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Original research

Subscapularis tendinopathy is highly prevalent in elite swimmer's shoulders: an MRI study

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ABSTRACT

Objectives: The aim of this study is to determine the prevalence of abnormal anatomical change present on MRI in elite swimmers' shoulders compared to age-matched controls.
Design: Descriptive epidemiological study.
Methods: Sixty (aged 16–36 years) elite Australian swimmers and 22 healthy active, age and gender matched controls (aged 16–34 years). All participants completed a demographic, and training load and shoulder pain questionnaire and underwent shoulder MRI. Tests for differences in the population proportion was used for comparison between swimmers dominant and non-dominant shoulders and those of the controls.
Results: Subscapularis and supraspinatus tendinopathy was the most common tendon abnormality identified in swimming participants, being reported in at least one shoulder in 48/60 (73 %) and 46/60 (70 %) swimmers, respectively. There was no significant difference between dominant and non-dominant shoulders for either tendinopathy, however, grade 3 tendinopathy was significantly more prevalent in subscapularis than in supraspinatus ($P < 0.01$). Compared with controls, significantly more abnormalities were reported in swimmers' shoulders in both subscapularis and supraspinatus tendons along with the labrum and acromioclavicular joint. Pathology was not a predictor of current pain.
Conclusions: This data confirms that tendon abnormality is the most common finding in elite swimmers' shoulders. Furthermore, that subscapularis tendinopathy is not only as common as supraspinatus but has a greater prevalence of grade 3 tendinopathy. With significant varied abnormalities including tendinopathy being so common in both symptomatic and asymptomatic shoulders of swimming athletes', clinicians should consider imaging findings alongside patient history, symptom presentation and clinical examination in determining their relevance in the presenting condition.

Practical implications

- In this study subscapularis tendon abnormality was as prevalent as supraspinatus tendon abnormality in elite swimmers, and as such clinicians should consider assessing function of both subscapularis and supraspinatus.
- Given the prevalence of tendon abnormality in swimmers shoulder management strategies should consider the tendinopathy model of rehabilitation. i.e., graded loading approach to meaningful load.
- The broad spectrum of pathological change found in this study in elite swimmer's shoulders, who were all performing at a high functional level with pathological change not predictive of pain, suggests clinical correlation is highly recommended when interpreting imaging findings.
- The anatomical abnormities found on MRI in elite swimmers' shoulder maybe indicative of normal adaptive changes, suggests judicious use of MRI on elite swimmers is recommend and should be reserved for athletes who are not responding to conservative management.

1. Introduction

'Swimmer's shoulder' is a common and debilitating condition in elite swimming athletes. Training volumes associated with this level of swimming equate to an extraordinary number of overhead shoulder cycles, as many as 1.5 million strokes per year.[1] Consequently, the shoulder is the

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most injured joint in elite swimmers. Its prevalence has been estimated to be between 40 and 91 %, and accounts for the most lost time from training for elite swimmers.[2–5]

While an examination of the literature studying pathology in swimmers' shoulders shows the supraspinatus tendon and subacromial bursa to have received the greatest attention in research,[5–8] a small number of studies have identified other pathologies present in swimmers' shoulders. These studies have utilised arthroscopy,[9–12] ultrasound[2,4,6] and 1.5 T MRI[5,7,13,14] for diagnosis, and reported significant other pathological changes including subscapularis tendinopathy, long head biceps (LHB) tendinopathy, instability and sheath effusions, labral tears including Bankart and SLAP lesions, subacromial/subdeltoid fluid, and a variety of acromioclavicular joint (AC) changes. Despite these findings, the attention to these other pathologies has been limited.

Most recent of these has been a study conducted by Celliers et al.,[7] in which both shoulders of 20 swimmers were examined using 1.5 T MRI. These authors describe many different pathologies present in the shoulders of their participants, however, again primarily focussed on supraspinatus tendinopathy concluding that if pre-season MRIs identified supraspinatus tendinopathy, then follow-up MRIs should be performed. After dividing the participants into symptomatic and asymptomatic groups they determined that there was no significant difference in the degree of pathology between the groups and concluded that pathology may be independent of symptom presentation. While recognising that a greater understanding of pathological change is important in improving management of swimmers with shoulder pain, this study did have several limitations including limited participant numbers, a relatively young cohort with a mean age of 18.9 years, and some with as little as 3 hours per week training being included in the study, suggesting a relatively sub-elite cohort. It also lacked a control group of non-swimmers to assist in determining the extent of change related to swimming.

As a result, the full extent of pathology that exists in elite swimmer's shoulders, is not well established. We hypothesise that pathologies in regions other than in the supraspinatus are prevalent. The aim of the current study is therefore to examine anatomical changes in a large cohort of elite swimmers using 3 T MRI technology and compare those with a cohort of healthy age and gender matched non-upper limb athletes, to determine the full extent of pathological change present in the shoulders of elite level swimmers independent of symptoms.

2. Methods

Sixty (35 male: 25 female) elite Australian swimmers aged 16–36 years were recruited. The athletes were considered elite if they had qualified to compete in a minimum of two open national championships. This cohort included 44 members of the Australian Swimming Team who have competed at Olympic Games and/or World Championships. Remaining athletes were recruited from Institute of Sport swimming programs around Australia. All athletes completed a standardised questionnaire capturing descriptive characteristics: age, sex, hand dominance, height, and weight as well as swimming related data: swimming event (stroke and distance), training history (years of squad training) and self-estimated average weekly training volume (total hours and kms). Participants experiencing current pain during swimming training, at the time of testing recorded a pain score out of 10 using a visual analogue scale (VAS) for the purpose of establishing a relationship between current pain and any pathologies present.

Twenty-two healthy active, age and gender matched controls (13 male: 9 female) were recruited. The controls comprised primarily elite middle-distance runners and race walkers but included hockey and soccer players. All controls completed a standardised questionnaire capturing descriptive characteristics such as age, sex, hand dominance, height and weight. Exclusion criteria for controls were a history of participation in overhead sports, shoulder pain or shoulder surgery.

This study was approved by the Australian Institute of Sport Ethics Committee (Approval Number: 20150602) and informed consent was obtained from each athlete and guardian, if applicable, prior to data collection.

Bilateral shoulder MRIs without arthrography were performed on all 60 swimmers ($n = 120$), while MRIs were only performed on a randomly selected shoulder for each of the controls ($n = 22$). Scans were performed a minimum of 4 hours post training to minimise any acute tendon abnormality changes that have been shown to occur immediately post training.[15] All MRIs were conducted with the arm in adduction and neutral rotation for the following sequences: oblique coronal proton-density and fat-suppressed T2, sagittal fat-suppressed T2 and axial proton-density. In addition, a further fat suppressed proton density sequence was performed in a standardised abduction external rotation (ABER) position.

Four MRI machines were utilised across four sites due to the geographical locations of the athletes however, all were 3 T Siemens machines with protocols and contrast factors being standardised. The swimmers' shoulders were examined with one of four magnets: 3 T Siemens Magnatrom Spectra, (Siemens Healthineers, Germany) and software version syngo MRI E11 (33 mT/m @ 125 T/m/s) with a sixteen-channel phased-array shoulder coil (Siemens Healthineers, Germany); 3 T Siemens Magnatrom Skyra, (Siemens Healthineers, Germany) and software version syngo MRI E11 (45 mT/m @ 200 T/m/s) with a sixteen-channel phased-array shoulder coil (Siemens Healthineers, Germany); 3 T Siemens Magnatron Spectra, (Siemens Healthineers, Germany) and software version syngo MR D12 (33 mT/m @ 125 T/m/s) with a sixteen-channel phased-array shoulder coil (Siemens Healthineers, Germany); 3 T Siemens Magnatrom Skyra, (Siemens Healthineers, Germany) and software version syngo MRI E11 (45 mT/m @ 200 T/m/s) with a sixteen-channel phased-array shoulder coil (Siemens Healthineers, Germany).

MRIs were evaluated by two independent musculoskeletal (MSK) specialist radiologists who were blinded to both the participants and each other. Each radiologist utilised a standardised 93-point proforma to assess the rotator cuff and long head bicep tendons for tendinopathy and tears, acromioclavicular joint, labral, articular cartilage changes, glenohumeral ligaments and rotator interval along with osseous and muscle oedema and subacromial and subdeltoid bursal thickness. Each proforma item was scored with either a “0” or “1” with any unresolved discordance being referred to a third experienced MSK specialist radiologist.

Tendon abnormality was assessed using a 0–3 grading scale as per Sein et al.[16] For grade 0 (normal), the tendon showed complete homogeneous low intensity on all pulse sequences. For grade 1 (mild tendon abnormality) there was a mild focal increase in tendon signal on proton density (PD) and fat suppressed T2 sequencing not equal to that of fluid. Grade 2 (moderate tendon abnormality) represented a moderate focal increase in tendon signal on PD and fat suppressed T2 sequencing not equal to that of fluid. For grade 3 (marked tendon abnormality), the tendon showed a marked generalised increase in tendon signal without frank fluid signal intensity. Radiologically subscapularis tears were described as either partial (minor fraying or partial tears of the deep fibres at the insertion site of lesser tuberosity) or full thickness tears (complete tear of either superior 1/3, superior2/3 or complete retraction tear with or without anterior subluxation of the humeral head) according to Garavaglia et al.,[17] which has been shown to have moderate reliability between MRI and arthroscopic findings.[18] The subacromial and subdeltoid bursae were measured immediately under the acromion and under the deltoid by identifying the thickest area of high signal on fat-suppressed T2-weighted coronal sequences to represent the fluid within the bursa, measured in millimetres by electronic callipers.

Data was summarised by frequencies, percentages, means, and standard deviations through the statistical software package R Core Team.[19] As both shoulders of each swimmer were scanned, results were therefore collated using dominant (D) and non-dominant (ND) sides. For comparisons between the dominant and control shoulders as well as the non-dominant and control shoulders, Fisher's exact test is employed as a test

of independence. A test for a difference in the population proportions was initially used, however due to the small number of observed successes/failures, the assumptions of that test were not met. An exact version of McNemar's test was employed for the comparisons between dominant and non-dominant shoulders. Some *p*-values were unable to be calculated due to there being no observed pathologies. For an analysis of whether particular pathologies were good predictors of current pain, mixed effects logistic regression models were applied with a random effect to account for the repeated measure of each shoulder.

3. Results

The mean age of the swimmers was 22.9 years and all swimmers had competed in at least two open national championships (35 male: 25 female). Forty-three (72 %) of the swimmers had competed at open international level, 11(18 %) junior international level and 6 (10 %) at open national level. The mean squad training years (±SD) was 12.6 years (±4.6) with a mean weekly training time (±SD) of 19 hours per week (±4.3). Weekly distance trained was categorised into 10 km intervals, with 45 (75 %) of swimmers swimming between 30 and 60 km/week. Regarding the swimmer's main stroke, 43 % swam freestyle, 20 % butterfly, 10 % breaststroke, 17 % backstroke and 20 % individual medley (Table 1). At the time of scanning, 15 swimmers (18 of 120 shoulders) had current shoulder pain, with the mean VAS score reported as 5.78/10. All participants were considered as highly functioning, as symptoms (15 athletes with current unilateral pain at the time of testing) were not sufficient to prevent training and competing at an elite level. The control participants were mean age (±SD) of 22.7 years (±4.6).

Supraspinatus tendon abnormality was present in 84 of the 120 shoulders (70 % of shoulders) accounted equally between the dominant and non-dominant shoulders (Table 3). Forty six of the 60 swimmers (77 %) having supraspinatus tendon abnormality in at least one shoulder, while it was found bilaterally in 39 (65 %) swimmers. Of those reported, 38 (45 %) were assessed as grade I, 36 (43 %) grade II and 10 (12 %) as grade III (Table 3). When breaking down the population, supraspinatus tendon abnormality was prevalent in 36/43 (84 %) of swimmers at senior international level, 8/11 (73 %) of swimmers at junior international level and 2/6 (33 %) of national level swimmers. Subscapularis tendon abnormality was reported in 46 (77 %) of dominant and 42 (70 %) of non-dominant shoulders (Table 3), with 48 of 60 swimmers (80 %) having a least one shoulder with subscapularis tendon abnormality. Of those reported, 30 (34 %) were assessed as grade I, 23 (26 %) grade II and 35 (40 %) as grade III. Furthermore, subscapularis tendon abnormality was prevalent in 39/43 (91 %) of swimmers at senior international level, 7/11 (64 %) of swimmers at junior international level and 3/6 (50 %) of national level swimmers. The tendon abnormalities found in the supraspinatus and subscapularis were both significantly different to the age matched control group subjects. Labral tears were found to be significantly different to the control group being reported in 26 of the 120 (22 %) swimmers' shoulders with no significant difference between dominant and non-dominant shoulders with 14/60 (24 %) and 12/60 (20 %) respectively. Of the 26 labral tears, 20 (77 %) were type 2 SLAP lesions. The AC joint was considered normal in 73 % of swimmers' shoulders, however 17 % had AC osteophytes being significantly more than the control group subjects.

Other abnormal anatomical findings found in our swimmer's shoulders included infraspinatus tendon abnormality which was reported in 19/60 (32 %) swimmers, and LHB tendon abnormality was prevalent in 11/60 (18 %) swimmers. Partial tears were reported in 10 % and 6 % of supraspinatus and subscapularis tendons, respectively. Effusion completely surrounding the LHB was found in 19 % of swimmer's shoulders. Bony oedema of the clavicle was found in 16 % of the subjects. Lessor tuberosity oedema was found in 16 of 120 (13 %) swimmers' shoulders again with no significant difference between dominant and non-dominant shoulders, 9/60 (15 %) and 7/60 (12 %). No labral tears, bone oedema or AC joint pathology were found amongst the controls. There was no significant difference in the mean subacromial bursal size (±SD) between swimmer's dominant (0.79 ± 0.46) and non-dominant (0.76 ± 0.56) sides or with controls (0.85 ± 0.61) (Table 2). Rotator cuff muscle oedema was found in 8/60 (17 %) of dominant shoulders and 11/60 (18 %) of non-dominant shoulders. The presence or severity of any of the observed anatomical abnormalities was not a predictor for pain ($P > 0.05$) (Table 4).

Table 1
Demographic table.

Demographic table	
Gender - male/female	35(58 %)/25(42 %)
Age (mean ± SD)	22.9 ± 4.0
Weight (kg) (mean ± SD)	75.8 ± 11.1
Height (cm) (mean ± SD)	181.9 ± 8.6
Hand dominance	
Left	6(10 %)
Right	54(90 %)
Competitive level	
Senior international	43(72 %)
Junior international	11(18 %)
National	6(10 %)
Training years (mean ± SD)	12.6 ± 4.6
Hrs/Week (mean ± SD)	19 ± 4.3
Kms/week	
20–30	6(10 %)
30–40	14(23 %)
40–50	19(32 %)
50–60	12(20 %)
60+	9(15 %)
Competition stroke	
Freestyle*	26(43 %)
Butterfly*	12(20 %)
Breaststroke	6(10 %)
Backstroke	10(17 %)
Individual medley	12(20 %)
Event distance (metres)	
50–100	14(23 %)
100–200	24(40 %)
200–400	13(22 %)
400+	6(10 %)

Legend: * indicates 6 swimmers nominated both freestyle and butterfly as their main stroke, make the total percentage equal to 110 %.

4. Discussion

The present study of elite swimmers currently competing is the largest reported to date and aimed to determine the degree of abnormal anatomical changes on MRI when compared to age matched healthy controls. The current study confirms that tendon abnormality is the most prevalent anatomical abnormality present in the shoulders of swimmers. Although these findings support those of previous researchers in identifying supraspinatus tendon abnormality as a primary condition in this athletic group, it is the first paper to identify the extent to which subscapularis tendon abnormality is present in the shoulders of "truly elite" level swimmers. While the prevalence of tendinopathy in subscapularis (73 %) was equivalent to that of supraspinatus (70 %), a significantly larger proportion of subscapularis tendon abnormalities were grade 3 (29 % vs 8 % supraspinatus, $P < 0.001$). In addition, the presence of oedema within the lesser tuberosity suggests that the subscapularis tendon and its insertion are subjected to significant load throughout the swimming stroke.

Interestingly, subscapularis tendon abnormality has been reported as present (to a lesser extent) in previous studies but has been largely ignored during any discussion. Klein et al[13] reported 46 % subscapularis tendinopathy in the throwing shoulder and 28 % in the non-throwing shoulder of water polo players, going on further to comment that these findings were not typical abnormalities found in swimmers. Sein et al[5] reported it as present in 2 out of 52 swimmers, while Celliers et al[7] graphically demonstrated 18 % and 14 % of asymptomatic and

Table 2
MRI pathoanatomical findings.

Pathoantomical findings						
	Dominant n (%)	p-value (95%CI) D v ND	Non-dominant n (%)	p-value (95%CI) ND v C	Control n (%)	p-value (95%CI) D v C
Supraspinatus						
Tendinopathy	42 (70)	1 (0.13–7.47)	42 (70)	0** (0–0.08)	0 (0)	0** (0–0.08)
Partial-tear	6 (10)	1 (0.19–5.40)	6 (10)	0.19 (0–2.00)	0 (0)	0.19 (0–2.00)
Full-tear	0 (0)	<NA>	0 (0)	1 (0-∞)	0 (0)	1 (0-∞)
Infraspinatus						
Tendinopathy	14 (23.3)	1 (0.20–3.28)	13 (21.7)	0.1 (0.01–1.32)	1 (4.5)	0.06 (0.01–1.18)
Partial-tear	1 (1.7)	1 (0-∞)	1 (1.7)	1 (0–51.82)	0 (0)	1 (0–51.82)
Full-tear	0 (0)	<NA>	0 (0)	1 (0-∞)	0 (0)	1 (0-∞)
Subscapularis						
Tendinopathy	46 (76.7)	0.29 (0.03–1.86)	42 (70)	0** (0–0.08)	0 (0)	0** (0–0.06)
Partial-tear	3 (5)	1 (0.03-∞)	4 (6.7)	0.57 (0–3.04)	0 (0)	0.56 (0–4.70)
Full-tear	0 (0)	<NA>	0 (0)	1 (0-∞)	0 (0)	1 (0-∞)
Long head biceps						
LHB-tendinopathy	8 (13.3)	1 (0.11–4.43)	7 (11.7)	0.18 (0–1.53)	0 (0)	0.10 (0–1.29)
LHB-anchor/sulcus	6 (10)	0.38 (0.40–197)	9 (15)	0.10 (0–1.34)	0 (0)	0.19 (0–2.00)
LHB-effusion	9 (15)	0.30 (0.62–7.46)	14 (23.3)	0.06 (0.01–1.18)	1 (4.5)	0.28 (0.01–1.95)
Labrum						
Tears	14 (23.3)	0.73 (0.32–10.73)	12 (20)	0.03* (1.18-∞)	0 (0)	0.02* (1.49-∞)
Bursal thickness						
Subacromial mean (±SD)	0.79 (±0.46)	0.48	0.76 (±0.56)	0.47	0.85 (±0.61)	0.71
Subdeltoid mean (±SD)	0.94 (±0.51)	0.90	0.94 (±0.48)	0.26	0.82 (±0.43)	0.3
Cartilage lesions						
Humeral head	3 (5)	0.50 (0–5.33)	1 (1.7)	1 (0–51.82)	0 (0)	0.56 (0–4.70)
Glenoid	1 (1.7)	<NA>	0 (0)	1 (0-∞)	0 (0)	1 (0–51.82)
AC Joint						
AC-Synovitis	3 (5)	1(0.17–17.96)	4 (6.7)	0.57(0–3.04)	0 (0)	0.56(0–4.70)
Osteophytes	9 (15)	0.77(0.38–5.59)	11 (18.3)	0.03*(0–0.97)	0 (0)	0.10(0–1.34)
AC-Clav Oed	9 (15)	1(0.23–9.10)	10 (16.7)	0.06 (0–1.23)	0 (0)	0.10(0–1.34)
AC-Acro Oed	3 (5)	1(0.10–117.99)	4 (6.7)	0.57(0–3.04)	0 (0)	0.56(0–4.70)
Osseous Oedema						
Humeral head	2 (3.3)	1(0–39.00)	1 (1.7)	1(0–51.82)	0 (0)	1(0–9.54)
Greater tuberosity	2 (3.3)	<NA>	0 (0)	1(0-∞)	0 (0)	1(0–9.54)
Lessor tuberosity	9 (15)	0.63(0.01–4.15)	7 (11.7)	0.18(0–1.53)	0 (0)	0.10(0–1.34)
Other						
Gang cysts	4 (6.7)	0.50(0.19-∞)	2 (3.3)	1(0.11-∞)	0 (0)	0.57(0.33-∞)
Rotator interval	2 (3.3)	1(0.10–118)	1 (1.7)	1(0.02-∞)	0 (0)	1(0.11-∞)
GH ligaments	5 (8.3)	0.63(0.24–157.49)	3 (5)	0.56(0.21-∞)	0 (0)	0.32(0.34-∞)
Cuff muscle oedema	8 (13.3)	0.51(0.08–2.34)	11 (18.3)	0.5(0.44–15.18)	2 (9.1)	0.72(0.31–10.82)

Legend: <NA> no probability due to no observed pathologies recorded.
Significance codes.
* P ≤ 0.05.
** P ≤ 0.01.

Table 3
Tendinopathy breakdown.

Tendinopathy						
	Dom (n)	(%)	Non-Dom (n)	(%)	Total (n)	Total (%)
Supraspinatus						
Gr I	17	28.3	21	35.0	38	45
Gr II	21	35.0	15	25.0	36	43
Gr III	4	6.7	6	10.0	10	12
Totals	42	70.0	42	70.0	84	100
Subscapularis						
Gr I	14	23.3	16	26.7	30	34
Gr II	11	18.3	12	20.0	23	26
Gr III	21	35.0	14	23.3	35	40
Totals	46	76.7	42	70.0	88	100
Infraspinatus						
Gr I	13	21.7	13	21.7	26	96
Gr II	1	1.7	0	0.0	1	4
Gr III	0	0.0	0	0.0	0	0
Total	14	23.3	13	21.7	27	100

symptomatic shoulders respectively had subscapularis tendinosis, however they failed to mention this within the results or discussion sections of the paper. A plausible reason for higher prevalence in our cohort is they are a "truly elite" group of swimmers, with more training hours and years of competing. We also found the subacromial and subdeltoid bursal measurements to be within normal ranges with no difference to

Table 4
Pathology as a predictor of pain (P-values).

Supraspinatus	P - value
Tendinopathy	0.414
Tear	0.159
Subscapularis	
Tendinopathy	0.754
Tear	0.327
Long head biceps	0.810
Labrum	0.275
AC Joint	0.213

Significance codes P ≤ 0.05.

the control group. This would suggest that an enlarged bursal finding on MRI may be clinically significant.

Sein et al[5] found supraspinatus tendinopathy the most common abnormality present in swimmer's shoulders (69 %) with a relationship to time spent in training, distance swum and level of competition. While the present study reported a similar level of supraspinatus pathology (71.7 %) there were no correlations with any of the training variables. Supraspinatus tendinopathy was a lower, but dominant finding by Celliers et al.,[7] with 36 % and 10 % of symptomatic and asymptomatic swimmer's shoulders, respectively. The most prevalent finding from Celliers et al[7] of supraspinatus partial tears (45.5 % symptomatic: 20.7 % asymptomatic shoulders) was significantly higher than both our findings of 10 % and Sein et al[5] with 6 %. Conversely, both the current study's finding of labral tears being present (21.6 %) and Sein et al[5] (19 %) were double those reported by Celliers et al[7] (10 %). Acromioclavicular joint pathology was a common finding in the current study with osteophytes present in 16.6 %, and bone oedema within the clavicle and acromion in 21.6 % of shoulders. This is lower than previously reported by Celliers et al.,[7] where AC joint arthrosis was found equally in 36.4 % and 34.5 % of symptomatic and asymptomatic shoulders, respectively. These latter, higher scores are surprising given the lower average age of the cohort. Both studies however reported the same prevalence of LHB tendinopathy (12.5 %). While it was not reported by Celliers et al.,[7] the current study demonstrated that 15 % of dominant and 23.7 % of non-dominant shoulders had an effusion completely surrounding the LHB tendon. Sein et al[5] reported 6 % of swimmer's shoulders had an effusion, however it was not clear if these were completely surrounding the tendon.

The differences in the demographics of the cohorts previously investigated may explain the variation between the current study and previous results. Earlier studies investigated smaller cohorts which were younger, completing fewer kilometres per week of training and at a sub elite level.[5,7] Furthermore, while previous studies have found similar pathologies when comparing to asymptomatic shoulders of the same athlete,[7,13,20] our age matched controls of healthy non upper limb elite athletes demonstrated virtually no abnormal anatomical findings. This may be because the asymptomatic shoulders previously studied were still from upper limb athletes participating in swimming, water polo and baseball which still involve substantial upper limb activity.

It is worth recognising that despite the degree of abnormal anatomical change present in the shoulders of the swimming participants of this study, at the time of imaging all participants were still managing to train and compete at the highest level, with 72 % of the cohort preparing for the 2016 Rio Olympics at the time of MRI. While 50 of our 60 swimmers at the time of testing were reporting to be pain-free, it is acknowledged that many of these participants will have experienced shoulder pain at some stage, which is common for these athletes.[21] Similar to the findings of anatomical abnormalities in asymptomatic shoulders by Celliers 2017, the current study showed the presence of anatomical abnormalities was not a predictor of current pain. This may indicate that many of the observed anatomical changes may well be normal adaptive changes related to the demands on the shoulder in an elite swimmer. Future research should look to correlate these abnormal findings in swimmer's who fail to continue competing due to shoulder pain.

Past literature has focused on a secondary external impingement model in the aetiology of swimmers' shoulder, and hence the supraspinatus tendon and subacromial bursa has been a major focus of research in this area. However, with subscapularis tendon abnormality occurring in the vast majority of the swimming participants in this study and as commonly as supraspinatus tendon abnormality, along with a significant number of intra-articular pathologies (labral), reconsideration of the likely precipitating mechanisms for pathological change in swimmers' shoulders seems warranted. Tendinopathy research over the past decade has provided insight into the type of loading associated with tendinopathy, with evidence supporting mixed loading (compression, tensile and shear) as a significant factor. With this research in mind recent hypotheses have been made suggesting that the shoulder range required during the swimming cycle, local anatomy, and tendon architecture make mixed loading through the entire rotator cuff highly likely.[22–24] Further, they suggest that the extraordinarily high number of shoulder cycles associated with elite swimming have the potential to drive maladaptive tendon response, precipitating tendinopathic change.

It is acknowledged that a limitation of this study is that the 120 shoulder MRIs were from both shoulders of 60 swimmers and that this may potentially increase the frequency of abnormal anatomical findings. However, the participants in this study were a truly elite cohort, and relative to participant numbers used in similar studies examining swimmer's shoulder,[4,7,8,13] 60 participants is a large sample size. Further, unlike previous studies we used an age matched healthy control group which allowed us to demonstrate that several of the abnormal anatomical changes observed (supraspinatus and subscapularis tendon abnormality, labral tears, AC joint osteophytes) are characteristic of elite swimmer's shoulder and significantly different to the healthy (non-upper limb athlete) population. When considering MRI technology, this study utilised the updated 3 T MRI, compared to earlier studies conducted on 1.5 T MRI, which has been shown to have inferior resolution, and may be a factor related to the higher prevalence of abnormities identified in this cohort.

5. Conclusion

This study provides increased understanding of the types and frequency of abnormal anatomical change in the shoulders of swimmers. Most significant of these findings is that subscapularis tendon abnormality is as prevalent as supraspinatus tendon abnormality in elite swimmers, with grade 3 tendon abnormality significantly more prevalent in subscapularis than in supraspinatus. This new knowledge will significantly improve decision-making when diagnosing and treating swimmers with shoulder pain. It also sheds new light on the possible mechanisms and pathological processes involved in the development of 'swimmer's shoulder'.

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Declaration of Interest Statement

The authors have no competing interests to declare.

Confirmation of Ethical Compliance

Ethic Committee: Australian Institute of Sport Ethics Committee.
Ethics Approval Number: 20150602.

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