E-ISSN: 3068-3726
Journal of Orthopaedic Science and Research

ATHENAEUM SCIENTIFIC PUBLISHERS

Review Article

Muscle Fat Infiltration, Atrophy and Glenohumeral Rotator Cuff Pathology Associations

Ray Marks[1*]

[1]Osteoarthritis Research Center, Canada

***Correspondence author:** Ray Marks, Osteoarthritis Research Center, Canada; Email: rm226@columbia.edu; Dr.RayMarks@osteoarthritisreserachcenter.com

Citation: Marks R. Muscle Fat Infiltration, Atrophy and Glenohumeral Rotator Cuff Pathology Associations. J Ortho Sci Res. 2025;6(2):1-13.

https://doi.org/10.46889/JOSR.2025.6209

Received Date: 08-07-2025

Accepted Date: 22-07-2025

Published Date: 31-07-2025

Abstract

Osteoarthritis, a disabling disease induces high degrees of disability among many older adults in all parts of the globe despite years of study, advanced surgical joint replacement and remedial procedures. Deemed incurable, it now appears it may be possible to mitigate this condition wherein an increasing role is observed for muscle atrophy, as well as the encroachment or expansion of excess fatty tissue into vulnerable or injured muscles or tendons. This report focuses on observations from studies examining rotator cuff muscle dysfunction, lesions or damage and its potential impact on shoulder arthritis, but may be overlooked. In this regard, it appears that with few exceptions and regardless of article examined a possible bi-directional role for muscle fat mass infiltration or presence alongside muscle atrophy that may have a strong bearing on the shoulder osteoarthritis disability cycle. Often overlooked, we conclude muscle fat infiltration appears of high clinical significance in influencing the disease progression and its observed reversibility challenges.

Keywords: Fatty Infiltration; Muscle; Muscle Atrophy; Muscle Mass; Osteoarthritis; Rotator Cuff; Shoulder

Introduction

The disabling joint condition termed osteoarthritis remains an intractable problem affecting many older adults in all parts of the world despite decades of study and investigations of the many possible causes thereof. While no successful intervention approach has been universally forthcoming, a highly important role for muscle as a disease precursor, modifier or mediator has been emerging for some time. This includes but is not limited to a specific condition termed sarcopenia and denoting muscle wasting, as well as alterations in the desired muscle fat ratio due to excess fat accumulation. Their unique and interactive role as consistent features in many osteoarthritis cases and the knowledge that their presence may expose the associated joint tissues and cartilage lining to undue injurious impacts and forces is hence a key focal point of multiple current pathogenic oriented studies. In addition, associated muscle volume declines may have enormous functional implications for joint stability, joint mobility, joint proprioception and pain that may all clearly exacerbate osteoarthritis disability significantly and progressively in their own right [1-7].

To this end, this brief specifically examines, a) the notion of a subnormal muscle fat ratio as a feature or predictor of osteoarthritis in general, b) possible insights from rotator cuff shoulder related studies, c) the proposed mechanisms for fatty infiltration other than age and obesity at the shoulder joint. It was felt this information would be of interest to orthopaedic clinicians and therapists as well as those who contemplate surgical solutions for shoulder osteoarthritis, as well as in other shoulder surgical needs. It was anticipated the literature review would reveal a variety of studies highlighting the potential impact of excess muscle fat mass encroachment on muscle fibrosis and atrophy in the face of joint injury and possibly pathology and surgery. One or more shoulder rorator cuff muscle studies were selected for review rather than all shoulder dysfunction sources as these commonly present enormous degrees of disablement and treatment challenges due to muscle atrophy and degeneration, fatty infiltration, immense pain and muscle fibrosis [8-11]. Here Merriman, et al., point out that that “fat infiltration” has been used to described fatty changes in muscle after rotator cuff tears, but it is possible fat expansion is a more appropriate descriptor for the appearance

of fatty rotator cuff tear pathological changes [12,13].

Drawn largely from the PUBMED, database, the largest peer reviewed medically oriented database, the overview aimed to provide the interested reader a general view of past work as well as current trends and gaps plus opportunities in the orthopedic and aging health care field.

The focus was on shoulder osteoarthritis, one mode of this condition assuming epidemic proportions [possibly due to increases in shoulder athletic or occupational associated injuries in young adult populations] and which may occur independently or in conjunction with one or more chronic health conditions. Responsible for most daily functional needs, a painful shoulder joint may pose an enormous activity obstacle and deterrent to successful aging goals due to its many adverse impacts on life quality, sleep quality and daily functioning, plus immense self-care challenges and socioeconomic losses. While the world awaits a possible antidote in this regard, mounting evidence points to a possible role for the presence of muscle fat and its expansion post injury as well as aging impacts as a causative factor.

Although clinical research in this realm is however limited with the exception of a widespread focus on shoulder repair or replacement surgery, not all older adults at high risk for shoulder dysfunction can withstand surgery. Those who exhibit high muscle fat volumes also pose multiple post operative challenges in a significant proportion of cases, regardless of age [14-16]. As such, if found to be influential in any way, intervention in this regard may provide one avenue that is reasonably practical for purposes of securing the well-being of the aging person, especially in the case of the chronically ill older adult that lives in the community and must be self reliant.

This work is significant because the ability to minimize osteoarthritis severity is currently of the highest import, especially among those older adults who wish to reside in the community zx well as providers as regards challenges and costs.

Specific Aims

This overview aimed to specifically examine the value of joint protection for minimizing the risk of acquiring and suffering from progressive bouts of osteoarthritis pain that are potentially compounded by declines in muscle volume and increases in muscle fat mass, for example at the shoulder, a non-weightbearing joint, but one essential for most life functions. Secondly, it aimed to offer insight into the processes underpinning the presence of muscle fat on a joint, an emergent topic of increasing interest. Third it aimed to provide recommendations for future consideration by clinicians and researchers in the field based on these findings.

Materials and Methods

To examine this issue we elected to employ the PUBMED, PubMed Central and Google Scholar data bases largely using the 'best match' and 'most current' prompts and covering data published largely between January 2010-June 30 2025, using the key words: osteoarthritis, shoulder, muscle, muscle fat mass, atrophy and infiltration. Only articles focusing on osteoarthritis and some form of shoulder muscle fat presence in various shoulder cuff lesions were deemed acceptable. Described in narrative form, are some general results of studies of all types that have chosen to examine aspects of the muscle fat fraction relative to lean muscle mass ratios in osteoarthritis or shoulder injury contexts using those data that pertain to one or more rotator cuff muscles which are four upper shoulder muscles (supraspinatus, infraspinatus, teres minor, subscapularis) and their tendons designed to permit shoulder movements and stability via their attachments to the upper aspect of the main shoulder bone or humerus. No systematic review was conducted, however and points made are those that have emerged over time and comport with the author's 25 years of research showing high degrees of obesity in cases with disabling hip and knee osteoarthritis, many young adults as well as older adults with shoulder dysfunction and the fact that muscle fat mass volumes that are deemed 'unhealthy' are commonly seen in many young adults who are otherwise healthy as demonstrated by body impedance measures. The article is based on careful data extraction and author selection and no AI technology was employed at any point. The content chosen focuses largely on intramuscular fat mass, as opposed to subcutaneous fat and muscle atrophy or mass as this realm of exploration is quite new and might prove insightful in understanding arthritis manifestations in the frail underweight person, as well as in the importance of primary prevention efforts to counter progressive disabling in the overweight adult.

Accordingly, this narrative review first highlights some general current 2025 reports and some reported recently that allude to an array of observed muscle influences or responses in the osteoarthritis disease cycle, rather than all those that have been

published to date. Second, studies that examine muscle fat attributes as this pertains to osteoarthritis as well as the traumatized shoulder joint are discussed. Third, some emerging evidence of a key role for preventive health behaviors and injury prevention approaches as a mediating or moderating pathogenic factor in this respect is discussed. The items chosen were those thought to have a strong bearing on pain and the related hallmark of osteoarthritis, namely cartilage tissue degeneration and destruction and ensuing functional declines as experienced in the case of shoulder arthritis. Reported on are largely publications listed as of June 30, 2025 that best matched or were potentially relevant to the current topic only selected after a careful examination of their contents and if they had support from other related studies and joints of the knee and hip that have been studied and addressed one of the author's premises enumerated below [17-20].

Excess muscle fat mass may adversely impact articular cartilage structural features as well as muscle composition, strength, and joint stability.
A failure to prevent repetitive or acute injuries may cause a reactive form of degrading arthritis in its own right and one involving muscle composition abnormalities
Obesity, aging and other chronic inflammatory diseases are associated with increased fat mass and decreased muscle mass and function that may invoke joint damage.
Lean muscle mass declines and increases in muscle fat ratios may provoke and perpetuate an array of unwanted joint functional outcomes and pain provoking adaptations both before as well as after surgical joint replacement.

Table 1: Patient details.

Findings

In terms of the topic specifically sought, it is safe to say most listed publications that initially appeared potentially relevant did not meet the desired criteria for this review that excluded invasive intervention discussions, diagnostic imagery studies, proposals for study or studies on other forms of shoulder dysfunction other than rotator cuff lesions. Moreover, among those deemed relevant, few were based on prospective or targeted studies of the older adult with forms of osteoarthritis other than the knee and hip and even then these were predominantly observational studies conducted with varied measurement tools, diverse imaging approaches, timing and samples. These were also generally non uniform in multiple design respects, exploratory or atheoretical, not necessarily commensurate with the human condition or only in the proposal rather than assessment stage. Bearing all the above limitations three categories of publication emerged as follows.

a. *General observations:* As outlined in increasing numbers of global reports the aging population and their common declines in wellbeing and challenges including those due to osteoarthritis remain a foremost public health issue seeking resolution. In this regard, past research shows that many aging adults who have a high risk of incurring sarcopenia and related muscle mass and strength declines often go on to manifest severe osteoarthritis, but this is not the only determinant. A disease with enormous disabling features in its own right, health conditions such as sarcopenic obesity, where lean muscle mass is replaced by fat mass, many with painful disabling osteoarthritis, as well as direct or chronic injuries are also potential factors. Others include obesity, chronic pain and often a reluctance to exercise even if advocated. The muscles around an osteoarthritic joint may undergo multiple degenerative features that also have a bearing on the emergent movement patterns, for example at the diseased shoulder joint pervasive mixotrauma incidents may induce intrinsic muscle fat impact alterations that perpetuate muscle dysfunction and pathology [11].
b. *Muscle fat mass observations:* Increasing numbers of studies confirm muscle fat deposition or infiltration may be a potent osteoarthritis disease muscle mediator [eg., 21-24]. Indeed, while neither well documented, nor universally applied to a degree commensurate with its possible diverse pathogenic disease ramifications, attributes and associations, shoulder muscle abnormalities potentially explain and may predict diverse aspects of the shoulder osteoarthritis progression phases. As distinct from obesity and its implications for both joint loading and inflammation, an independent role for muscle adipose tissue composition alterations including muscle fat deposition and possible changes in muscle mass or sarcopenia [a progressive muscle mass declining state] may not only impact mobility but may set the stage for inactivity and muscle fat fraction increases [12,25-27]. Others show a role for deforming contractures, varying degrees of muscle spasm and subnormal

vector influences, plus functional changes in muscle biochemistry and muscle size [28] that have a unique or collective bearing on cartilage viability, and that may implicate or lead to muscle fat excesses and infiltration as well as deficits in lean muscle mass and muscle fibrosis [29,30].

Other data imply that there may be a progressively harmful adverse degree of joint loading that subsequently manifests as fully fledged osteoarthritis, as various degrees of muscle pathology and disordered biochemical expression and/or possible fears of moving due to pain. Collectively these factors have the potential to foster subnormal muscle forces and joint loading or attenuation responses, joint stability and the ability of an injured muscle to regenerate. As outlined by Chen, et al., osteoarthritis is a disease of joint degeneration and impaired function where muscle atrophy, fatty infiltration and fibrosis are degenerative features of muscle injury and predict poor outcomes in some instances [31]. Unsurprisingly, patients with glenohumeral joint osteoarthritis usually exhibit rotator cuff muscle degeneration, even though the rotator cuff may remain intact.

c. Rotator cuff fat mass attributes: Rotator cuff tears, also referred to as rotator cuff ruptures or tendon tears, represent prevalent orthopedic injuries associated with substantial discomfort and functional impairment and possible arthritis, fat accumulation within the muscle and muscle atrophy have been correlated with high re-tear rates after surgical repair of the rotator cuff possibly due to increased immune cell crosstalk and interactions with the extracellular matrix [9-12].

Furthermore, fatty infiltration of the rotator cuff evaluated with computerized tomography has been associated with asymmetric glenoid or shoulder joint cavity wear as well as humeral head subluxation in those patients with confirmed glenohumeral arthritis. This relationship arguably plays an important role in determining the optimal surgical management of advanced glenohumeral osteoarthritis, as well as its management in general. Compared with concentric forms of wear, posteriorly worn glenoids appear to have an imbalance in axial-plane rotator cuff fatty infiltration and an increased amount of fatty infiltration of the infraspinatus and teres minor compared with the subscapularis. The authors suggest these imbalances may contribute to the higher rates of failure after anatomic total shoulder arthroplasty in patients with posterior wear compared with those with concentric wear, but may also cause initial damage to a vulnerable joint [31].

Other data show rotator cuff muscle degeneration, bone morphology and humeral head subluxation are known risk factors for failure of anatomic total shoulder arthroplasty in patients with B-glenoid shoulder osteoarthritis [32]. Others reveal observations of prevailing or emergent muscle fat associated metabolic muscle alterations, alterations in muscle quality, volume, mitochondrial muscle energetic alterations and/or myopathy in the face of any perpetual state of undue soft tissue damage, persistent joint stresses, pain and signs of muscle weakness. According to Merriman, et al., it appears numerous articles have supported the role of muscle resident fibroadipogenic cells as a contributor to rotator cuff tear pathological changes, suggesting aime firm of regenerative engineering solution may hence improve the pathological changes associated with rotator cuff tears [12]. However, without insightful intervention, there may be progressive and emergent indications of a gradually diminishing joint range of motion and stiffness, plus an abnormal degree of muscle related joint biomechanics, plus an ongoing cycle of disordered joint destruction and inflammation [33-36].

In cases where rotator cuff tears prevail in particular, these are commonly accompanied by fatty infiltration or expansion of the associated muscles to varying degrees depending on the tear pattern and that can substantially limit daily activities and affect quality of life. Ample research also shows that after a tendon tear, the rotator cuff muscle will commonly undergo fatty infiltration within and around the muscles as well as declines in muscle volume often associated with poorer functional and surgical outcomes after reconstruction surgery [37,38]. In this regard, the etiology of rotator cuff disease is likely to be a multi-factorial one due to age-related degeneration, genetics, high choledterol levels, smoking, micro- and macro trauma Those classified as having full-thickness rotator cuff tears tend to show a progressive fat encroachment that enlarges over time, as does pain or worsening pain (Fig. 1) [39].

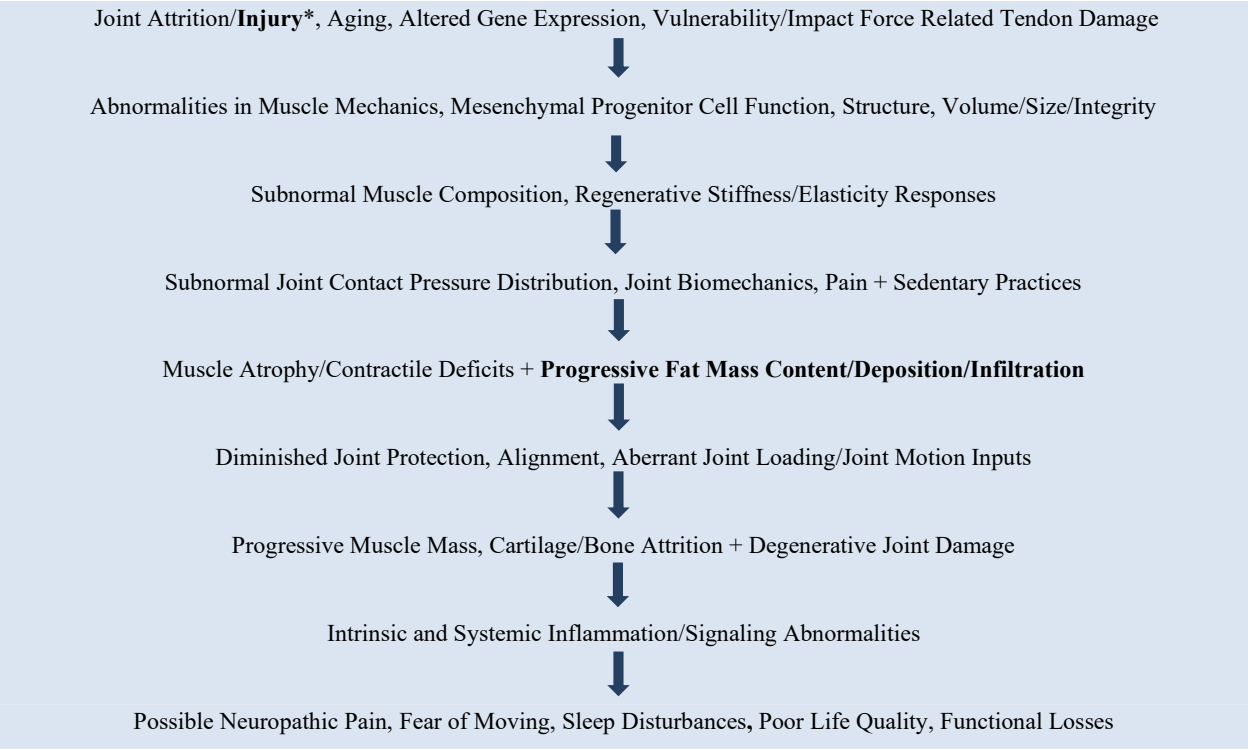

Figure 1: Hypothetical upstream and downstream rotator cuff injury, pathology, pain and muscle fat deposition factors-depicted linearly but where all features are potentially interactively associated in the long term [10,17,26,36-37,40-46]. Note *- denotes injury degree that yields 'dose' response poor outcome trends.

In essence, this envisioned series of adverse interactions including heightened decrements in muscle force capacity and contractile responses and others may induce or be mediated by various degrees of muscle fat encroachment and/or enlargement plus progressively subnormal joint biodynamical, structural changes in muscle architecture, mobility and functional ability and largely irreversible osteoarthritis joint destruction processes [42]. As well, this idea is consistent with known age associated degrees of the gradual onset of subnormal muscle metabolic physiological states, losses of lean muscle mass, increasingly sedentary behaviors, those comorbid osteoarthritis associated health conditions such as diabetes and the loads needed for muscle regeneration that are increasingly excessive in the face of associated muscle volume declines [43]. In addition, there may be a reactive or causative array of intrinsic muscle cell mesenchymal alterations that prevent, inhibit or impair muscle repair [44].

Underlying local factors may also include possible painful muscle reactive adaptations due to persistent abnormal sensory inputs from one or more of the surrounding tissues in the presence of an increased muscle fat mass and that may impact the ability to attenuate joint loads significantly and effectively. Also known as myosteatosis, its presence in muscle exhibits a negative correlation with muscle mass, strength, mobility and a decrement in muscle quality, while serving as a biomarker for sarcopenia, cachexia and metabolic syndromes. It also induces pro-inflammatory changes that clearly contribute to declines in muscle function, compromise mitochondrial function and increase muscle inflammatory responses and possibly postural stability [35,46]. Worse effects occur in cases of higher muscle fat content and area presence [47].

Others show the presence of muscle fat derived inflammation that may not only evoke local muscle pain signals, but may elicit more widespread pain, bone attrition and sensory sympathetic inputs that contribute to the arthritis disease and disability cycle [48]. It also appears both externally or extrinsic as well as internally or intrinsic induced muscle fat infiltration and subsequent fibrosis may have a separate or combined cumulative and progressive effect on osteoarthritis structural pathology of the shoulder joint and others and regardless of whether this situation is causative or reactive [49]. Moreover, unless identified early on, it may be highly challenging to heighten the subject's physical function or relieve longstanding pain, especially if there is progressive muscle atrophy plus a possible unwillingness or fear on the part of the affected adult to move their arm to counter increasing pain and stiffness, alongside muscle mass and sensory declines and joint stability deficits that may influence them to adopt abnormal and awkward movements to avoid pain and injury but that may place strain on the joints, further pain and possible

diverse muscle associated adverse structural alterations that may vary depending on the degree of muscular fat infiltration [49]. Other muscle based determinants that may be related to the degree of muscle fat encroachment and presence involved in osteoarthritis include sarcopenic obesity [42]. As well, there is the possible development of a muscle nerve entrapment process by osteoarthritis bone spurs, muscle fibrosis, muscle inflammation, joint instability and an altered cartilage structure with the added risk of incurring more extensive joint pathology and pain than those generated in the presence of sufficient fat free lean muscle mass [50,56].

Adding to this subnormal cycle is a possible role for osteoarthritis associated muscle fat infiltration impacts on muscle metabolic alterations alongside widespread pain, atrophic muscle weaknesses, muscle volume deficits, inflammation and muscle architecture alterations [57,58]. At the shoulder joint, it appears muscle resident fibroadipogenic cells do serve as a key a contributor to rotator cuff tear pathological changes that cause immense pain and disability [13].

While mechanical injury is the most likely cause of osteoarthritis in most instances, emerging evidence showing functional ability declines in the presence of excess muscle fat could be causative or reactive or both. As well, those cases displaying atrophic muscle weakness are likely to also display disruptions in muscle genetics and muscular alterations such as declines in muscle force capacity and responsiveness that may further amplify the pain and dysfunction attributable to the local condition even after surgery where muscle fatty tissue infiltration and deposition may emerge or persist [59,60].

Discussion

As per a number of diverse current and past studies, it can be concluded shoulder osteoarthritis and impaired function can be significantly influenced by muscle atrophy, fatty muscle infiltration and fibrosis usually accompanying rotator cuff muscle degeneration [31]. In addition, MRI type scans and analyses, further reveal increased fatty infiltration in the infraspinatus muscle with age [41]. Moreover, in full-thickness rotator cuff tears in human samples tears of all sizes show significantly greater lipid content and smaller myofiber cross-sectional areas compared with partial-thickness tears and control muscles [61]. As well, a preclinical study of rabbit supraspinatus tenotomy outcomes showed this artificial injury to recapitulate key features of the pathophysiology of human rotator cuff tears, including muscle atrophy and degeneration, a lack of regenerative ability, fat accumulation and fibrosis [13]. Indeed, despite years of study and insights, what remains is a high re-tear rate after a successful repair of the rotator cuff that is a major clinical challenge attributed to muscle atrophy and fat accumulation of rotator cuff muscles over time [62]. Moreover, after successful arthroscopic repair, even if tendon tear-induced fatty infiltration can be almost negated and muscle atrophy slightly reversed in the case of a failed repair, these changes are further pronounced during the first 3 postoperative months [63].

Gerber, et al., note that it appears that muscle changes that occur after tendon release, may be exacerbated in the presence of denervation of the muscle due to an ensuing decrease in the pennation angle of lengthened muscle fibers, with a reduced mean cross-sectional area of pooled muscle fibers, a slow- to fast-type transformation and an increase in the area percentage of hybrid fibers, leading to overall significantly greater atrophy of the corresponding muscle [45].

Remarkably, the pattern of fat infiltration within the supraspinatus muscle of the shoulder joint changes from a laterally based location around the muscle-tendon junction to a more diffuse, global infiltration pattern when the whole muscle fat content exceeds 10% according to Wallenberg, et al., [58]. Hence, more translational research on diverse osteoarthritis samples of higher ages may prove promising in stressing a role for fat mass correlates that can foster extensive alterations in muscle force capacity and immense irreparable disabling outcomes if overlooked.

In addition, even though the source of muscle fat infiltration and why this may enlarge and become pathogenic is not well clarified and nutrition and physical activity behaviors may clearly be of high import, basic preclinical studies speak to a series of adaptive responses in the face of joint damage including those induced by neurological nerve damage as one factor, even if this does not apply in all cases [17,45]. As well, even if fatty tissue can be removed artificially the underlying muscle abnormalities, for example fibrosis may prevail [17]. The rotator cuff tendons can degenerate and/or tear from the greater tuberosity of the humerus, which is associated with several anatomical, physiological, biochemical and molecular changes in tendon and muscle. immunobiological responses following the rotator cuff lesion and the inherent repair mechanisms elicited by the body. The greatest difficulty in treating this pathology is that the muscle can undergo irreversible fatty infiltration in the setting of chronic

tears that is associated with poor surgical outcomes. The article also investigates the key molecular pathways of the muscle homeostasis and energy metabolism to propose a possible mechanism for fatty infiltration [64]. The radiologic grading of muscle fat content was associated with the expression of various genes, including adipogenic, fibrotic, inflammatory and atrophy-related genes and these genes were closely correlated with each other in terms of expression [65].

Siso, et al., who studied patients with primary glenohumeral osteoarthritis awaiting total shoulder arthroplasty showed B3 glenoids had the greatest degree of fat presence of all rotator cuff muscles, implying a possible disabling correlation [14]. Although the cause effect association is unclear, high-grade rotator cuff muscle fatty infiltration is associated with B3 glenoids-subtype of arthritis, increased pathologic glenoid retroversion and increased joint-line medialization [25]. An additional thought is that posterior humeral head subluxation and glenoid retroversion, which are pathognomonic of the Walch type B shoulder, may lead to a disturbance in the length-tension relationship of the posterior rotator cuff, causing fatty infiltration [66].

Review of Key Points

What can be gleaned from the presently retrieved sources of peer reviewed information on osteoarthritis of the shoulder as well as osteoarthritis of other joints is as follows in our view:

i) How injury to a vital muscle group or its attachments to bone can independently as well as collectively induce joint attrition and immense immobilizing pain, as can the presence of excess muscle fat in its own right in cases of low muscle mass [42]

ii) The challenges in reversing the above interactions compound as time progresses. Moreover if this process is not halted or retarded in a timely manner reparative surgical interventions may fail

Indeed, failing to appreciate the complexity of what we do know about the triadic association of fatty tissue, sarcopenia and their osteoarthritis associations implies a lack of insightful analysis and intervention applications and/or especially a failure to appreciate the toxicity of muscle fat infiltration that may conceptually do much harm in multiple respects [42].

Fortunately, these clinical determinants of some forms of osteoarthritis do show this idea is not just theoretical but that fibro-adipogenic progenitors, a major population of resident muscle stem cells constitute the main source of intramuscular fibrosis and fatty infiltration that are key features of rotator cuff muscle degeneration and possible further joint tendon tears [23].

The implications here are that strategies to reduce injury exposure and others directed across adulthood towards enhancing or maintaining an optimally desired state of muscle mass, strength and endurance, plus efforts towards avoidance of repetitive or acute injurious joint loading activities appear highly indicated, as do routine body composition measures [42]. Muscle stress protection seems especially crucial as recent analyses reveal that in the presence of an injury, a hostile microenvironment characterized by heightened inflammation, fibrosis and muscle weakness may be forthcoming and is one shown to stimulate intramuscular adipose tissue expansion and impair its regeneration capacity and contractile functions especially in the face of a sedentary lifestyle [66].

In adults with rotator cuff tears and shoulder osteoarthritis, as well as instability, clinicians can track or look out for these features and work with the client to mitigate their impacts. Finally, rehabilitation programs targeting shoulder muscle function and pain should not neglect to examine shoulder proprioception that is showing promising initial results in restoring function and returning injured athletes to play.

At present, it appears safe to say that even though more solid uniform information on this topic is needed in multiple realms [25], most clinical researchers currently assume muscle problems of some sort underlie and impact osteoarthritis pain and if minimized will prove to have a bearing on osteoarthritis outcomes. However, distinctive efforts to study and address muscle fat deposition origins and impacts such as fears of movement, muscle deconditioning muscle strength declines, pain and pain behaviours are desirable due to their possible cross talk effect between fat and body tissues and pain [68-74]. Developing effective osteoarthritis preventive as well as treatment strategies using more advanced technologies and analyses can also help to greatly advance the field as cited by most current researchers, including those who conduct joint replacement surgery, especially at the shoulder joint [25].

Limitations

Although publication bias may prevail, these aforementioned observations are largely in agreement regarding a negative role for the presence of fatty tissue in muscles around a healthy as well as an osteoarthritis damaged joint. While not carefully explored to date, its enlargement in the face of poor management and non protective behaviors appears to have muscle as well as joint functional implications. At the shoulder, despite a lack of longitudinal study, it appears avoidance of an acute injury will favour the ability to resist muscle fat infiltration and/or its persistence and adverse signalling influences [75]. Hence although osteoarthritis is currently deemed a chronic progressively disabling condition with no known cure, research over the past 15 years or more has indicated that there is strong possibility that an array of muscle related factors that can contribute to the osteoarthritis pain and disease cycle and muscle fat infiltration perspectives appear to hold great promise, although not evident overall to date.

Moreover, while a diverse array of intervention approaches that focus on maximizing muscle structure and function appear advocated to potentially reduce the degree of disability in knee and hip osteoarthritis, that at the shoulder is rarely alluded to except for surgical research purposes. Muscle fat mass, a predictor of adverse joint events [68] alongside pain, regardless of its precise muscle site and disease severity is also not well articulated or even mentioned in the realm of shoulder exercise therapies.

This lack of attention to a topic of high explanatory and therapeutic potential with excellent preclinical model reports may inadvertently impact outcomes of life in general for many, especially those undergoing surgery to replace a diseased joint. Studies that combine both clinical as well as biopsy type analyses and those that foster well conceived sensorimotor, strength training and joint protection are likely to prove highly insightful and are urgently needed [10].

Additionally, symptomatic cases who do not receive careful instructions as to the considerable care they must take versus those who do should be studied. In this regard careful monitoring of the client and helping those with severe overweight to lose weight and avoid excessive muscle stretching and high frequency loading activities after periods of immobilization found to hasten cartilage destruction appear indicated and should be validated [75].

Conclusion

Although modern medicine has been quite successful in managing many acute health conditions and reducing infection and injury risk and magnitude in the older adult population, no similar simple solution prevails against osteoarthritis especially among those older adults who are possibly either over- or underweight. There may also be associated emergent signs of pain and need for surgery if nothing is done to avert preventable disease correlates. Meantime it appears:

a. High degrees of shoulder osteoarthritis impairment and suffering will persist among older adults if a role for muscle fat mass as both a precursor and progenitor remains fragmentary or unaccounted for
b. Dedicated efforts to assess and unravel the origins and profound impact of muscle fat mass deposition on shoulder joint health and how exercise may exacerbate this, rather than thwart this if poorly devised and implemented are indicated
c. Injuries involving macro or micro tears of the shoulder rotator cuff along with subsequent or pre-existing deconditioning and poor health and are poorly treated are likely to prove key contributors to shoulder osteoarthritis rather than age alone and warrant upstream and downstream prevention efforts
d. Research that examines the link between intramuscular fat and joint sensory functions and pain will prove insightful
e. A study of disease free older adults and how muscle fat affects joint loading and muscle regeneration and how this knowledge can be applied pre-emptively would also greatly advance the field

Current findings also strongly highlight the degree to which muscle fat infiltration and/or enlargement as well as muscle atrophy may play a disabling role in the osteoarthritis disease cycle, as well fostering multiple levels of focal and systemic dysfunction, severe pain and biomechanical and metabolic challenges. Moreover, at the shoulder it can be observed that although the temporal and causal relationships of these associations remain ambiguous, muscle atrophy and fat infiltration can be considered two discrete processes in the natural history of gleno humeral osteoarthritis and without due care and accurate assessments of fat extent, muscle joint stability, exercise may fail to achieve the desired outcome [41,69,79,80].

Indeed, it appears increasingly difficult to ignore a role for muscle fat mass deposition and/or its expansion as a potential pathogenic factor or co-factor in the cycle of progressive shoulder pathology and requiring attention even if surgery is

contemplated [4]. Additionally, it appears that rotator cuff muscle volume is significantly decreased in those adults with definitive osteoarthritis and can thereby impact outcomes including muscle force capacity and joint protection [60].

However, physical therapies that address this pathogenic disease feature may greatly help to mitigate this disabling condition especially if they encourage efforts to safely increase lean muscle mass and balanced well modulated movements and stability, while reducing pain and the risk of further joint destruction [50]. In particular, to avert rapid or excess disease progression and disability and its association with immense social and mobility-related losses, there appears to be an increasing body of research that supports the view that efforts to reduce muscle fat encroachment is a highly salient osteoarthritis disease correlate and possible efficacious mitigation strategy.

Unfortunately, since this idea is by no means a universally accepted or practice based one, more studies that tease out the possible relationship between muscle factors and osteoarthritic pain along with central factors that affect pain and muscle fat encroachment may enable progress especially if conducted over extensive time periods utilizing a variety of possible patient specific interventions. As shown in Fig. 1 efforts to untangle these interactive reactionary states may prove especially revealing and help in the design of optimal osteoarthritis rehabilitation plans as well as reducing the need for surgery and its oftentimes less than optimal outcomes. Similarly conservative treatments of the shoulder and their scope and sequence may be enhanced such that: 1) older adults and those at risk for excess reactive or age associated muscle atrophy declines or both will suffer less in multiple respecrs; 2). It may be possible to elucidate how adverse joint impact generated internally or externally or both can induce of exacerbate osteoarthritis joint damage; 3). while not definitive, prevention appears highly promising as an adjunctive approach to avert and ameliorate the magnitude of muscle mass declines and attrition generated by injury both macro and micro and osteoarthritis disease severity even in cases that undergo joint replacement surgery; 4) securing an ample or desired level of motion as regards muscle health in a controlled progressive manner and avoiding excessive joint stretches may yet help to protect against excess movement generation, age related joint damage and injury, while speeding up recovery.

In this sense, regardless of the great benefits of current forms of rotator cuff diagnostics and repair, concerted parallel efforts to educate as to the role of excess weight and protect against cartilage damage and injury due to acute injuries and/or chronic loading, including repetitive joint loading impacts may be of the highest import in allaying the onset or progression of excess muscle fat infiltration at the shoulder joint and others [81,82]. Another point is that the presence of any subnormal muscle composition has previously been linked not only to poor function, but comorbidity and increased hospitalization and all cause mortality [76].

However, to advance this field successfully, well-powered and carefully conceived long term clinical, as well as epidemiological and basically oriented studies that employ advanced technologies, careful subgroup allocation and healthy as well as impaired subjects are sorely needed and strongly encouraged. Until more is known, we further assert shoulder osteoarthritis determinants that affect many older adults adversely are possibly compounded by modifiable muscle fat mass invasion processes and/or reactive infiltration and enlargement that can evoke a variety of increasingly subnormal bone-muscle interactions.

Moreover, since the presence of sarcopenic obesity may play a major role in diminishing the health of older adults, neglecting or overlooking the relevance of possible associated increases in disordered muscle and tendon composition, especially that attributable to age as well as injury risk and related fatty infiltration or expansion processes may duly hasten the onset of progressive muscle weakness, dysfunction, neuropathic pain, cartilage failure and its disabling life quality and functional implications quite markedly and significantly [83,84].

By contrast, possible long lasting adverse clinical implications can be offset by early detection, careful follow ups and record keeping and primary preventive [including robotic arm assistance and protective devices], rather than relying on late end stage interventions where possible surgery is often fraught with its own challenges, persistent pain, muscle strength decreases and long recovery periods [75]. Careful primary and secondary preventive measures against poor outcomes may also affect the need for reparative surgery or enable more practical solutions that reduce the spread of muscle fat to unaffected shoulder cuff muscles in the case of further muscle attrition and inflammation that heightens sarcopenic obesity risk [76,77,83,85]. It may help avoid or reduce the negative impact of excess body weight as well as frailty and poor proprioception on muscle fat mass, especially among those deemed at risk [78].

Final Comments

Although seemingly an obscure topic in the realm of osteoarthritis mitigation- it is apparent much osteoarthritis suffering can be avoided or undone. In particular, a concerted desire to identify, track and examine muscle factors such as muscle fat mass in general in the realm of both osteoarthritis research and specific research at the shoulder joint-a common site of musculoskeletal pain, the design of optimal shoulder rehabilitation plans and their scope and sequence may well be vastly improved as well as far reaching at low cost and generate much success. However, as of 2025 with the exception of the knee joint, the predominant joint studied in this regard, its role, promise and life affirming potential remains speculative.

Conflict of Interests

The author declares no conflict of interest.

Funding/Sponsorship

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Consent for Publication

Not applicable

Acknowledgments

None.

References

1. Nguyen A, Lee P, Rodriguez EK. Addressing the growing burden of musculoskeletal diseases in the ageing US population: challenges and innovations. Lancet Healthy Longev. 2025.
2. Ma W, Chen H, Yuan Q. Global, regional and national epidemiology of osteoarthritis in working-age individuals: insights from the global burden of disease study 1990-2021. Sci Rep. 2025;15(1):7907.
3. Wang F, Cao Y, Lu H. Osteoarthritis incidence trends globally, regionally and nationally, 1990-2019: an age-period-cohort analysis. Musculoskeletal Care. 2025;23(1):e70045.
4. Wu Q, Xu Z, Ma X. Association of low muscle mass index and sarcopenic obesity with knee osteoarthritis: a systematic review and meta-analysis. J Int Soc Sports Nutr. 2024;21(1):2352393.
5. Collins KH, Lenz KL, Pollitt EN. Adipose tissue is a critical regulator of osteoarthritis. Proc Natl Acad Sci USA. 2021;118(1):e2021096118.
6. Okada S, Taniguchi M, Yagi M. Degeneration of the cartilage quality is correlated with a higher intramuscular fat infiltration of the vastus medialis in older adults with pre-to-mild knee osteoarthritis. Eur J Radiol. 2025;183:111930.
7. Svarnas G, Popa V, Patsiou TS. Postoperative muscle atrophy and fatty degeneration with respect to surgical approaches in total hip arthroplasty. Arch Orthop Trauma Surg. 2025;145(1):177.
8. Chalmers PN, Beck L, Miller M. Glenoid retroversion associates with asymmetric rotator cuff muscle atrophy in those with Walch B-type glenohumeral osteoarthritis. J Am Acad Orthop Surg. 2020;28(13):547-55.
9. Karapınar M, Ayyıldız VA, Unal M. Effect of intramuscular fat in the thigh muscles on muscle architecture and physical performance in the middle-aged women with knee osteoarthritis. J Orthop Sci. 2024;29(1):194-9.
10. Longo UG, Mazzola A, Magrì F. Histological, radiological and clinical analysis of the supraspinatus tendon and muscle in rotator cuff tears. BMC Musculoskelet Disord. 2023;24(1):127.
11. Liu W, Wang X, Yu Y. Inflammatory cell interactions in the rotator cuff microenvironment: insights from single-cell sequencing. Int J Genomics. 2025;2025:6175946.
12. Merriman MA Jr, Chapman JH, Whitfield T. Fat expansion not fat infiltration of muscle post rotator cuff tendon tears of the shoulder: regenerative engineering implications. Regen Eng Transl Med. 2025;11(1):1-14.
13. Vargas-Vila MA, Gibbons MC, Wu IT. Progression of muscle loss and fat accumulation in a rabbit model of rotator cuff tear. J Orthop Res. 2022;40(5):1016-25.
14. Siso D, Wee H, Ponnuru P. The association of rotator cuff muscle morphology and glenoid morphology in primary glenohumeral osteoarthritis. Shoulder Elbow. 2024:17585732241269193.
15. Wu HT, Liu Q, Lin JH. Fatty infiltration predicts retear and functional impairment following rotator cuff repair: systematic

review and meta-analysis. J Orthop Surg Res. 2025;20(1):303.
16. Weng Q, Jiang T, Yang T. Association of thigh intramuscular fat infiltration with incident knee and hip osteoarthritis: A longitudinal cohort study. Arthritis Rheumatol. 2025.
17. Norris AM, Fierman KE, Campbell J. Studying intramuscular fat deposition and muscle regeneration: insights from a comparative analysis of mouse strains, injury models and sex differences. Skelet Muscle. 2024;14(1):12.
18. Joseph GB, Akkaya Z, Sims WM. MRI-based analysis of thigh intramuscular fat and its associations with age, sex and BMI using data from the osteoarthritis initiative data. Sci Rep. 2025;15(1):6188.
19. Zhang X, Pan X, Deng L. Relationship between knee muscle strength and fat/muscle mass in elderly women with knee osteoarthritis based on dual-energy X-ray absorptiometry. Int J Environ Res Public Health. 2020;17(2):573.
20. Raynauld JP, Pelletier JP, Roubille C. Magnetic resonance imaging-assessed vastus medialis muscle fat content and risk for knee osteoarthritis progression: Relevance from a clinical trial. Arthritis Care Res. 2015;67(10):1406-15.
21. Inhuber S, Sollmann N, Schlaeger S. Associations of thigh muscle fat infiltration with isometric strength measurements based on chemical shift encoding-based water-fat magnetic resonance imaging. Eur Radiol Exp. 2019;3(1):45.
22. Eckers F, Loske S, Ek ET. Current understanding and new advances in the surgical management of reparable rotator cuff tears: a scoping review. J Clin Med. 2023;12(5):1713.
23. Agha O, Diaz A, Davies M. Rotator cuff tear degeneration and the role of fibro-adipogenic progenitors. Ann N Y Acad Sci. 2021;1490(1):13-28.
24. Aily JB, de Noronha M, Ferrari RJ. Differences in fatty infiltration in thigh muscles and physical function between people with and without knee osteoarthritis and similar body mass index: A cross-sectional study. BMC Musculoskelet Disord. 2025;26(1):109.
25. Donohue KW, Ricchetti ET, Ho JC. The association between rotator cuff muscle fatty infiltration and glenoid morphology in glenohumeral osteoarthritis. J Bone Joint Surg Am. 2018;100(5):381-7.
26. Papalia GF, Franceschetti E, Giurazza G. MicroRNA expression changes in the development of rotator cuff tendon injuries. JSES Rev Rep Tech. 2023;3(3):343-9.
27. Luo J, Xiang Q, Lin T. Associations between total and regional fat-to-muscle mass ratio and osteoarthritis incidence: A prospective cohort study. Osteoarthritis Cartilage. 2025.
28. Aleem AW, Chalmers PN, Bechtold D. Association between rotator cuff muscle size and glenoid deformity in primary glenohumeral osteoarthritis. J Bone Joint Surg Am. 2019;101(21):1912-20.
29. Grondin F, Freppel S, Jull G. Fat infiltration of multifidus muscle is correlated with neck disability in patients with non-specific chronic neck pain. J Clin Med. 2022;11(19):5522.
30. Wang L, Valencak TG, Shan T. Fat infiltration in skeletal muscle: Influential triggers and regulatory mechanism. iScience. 2024;27(3):109221.
31. Chen C, Zhou H, Yin Y. Rotator cuff muscle degeneration in a mouse model of glenohumeral osteoarthritis induced by monoiodoacetic acid. J Shoulder Elbow Surg. 2023;32(3):500-11.
32. Hartwell MJ, Harold RE, Sweeney PT. Imbalance in axial-plane rotator cuff fatty infiltration in posteriorly worn glenoids in primary glenohumeral osteoarthritis: an MRI-based study. Clin Orthop Relat Res. 2021;479(11):2471-9.
33. Elliott J, Jull G, Noteboom JT. Fatty infiltration in the cervical extensor muscles in persistent whiplash-associated disorders: a magnetic resonance imaging analysis. Spine. 2006;31(22):E847-55.
34. Daneshvarhashjin N, Debeer P, Matthews H. Covariation between rotator cuff muscle quality and shoulder morphometric bony features in B-glenoids: a statistical modeling approach. Biomech Model Mechanobiol. 2025.
35. Garcia-Diez AI, Porta-Vilaro M, Isern-Kebschull J. Myosteatosis: diagnostic significance and assessment by imaging approaches. Quant Imaging Med Surg. 2024;14(11):7937-57.
36. Ruoss S, Möhl CB, Benn MC. Costamere protein expression and tissue composition of rotator cuff muscle after tendon release in sheep. J Orthop Res. 2018;36(1):272-81.
37. Feuerriegel GC, Fritz B, Marth AA. Assessment of the rotator cuff muscles: state-of-the-art MRI and clinical implications. Radiology. 2025;315(2):e242131.
38. Deraedt L, Diependaele C, Cardon D. 3D quantitative CT study to assess rotator cuff muscle fatty infiltration. Acta Orthop Belg. 2024;90(2):221-7.
39. Tashjian RZ. Epidemiology, natural history and indications for treatment of rotator cuff tears. Clin Sports Med. 2012;31(4):589-604.
40. Gorski T, Casartelli NC, Fitzgerald G. Intramuscular fatty infiltration and its correlation with muscle composition and

function in hip osteoarthritis. Skelet Muscle. 2024;14(1):32.
41. Riem L, Blemker SS, DuCharme O. Objective analysis of partial three-dimensional rotator cuff muscle volume and fat infiltration across ages and sex from clinical MRI scans. Sci Rep. 2023;13(1):14345.
42. Polat Y, Karaduman D, Karanfil Y. Independent association between sarcopenic obesity and knee osteoarthritis: A cross-sectional study. J Sports Sci. 2025:1-13.
43. Safran O, Derwin KA, Powell K. Changes in rotator cuff muscle volume, fat content and passive mechanics after chronic detachment in a canine model. J Bone Joint Surg Am. 2005;87(12):2662-70.
44. Liu X, Ning AY, Chang NC. Investigating the cellular origin of rotator cuff muscle fatty infiltration and fibrosis after injury. Muscles Ligaments Tendons J. 2016;6(1):6-15.
45. Gerber C, Meyer DC, Flück M. Muscle degeneration associated with rotator cuff tendon release and/or denervation in sheep. Am J Sports Med. 2017;45(3):651-8.
46. Mitsutake T, Sakamoto M, Chyuda Y. Greater cervical muscle fat infiltration evaluated by magnetic resonance imaging is associated with poor postural stability in patients with cervical spondylotic radiculopathy. Spine. 2016;41(1):E8-14.
47. Yang Y, Qiu L, Gu X. Monitoring rotator cuff muscle fatty infiltration progression by magnetic resonance imaging T1 mapping: correlation with direct evaluation findings in rats. Am J Sports Med. 2022;50(4):1078-87.
48. Mohajer B, Dolatshahi M, Moradi K. Role of thigh muscle changes in knee osteoarthritis outcomes: Osteoarthritis initiative data. Radiology. 2022;305(1):169-78.
49. McCrum E. MR imaging of the rotator cuff. Magn Reson Imaging Clin N Am. 2020;28(2):165-79.
50. Millett PJ, Schoenahl JY, Allen MJ. An association between the inferior humeral head osteophyte and teres minor fatty infiltration: Evidence for axillary nerve entrapment in glenohumeral osteoarthritis. J Shoulder Elbow Surg. 2013;22(2):215-21.
51. Sepúlveda-Loyola W, Silva-Díaz YA, Molari M. Association between the fat mass/fat-free mass ratio and muscle strength, static balance and exercise capacity in older adults: a cross-sectional study. Nutr Hosp. 2025;43(3):464-9.
52. Loureiro A, Constantinou M, Beck B. A 12-month prospective exploratory study of muscle and fat characteristics in individuals with mild-to-moderate hip osteoarthritis. BMC Musculoskelet Disord. 2019;20(1):283.
53. Seyedhoseinpoor T, Taghipour M, Dadgoo M. Alteration of lumbar muscle morphology and composition in relation to low back pain: a systematic review and meta-analysis. Spine J. 2022;22(4):660-76.
54. Snodgrass SJ, Stanwell P, Weber KA. Greater muscle volume and muscle fat infiltrate in the deep cervical spine extensor muscles (multifidus with semispinalis cervicis) in individuals with chronic idiopathic neck pain compared to age and sex-matched asymptomatic controls: a cross-sectional study. BMC Musculoskelet Disord. 2022;23(1):973.
55. Drummer DJ, McAdam JS, Seay R. Perioperative assessment of muscle inflammation susceptibility in patients with end-stage osteoarthritis. J Appl Physiol (1985). 2022;132(4):984-94.
56. Fox JA, Luther L, Epner E. Shoulder proprioception: A review. J Clin Med. 2024;13(7):2077.
57. Miljkovic I, Kuipers AL, Cauley JA. Greater skeletal muscle fat infiltration is associated with higher all-cause and cardiovascular mortality in older men. J Gerontol A Biol Sci Med Sci. 2015;70(9):1133-40.
58. Wallenberg RB, Belzer ML, Ramsey DC. MRI-based 3-dimensional volumetric assessment of fatty infiltration and muscle atrophy in rotator cuff tears. J Shoulder Elbow Surg. 2022;31(6):1272-81.
59. Werthel JD, Boux de Casson F, Burdin V. CT-based volumetric assessment of rotator cuff muscle in shoulder arthroplasty preoperative planning. Bone Jt Open. 2021;2(7):552-61.
60. Ruderman L, Leinroth A, Rueckert H. Histologic differences in human rotator cuff muscle based on tear characteristics. J Bone Joint Surg Am. 2022;104(13):1148-56.
61. Shemshaki NS, Kan HM, Barajaa MA. Efficacy of a novel electroconductive matrix to treat muscle atrophy and fat accumulation in chronic massive rotator cuff tears of the shoulder. ACS Biomater Sci Eng. 2023;9(10):5782-91.
62. Wieser K, Joshy J, Filli L. Changes of supraspinatus muscle volume and fat fraction after successful or failed arthroscopic rotator cuff repair. Am J Sports Med. 2019;47(13):3080-8.
63. Thankam FG, Dilisio MF, Agrawal DK. Immunobiological factors aggravating the fatty infiltration on tendons and muscles in rotator cuff lesions. Mol Cell Biochem. 2016;417(1-2):17-33.
64. Ki SY, Lee YS, Kim JY. Relationship between fatty infiltration and gene expression in patients with medium rotator cuff tear. J Shoulder Elbow Surg. 2021;30(2):387-95.
65. Arenas-Miquelez A, Liu VK, Cavanagh J. Does the Walch type B shoulder have a transverse force couple imbalance? A volumetric analysis of segmented rotator cuff muscles in osteoarthritic shoulders. J Shoulder Elbow Surg. 2021;30(10):2344-

54.
66. Brioche T, Pagano AF, Py G. Muscle wasting and aging: Experimental models, fatty infiltrations and prevention. Mol Aspects Med. 2016;50:56-87.
67. Melis B, DeFranco MJ, Chuinard C. Natural history of fatty infiltration and atrophy of the supraspinatus muscle in rotator cuff tears. Clin Orthop Relat Res. 2010;468:1498-505.
68. Moverman MA, Puzzitiello RN, Menendez ME. Rotator cuff fatty infiltration and muscle atrophy: Relation to glenoid deformity in primary glenohumeral osteoarthritis. J Shoulder Elbow Surg. 2022;31(2):286-93.
69. Jiang Z, Wang K, Zhang H. Correlation between paraspinal muscle fat infiltration and thoracic vertebral degeneration based on phantom-less QCT: A novel insight into thoracic vertebral degeneration. Eur Spine J. 2025;34(3):837-52.
70. Al Saedi A, Duque G, Stupka N. Targeting intramuscular adipose tissue expansion to preserve contractile function in volumetric muscle loss: a potentially novel therapy? Curr Opin Pharmacol. 2021;58:21-6.
71. Huang Y, Wang L, Luo B. Associations of lumbar disc degeneration with paraspinal muscles myosteatosis in discogenic low back pain. Front Endocrinol (Lausanne). 2022;13:891088.
72. Zhu Y, Hu Y, Pan Y. Fatty infiltration in the musculoskeletal system: Pathological mechanisms and clinical implications. Front Endocrinol (Lausanne). 2024;15:1406046.
73. Theret M, Rossi FMV, Contreras O. Evolving roles of muscle-resident fibro-adipogenic progenitors in health, regeneration, neuromuscular disorders and aging. Front Physiol. 2021;12:673404.
74. Bowen E, Waque A, Su F. Muscle health & fatty infiltration with advanced rotator cuff pathology. Curr Rev Musculoskeletal Med. 2025 Feb 26:1-3.
75. Lopiz Y, Herzog R, Arvinius C. Functional outcomes and complications of classic Grammont-style reverse shoulder arthroplasty in patients with Os Acromiale: a retrospective case-control study. Int Orthop. 2025.
76. Linge J, Petersson M, Forsgren MF. Adverse muscle composition predicts all-cause mortality in the UK Biobank imaging study. J Cachexia Sarcopenia Muscle. 2021;12(6):1513-26.
77. Herve A, Thomazeau H, Favard L. Clinical and radiological outcomes of osteoarthritis twenty years after rotator cuff repair. Orthop Traumatol Surg Res. 2019;105(5):813-8.
78. Ager AL, de Oliveira FCL, Roy JS. Effects of elastic kinesiology taping on shoulder proprioception: A systematic review. Braz J Phys Ther. 2023;27(3):100514.
79. Mattar LT, Mahboobin AB, Popchak AJ. Individuals with rotator cuff tears unsuccessfully treated with exercise therapy have less inferiorly oriented net muscle forces during scapular plane abduction. J Biomech. 2024;162:111859.
80. Menze J, Croci E, Andersen MS. Advancing musculoskeletal shoulder modeling: reflecting glenohumeral translation with bony, ligamentous and muscular stability constraints. Front Bioeng Biotechnol. 2025;13:1441530.
81. Ferrell JL, Dodson A, Martin J. Microfragmented adipose tissue in the treatment of a full-thickness supraspinatus tear: A case report. Regen Med. 2023;18(10):773-80.
82. Mahna A, Boulanger SM, Gatti AA. Investigating the relationship between quantitative-based ultrasound and MRI estimations of rotator cuff fatty infiltration. J Clin Ultrasound. 2024;52(4):343-52.
83. Jung UJ. Sarcopenic obesity: involvement of oxidative stress and beneficial role of antioxidant flavonoids. Antioxidants (Basel). 2023;12(5):1063.
84. Mori D, Kagaku A, Wang KY. Histologic and ultrastructural degeneration of rotator cuff tendons in elderly patients with glenohumeral arthritis and cuff tear arthropathy. BMC Surg. 2025;25(1):281.
85. Shah S. Editorial commentary: partial rotator cuff repair and deltoid retraining shows positive outcomes for large to massive rotator cuff tear. Arthroscopy. 2025;41(7):2197-9.