Shoulder Pain in Elite Swimmers: Primarily Due to Swim-volume-induced Supraspinatus Tendinopathy

Mya Lay Sein, Judie Walton, James Linklater, Richard Appleyard, Brent Kirkbride, Donald Kuah and George AC Murrell

Br. J. Sports Med. published online 7 May 2008;
doi:10.1136/bjsm.2008.047282

BJSM Online First, published on May 7, 2008 as 10.1136/bjsm.2008.047282

Shoulder Pain in Elite Swimmers: Primarily Due to Swim-volume-induced Supraspinatus Tendinopathy

Mya Lay Sein[*], MBBS, MSpMed, PhD; Judie Walton[*], BSc, PhD; James Linklater‡, MBBS, BMedSci, FRANZCR; Richard Appleyard[*], BEng, PhD; Brent Kirkbride[†], MAppSci (PT); Donald Kuah[†], MBBS, FACSP; George A.C.Murrell, [*] MD, DPhil

[*]Orthopaedic Research Institute, University of New South Wales, St George Hospital and the [†]New South Wales Institute of Sport, Sydney, Australia

Address for correspondence:

Professor George AC Murrell,

Department of Orthopaedic Surgery

St. George Hospital Campus,

University of New South Wales,

Kogarah, Sydney, 2217, NSW, Australia

Phone: 61 2 9350-2088

Fax: 61 2 9350-3967

Email: **murrell.g@ori.org.au**

Abstract

Background/Hypothesis: Shoulder pain in elite swimmers is common and its pathogenesis is uncertain.

Hypothesis/Study Design: We used a cross-sectional study design to test Jobe's hypothesis that repetitive forceful swimming leads to shoulder laxity which in turn leads to impingement pain.

Methods: Eighty young elite swimmers (13-25 years of age) completed questionnaires on their swimming training, pain, and shoulder function. They were given a standardized clinical shoulder examination, and tested for glenohumeral joint laxity using a non-invasive electronic laxometer. 52/80 swimmers also attended for shoulder magnetic resonance imaging (MRI).

Results: 73/80 (91%) swimmers reported shoulder pain. Most (84%) had a positive impingement sign and 69% of those examined with MRI had supraspinatus tendinopathy. The impingement sign and MRI-determined supraspinatus tendinopathy correlated strongly (r_s=0.49, $p<0.00001$). Increased tendon thickness correlated with supraspinatus tendinopathy (r_s=0.37, $p<0.01$). Laxity correlated weakly with impingement pain (r_s=0.23, $p<0.05$) and was not associated with supraspinatus tendinopathy (r_s=0.14, p=0.32). The number of hours swum/week (r_s=0.39, $p<0.005$) and weekly mileage (r_s=0.34, p=0.01) both correlated significantly with supraspinatus tendinopathy. Swimming stroke preference did not.

Conclusions: These data indicate: (1) supraspinatus tendinopathy is the major cause of shoulder pain in elite swimmers; (2) this tendinopathy is induced by large amounts of swimming training; and (3) shoulder laxity *per se* has only a minimal association with shoulder impingement in elite swimmers. These findings are consistent with animal and tissue culture findings which support an alternate hypothesis: the intensity and duration of load to tendon fibers and cells causes tendinopathy, impingement, and shoulder pain.

Keywords: shoulder pain; shoulder laxity; laxometer; swimming; impingement; supraspinatus tendinopathy; injury prevention

The authors have no actual or potential conflict of interest in this manuscript or in the work that is the subject of this manuscript. No commercial entity paid or directed, or agreed to pay or direct, any benefits to the authors or to any research fund, foundation, educational institution, or other charitable or non-profit organization with which the author is affiliated or associated.

Introduction

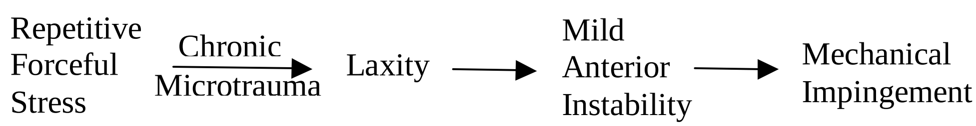
Millions of people participate in and enjoy aquatic sports, mostly swimming for fitness or competition. Sports injury surveys confirm that recreational swimmers have a low injury potential because of the buoyant effect of water.[60] However, as in any competitive sport, injury and pain can affect the elite swimmer. Swimming training involves repetitive overhead movement. In all the main swimming strokes (freestyle, backstroke, breaststroke and butterfly), the swimmer uses large moment arm forces to reach forward to drag the water. Where the training is intense, these factors may all contribute to shoulder injury and pain.[12,35,38,44] Competitive swimmers begin their careers as early as age 7 and most of them train and take part in year-round competitions. Competitive swimmers typically complete 2500 or more shoulder revolutions per day.[42]

The condition of “swimmer's shoulder” was first described by Kennedy et al.[24] in 1974 as a "common, painful syndrome of repeated shoulder impingement in swimmers.” Many studies have reported shoulder pain in elite swimmers.[11,13,24,25,31-35,45] The prevalence of shoulder pain in swimmers was 3% in a study published in 1974[24] and has increased in recent publications: 42% in 1980,[36,45] 68% in 1986,[33] 73% in 1993,[35] 40% to 69% in 1994,[1] and 5% to 65% in 1996.[3]

Proposed causes of swimmer’s shoulder

A clear consensus is lacking as to the cause(s) of shoulder pain in swimmers. Suggestions have been made that swimmer's shoulder represents a part of the impingement syndrome complex, rotator cuff tendonitis, biceps tendonitis, and shoulder instability.[16,17,36,56] Other pathological shoulder conditions, such as labral tears and acromioclavicular joint disruption, have also been observed in swimmers.[6,46]

As swimmers engage in repetitive overhead motion, it is possible that swimmer's shoulder may involve glenohumeral joint laxity and the "instability complex" described by Jobe[4,21,27] Jobe et al.[21] hypothesized that repetitive and forceful overhead activity causes a gradual stretching out of the anteroinferior capsuloligamentous structures leading to mild laxity, instability, and impingement. The following diagram summarizes their hypothesis.



This hypothesis has been quoted extensively and has been used to design protocols to treat swimmers and other overhead athletes with shoulder pain. However, on review of the literature there is little information that supports or refutes this hypothesis.

Laxity measurement

The normal range of glenohumeral joint laxity is unknown.[64] Excessive translation of the humeral head on the glenoid is normally prevented during athletic activities by static stabilizers of the glenohumeral joint, especially the glenoid labrum and capsular ligaments. Repetitive stretch injury or traumatic injury to the capsular ligaments may adversely affect static stabilization and increase the risk of instability to the joint. Instability has been defined as a symptom secondary to increased laxity.[5]

Many clinical tests have been described for assessing shoulder laxity and instability.[57,58] However, most of these tests are subjective, do not provide a continuous scale of numeric values, and have poor inter- and intra-observer reliability.[30] Neer and Foster[37] originally described the sulcus sign test as a test for multidirectional instability. A sulcus sign is a palpable and visible dimple created beneath the acromion when applying an inferior force, pulling downward on the subject's arm while in a sitting position.[37,52] Tzannes & Murrell studied multidirectional instability which was defined as the presence of grade 2 or greater laxity in more than one direction, in a symptomatic shoulder, on examination under anaesthesia. They confirmed that the sulcus sign is a good indicator of multidirectional instability.[57,58] However, when performing this test, the inferior force applied is not standardized and assessment of the dimple size is subjective. With this manual assessment, inter-observer reliability is only fair (ICC = 0.60).[58]

Electronic quantification of translation in the knee joint, using the KT-1000[20] is well established. However, there have been few attempts to quantify shoulder laxity with instrumentation. Jörgensen and Bak[22] have applied the use of a Donjoy knee laxity tester (dj Orthopaedics, Vista, California) to study anteroposterior translation of the glenohumeral joint. Sauers et al.[47] developed an instrumented arthrometer to assess glenohumeral joint laxity anteriorly and posteriorly at four force levels. As noted by Tibone et al.,[57] it has been very difficult to find a clinical device that provides an easy-to-use, objective, accurate, noninvasive, and reproducible measurement of shoulder laxity. We recently described an instrumental shoulder laxometer with these features that can measure inferior translation of the glenohumeral joint.[51] In the present study, we used this laxometer to test the laxity of shoulders in elite swimmers.

Supraspinatus tendinopathy

Supraspinatus tendinopathy (i.e. supraspinatus tendinosis or tendinitis) is another candidate cause of swimmer's shoulder. The supraspinatus is the major rotator cuff muscle responsible for securing the humeral head in the glenoid and its tendon is susceptible to tendinopathy in swimming and other overhead sports.[21,28,38,39,44,56] The normal tendon appears yellow-white. When magnified, quiescent rows of tenocytes (tendon cells of fibroblast origin) can be seen interspersed amongst the compact parallel bundles of collagen fibers which in turn are comprised of collagen fibrils. In supraspinatus tendinopathy, the tendon usually changes from yellow-white to gray, becoming dull and edematous, often with the bursa appearing inflamed. Microscopically, the tissue appears disrupted. Although there is little evidence of inflammatory cells, the tissue appears hypercellular with fibroblastic cells in varying states of cell health.[39]

The aim of this study, therefore, was to investigate the pathogenesis of swimmer's shoulder. To do this, we examined (1) which clinical sign best predicts shoulder pain in swimmers; (2) which anatomical structure(s) is/are associated with this shoulder pain; (3) whether repetitive swimming movements are associated with increased shoulder laxity; (4) whether increased laxity is associated with impingement pain; and (5) the extent, if any, to which training characteristics are associated with supraspinatus tendinopathy, laxity, or both, in elite swimmers.

METHODS

Swimmer recruitment

Under ethics approval, 86 elite swimmers were recruited from four competitive swimming clubs. All swimmers met the following inclusion criteria: (1) aged between 13 and 25 y; (2) having trained with a coach for a minimum of 2.5 y; and (3) swimming for at least 6 hr/wk. Exclusion criteria were: (1) previous surgery on the involved shoulder; (2) previous fracture of the shoulder; and (3) inability or unwillingness to participate in the MRI and clinical shoulder examination. Six elite swimmers were excluded from the study. Two were older than the age inclusion criterion, one had a dislocated shoulder, one inadvertently enrolled twice, and two failed to sign the consent form, leaving a total of 80 swimmers for the study.

Swimming training

A standardized self-administered swimming training questionnaire was completed by each study participant which requested the following information: number of years with coaching; hours per week in swimming training; level of competition (international, national, state or club level); weekly swimming distance; personal best in highest ranked event; and percentage of time in training spent in each stroke over the previous three months.

Shoulder pain and function

Descriptive characteristics were obtained for each swimmer using a standardized Shoulder Service Questionnaire, which is a modification of a validated questionnaire.[31]

Items pertaining to the subject included age, gender, birth date, occupation, arm dominance (right, left, or ambidextrous) and an overview of general health. Clinical parameters of the shoulder condition included affected shoulder (right or left), date of injury onset, mechanism of onset (whether traumatic or insidious), presence of an insurance claim, level of activity at work and highest exercise level before shoulder problem. All items contained in the Shoulder Service Questionnaire were scored on a five-point rating scale (0 to 4) except for the current level of activity, which was scored on a four-point rating scale (1 to 4). This questionnaire was designed to indicate frequency and severity of the shoulder pain, (with activity, at night, and at rest), stiffness of the shoulder, difficulty in reaching behind the back, difficulty with activities above the head, overall shoulder status (very bad, bad, fair, good), current level of activity (none, light activity, moderate activity and strenuous labor), and highest level of sport at the time of examination (none, hobby sport, club sport, state sport, national and international sport).

Clinical examination

Clinical shoulder examinations were performed on all 80 subjects. For the analysis, data from one shoulder of each subject were included. The shoulder chosen was the affected shoulder if symptomatic or the dominant shoulder if asymptomatic. Each athlete was given a systematic clinical shoulder examination which included 23 clinical tests. The results of each of these tests were recorded on a standardized Examiner's Form. The validity, reliability, and clinical usefulness of the majority of these tests have been previously described.[21-22,40,58,62]

Laxometer

We used a non-invasive electronic device for testing inferior translation of the humeral head in humans which has been shown to have excellent reliability and validity, i.e. patients with shoulder instability have higher laxity readings.[51] All subjects in this study were tested with the laxometer for inferior translation of their glenohumeral joint.

Magnetic resonance imaging (MRI)

Fifty-three swimmers were available for magnetic resonance imaging (MRI). One was unable to tolerate more than two pulse sequences and was excluded from the MRI study, leaving 52 swimmers for this aspect of the study.

MRI was used in a systematic fashion by a single examiner trained in musculoskeletal MRI (JL) using a standardized form to determine the minimum thickness of the supraspinatus tendon and to assess for acromion shape, tendinopathy, tears, acromioclavicular joint arthritis, and other pathological conditions in the shoulder. Minimum tendon thickness was measured immediately medial to the insertion of the supraspinatus tendon into the greater tuberosity of the humerus, 1 cm posterior to the biceps tendon.

The swimmers' shoulders were examined with a 1.5-Tesla (T) magnetic resonance imaging system (Signa; GE Medical Systems, Milwaukee, Wisconsin) and system software 9.1 (slew rate, 77 T/m/sec, 33-mm T gradient amplitude), with use of a high-resolution, non-arthrographic technique with a 4-channel phased-array shoulder coil (Medical Advances, Milwaukee, Wisconsin). Oblique coronal proton-density and fat-suppressed T2, sagittal T2, and axial proton-density sequencing were performed (***Table 1***).

MRI-assessed supraspinatus tendinopathy

Supraspinatus tendinopathy was graded using a modified four-point scale from 0 to 3.[52]. For Grade 0 (normal), the tendon showed complete homogeneous low intensity on all pulse sequences. For Grade 1 (mild tendinopathy,) there was a mild focal increase in tendon signal on PD and fat suppressed T_2 sequencing not equal to that of fluid. Grade 2 (moderate tendinopathy) represented a moderate focal increase in tendon signal on PD and fat suppressed T_2 sequencing not equal to that of fluid. For Grade 3 (marked tendinopathy), the tendon showed a marked generalized increase in tendon signal without frank fluid signal intensity. The grading of MRI-determined supraspinatus tendinopathy is very reliable (intraclass correlation coefficient = 0.75) when assessed by a single well-trained observer.[52]

Statistical analyses

Statistical tests were carried out using SigmaStat® software (Systat Software Inc., Point Richmond, CA). Multiple logistic regression analysis was performed with supraspinatus tendinopathy as the dependent variable and training regime parameters as independent variables. Statistical analyses that compared laxity with training and symptoms were based on data from all 80 swimmers whereas the MRI analyses were based on a subset of 52 swimmers.

RESULTS

Demographics of the swimmers

The age range of the swimmers in this study was 13-25 y. The mean (±SD) age was 15.9 (± 2.7) y and the median age was 16 y. Forty-two (53%) of the elite swimmers were male and 38 (47%) were female**.** Of the total participants, 13/80 represented their country at an international level, 41/80 swam at the national level, 24/80 were at the state level and 2/80 swimmers were at the club level. With regards to the swimmers' main event, 35% specialized in freestyle, including two open water swimmers. 19% specialized in butterfly stroke. 24% in backstroke, 18% in breaststroke, and 5% were individual medley swimmers. ***Table 2*** shows the percentage of training time spent in each of the four strokes.

All swimmers had been coached for at least 2.5 years, ranging upwards to a maximum of 17 years, with a median time of 8 years ***(Fig. 1)***. According to their questionnaire responses, the amount of time the swimmers practiced in the water varied between 8-29 hr/wk with the median number of hours they spent in water training was 16 hr/wk ***(Fig. 2)***. They swan between 9-110 km/wk with a median distance of 40 km/wk ***(Fig. 3)***. Ninety percent (72/80) of the swimmers spent more than 50% of their training time in freestyle. On average, they estimated that they spent approximately 13% of their swimming time in butterfly stroke, 21% in back stroke, and 13% in breaststroke ***(Fig. 4)***.

Shoulder pain and function

Of the total study group of 80 swimmers, 43 (54%) reported unilateral shoulder pain (affecting 28 right shoulders and 15 left shoulders) and 30 (37%) others reported bilateral shoulder pain. The remaining seven swimmers (9%) stated they had no shoulder pain. Sixty-four swimmers (80%) reported they had pain during activity and, of these, 25 had activity pain monthly, 22 had it weekly, 14 had it daily, and three stated they always had shoulder pain during activity. 56/80 (70%) swimmers specified that they had shoulder pain with activity above their head. Twenty-two (28%) swimmers had shoulder pain at night. Of this group, 14 swimmers had night pain monthly, six had it weekly, one had pain every night, and another stated she had pain continuously throughout the night. Twenty-two (28%) swimmers rated the level of their night pain as mild to moderate. 24/80 (30%) swimmers reported they had extreme pain monthly while nine had it weekly. The same proportion of swimmers indicated they had mild to moderate shoulder pain even while resting.

54/80 (68%) swimmers indicated they had shoulder stiffness. Of these, 42 (52%) had difficulty reaching behind their back and 43 (54%) had difficulty with activities above their head. Four (5%) of the 80 swimmers had severe to very severe difficulty with overhead activities whereas the remainder had mild to moderate difficulty.

Of the total study group, 41/80 (51%) self-assessed the over-all condition of their shoulder as fair (4) on a scale of 1-5 (very bad to good). 10/80 (12.5%) reported their shoulder to be in poor condition (3), and 5/80 (6%) in bad condition (2). None of the swimmers permanently discontinued swimming because of their shoulder problem. Eighty-nine percent had sought treatment for their shoulder pain: 16/80 (20%) with chiropractic therapy, 47/80 (59%) with physiotherapy, 9/80 (11%) with acupuncture while 8/80 (10%) had no treatment for their pain.

Clinical examination of the shoulder

Details regarding the clinical examination findings in swimmers are shown in ***Table 3***. The most common positive findings were a positive impingement sign (90%) and signs related to the biceps and A-C joint: biceps tenderness (45%), Paxinos sign – compression of the clavicle against the acromion (34%)[62], O'Brien's sign (25%), A-C joint tenderness (10%).

Glenohumeral joint laxity in swimmers

Glenohumeral joint laxity was manually assessed during the clinical examination for a sulcus sign. None of the swimmers had a grade 3+ sulcus sign. In 11 (14%) of simmers, the sulcus sign was assessed as grade 2+, and in 41 (51%) as grade 1+. Forty-nine (61%) swimmers had grade 1+ and eight (10%) had grade 2+ anterior translation. Twenty-six (33%) swimmers had grade 1+ and four (5%) had grade 2+ posterior translation.

An instrumented laxometer was also used to test inferior translation of the humerus in both shoulders of the 80 swimmers. With this technique, the range of inferior translation of the glenohumeral joint was 0.5-5.9 mm with a median value of 1.5 mm for both male and female shoulders. For the age range studied here, there was no significant correlation between swimmer age and shoulder laxity ($r = 0.097$, $p = 0.49$).

Within the entire group of swimmers, shoulder laxity correlated positively with a greater range of motion in internal rotation (Pearson's product moment correlation, $r = 0.33$, $p < 0.05$). The entire group had more inferior glenohumeral joint translation in the left shoulder than in the right (paired t-tests, $p<0.01$). Significantly more laxity was also noted in the left shoulders of females than in their right shoulders (paired t-tests, $p<0.05$). Females had significantly more laxity than males (1.72 mm versus 1.28 mm; t-test, $p< 0.05$. However, the difference in glenohumeral joint laxity between male left and right shoulders was insignificant.

MRI findings in elite swimmers

Supraspinatus tendinopathy

Thirty-six (25 male, 11 female) or 69% of the 52 swimmers that were imaged by MRI showed evidence of supraspinatus tendinopathy. Of those with tendinopathy, 27/36 (75%) had their tendinopathy assessed as grade 1, 8/26 (22%) as grade 2, and 1/36 (3%) as grade 3 ***(Fig. 5)***. Of the imaged population, 4/4 (100%) swimmers at the international level exhibited supraspinatus tendinopathy, 24/27 (89%) swimmers at the national level, 8/20 (40%) swimmers at the state level, and 0/1 (0%) swimmers at the club level had supraspinatus tendinopathy ***(Fig. 6)***. This condition was found in 13/24 (54%) of swimmers aged between 13-14 years, 10/13 (77%) between 15-16 years, 8/8 (100%) between 17-18 years, and 5/7 (71%) between 19-22 years of age. This study found no association between preferred swimming stroke and supraspinatus tendinopathy.

Other MRI findings

For all swimmers, MRI-determined minimum supraspinatus tendon thickness ranged from 6 to 10 mm with a median value of 8 mm. 27% of the swimmers had thickened supraspinatus tendons. There was no relationship between tendon thickening and the swimmers' ages.

The biceps tendon was normal in 46 imaged shoulders. The biceps anchor was reported to be unstable in three swimmers' shoulders and three other shoulders were found to have biceps sheath effusion. Tears of the supraspinatus tendon were found in three swimmers: two were reported as having a delaminated intrasubstance tear and one had a partial 3mm articular side tear. Two had subscapularis tendinopathy, and one had infraspinatus tendon thickening, but no change was reported for the teres minor tendon.

Labral tears were detected in 10/52 swimmers whose shoulders were imaged. Eight swimmers had SLAP (Superior Labrum Anterior Posterior) lesions. One had a SLAP lesion and Bankart lesion and another had a Bankart lesion alone. Mucoid changes of the labrum were noted in five swimmers' shoulders and two were noted to have a paralabral cyst. Five sublabrum-foramens and one Buford complex were also observed.

The acromion shape was assessed as type I in 20/52 (38%) swimmers, type II in 29/52 (56%), and type III in 3/52 (6%). Thickening of the subacromial bursa was reported in 33/52 (63%) swimmers, and thickness of the subscapularis bursa affected 3/52 (6%) others. Minimal to moderate increase in subacromial and subdeltoid fluid was observed in 21/52 (40%) swimmers' shoulders while 7/52 (13%) of the swimmers had acromioclavicular joint arthritis. In 17/52 (33%) of the swimmers' shoulders, the acromion ossification centre had not yet fused.

Statistical Correlations

Laxity correlated modestly with impingement ($r_s = 0.28$; $p < 0.05$) but lacked significant association with supraspinatus teninopathy. There were significant relationships between laxity and extreme pain ($p<0.05$) and between impingment during external rotation and shoulder pain (r_s = 0.25, p< 0.05). The correlation between laxity and pain during activity approached but did not reach significance (r_s = 0.21; p = 0.065). There was no correlation between laxity and level of competition, hours of training, or weekly mileage.

In contrast, the swimmers' supraspinatus tendon thickness correlated significantly with their (1) level of training (r = 0.28, p<0.05), (2) years in training (r = 0.35, p <0.01), (3) hours/week in training (r = 0.33, p<0.05), and (4) cumulative use (weekly distance x 48 wk x No. y with coach) (r = 0.37, p<0.01). All swimmers with increased tendon thickness had impingement pain and supraspinatus tendinopathy.

All but one of the 36/52 (69%) swimmers who had supraspinatus tendinopathy indicated they had shoulder pain on a diagram included in the shoulder pain and function questionnaire. However, correlations were lacking between supraspinatus tendinopathy and severity or frequency of shoulder pain during activity, at night, or at rest (p> 0.05). Stepwise multiple regression analysis showed that the dependent variable 'pain during activity' could be predicted from a linear combination of two independent variables: impingement sign (in either direction) r^2 = 0.16 (p <0.05) and the Paxinos sign r^2 = 0.10 (p <0.05) in the elite swimmers.

A positive impingement sign correlated strongly with the presence of supraspinatus tendinopathy (r_s=0.49, p<0.00001). A positive impingement sign had 100% sensitivity and 65% specificity for diagnosing supraspinatus tendinopathy. The presence of a positive impingement sign failed to correlate significantly with other shoulder pathologies including labral tear, biceps instability, MRI-determined acromial types (1,2,3), supraspinatus tendon thickness, bursal thickness, sublabral-foramen, acromioclavicular joint arthritis, or an unfused acromion ossification centre. A significant inverse association was noted between supraspinatus tendinopathy and a positive apprehension sign (r_s = -0.33, p<0.05). Apart from tests for the impingement sign and apprehension, there were no significant correlations between supraspinatus tendinopathy and any of the clinical tests (see Appendix 1).

Spearman correlation coefficients were used to test the relationships between supraspinatus tendinopathy and the swimming training (years of coaching, percentage of stroke specialty, level of competition, hours of training, and weekly mileage). There was no association between supraspinatus tendinopathy and the number of years coached (p >0.05*)*.

The swimmers stated that their percentage of swimming training time spent with each of the four main strokes were: freestyle (25% to 95%) with a median percentage of 50%; butterfly (0% - 40%) with a median percentage 11.25%; backstroke (2% - 70%) with a median percentage of 20%; and breaststroke (0% - 45%) with a median percentage of 10%. Except for inverse relationships between the percentage of swimming time spent in butterfly stroke and supraspinatus tendon thickness (r_s = -0.30, p< 0.05), or between supraspinatus tendinopathy (r = -0.30, p< 0.05) and the number of years coached (r_s = -0.29, p < 0.05), there were no significant associations for freestyle, backstroke, or breaststroke.

There was a highly significant association between supraspinatus tendinopathy and competition level ***(Fig. 6)***. Athletes at higher levels of competition were more likely to have supraspinatus tendinopathy than those at lower levels of competition ($r_s = 0.56$, $p < 0.0001$).

A significant association was also found between supraspinatus tendinopathy and the number of hours swum/week ($r_s = 0.36$, $p < 0.01$). Swimmers who swam more than 15 hours per week were more likely to have supraspinatus tendinopathy than those who swam fewer hours, ($r_s = 0.48$, $p < 0.0005$). All swimmers who swam more than 20 hours per week had supraspinatus tendinopathy ***(Fig7).*** Elite swimmers who trained for more than 15 hours per week were twice as likely to have tendinopathy as those who trained for less time ($r_s = 0.48$, p <0.0001).

There was also a significant association between supraspinatus tendinopathy and the kilometers swum per week ($r_s = 0.33$, $p < 0.05$). The swimmers who swam more than 35 kilometers per week were more likely to have supraspinatus tendinopathy than those who swam fewer kilometers ($r_s = 0.37$, $p < 0.01$). Importantly, all swimmers who swam more than 60 kilometers per week exhibited supraspinatus tendinopathy ***(Fig 8).*** Athletes who swam more than 35 kilometers per week ($r = 0.37$, $p < 0.01$) were four times more likely to have tendinopathy than those who swam fewer kilometers.

Stepwise multiple regression analysis was used to determine the relationship between supraspinatus tendinopathy and the significant independent variables of swimming training: the number of hours swum per week and the weekly mileage. The level of competition was omitted since this is not a variable that the swimmers could voluntarily change. This analysis showed that the dependent variable ‘supraspinatus tendinopathy’ could be predicted from the independent variable ‘hours swum per week’ ($r^2 = 0.131$, $p < 0.01$). Multiple logistic regression analysis showed that supraspinatus tendinopathy could be correctly predicted in 85% of elite swimmers from either the number of hours swum per week alone or in combination with the swimmer's weekly mileage. In essence, swimmers who train more than 15 hours/week and swim a weekly distance of more than 35 kilometers are at increased risk for supraspinatus tendinopathy.

Stepwise multiple regression analysis showed that the dependent variable 'pain during activity' in the elite swimmers could be predicted from a linear combination of two independent variables: a positive impingement sign (in either direction) $r^2 = 0.16$ ($p < 0.05$) and the Paxinos sign $r^2 = 0.10$ ($p < 0.05$). Multiple logistic regression analysis, performed with pain during activity as the dependent variable and the impingement sign and the Paxinos sign as independent variables, showed that the presence of absence of these signs could predict pain during activity in 84% of elite swimmers.

Similarly, the best predictors for pain level with activities above the head in elite swimmers were the impingement sign ($r^2 = 0.11$; $p < 0.05$) and O'Brien's test ($r^2 = 0.17$; $p < 0.05$). Multiple logistic regression analysis demonstrated that the impingement sign and O' Brien's sign could predict pain level with activities above the head in 75% of elite swimmers.

In summary, of the elite swimmers in our imaging study, 36/52 (69%) had supraspinatus tendinopathy. Each swimmer with supraspinatus tendinopathy had a positive impingement sign. The positive impingement sign correlated strongly with MRI assessment of tendinopathy. Moreover, all swimmers with supraspinatus tendon thickening had impingement and supraspinatus tendinopathy. There was a modest, yet significant, correlation between glenohumeral joint laxity and impingement pain. There were highly significant associations between supraspinatus tendinopathy and competition level, and the number of hours swum per week. Supraspinatus tendinopathy also correlated significantly with the swimmers' weekly mileage.

DISCUSSION

An evaluation of Jobe's hypothesis that glenohumeral joint laxity is a cause of shoulder pain

Using manual laxity tests, several authors have examined glenohumeral joint laxity in recreational and/or competitive swimmers and concluded that competitive swimmers have greater glenohumeral and general joint laxity.[3,11,34,42,67] Borsa et al.[7] disagreed. Using sonographic imaging to quantify glenohumeral joint laxity under stress and non-stress conditions, they found that elite swimmers do not have excessive glenohumeral joint translation compared to age-matched non-swimming controls. Nor do they acquire excessive laxity as a result of their long-term participation in a sport with repetitive overhead activity.

Our results concur with the findings of Borsa et al.[7] that repetitive swimming does not increase shoulder joint laxity in elite swimmers. Our data also oppose the hypothesis that cumulative swimming time (weekly mileage x 48 weeks x number of years coached) in the form of swimming training leads to glenohumeral joint laxity in swimmers. Hence, we doubt that laxity is a major contributor to swimmer's shoulder.

On the other hand, our results showed a modest yet significant correlation between glenohumeral joint laxity and either impingement pain ($r_s = 0.23$, $p < 0.05$) or extreme pain ($r_s = 0.26$, $p < 0.05$). Similarly, McMaster et al.[34] found a statistically significant correlation between a clinical score of glenohumeral joint laxity and the presence of interfering shoulder pain in senior national and elite swimmers.

An alternate interpretation of swimmer's shoulder

We found a high incidence of MRI-assessed symptomatic supraspinatus tendinopathy in elite swimmers. The incidence of tendinopathy was related to the time spent in training (hours swum per week) and the distance swum per week. Elite swimmers who trained for more than 15 hours per week were twice as likely to have tendinopathy as those who trained for less time. Similarly, elite athletes who swam more than 35 kilometers per week were four times more likely to have tendinopathy as those who swam fewer kilometers. The level of competition also correlated with supraspinatus tendinopathy, with a higher proportion of swimmers at the higher competition level having supraspinatus tendinopathy than swimmers at lower competitive levels.

An association between the extent of training and shoulder pain has been identified by Pollard[43] when evaluating the prevalence of shoulder pain in elite British swimmers. Specifically, he found a correlation between the number of kilometers the athletes swam per week and shoulder pain ($p < 0.001$). A study involving German triathletes also noted a significant relationship between the number of weekly training hours and incidence of muscle and tendon injuries ($p < 0.05$).[14] Thus, shoulders of the elite swimmer are, in the long-term, susceptible to the continuous overloading associated with overuse activity.[9,23,] Our study indicates that supraspinatus tendinopathy in swimmers is related both to the amount and the duration of load on supraspinatus tendons. The high incidence of biceps tenderness and A-C joint pain suggest that these areas may also be subject to overload stress in elite swimmers.

Despite some evidence to the contrary by others,[43,45] our data indicated that specific swimming strokes have little effect in predisposing elite swimmers to shoulder pain. In our view, the following diagram more accurately describes what generally happens to cause shoulder pain in elite swimmers.

Excessive time swimming/ mileage → Supraspinatus tendinopathy (including tendon thickening) → Shoulder pain

In this model, repetitive movement causes tendinopathy with an associated increase in tendon thickness. Tendinopathy leads to pain when the thickened tendon and associated bursa is repeatedly squashed under the bony arch of the acromion during swimming as in impingement testing.

Our results showed that intensive training increases tendon thickness, a feature that is significantly associated with supraspinatus tendinopathy. Animal studies have also shown that prolonged mechanical loading increases tendon thickness and alters its mechanical properties[8,15,26,56,61,65] For example, the cross-sectional area of the supraspinatus tendon was larger than normal in rats exercised for 4 weeks and continued to increase in size up to 16 weeks, its elasticity amounted to only 52-61% of the control (non-exercised) value and less stress was required to rupture it. Following this overuse protocol, more substance of lesser quality was present in the injured tendons.[56]

In a study with horses, 18 months of training increased the cross-sectional area of the Achilles tendon whereas five months of training did not.[41] In another study with exercised rats, the cross-sectional area of the Achilles tendon was increased at four months of running training.[15] In a guinea fowl study, 8-12 weeks of mechanical loading was sufficient to increase tendon stiffness.[8]

A possible cellular mechanism for swimmer's shoulder

Experimental studies suggest a mechanistic basis that may explain the supraspinatus tendinopathy we observed in the swimmers' shoulders. For evaluating supraspinatus tendinopathy, the running rat model has been particularly informative.[10,54] Supraspinatus tendon overuse was modeled by subjecting the rats to repetitive exercise, consisting of treadmill running at 17 meters per minute at a 10º decline for 1 hour per day over 5 days per week.[54] This protocol results in approximately 7500 strides per day.

In running rats, the supraspinatus tendon can be damaged either by overuse and intrinsic factors, overuse and extrinsic factors, or by overuse alone.[10,48,55] Specifically, extrinsic factors such as the coracoacromial arch can cause repetitive mechanical impingement of the rotator cuff. Intrinsic factors originate within the tendon as from tendon overload or tendon thickening. In combination, overuse plus extrinsic compression dramatically increased the incidence of significant injury in the rat supraspinatus tendon.[55]

Normally, the spindle-shaped tendon cells or tenocytes lie in rows surrounded by parallel bundles of collagen fibrils. Early microscopic change in rat supraspinatus tendons after running for 12 weeks, is characterized by tenocyte rounding and proliferation. The cell machinery of the tenocytes is diverted from their differentiated task of maintaining the collagen fibrils and the extracellular matrix to mitosis. Under these conditions, the matrix accumulates water, glycosaminoglycans and other ground substance while collagen frays and becomes disorganized, and the tendon thickens. Loss of tendon biomechanical integrity occurs early, coinciding with tendon thickening. The structural changes are accompanied by decreased elasticity in the tendon and decreased load for its rupture.[56]

Mixed findings have been reported in regard to apoptosis and cellular inflammation in the tendons of rats that ran on a treadmill for 1 h/day over 1-3 months[49,] (authors, manuscript in preparation). Excessive apoptosis was induced in isolated rat tibialis anterior tendons loaded with a high (20%) strain for 6 hours. Apoptotic rates were 20 times higher than in control tendons.[50] The biochemical basis for this apoptosis effect was examined using canine patellar tendon cells in tissue culture. The amount of stress applied directly to the tendon cells showed a positive relationship with the induction of a stress-activated protein kinase (c-Jun N-terminal kinase or JNK) within the cells.[2] Cyclic strain (analogous to that in swimming movements) results in immediate activation of JNK, peaking at 30 minutes. This activation is regulated by a magnitude-dependent but not frequency-dependent, calcium-dependent mechanotransduction pathway. Whereas transient JNK activation is associated with normal cell metabolism, persistent JNK activation can initiate apoptosis.[2]

In our study, the number of strokes per day calculated to be made by elite athletes who swam more than 15 hours per week was similar to the number of strides per day made by the running rat model.[54,55] At this activity level, significant change can occur in the thickness of the tendon and in supraspinatus tendinopathy. In the swimmers and the running rats, overuse activity can affect factors that are intrinsic (e,g, tendon thickening) and extrinsic (e,g, tendon impingement by the acromial arch). A weakness in our hypothesis is that we did not test whether scheduled rests can prevent or reverse supraspinatus tendinopathy in athletes. Soslowsky has shown this condition to be reversible in the running rat model (personal communication).

Although biopsies of the athletes' tendons were not available to determine whether apoptosis occurs in human tendon cells stressed by over-exercise, other studies in our laboratory based on discarded human tendon tissue obtained during surgery found that JNK is expressed, cells proliferate, collagen fibrils become disorganized, and apoptosis occurs in human rotator cuff tissue exposed to oxidative stress.[63,66]

In summary, these animal, *ex vivo*, and *in vitro* findings help to explain our results in the elite athletes (Fig. 9). Both the swimming human and running rat systems showed significant associations between the amount of exercise (mileage/time) and the development of tendinopathy, supporting the hypothesis that tendinopathy relates to the amount (mileage and hours) of load on the tenocytes that normally secrete pro-collagen and maintain the collagen fibrils. Under conditions of excessive stress, the homeostatic equilibrium may be lost, ultimately resulting in increased apoptosis, fraying and disorganization of the collagen fibrils, water accumulation, tendon deterioration, and pain. These changes could thus lead to the presence of incipient tears evident in some of the swimmers' tendons.

What is already known on this topic?

Elite swimmers are prone to shoulder pain, the cause of which is unknown. Rats subjected to excessive activity involving similar shoulder action develop supraspinatus tendinopathy.

What this study adds:

Swimmers who overexercise, either by swimming more than 15 hours per week or more than 35 miles per week, have a propensity for developing supraspinatus tendinopathy. This condition arises independently of shoulder laxity.

REFERENCES

1. Allegrucci, Whitney SL, Irrgang JJ. Clinical implications of secondary impingement of the shoulder in freestyle swimmers. *J Orthop Sports Phys Ther* 1994:20(6):307-18.
2. Arnoczky SP, Tian T, Lavagnino M, Gardner K, Schuler P, Morse P. Activation of stress-activated protein kinases (SAPK) in tendon cells following cyclic strain: the effects of strain frequency, strain magnitude, and cytosolic calcium. *J Orthop Res* 2002;20(5):947-956.
3. Bak K. Nontraumatic glenohumeral instability and coracoacromial impingement in swimmers. *Scand J Med Sci Sports* 1996;6(3):132-144.
4. Bak K, Faunø P. Clinical findings in competitive swimmers with shoulder pain. *Am J Sports Med* 1997;25(2):254-260.
5. Bigliani LU, editor. *The Unstable Shoulder*. Illinois: American Academy of Orthopaedic Surgery, 1996.
6. Blaznia ME, and Satzman, J.S. Recurrent anterior subluxation of the shoulder in athletics-a distinct entity. In Proceedings of the American Acadamy of Orthopaedic Surgeons. *J Bone Joint Surg Am* 1969;51:1037-1038.
7. Borsa PA, Scibek JS, Jacobson JA, Meister K. Sonographic Stress Measurement of Glenohumeral Joint Laxity in Collegiate Swimmers and Age-Matched Controls. *Am J Sports Med* 2005;33:1077-1084.
8. Buchanan CI, Marsh RL. Effects of long-term exercise on the biomechanical properties of the Achilles tendon of guinea fowl. *J Appl Physiol* 2001;90(1):164-171.

9. Budoff JE, Nirschl RP, Guidi EJ. Debridement of partial-thickness tears of the rotator cuff without acromioplasty. Long-term follow-up and review of the literature. J Bone Joint Surg Am 1998;80(5):733-748.

10. Carpenter JE, Flanagan CL, Thomopoulos S, Yian EH, Soslowsky LJ. The effects of overuse combined with intrinsic or extrinsic alterations in an animal model of rotator cuff tendinosis. *Am J Sports Med* 1998;26(6):801-807.

11. Ciullo JV. Swimmer's shoulder. *Clin Sports Med* 1986;5(1):115-37.

12. Ciullo JV, Stevens GG. The prevention and treatment of injuries to the shoulder in swimming. *Sports Med* 1989;7(3):182-204.

13. Dominguez RH. Shoulder pain in age group swimmers; in Erikson B, Furberg B (eds). S*wimming Medicine IV: Proc 4th Int Congress on Swimming Med, Stockholm, Sweden. Baltimore: University Park Press* 1978:105-109.

14. Egermann M, Brocai D, Lill CA, Schmitt H. Analysis of injuries in long-distance triathletes. *Int J Sports Med* 2003;24(4):271-276.

15. Enwemeka C, Maxwell L, Fernandes G. Ultrastructural morphometry of matrical changes induced by exercise and food restriction in the rat calcaneal tendon. *Tissue Cell* 1992;24(4):499-510.

16. Fowler PJ. Symposium: Shoulder problems in overhead-overuse sports. Swimmer problems. *Am J Sports Med* 1979;7(2):141-142.

17. Fu FH, Harner CD, Klein AH. Shoulder impingement syndrome. A critical review. Clin Orthop 1991(269):162-173.

18. Hayes K, Walton JR, Szomor ZR, Murrell GA. Reliability of five methods for assessing shoulder range of motion. *Aust J Physiother* 2001;47(4):289-294.

19. Hayes K, Walton JR, Szomor ZL, and Murrell GAC. Reliability of three methods for assessing shoulder strength. *J Shoulder Elbow Surg* 2002;11(1):33-39.

20. Huber FE, Irrgang JJ, Harner C, Lephart S. Intratester and Intertester Reliability of the KT-1000 Arthrometer in the Assessment of Posterior Laxity of the Knee. *Am J Sports Med*, July 1, 1997; 25(4): 479 - 485.

21. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthop Rev* 1989;18(9):963-975.

22. Jorgensen U, Bak K. Shoulder instability. Assessment of anterior-posterior translation with a knee laxity tester. *Acta Orthop Scand* 1995;66(5):398-400.

23. Kammer CS, Young CC, Niedfeldt MW. Swimming Injuries and illnesses. *The Physician Sports Med* 1999;27(4):51-60.

24. Kennedy JC, Hawkins R. Swimmers shoulder. *Physician Sports Med* 1974;2(4):34-38.

25. Kennedy JC, Hawkins R, Krissoff WB. Orthopaedic manifestations of swimming. *Am J Sports Med* 1978;6(6):309-322.

26. Kubo K, Kaneshisa H, Kawakami Y, Fukunaga T. Elastic properties of muscle-tendon complex in long-distance runners. *Eur J Appl Physiol* 2000;81(3):181-187.

27. Kvitne RS, Jobe FW, Jobe CM. Shoulder instability in the overhand or throwing athlete. *Clin Sports Med* 1995;14(4):917-935.

28. Leadbetter WB. Cell-matrix response in tendon injury. *Clin Sports Med* 1992;11:533-578.

29. L'Insalata JC, Warren RF, Cohen SB, Altchek DW, Peterson MG. A self-administered questionnaire for assessment of symptoms and function of the shoulder. *J Bone Joint Surg Am* 1997;79:738-748.

30. Lippitt SB, Harris SL, Harryman DTII, et al. In vivo quantification of the laxity of normal and unstable glenohumeral joints. *J Shoulder Elbow Surg* 1994;3:215-223.

31. McMaster WC. Painful shoulder in swimmers: A diagnostic challenge. *Physician Sports Med* 1986;14(12):108-122.

32. McMaster WC. Anterior glenoid labrum damage: a painful lesion in swimmers. *Am J Sports Med* 1986;14(5):383-387.

33. McMaster WC. Diagnosing swimmer's shoulder. *Swimming Technique* 1987 (February-April):17-24.

34. McMaster WC, Roberts A, Stoddard T. A correlation between shoulder laxity and interfering pain in competitive swimmers. *Am J Sports Med* 1998;26(1):83-86.

35. McMaster WC, Troup J. A survey of interfering shoulder pain in United States competitive swimmers. *Am J Sports Med* 1993;21(1):67-70.

36. Neer CS, 2nd. Impingement lesions. *Clin Orthop* 1983(173):70-77.

37. Neer CS, 2nd, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. A preliminary report. *J Bone Joint Surg Am* 1980;62(6):897-908.

38. Neer CS, 2nd, Welsh RP. The shoulder in sports. *Orthop Clin North Am* 1977;8(3):583-591.

39. Nirschl RP. Rotator cuff tendinitis: basic concepts of pathoetiology. *Instr Course Lect* 1989;38:439-445.

40. O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. *Am J Sports Med*. 1998;2:610-3.
41. Patterson-Kane JC, Firth EC, Parry DA, Wilson AM, Goodship AE. Effects of training on collagen fibril populations in the suspensory ligament and deep digital flexor tendon of young thoroughbreds. *Am J Vet Res* 1998;59(1):64-68.
42. Pink MM, Tibone JE. The painful shoulder in the swimming athlete. *Orthop Clin North Am* 2000;31(2):247-261.
43. Pollard B. *The Prevalence of Shoulder Pain in Elite British Swimmers and the Effects of Ttraining Technique*. B.Sc Thesis, University of Huddersfield, (unpublished) 2001.
44. Renstrom PA, Johnston RJ. Overuse injuries in sports: A review. *Sports Med* 1985;2:316-333.
45. Richardson AB, Jobe FW, Collins HR. The shoulder in competitive swimming. *Am J Sports Med* 1980;8(3):159-163.
46. Rowe CR, Zarins B. Recurrent transient subluxation of the shoulder. *J Bone Joint Surg Am* 1981;63(6):863-872.
47. Sauers EL, Borsa PA, Herling DE, Stanley RD. Instrumented measurement of glenohumeral joint laxity and its relationship to passive range of motion and generalized joint laxity. *Am J Sports Med* 2001;29(2):143-150.
48. Schneeberger AG, Nyffeler RW, Gerber C. Structural changes of the rotator cuff caused by experimental subacromial impingement in the rat. *J Shoulder Elbow Surg* 1998;7(4):375-380.

49. Scott A, Cook JL, Hart DA, Walker DC, Duronio V, Khan KM. Tenocyte responses to mechanical loading in vivo: a role for local insulin-like growth factor 1 signaling in early tendinosis in rats. *Arthritis Rheum* 2007;56(3):871-881.

50. Scott A, Khan KM, Duronio V. IGF-1 activates PKB and prevents anoxic apoptosis in Achilles tendon cells. *J Orthop Res* 2005;23(5):1219-1225.

51. Sein ML, Appleyard RC, Walton JR, Bradley T, Murrell GA. Reliability of a new shoulder laxometer to assess inferior glenohumeral joint translation. Br J Sports Med. 2008 Mar;42(3):178-82. Epub 2007 Aug 30.

52. Sein ML, Walton J, Linklater J, Harris C, Dugal T, Appleyard R, Kirkbride B, Kuah D, Murrell GA. Reliability of MRI assessment of supraspinatus tendinopathy. Br J Sports Med. 2007 Aug;41(8):e9. Epub 2007 Feb 8.

53. Silliman JF, Hawkins RJ. Classification and physical diagnosis of instability of the shoulder. *Clin Orthop* 1993(291):7-19.

54. Soslowsky LJ, Carpenter JE, DeBano CM, Banerji I, Moalli MR. Development and use of an animal model for investigations on rotator cuff disease. *J Shoulder Elbow Surg* 1996;5(5):383-392.

55. Soslowsky LJ, Thomopoulos S, Esmail A, Flanagan CL, Iannotti JP, Williamson JD 3rd, Carpenter JE. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. *Ann Biomed Eng* 2002;30(8):1057-1063.

56. Soslowsky LJ, Thomopoulos S, Tun S, Flanagan CL, Keefer CC, Mastaw J, et al. Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. *J Shoulder Elbow Surg* 2000;9(2):79-84.

57. Tibone JE, Jobe FW, Kerlan RK, Carter VS, Shields CL, Lombardo SJ, Yocum LA. Shoulder impingement syndrome in athletes treated by an anterior acromioplasty. *Clin Orthop* 1985;(198):134-140.

58. Tzannes A, Murrell GAC. Clinical examination of the unstable shoulder. *Sports Med* 2002;32(7):1-11.

59. Tzannes A, Paxinos A, Callanan M, Murrell GA. An assessment of the interexaminer reliability of tests for shoulder instability. *J Shoulder Elbow Surg* 2004;13(1):18-23.

60. US Bureau of Labour Statistics, *Occupational Injuries and Illnesses in the United States by Industry*. Bulletin 2368, 1990 (August).

61. Viidik A. The effect of training on the tensile strength of isolated rabbit tendons. *Scand J Plast Reconstr Surg* 1967;1(2):141-147.

62. Walton J, Mahajan S, Paxinos A, Marshall J, Bryant C, Shnier R, Quinn R, Murrell GA. Diagnostic values of tests for acromioclavicular joint pain. *J Bone Joint Surg Am*. 2004;86(4):807-812.

63. Wang F, Murrell GA, Wang MX. Oxidative stress-induced c-Jun N-terminal kinase (JNK) activation in tendon cells upregulates MMP1 mRNA and protein expression. *J Orthop Res* 2007;25(3):378-389.

64. Warren RF. Subluxation of the shoulder in athletes. *Clin Sports Med* 1983;2(2):339-54.

65. Woo SL RM, Amiel D, Sanders TM, Gomez MA, Kuei SC, Garfin SR, Akeson WH. The biomechanical and biochemical properties of swine tendons: long term

effects of exercise on the digital extensors. *Connect Tissue Res* 1980;7(3):177-183.

66. Yuan J, Murrell GA, Wei AQ, Wang MX. Apoptosis in rotator cuff tendonopathy. *J Orthop Res* 2002;20(6):1372-1379.

67. Zemek MJ, Magee DJ. Comparison of glenohumeral joint laxity in elite and recreational swimmers. *Clin J Sport Med* 1996;6(1):40-47.

Legends for Tables and Figures

Table 1: MRI shoulder protocol. The swimmers underwent an MRI of a single shoulder scanned, either the dominant shoulder if asymptomatic or the affected shoulder if symptomatic. Oblique coronal proton density (PD) and fat suppressed T2, Sagittal T2 and axial PD sequencing was performed on a Signa 1.5 Tesla superconducting magnet, Hi Speed MRI unit (General Electric Medical Systems, Milwaukee, Wisconsin), using system software 9.1, slew rate 77 Tesla/meter/sec, 33-millimetre Tesla gradient amplitude, utilizing high resolution, non-arthrographic technique with a 4-channel phased array shoulder coil (Medical Advances, Milwaukee, Wisconsin).

Table 2: Elite swimming training profiles.

Table 3: Clinical examination results.

Figure 1. Number of years with coaching, median 8 years.

Figure 2. Number of hours swum per week, median 16 hours.

Figure 3. Number of kilometers swum per week, median 40 kilometers.

Figure 4. Percentages of the total swimming training time spent with each of the four main strokes.

Figure 5. MRI-determined supraspinatus tendinosis in elite swimmers.

Figure 6. The relationship between competition level and supraspinatus tendinosis in elite swimmers (r_s = 0.56, $p < 0.00005$, using Spearman's rank order correlation).

Figure 7. The relationship between hours swum per week and supraspinatus tendinosis in elite swimmers (r_s=0.39, $p<0.005$, using Spearman's rank order correlation).

Figure 8. The association between kilometres swum per week and supraspinatus tendinosis in elite swimmers (r_s=0.34, p=0.01 using Spearman's rank order correlation).

Figure 9. A diagram showing how tendinopathies may arise.

Table 1. MRI protocol

	Oblique coronal proton density	Coronal fat suppressed sagittal T2	Sagittal T2	Axial proton density	Axial proton density fat suppressed
Swimmers	Adduction	Adduction	Adduction	Adduction	Adduction
Position	neutral rot.	neutral rot.	neutral rot.	neutral rot.	neutral rot.
FOV (cm)	13	13	13	15	15
TR (ms)	3500	3500	3500	3500	3500
TE (eff) (ms)	34	90	50	34	34
Slice thickness	3	3	3	3	3
Matrix size	512 x 256	256 x 192	512 x 256	384 x 320	384 x 256
Echo train(ET)	8	10	10	8	8
Bandwidth (kHz)	25	20	20	20	2
NEX	2	3	2	2	2

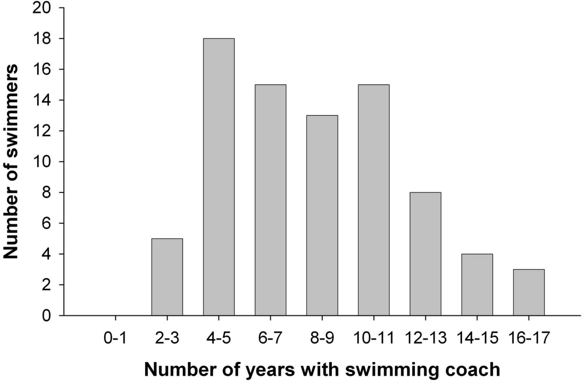
FOV = field of view; TR = repetition time; TE (eff) = Echo time (effective); NEX = no. of excitations

Table 2. Elite swimming training profiles

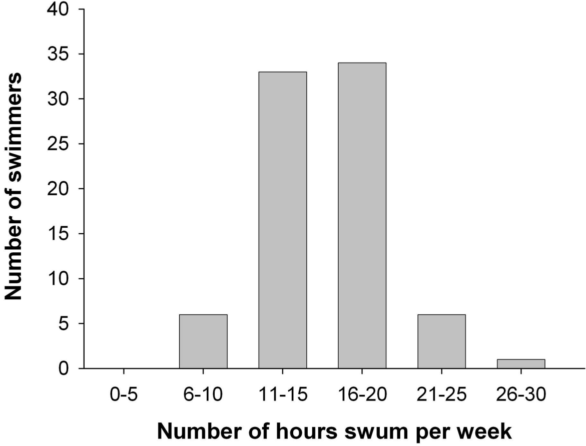
	Percentage of training time		
Swimming Strokes	Minimum (%)	Maximum (%)	Mean (%)
Freestyle	25	95	53
Butterfly	0	40	13
Backstroke	2	70	21
Breaststroke	0	50	13

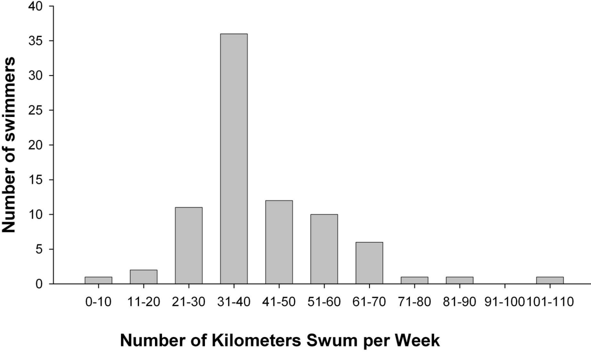
Table 3. Clinical examination results

	Positive	Negative
Drop arm sign	0 (0%)	52 (100%)
Impingement (IR)	44 (85%)	8 (15%)
Impingement (ER)	19 (37%)	33 (64%)
Paxinos sign	23 (44%)	29 (56%)
O'Brien's sign	16 (31%)	36 (69%)
Apprehension sign	23 (44%)	29 (56%)
ER Lag	5 (10%)	47 (90%)
Ab ER Lag	5 (10%)	47 (90%)
Speed's	10 (19%)	42 (81%)
Cross Arm	13 (25%)	39 (75%)
Scoliosis	2 (4%)	50 (96%)

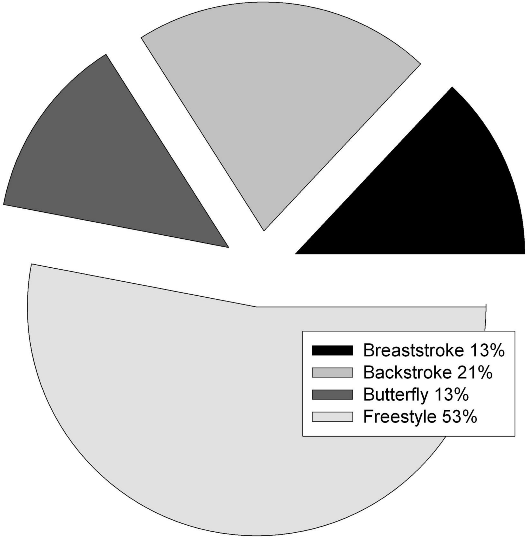


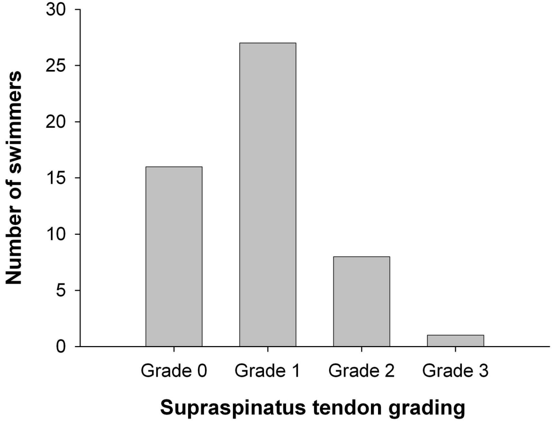
Number of swimmers
20
18
16
14
12
10
8
6
4
2
0
0-1
2-3
4-5
6-7
8-9
10-11
12-13
14-15
16-17
Number of years with swimming coach

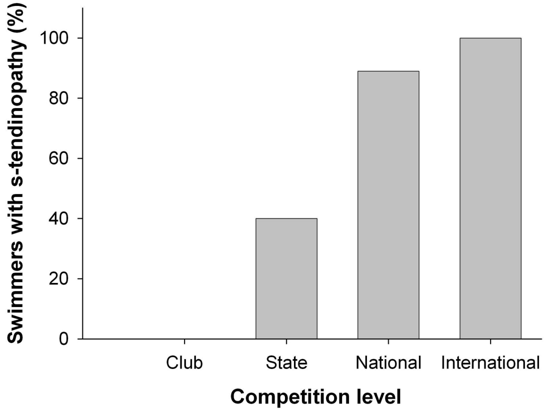

40
35
30
25
20
15
10
5
0
Number of swimmers
0-5
6-10
11-15
16-20
21-25
26-30
Number of hours swum per week

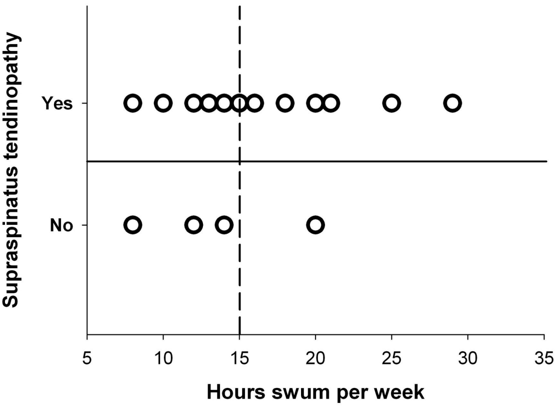


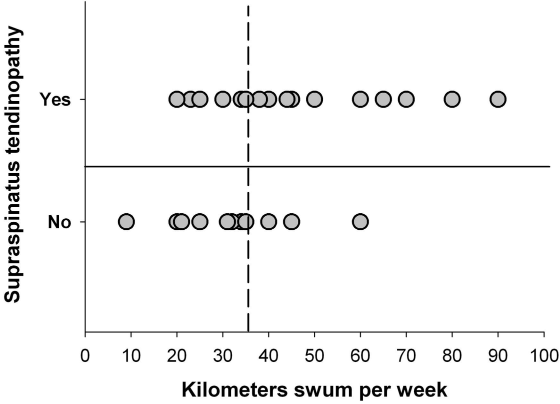
40
35
30
25
20
15
10
5
0
Number of swimmers
0-10
11-20
21-30
31-40
41-50
51-60
61-70
71-80
81-90
91-100
101-110
Number of Kilometers Swum per Week

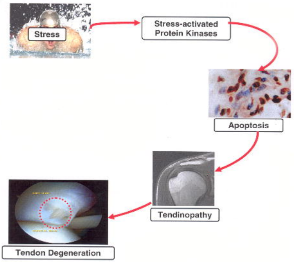

Breaststroke 13%
Backstroke 21%
Butterfly 13%
Freestyle 53%


30
25
20
15
10
5
0
Number of swimmers
Grade 0
Grade 1
Grade 2
Grade 3
Supraspinatus tendon grading


Swimmers with s-tendinopathy (%)
100
80
60
40
20
0
Club
State
National
International
Competition level


Supraspinatus tendinopathy
Yes
No
5
10
15
20
25
30
35
Hours swum per week


Supraspinatus tendinopathy
Yes
No
0
10
20
30
40
50
60
70
80
90
100
Kilometers swum per week



Stress
Stress-activated
Protein Kinases
Apoptosis
Tendinopathy
Tendon Degeneration