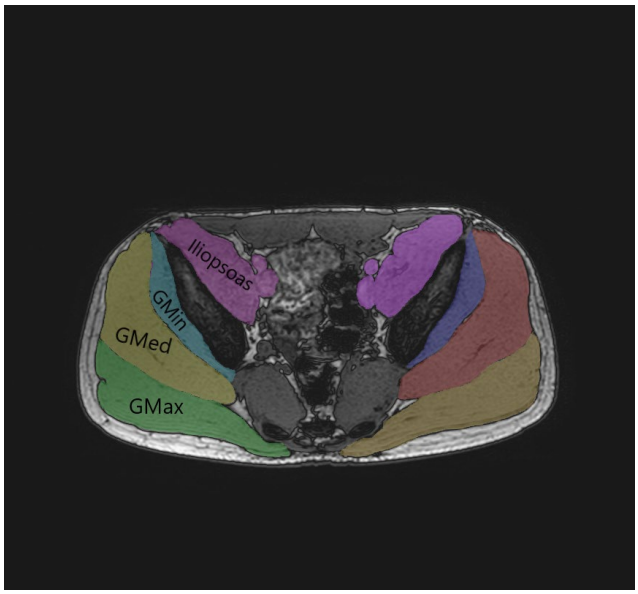


Figure 7. Typical T1-weighted magnetic resonance image (MRI) displaying the cross-sectional areas (CSAs) of the gluteus maximus (GMAX), gluteus medius (GMED), gluteus minimus (GMIN), and iliopsoas (ILIOP).

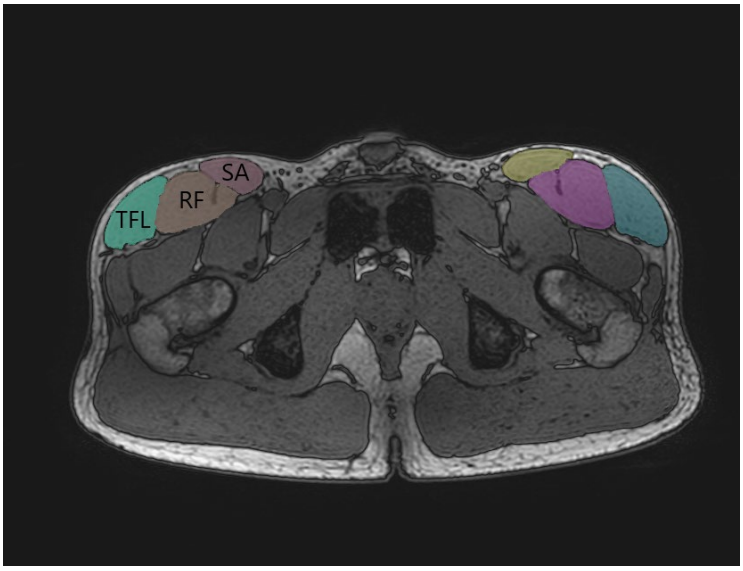


Figure 8. Axial MR images tensor fascia latae (TFL), Rectus Femoris (RF), Sartorius (SA).

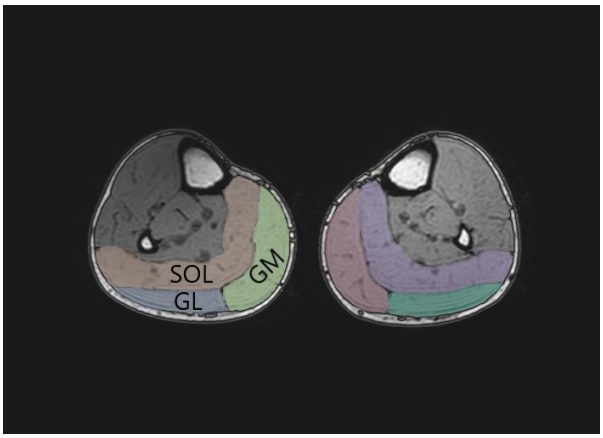


Figure 10. Axial MR images of gastrocnemius lateral head (GL), gastrocnemius medial head (GM), soleus (SOL).

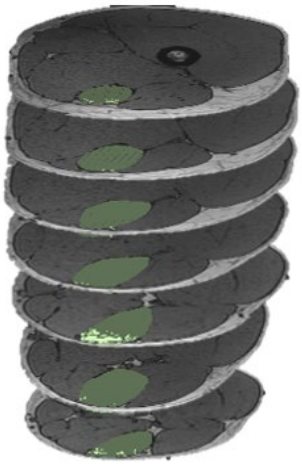


Figure 11. Segmented axial slices of semitendinosus used to calculate normalised muscle volume.

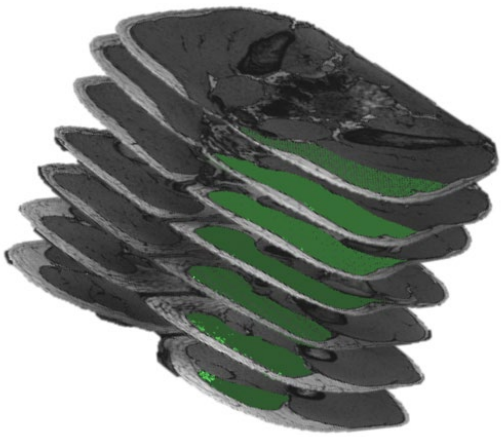


Figure 12. Segmented axial slices of gluteus maximus used to calculate normalised muscle volume.

3.4 Sprint Performance

All over-ground sprint testing was completed on the same well-maintained natural grass surface. Participants were required to wear sprigged training shoes and suitable training attire. Each participant performed a 15-min on-field dynamic warm-up protocol which closely reflected the warm-up used before field-based training sessions. Participants performed two linear 30-m sprints and sprint times were recorded by infrared timing gates (Smartspeed, Fusion Sport, Australia) with split times every 5-m. The two 30-m sprints were interspersed with 5 minutes recovery in order to minimise fatigue. All starts commenced from a static position and the upper body of each participant was positioned as close as possible to the inter-gate beam of the first timing gate which was placed on the starting line. The timing gates at the start line and 5 m line were mounted on separate tripods 1.00/1.20 m above the ground level, while the remaining gates were mounted

1.30/1.50 m above the ground level. The mean split times from both sprints were used for all analyses in order to reduce the typical error associated with using a single sprint (Haugen & Buchheit, 2016). This starting procedure and the use of infrared timing gates have been shown to provide a reliable measure for sprint times when compared to other commonly used methods (Earp & Newton 2012; Duthie et al., 2006).

3.5 Statistical Analysis

All statistical analyses were undertaken using SPSS version 25 (Chicago, USA). The values presented are expressed as mean ± SD and range unless otherwise stated. The Shapiro-Wilk test was used to assess the distribution of normality amongst the variables. Relationships between individual muscle characteristics (normalised volumes and peak anatomical CSAs) and performance variables (10-m and 30-m sprint times and maximal velocity) were explored using Pearson's product-moment correlation coefficients in order to indicate the magnitude and direction of univariate associations for normally distributed variables. Spearman's Rho was used to examine the magnitude and direction of univariate associations for non-normally distributed variables. Muscular characteristics and performance variables were subsequently entered into stepwise multiple linear regression models to explore which combinations of muscle characteristics explained the most variation in sprint performance. The Variance Inflation Factor (VIF) was used to assess the multi-collinearity between the independent variables with a value of <5 being acceptable. A two-tailed P-value <0.05 was considered to be statistically significant.

3.6 Results

The peak CSA values ranged from 11.31cm^2 to 147.25cm^2 (Table 1 and Figure 13). The normalised muscle volumes ranged from 1.14 cm^3·kg·m to 17.26 cm^3·kg·m (Table 1 and Figure 14).10-m sprint times ranged from 1.39s to 1.70s, 30-m sprint times ranged from 3.69s to 4.21s, and peak sprinting velocity ranged from 8.18m.s^{-1} to 9.80m.s^{-1} (Table 2).

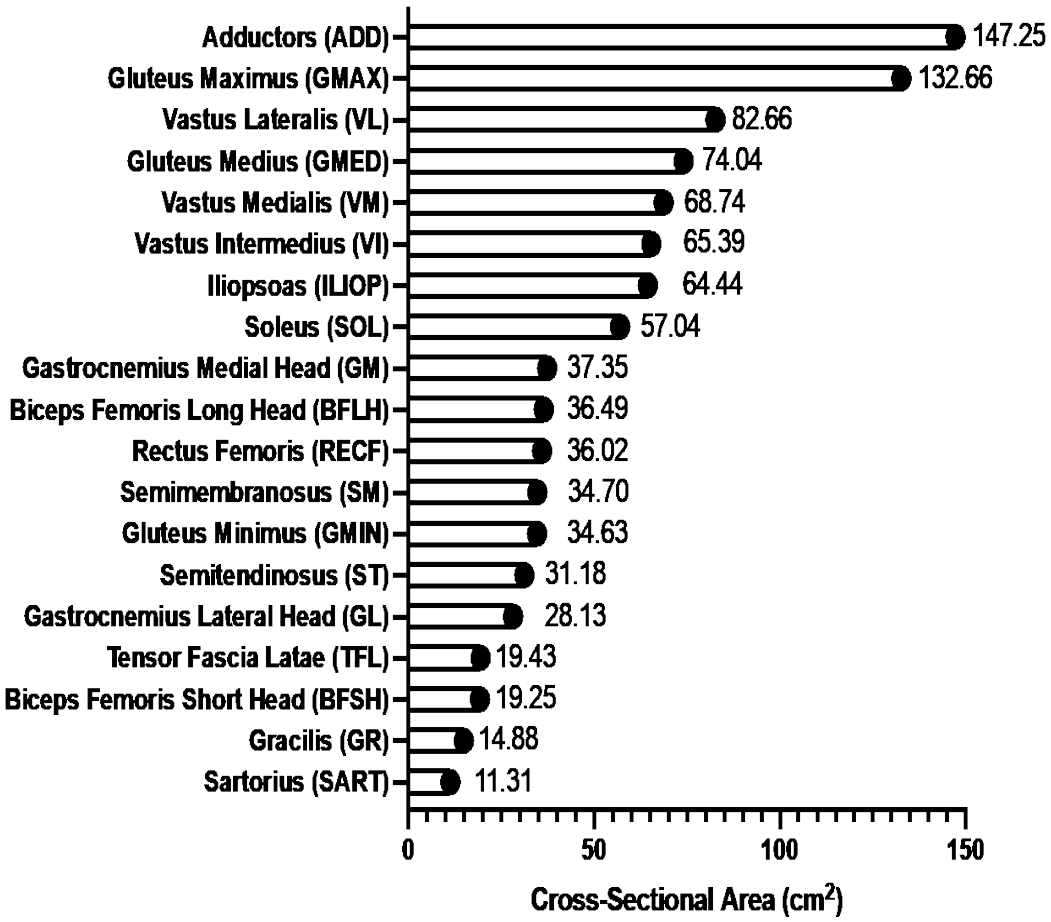


Figure 13. Average peak cross-sectional area (CSA) of the lower limb muscles. Values represent the sum of the left and right limb.

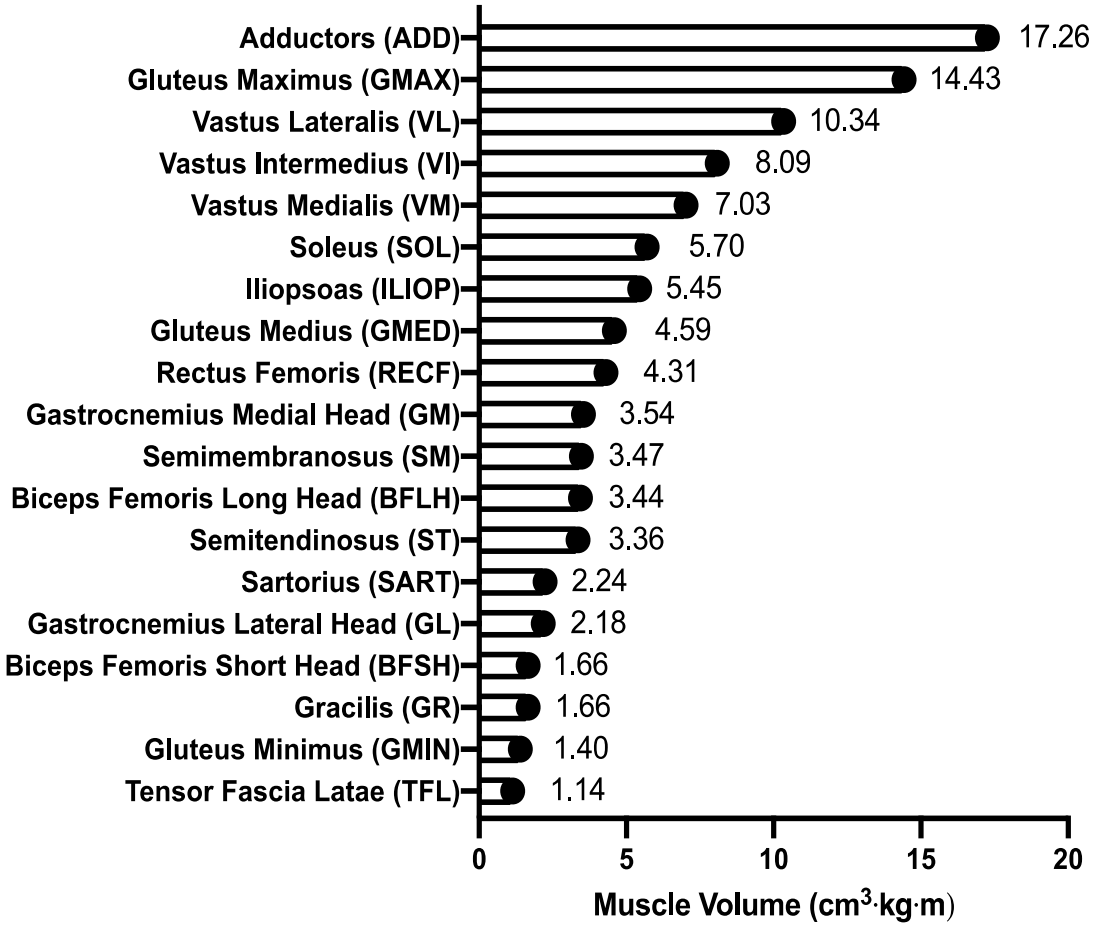


Figure 14. Average muscle volumes for the lower limb, normalised to the height-mass product. Values represent the sum of the left and right limbs.

Table 1. Normalised muscle volumes and peak cross-sectional areas (CSAs) for the lower limb. Values represent the sum of the left and right limb.

Variable	Normalised Volume Mean ± SD	Normalised Volume Range	Peak CSA Mean ± SD	Peak CSA Range
Semimembranosus (SM)	3.47 (0.57)	2.27 – 4.51	34.70 (4.41)	27.20 – 43.24
Semitendinosus (ST)	3.36 (0.44)	2.21 – 3.96	31.18 (4.69)	23.57 – 40.62
Biceps Femoris Long Head (BFLH)	3.44 (0.44)	2.86 – 4.22	36.49 (4.28)	31.82 – 46.13
Biceps Femoris Short Head (BFSH)	1.66 (0.24)	1.33 – 2.15	19.25 (1.79)	15.41 – 22.38
Gracilis (GR)	1.66 (0.19)	1.29 – 2.16	14.88 (2.55)	12.45 – 21.18
Gluteus Maximus (GMAX)	14.43 (1.05)	13.29 – 16.22	132.66 (13.13)	111.93 – 169.32
Gluteus Medius (GMED)	4.59 (0.56)	3.56 – 5.64	74.04 (7.74)	63.88 – 90.28
Gluteus Minimus (GMIN)	1.40 (0.30)	0.74 – 1.86	34.63 (4.07)	28.99 – 44.09
Adductors (ADD)	17.26 (1.53)	15.36 – 20.46	147.25 (15.62)	123.40 – 175.55
Rectus Femoris (RF)	4.31 (0.31)	3.82 – 4.95	36.021 (3.98)	28.82 – 44.55
Vastus Lateralis (VL)	10.34 (0.99)	9.24 – 13.04	82.66 (6.53)	71.83 – 95.43
Vastus Medialis (VM)	7.03 (0.83)	5.73 – 8.52	68.74 (6.63)	60.24 – 86.81
Vastus Intermedius (VI)	8.09 (1.29)	5.23 – 10.35	65.39 (6.48)	56.47 – 77.98
Tensor Fascia Lata (TFL)	1.14 (0.23)	0.81 – 1.63	19.43 (4.08)	15.26 – 28.93
Sartorius (SART)	2.24 (0.40)	1.57 – 3.06	11.31 (1.89)	8.32 – 16.23
Iliopsoas (ILIOP)	5.45 (0.87)	4.10 – 6.91	64.44 (6.33)	55.24 – 78.03
Soleus (SOL)	5.70 (0.64)	4.85 – 7.28	57.04 (7.53)	46.91 – 69.99
Gastrocnemius Medial Head (GM)	3.54 (0.51)	2.60 – 4.47	37.35 (6.49)	21.43 – 49.59
Gastrocnemius Lateral Head (GL)	2.18 (0.43)	1.52 – 2.82	28.13 (3.88)	22.53 – 35.43

Muscle volumes are normalised to the height-mass product and reported in $cm^3 \cdot kg \cdot m$.
Peak cross-sectional area (CSA) values are reported in cm^2.

Table 2. Values for 10-m sprint times, 30-m sprint times and peak sprinting velocity (m.s-1)

Variable	Mean ± SD	Range
10-m sprint time (s)	1.55 (0.84)	1.39 – 1.70
30-m sprint time (s)	3.97 (0.15)	3.69 - 4.21
Peak Velocity ($m.s^{-1}$)	9.05 (0.48)	8.18 – 9.80

Sprint times reported in seconds (s). Peak velocity reported in metres per second ($m.s^{-1}$)

Table 3. Pearson's correlations for peak cross-sectional area (CSA) (cm^2) of the lower limb muscles with 10-m sprint times, 30-m sprint times and peak velocity ($m.s^{-1}$)

Variable	30-m Sprint time (r)	P Value	10-m Sprint Time (r)	P value	Peak Velocity (r)	P value
Peak CSA (cm^2)						
Semimembranosus (SM)	-0.096	0.713	-0.195	0.452	0.052	0.843
Semitendinosus (ST)	0.231	0.373	0.100	0.702	-0.043	0.870
Biceps Femoris Long Head (BFLH)	-0.520*	0.032*	-0.570*	0.017*	0.465	0.060
Biceps Femoris Short Head (BFSH)	-0.474	0.054	-0.451	0.069	0.155	0.553
Gluteus Maximus (GMAX)	-0.174	0.505	-0.377	0.136	-0.036	0.892
Gluteus Medius (GMED)	-0.074	0.778	-0.117	0.655	0.108	0.108
Gluteus Minimus (GMIN)	-0.277	0.282	-0.320	0.210	0.555*	0.021
Adductors (ADD)	-0.573*	0.016*	-0.768**	0.000**	0.268	0.298
Rectus Femoris (RF)	-0.170	0.530	-0.410	0.115	-0.156	0.564
Vastus Lateralis (VL)	-0.207	0.442	-0.395	0.130	0.109	0.687
Vastus Intermedius (VI)	-0.431	0.096	-0.412	0.112	0.412	0.112
Sartorius (SART)	-0.466	0.069	-0.467	0.068	0.158	0.558

Iliopsoas (ILIOP)	-0.154	0.568	-0.116	0.670	-0.085	0.754
Soleus (SOL)	-0.135	0.618	-0.074	0.785	-0.047	0.862
Gastrocnemius Medial Head (GM)	-0.170	0.528	-0.264	0.324	0.132	0.625
Gastrocnemius Lateral Head (GL)	0.351	0.183	0.360	0.171	-0.391	0.135
Gracilis (GR) ≠	-0.331	0.195	-0.459	0.064	0.223	0.390
Vastus Medialis (VM) ≠	-0.053	0.845	-0.362	0.168	0.021	0.938
Tensor Fascia Latae (TFL) ≠	-0.331	0.210	-0.433	0.094	0.171	0.528

**. Correlation is significant at the p<0.01 level
*. Correlation is significant at the p<0.05 level
≠. Spearman correlation for non-normally distributed variables

Table 3. displays the results of the univariate analyses exploring the association between peak CSA and 10-m and 30-m sprint times as well as peak sprint velocity. BFLH ($r = -0.520$, $p = 0.032$) and ADD peak CSA ($r = -0.573$, $p = 0.016$) showed significant moderate to large negative correlations with 30-m sprint times. Moderate to large negative correlations were also observed for BFLH ($r = -0.570$, $p = 0.017$) and ADD peak CSA ($r = -0.768$, $p < 0.001$) with 10-m sprint times. GMIN peak CSA was the only variable to demonstrate a significant positive correlation with peak velocity ($r = 0.555$, $p = 0.021$).

The stepwise multiple linear regression revealed that ADD peak CSA was able to best explain the variation in 10-m sprint times (r^2 = 0.582, p = 0.005) (Table 4 and Figure 15). The combination of ADD, VI and VM peak CSA were able to best explain the variation in 30-m sprint times (r^2 = 0.751, p = 0.030) (Table 5 and Figure 16).

Table 4. Multiple linear regression model for the relationship between peak cross-sectional area (cm^2) of the lower limb muscles and 10-m sprint times

Model 1:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
ADD Peak CSA	0.582	-0.004 (-0.006 to -0.002)	0.005

Adductors (ADD).

Table 5. Multiple linear regression model for the relationship between peak cross-sectional area (cm^2) of the lower limb muscles and 30-m sprint times

Model 3:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
ADD Peak CSA		-0.010 (-0.013 to -0.006)	
VM Peak CSA		0.016 (0.008 to 0.025)	
VI Peak CSA	0.751	-0.008 (-0.014 to -0.001)	0.030

Adductors (ADD), vastus medialis (VM), vastus intermedius (VI)

Table 6 and Figure 17 display the results of the stepwise multiple linear regression model for peak sprinting velocity and peak CSA. The combination of GMIN and GL peak CSA best explained the variance in peak sprinting velocity (adjusted r^2 = 0.445, p = 0.029).

Table 6. Multiple linear regression model for the relationship between peak cross-sectional area (cm^2) of the lower limb muscles and peak velocity ($m.s^{-1}$)

Model 2:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
GMIN Peak CSA		0.064 (0.020 to 0.108)	
GL Peak CSA	0.445	-0.053 (-0.100 to -0.007)	0.029

Gluteus minimus (GMIN), gastrocnemius lateralis (GL).

The univariate analyses displayed in Table 7 show the correlations for 10-m and 30-m sprint times as well as peak sprint velocity with muscle volume. Significant moderate to large negative correlations were seen for BFLH (r = -0.536, p = 0.027), ADD (r = -0.533, p = 0.028), VI (r = -0.509, p = 0.044), TFL (r = -0.688, p = 0.005) and ILIOP (r = -0.599, p = 0.014) muscle volume with 10-m sprint times. Further, significant moderate to large negative correlations were observed for BFLH (r = -0.654, p = 0.004), ADD (r = -0.564, p = 0.018), VI (r = -0.659, p = 0.005), TFL (r = -0.613, p = 0.012), SART (r = -0.550, p = 0.027) and ILIOP (r = -0.753, p = 0.001) muscle volume with 30-m sprint times. The muscle volumes of VI (r = 0.590, p = 0.016) and ILIOP (r = 0.648, p = 0.007) demonstrated significant positive correlations with peak sprinting velocity.

Table 7. Pearson's correlations for normalised muscle volume with 30-m and 10-m sprint times and peak sprint velocity ($m \cdot s^{-1}$).

Variable	30-m Sprint Time (r)	P value	10-m Sprint Time (r)	P Value	Peak Velocity (r)	P value
Muscle volume ($cm^3 \cdot kg\text{-}m$)						
Semimembranosus (SM)	-0.284	0.270	-0.082	0.754	0.184	0.481
Biceps Femoris Long Head (BFLH)	-0.654**	0.004	-0.536*	0.027	0.308	0.228
Biceps Femoris Short Head (BFSH)	-0.331	0.194	-0.073	0.782	0.012	0.964
Gracilis (GR)	-0.304	0.235	-0.151	0.563	0.146	0.575
Gluteus Maximus (GMAX)	-0.224	0.388	-0.254	0.325	0.018	0.944
Gluteus Medius (GMED)	-0.049	0.851	0.138	0.597	0.180	0.490
Gluteus Minimus (GMIN)	-0.021	0.657	0.012	0.962	0.477	0.053
Adductors (ADD)	-0.564*	0.018	-0.533*	0.028	0.383	0.129
Rectus Femoris (RF)	-0.335	0.205	-0.385	0.141	-0.242	0.367
Vastus Lateralis (VL)	0.037	0.890	-0.102	0.706	-0.259	0.332
Vastus Medialis (VM)	-0.138	0.609	-0.425	0.101	-0.162	0.549
Vastus Intermedius (VI)	-0.659**	0.005	-0.509*	0.044	0.590*	0.016*

Tensor Fascia Lata (TFL)	-0.613*	0.012	-0.688**	0.005	0.403	0.122
Sartorius (SART)	-0.550*	0.027	-0.486	0.056	0.117	0.666
Iliopsoas (ILIOP)	-0.753**	0.001	-0.599*	0.014	0.648**	0.007**
Gastrocnemius Medial Head (GM)	-0.339	0.199	-0.283	0.289	0.287	0.281
Gastrocnemius Lateral Head (GL)	-0.002	0.996	0.094	0.729	-0.216	0.421
Soleous (SOL) ≠	-0.187	0.489	-0.165	0.540	-0.147	0.586
Semitendinosus (ST) ≠	0.152	0.561	0.234	0.366	0.006	0.981

**. Correlation is significant at the 0.01 level.
*. Correlation is significant at the 0.05 level.
≠. Spearman Correlation for non-normally distributed variables.

The multiple linear regression models which can be seen in Table 8/Figure 15 and Table 9/Figure 16 show the combination of muscle volumes which best explain 10-m and 30-m sprint times, respectively. The variance in 10-m sprint times could be best explained by the combination of VI and TFL muscle volume ($r^2 = 0.570$, $p < 0.05$). The combination of ILIOP and TFL muscle volume explained most of the variance in 30-m sprint times ($r^2 = 0.672$, $p < 0.05$).

Table 8. Multiple linear regression model for the relationship between normalised muscle volume and 10-m sprint times

Model 2:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
Normalised TFL		-0.233 (-0.374 to -0.093)	
Normalised VI	0.570	-0.029 (-0.54 to -0.004)	0.026

Table 9. Multiple linear regression model for the relationship between normalised muscle volume and 30-m sprint times

Model 2:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
Normalised ILIOP		-0.103 (-0.159 to -0.046)	
Normalised TFL	0.672	-0.262 (-0.478 to -0.045)	0.021

Table 10. Multiple linear regression model for the relationship between normalised muscle volume and peak sprinting velocity ($m.s^{-1}$)

Model 4:

Independent Variables	Adjusted r^2	Regression Coefficient (95% CI)	p
ILIOP Volume		0.422 (0.285 to 0.558)	
GL Volume		-0.392 (-0.669 to -0.114)	
RECF Volume		-0.692 (-1.065 to -0.320)	
ADD Volume	0.820	0.082 (0.007 to 0.158)	0.035

The muscle volumes that could best explain the variance in peak sprinting velocity can be seen in Table 10 and Figure 17. The combination of ILIOP, GL, RECF and ADD muscle volumes best explained the variance in peak sprinting velocity (r^2 = 0.820, p = 0.035).

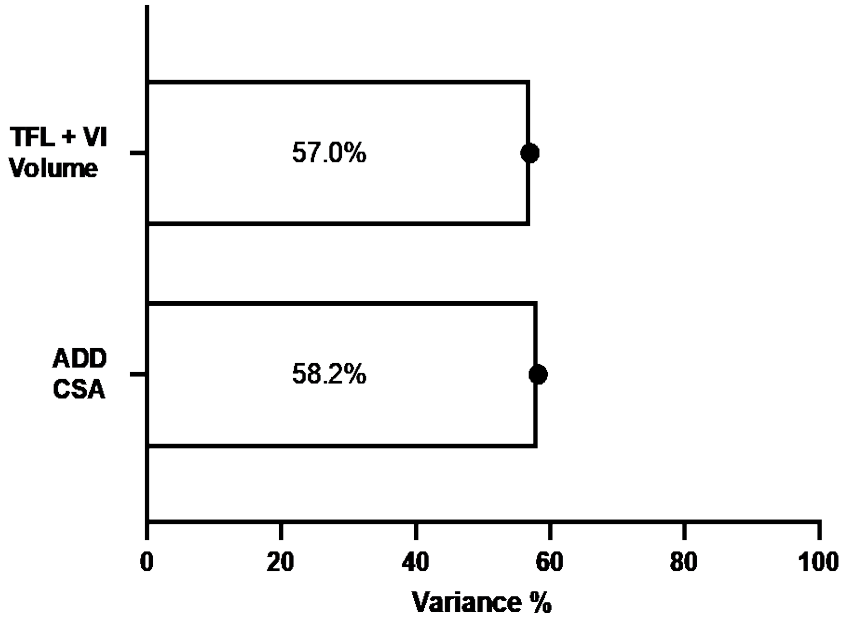


Figure 15. Muscular determinants explaining the variance in 10-m sprint times. Bars indicate the magnitude of explained variance (% r^2) obtained from multiple linear regression analyses with the combination of predictors presented on the Y axis. Tensor fascia latae (TFL), vastus intermedius (VI), adductors (ADD).

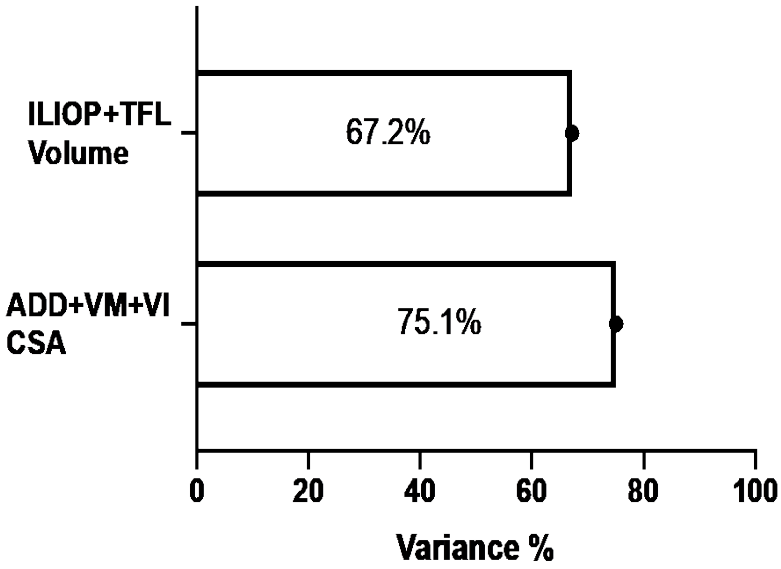


Figure 16. Muscular determinants explaining the variance in 30-m sprint times. Bars indicate the magnitude of explained variance (% r^2) obtained from multiple linear regression analyses with the combination of predictors presented on the Y axis. Iliopsoas (ILIOP), tensor fascia latae (TFL), adductors (ADD), vastus medialis (VM), vastus intermedius (VI).

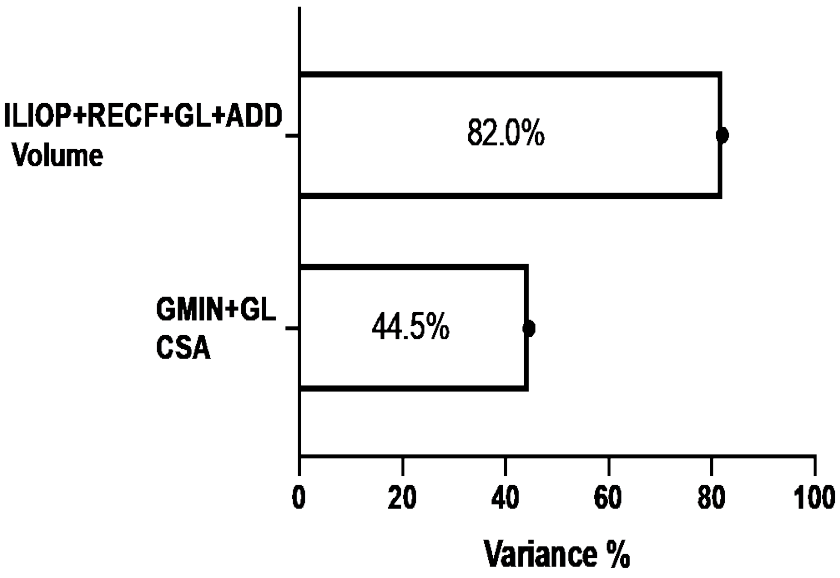


Figure 17. Muscular determinants explaining the variance in peak sprint velocity ($m.s^{-1}$). Bars indicate the magnitude of explained variance (% r^2) obtained from multiple linear regression analyses with the combination of predictors presented on the Y axis. Iliopsoas (ILIOP), rectus femoris (RECF), gastrocnemius lateral head (GL), adductors (ADD), gluteus minimus (GMIN).

4.0 Discussion

To the authors' knowledge, this study is the first to map the spatial patterns of lower limb muscle size in male rugby league athletes and determine if regional differences in muscle volume and peak CSA are associated with 10-m and 30-m sprint times and peak sprinting velocity ($m \cdot s^{-1}$). The major findings were that i) rugby league athletes display heterogenous patterns of lower limb muscle size that are similar to what has been observed in other running-based athletes; ii) ADD peak CSA and the normalised muscle volumes of TFL and VI were associated 10-m sprint times; iii) ADD, VM and VI peak CSA and the normalised muscle volumes of ILIOP and TFL were associated with 30-m sprint times and; iv) the peak CSA of GMIN and GL and the normalised muscle volumes of ILIOP, RECF, GL and ADD were associated with peak sprinting velocity ($m \cdot s^{-1}$). These data suggest that preferential hypertrophy of certain proximal hip- and knee-spanning muscles may have important implications for improving sprint acceleration and maximal velocity performance in semi-professional rugby league players.

4.1 Spatial Patterns of Lower Limb Muscle Size

The current study suggests that rugby league athletes display a unique distribution of lower limb muscle size whereby the ADD (17.26 $cm^3 \cdot kg \cdot m$) > GMAX (14.43 $cm^3 \cdot kg \cdot m$) > VL (10.34 $cm^3 \cdot kg \cdot m$) > VI (8.09 $cm^3 \cdot kg \cdot m$) > VM (7.03 $cm^3 \cdot kg \cdot m$) > all other muscles (1.14 – 5.70$cm^3 \cdot kg \cdot m$). In comparison, Handsfield et al. (2013) reported that the individual muscle volumes for recreationally active participants were greatest for GMAX (6.01 $cm^3 \cdot kg \cdot m$) > VL (5.89 $cm^3 \cdot kg \cdot m$) > adductor magnus (3.64 $cm^3 \cdot kg \cdot m$), adductor longus (1.19 $cm^3 \cdot kg \cdot m$) and adductor brevis (0.622 $cm^3 \cdot kg \cdot m$) (for a combined total of 5.45 $cm^3 \cdot kg \cdot m$) > VM (3.03 $cm^3 \cdot kg \cdot m$) > SOL (2.57 $cm^3 \cdot kg \cdot m$) > psoas (2.43 $cm^3 \cdot kg \cdot m$), with all other muscles ranging from 0.120 to 2.17 $cm^3 \cdot kg \cdot m$. These data suggest that rugby league athletes display greater normalised muscle volumes and may exhibit unique spatial patterns of lower limb muscle size (i.e., preferential hypertrophy of proximal hip- and knee-spanning muscles) in comparison to healthy non-trained individuals. Previous investigations have highlighted that it may be advantageous to have larger proximal but not distal lower limb muscles to optimise sprint performance (Hoshikawa et al., 2006; Sugisaki et al., 2018). For example, Handsfield et al. (2017) demonstrated that limb muscle size per height-mass product was 22% greater in sprinters than non-sprinters, with all but one of the larger muscles crossing the hip or knee. In this study (Handsfield et al., 2017), the largest differences between sprinters and non-sprinters were observed for the ST (54%), GR (42%), TFL (41%), RECF (40%), SART (37%), GMAX (31%) and VM (30%). Although this earlier work (Handsfield et al., 2017) reported the average muscle volumes of the two lower limbs and the current study summed the volumes from the left and right limbs, the distribution of muscle size appears similar between studies. For example, GMAX (9.0 $cm^3 \cdot kg \cdot m$), VL (8.5 $cm^3 \cdot kg \cdot m$), adductor magnus (5.8 $cm^3 \cdot kg \cdot m$), and VM (4.6 $cm^3 \cdot kg \cdot m$) were the largest muscle volumes for elite sprinters (Handsfield et al., 2017). The distribution of muscle size may also differentiate between performance

levels in the same sport. For example, Miller and colleagues (2020) demonstrated that the TFL, SART and GMAX muscles were 45-57% larger in elite sprinters (season best: $10.10 \pm 0.07s$) than sub-elite sprinters (season best: $10.80 \pm 0.30s$), whereas no difference was observed in the size of the plantar flexors between groups. Compared to track and field athletes, sprints that are performed in field-based sports such as rugby league, may last as brief as 2 seconds which emphasises the need to generate high rates of force development (Lockie et al., 2013). As a large portion of rugby league match-play requires athletes to generate momentum in order to create and tolerate the large collisions made (Till et al., 2017: Gabbett et al., 2011), the muscles which are primarily responsible for power generation and further aiding the ability to accelerate and reach peak sprinting velocity sooner, may be different in rugby league players.

The contribution of muscle CSA and volume to sprint performance has been examined in several studies (Sugisaki et al., 2011; Handsfield et al., 2017; Hoshikawa et al., 2018). Muscle CSA is associated with measures of maximal isometric and concentric torque (Trezise et al., 2016; Blazevich et al., 2009), while muscle volume is theoretically proportional to its power generating capacity (Lieber & Friden 2000). As joint torque is determined by the product of muscle force and moment arm length, increases in muscle size would presumably increase the capacity to develop joint torque, which may have implications for enhancing performance and reducing injury risk (Akagi et al., 2009; Fukunaga et al., 2001; Blazevich et al., 2009; Trezise et al., 2016). The findings of the present study suggest that sub-elite rugby league players are characterised by relatively large proximal but not distal lower limb muscles. Whether these spatial patterns of muscle size confer a sports-specific advantage via influencing the rate and magnitude of lower limb force development remains unclear and should be a focus of future work.

4.2 Association Between Lower Limb Muscle Size and 10-m Sprint Performance

The ability to rapidly accelerate across distances ≤10-m is an important performance criterion in rugby league (Till et al., 2017). In the present study, ADD peak CSA was able to explain 58.2% of the variance in 10-m sprint times. These findings are at least partly supported by previous investigations (Sugisaki et al., 2011). For example, Sugisaki et al. (2011) reported that in a group of male college short and middle-distance runners, ADD CSA was the only variable to show a positive effect on 30-m sprint times. It was suggested that due to the large degree of hip flexion that occurs during the early phase of acceleration, the moment arm length of the ADD may provide a mechanical advantage over other hip extensor muscles (Sugisaki et al., 2011; Yasuda 2019). The findings of Sugisaki et al. (2011) emphasise the role of the ADDs as hip extensors and thus their ability to greatly influence horizontal force production. We also observed associations between peak CSA of the BFLH (r = -0.570) and 10-m sprint times. In support of this, Morin et al. (2015[A]) identified that the combination of eccentric isokinetic knee flexor torque and BFLH EMG activity during the terminal-swing phase of sprinting, explained 49% of the variance in horizontal ground reaction force during 6-s sprints on a motorised treadmill. Although we did not measure EMG activity or muscle strength in this study, a larger BFLH muscle, with presumably greater force generating capacity, may be better able to decelerate the forward swinging shank during the late swing phase of running, thereby contributing to a greater stride rate (Alt et al., 2021). High levels of BFLH and ADD force development would also be expected to lead to an enhanced hip extensor moment upon ground contact (Howard et al., 2018; Morin et al., 2015[A]), which may

contribute to greater stride lengths (Dorn et al., 2012: Rabita et al., 2015); however, future biomechanical studies are needed to clarify these hypotheses.

When examining measures of muscle volume, the TFL and VI muscles were able to explain 57% of the variance in 10-m sprint times (r^2 = 0.570, p = 0.026). Negative associations were also observed between the volume of ILIOP (r = -0.599), BFLH (r = -0.536) and ADD (r = -0.533) and 10-m sprint performance. Fine-wire EMG analyses reveal that forward propulsion during sprinting is primarily due to increased hip flexor and knee extensor activity (Montgomery et al., 1994). In support of this, Miller et al. (2020) identified that TFL muscle volume was one of three individual muscles that differentiated elite sprinters from their sub-elite counterparts. Further, psoas major CSA to quadriceps femoris CSA ratio was shown to positively influence 100-m sprint performance in a group of junior sprinters (Hoshikawa et al., 2006). Acceleration in the early phase of overground sprinting is influenced to a greater extent by increases in stride length than stride rate, with stride rate reaching its maximum value by the fourth step while stride length reaches is maximum value by the twenty-fourth step (Nagahara et al., 2014). Theoretically, larger hip flexor muscles (i.e., TFL and ILIOP) may enable athletes to generate more hip flexor power during the swing phase of running which may allow for longer strides (Hoshikawa et al., 2006; Miller et al., 2020). In support of this, Dorn et al. (2012) demonstrated that ILIOP makes a significant contribution to increasing stride rate with faster sprint speeds. In addition to contributing to propulsive knee extensor forces, the VI appears to serve a unique role in decelerating knee flexion during the early swing phase of sprinting (Montgomery et al., 1994), which may explain its association with 10-m sprint times (Nagahara et al., 2014; Nagahara et al., 2018[A]). It remains unclear why other knee extensors were not also associated with 10-m sprint performance;

however, an increase in total limb muscle mass would result in an increase in inertia which may have a negative influence on sprint performance (Hoshikawa et al., 2006; Handsfield et al., 2017). Evidence that VI but not VL, VM, or RECF muscle volume was associated 10-m sprint time supports the hypothesis that preferential hypertrophy of certain muscle(s) within a muscle group may enhance sprint performance (Hoshikawa et al., 2006; Handsfield et al., 2017). Minimising ground contact time and effectively applying ground reaction force are two variables that are known to determine acceleration and maximal sprint speed (Wilkau et al., 2020; Nagahara et al., 2018[B]; Lockie et al., 2013). Collectively, the findings of the current study suggest that peak CSA of the ADD and the combination of TFL and VI muscle volume best predict 10-m sprint times. These data provide valuable insight into the muscular characteristics required for sub-elite rugby league players to accelerate optimally over short distances. Future studies are needed to determine whether interventions targeted at promoting hypertrophy in the aforementioned muscles lead to improvements in sprint acceleration performance.

4.3 Association Between Lower Limb Muscle Size and 30-m Sprint Performance

Although it has previously been shown that 67.5% of all sprints during rugby league match play are across distances ≤20-m, a large proportion of sprints still occur across distances greater than 20-m suggesting that the ability to sprint across longer distances may heavily influence playing performance in rugby league athletes (Gabbett 2012). Further, it has been recognised that for non-sprint specialists, as is the case for the rugby league athletes recruited for the current study, maximum sprint velocity is typically reached by 30-m (Haugen et al., 2019), supporting the need to understand the muscular contributions that lead to faster 30-m sprint times. For the current study, ADD, VM and

VI peak CSA explained 75.1% of the variance in 30-m sprint times. Interestingly, both ADD and VI peak CSA had a positive influence on 30-m sprint time, whereas VM peak CSA showed a negative influence on 30-m sprint times (Table 5). These data suggest that an increase in CSA of VM may result in slower 30-m sprint times, while increases in CSA of ADD and VI may lead to faster 30-m sprint times. These findings are in line with Sugisaki et al. (2011) who demonstrated that the ADD CSA explained 60% of the variance in 30-m sprint time. The mechanism by which ADD size contributes to 30-m sprint performance is unclear but it is possible that this muscle group makes a significant contribution to the hip extension torque required during the late-swing to early-contact phase of a sprint (Sugisaki et al., 2011). Hoshikawa et al. (2006) proposed that relatively larger quadriceps femoris muscles might negatively affect sprint velocity due to an increase in the moment of inertia of the lower limb. The findings of the current study suggest that greater VM CSA was associated with slower 30-m sprint times, however, greater VI CSA was associated with faster 30-m sprint times. Although VM and VI are both prime movers of knee extension, fine wire EMG analyses suggest that the VI is preferentially recruited in the swing phase of running and is coactivated with the RF and ILIAC muscles during hip flexion (Montgomery et al., 1994). A larger and presumably more powerful VI might therefore be expected to improve stride frequency via increasing the rate at which the flexing knee can be decelerated during swing. Whether each of the vasti muscles can be preferentially targeted via different strengthening exercises remains unclear and should be a focus of future work. The univariate analyses also revealed that BFLH ($r = -0.520$) peak CSA was correlated with 30-m sprint times. Although BFLH CSA did not contribute to the multiple linear regression model, these data support the hypothesis that the BFLH muscle is an important determinant of horizontal ground reaction forces in overground sprinting (Morin et al., 2015[A]).

When exploring measures of muscle volume, ILIOP and TFL size explained 67.2% of the variance in 30-m sprint times. These associations might reflect the requirement to generate large amounts of hip flexor power during relatively short sprints (Hoshikawa et al., 2006; Miller et al., 2020; Dorn et al., 2012). Dorn et al. (2012) demonstrated, with the combination of EMG data, kinematic data, ground contact data and musculoskeletal modelling, that the increase in stride frequency across progressively faster running speeds (3.5m•s^{-1}, 5.0 m•s^{-1}, 7.0 m•s^{-1} and ≥ 8.0 m•s^{-1}) paralleled the increase in force generated by the ILIOP, GMAX and the hamstring muscles.. These muscles also made a significant contribution to the hip and knee joint accelerations at faster running speeds. Future studies should seek to determine the mechanism(s) by which larger hip flexors contribute to improved sprinting performance. We also observed negative univariate associations between BFLH (r = -0.654), ADD (r = -0.564), VI (r = -0.659), TFL (r = -0.613), ILIOP (r = -0.753) and SART (p = -0.550) muscle volumes and 30-m sprint times. It is not surprising that BFLH, ADD, and VI were also associated with 30-m sprint times, given that these muscles are prime movers of hip and knee extension. The observation that SART muscle volume was correlated with 30-m sprint performance is in line with indirect evidence from previous studies (Miller et al., 2020; Handfield et al., 2017). For example, Handsfield et al. (2017) reported that SART muscle volume was 37% larger in elite sprinters than non-sprinters. Further, Miller et al. (2020) identified that SART muscle volume was one of three individual muscles that distinguished elite sprinters from sub-elite sprinters, with SART muscle volume being 28% larger in elite sprinters. Although research on the role of SART during sprinting is limited, the ability for SART to both flex the hip and knee may provide a beneficial role in the early swing phase of sprinting (Miller et al., 2020; Andersson et al., 1997). In summary, the peak CSA of ADD, VM and VI as

well as the normalised muscle volumes of ILIOP and TFL best explained the variance in 30-m sprint times for the rugby league athletes in the current study. Whether these findings are generalisable to other athletic cohorts is unknown and should be examined in future work (Ema et al., 2018: Takahashi et al., 2021: Miller et al., 2020).

4.4 Association Between Lower Limb Muscle Size and Peak Sprint Velocity

As team sport athletes typically reach their maximum sprint velocity by 30-m (Haugen et al., 2019) and due to the close proximity with opposing players during rugby league match play (Gabbett 2012), improving the ability to reach maximum sprint velocity sooner may further influence individual and team success (Till et al., 2017). Stepwise multiple linear regression revealed that the combination of GMIN peak CSA and GL peak CSA explained 44.5% of the variance in peak sprint velocity ($m \cdot s^{-1}$). The association between GMIN size and peak sprinting velocity is a novel finding and the mechanism(s) explaining this effect remain unclear. Sprinting at maximal velocity results in an increased demand for lateral hip stability (Kulmala et al., 2017) and the GMIN is an important hip stabiliser (Neumann 2010). Miller et al. (2020) reported that GMAX volume alone explained 34-44% of the variance in season best 100-m performance in a group of elite (season best: $10.10 \pm 0.07s$) and sub-elite (season best: $10.80 \pm 0.30s$) sprinters. However, in the current study, we did not detect any association between GMAX morphology and sprint performance. Part of the explanation for the differences between Miller et al. (2020) and the present study, could be that longer sprints (i.e., 100-m), may require a greater contribution from GMAX to maintain maximal sprinting velocity ($m \cdot s^{-1}$) (Lockie et al., 2013; Haugen et al., 2019). Another possible explanation for the positive relationship between GMIN and peak sprint velocity ($m \cdot s^{-1}$) may be the different physical

requirements in rugby league athletes compared to track and field athletes (Lockie et al., 2013; Haugen et al., 2019). It should be recognized that very limited research has been conducted on GMIN for its role in sprinting. The lack of research on GMIN has been partly highlighted in a systematic review by Howard et al. (2018). It was revealed that out of 18 articles that explored the musculature contributions to sprinting, 5 were dedicated to GMAX, 1 dedicated to GMED and none examined GMIN. Previous research suggests that greater GL muscle thickness and longer fibre lengths may separate faster from slow sprinters (Kumagai et al., 2000), however, EMG studies suggest that the plantar flexors may not make a significant contribution to forward propulsion during running (Montgomery et al., 1994). Although we did not measure muscle thickness, fibre length, or EMG in the current study, we observed a negative association between GL CSA and peak sprint velocity. These data suggest that rugby league athletes with smaller GL muscles are able to achieve faster peak sprint velocities. These findings are in line with previous investigations suggesting that smaller distal limb muscles, with less mass, may reduce the moment of inertia of the lower limb which may be beneficial in achieving faster sprint velocities (Sugisaki et al., 2011: Handsfield et al., 2017).

When only examining measures of muscle volume, the combination of ILIOP, RECF, GL and ADD muscle size explained 82% of the variance in peak sprint velocity. Interestingly, although ILIOP and ADD muscle volume both displayed a positive influence on peak sprinting velocity, RECF and GL muscle volume showed a negative association. These data suggest that an increase in muscle volume of RECF and GL would result in slower peak sprint velocities, while increases in ILIOP and ADD volume would result in greater peak sprint velocities. Previous studies have shown that the psoas major to quadriceps CSA ratio (Hoshikawa et al., 2006) and ADD CSA (Sugisaki et al., 2011) are determining

factors in 100-m and 30-m sprint performance, respectively. The negative interaction observed for GL is partly supported by Sugisaki et al. (2011) who reported that an increase in dorsiflexor CSA had a negative effect on 30-m sprint time. Handsfield et al. (2017) also found no significant difference in GL (7%) or GM (4%) muscle volumes (normalised to body size) when comparing sprinters to non-sprinters. The negative association between RECF and maximal sprint velocity can be supported by the findings from Sugisaki et al. (2018) and Hoshikawa et al. (2006) who both suggested that larger quadriceps femoris muscles may increase the inertial load placed on the lower limb when sprinting at faster speeds. It is also noteworthy that RECF is largely inactive during tasks involving simultaneous hip and knee extension (i.e., squatting) (Ploutz-Snyder et al., 1997), and perhaps as a consequence, is not heavily recruited during the late swing and stance phases of sprinting (Morin et al., 2015[A]). Collectively, these data support the hypothesis that a uniform increase in muscle mass in the lower limb may be disadvantageous for sprinting. The results in the present study suggest that an increase in the CSA and/or volume of more proximal lower limb muscles may be an important determinant of peak sprinting speed, at least in rugby league athletes.

4.5 Limitations

Despite strong associations between muscle size and 10-m, 30-m and peak sprint velocity ($m \cdot s^{-1}$), the cross-sectional design of this study makes it impossible to determine causal effects. Future longitudinal studies are needed to clarify whether training interventions targeted at promoting hypertrophy in select lower limb muscles are effective in improving sprinting performance. Further, we report a large number of

correlation analyses, which may result in type 1 error inflation, so the findings should be interpreted with caution. The present study used MR imaging to calculate peak CSA and muscle volume for the lower limb muscles. It should be recognized that muscle volume was estimated from the product of the interslice gap, and the sum of CSAs located at 20, 30, 40, 50, 60, 70 and 80% of the length of the muscle. Lund et al. (2002) previously reported that estimating muscle volume from 8 segmented CSA slices is valid and reliable and results in a difference of 0.004% compared to using 50 slices. The present study only examined the relationship between muscle size (peak CSA and volume) and sprint performance, although there are several other factors which were not investigated but may contribute to explaining the missing variance in the present study. Factors such as muscle fibre type, architecture, voluntary activation and sprinting technique are all likely to contribute to sprint performance and should be further investigated in combination with the findings of the present study. Lastly, due to the limited sample size, the current study may have been underpowered to detect small to moderate effects. Future research may benefit from employing larger sample sizes; however, given the high costs (>$500 per participant) of MR imaging, this may not be feasible. Further, due to the large number of correlation analyses reported, type 1 error inflation may be present and so interpretation of the results should be done with caution.

4.6 Practical Applications

In team-sports such as rugby league, the ability to generate high levels of muscular power during sprinting is paramount (Gabbett 2006). The ability to rapidly accelerate and reach peak sprinting speed is advantageous for generating momentum, creating tackles,

breaking tackles and creating try scoring opportunities (Chelly et al., 2010; Till et al., 2017, Gabbett 2006). Given the distinct physical requirements of rugby league (Gabbett 2006), understanding the muscular contributions to acceleration and peak sprint speed may enable coaches and practitioners to make better informed decisions regarding which muscle/s may be preferentially targeted for a given training intervention. The present study provides novel evidence to suggest that proximally located hip- and knee-spanning muscles make a significant contribution to sprint performance among semi-professional rugby league athletes. Specifically, greater CSA and normalised muscle volumes of the BFLH, ADD, ILIOP, TFL, VI, GMIN and SART were associated with faster 10-m sprint times, 30-m sprint times and/or peak sprint velocity ($m \cdot s^{-1}$). In contrast, larger peak CSAs and muscle volumes of RECF, VM and GL were associated with slower 30-m sprint times and peak sprint velocities ($m \cdot s^{-1}$). Multiple linear regression revealed that the combination of 1) ADD peak CSA (58.2%) and TFL and VI muscle volume (57%) best explain 10-m sprint times, 2) ADD, VM and VI peak CSA (75.1%) and ILIOP and TFL muscle volume (67.2%) best explain 30-m sprint times, and 3) GMIN and GL peak CSA (44.5%) and ILIOP, ADD, RECF and GL muscle volume (82%) best explain peak sprint velocity ($m \cdot s^{-1}$). Given the associations found in the present study between muscle size and sprint performance, future work is warranted to determine whether interventions targeted at promoting hypertrophy in these muscles leads to an improvement in sprint performance in this cohort.

4.7 Conclusion

This study aimed to map the spatial patterns of lower limb muscle size in under-20 male rugby league athletes and determine if regional differences in muscle volume and peak

CSA were associated with 10-m and 30-m sprint times and peak sprinting velocity ($m \cdot s^{-1}$). The results demonstrated that ADD CSA best explained 10-m sprint times, while the combination of ADD and VI CSA explained most of the variation in 30-m sprint times. Further, the CSA of GMIN was the best variable to explain peak sprint velocity ($m \cdot s^{-1}$). When normalised to the height-mass product, TFL and VI muscle volume as well as ILIOP and TFL muscle volume best explained 10-m and 30-m sprint times, respectively. In addition, peak sprint velocity ($m \cdot s^{-1}$) was best explained by the volumes of ILIOP and ADD. These data suggest the possibility that interventions targeted at promoting hypertrophy in certain proximal hip- and knee-spanning muscles might have important implications for improved sprint performance in semi-professional rugby league players.

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