



Review Article

Hip Muscle Atrophy and Hip Osteoarthritis-Evidence and Continuing Challenges

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Citation: Ray M. Hip Muscle Atrophy and Hip Osteoarthritis-Evidence and Continuing Challenges. J Ortho Sci Res. 2023;4(1):1-9.

<https://doi.org/10.46889/JOSR.2023.4105>

Received Date: 28-02-2023

Accepted Date: 14-03-2023

Published Date: 21-03-2023



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Abstract

Background: Hip osteoarthritis, a highly disabling disease with no distinct cause, and a disease associated with high degrees of pain, stiffness, and disability continues to affect the lives of many adults of all ages highly adversely and may be influenced adversely by some forms of muscle dysfunction, such as hip muscle atrophy.

Aim: To examine the prevailing research support for a mediating or causative role of muscle atrophy in hip osteoarthritis.

Methods: An in-depth literature review focusing on hip osteoarthritis muscle atrophy associations was conducted.

Results: A fair number of past and recent research reports indicate efforts to examine and intervene to minimize one or more attributes of hip muscle atrophy appears promising in the context of efforts to foster improved outcomes of hip joint osteoarthritis, regardless of the application of high-tech surgery.

Conclusion: Future research efforts are strongly indicated.

Keywords: Arthroplasty; Muscle Atrophy; Hip Osteoarthritis; Pathology; Prevention; Surgery

Introduction

Osteoarthritis, a chronic painful disabling joint disorder, often impacting both hip joints and where its slow progression often induces a high degree of progressive functional and social disability is generally considered a disease of the cartilage tissue lining the joint, rather than any other source. However, this commonly accepted belief fails to consider that mention has been made to view osteoarthritis as a disease affecting the 'whole joint' rather than a single component

of the affected joint[s] [1,2]. As well, more emphasis in this regard has been placed for some time on its biomechanical origins [3,4]. However, with no specific clearly defined agreed upon cause of this condition, and since cartilage is found poorly amenable to intrinsic regeneration or healing processes, and may be impacted adversely by various drugs that have no meaningful disease modifying effect [5,6], a host of non-pharmacologic approaches have been advocated. However, most are not universally accepted as mainstream solutions, nor studied to the same degree as cartilage molecular and biochemistry approaches. As a result, those who are most disabled may often suffer unduly perhaps, and must rely on various surgical interventions to replace the damaged hip joint tissues with a metallic or artificial prosthesis to offset their pain if this is not contra indicated. However, some recent discussions and findings that attempt to carefully examine osteoarthritis origins and progression, and that point to a possible key or influential role of muscles, such as those surrounding the hip joint in mediating or moderating the disease might be amenable to interventions that help improve joint status, or prevent it from worsening rapidly, regardless of the degree and duration and extent of any underlying joint pathology [7].

This possible and biologically plausible role for muscle in the osteoarthritis disease context has thus been quite carefully investigated of late as it appears that muscle abnormalities, for example those at the hip joint, may yet affect cartilage integrity,

for example if abnormal physical stresses are placed on joints as a result of muscle weakness, uncoordinated muscular forces, excessive muscular forces or by muscle contractile dysfunction [7,8]. In turn, damage caused by cartilage destruction that leads to joint inflammation and declining motivation for movement may impact muscle adversely, thus leading to a decline in joint protection mechanisms and hastening movement challenges that are more evident in cases with hip joint osteoarthritis than controls [9].

In view of these possibilities, and the very severe impact of hip joint osteoarthritis on the lives of many older adults, that appears to extend to the underlying bone tissues and generates consistent findings of muscle force capacity declines but that the disease symptoms do not correlate well with commonplace radiographic staged disease classifications, it thus seems plausible to examine if researchers are pursuing this line of inquiry implicating muscle in some way in the current era of efforts to advance the idea of joint and health protection rather than reliance on costly health interventions that do not account for upstream or salient physiological factors that may predict future joint problems and others [1,8]. The rationale for considering the importance of the muscle state at all stages of osteoarthritis disease as well as its prevention is the cross-cutting role played by muscle in generating movement, as well as joint protection. Given the enormous burden of the disease, and the fact age and a 'wear and tear' theory is often not a factor in explaining either the presence of the disease or why the disease manifests on one side and not the other, and surgery to reduce pain does not help to uniformly restore joint function, in all older adults, especially if they are de-conditioned it appears information on the state of the hip muscles in cases with varying complaints of persistent hip pain may help to resolve some of this distress, while yielding one or more cost savings [10].

Because of the strong possibility that muscular disturbances at the hip or those that impact hip joint function in some way may yet be implicated in the risk of incurring some degree of hip joint osteoarthritis, and/or a more detrimental outcome, especially pain, and that may not be evidenced via standard radiological examination alone, this brief explores research that may provide a clearer view of whether efforts to examine the possible role of muscle dysfunction at the hip joint has any bearing on the manifestation of hip osteoarthritis disability, and if so in what respect [11]. Using the PUBMED, PubMed Central, and Google Scholar data bases deemed to house salient representative data, and the terms hip muscle atrophy and hip osteoarthritis clinically oriented articles dating largely from the most studied period of 2000-2023 and specifically related to muscle atrophy as opposed to other forms of muscle dysfunction, this body of data was sought and carefully examined and reported in summary form only, and regardless of muscle discussed or method of assessment. For an in-depth analysis of muscle related facts in the context of hip osteoarthritis biomechanics, the discourse by Denham and the treatise by Maquet is highly recommended [4,12].

However, rather than any systematic analysis, this current scoping review of data that have emerged over the past 60 years was designed to embrace all data linking muscle mass correlates with hip osteoarthritis. Since certain forms of study, while conforming to solid design protocols may not capture all possibilities in this regard, this analysis accepted all forms of peer reviewed research pertaining to the topics of muscle atrophy and hip osteoarthritis as valid. Excluded were studies highlighting abnormal dynamic muscle forces and their manifestations, intervention associated studies and measurement studies, and foreign non-English studies. Also excluded were studies pertaining to other possible hip osteoarthritis pathogenic factors such as heredity, hormones, occupation, injury and diet, among other factors. The aim was to identify what the literature has revealed over time as to the idea that a role for muscle atrophy at the diseased hip joint is a clinically relevant one; as well as related research gaps, discrepancies and potential clinical and research opportunities. As outlined by Godziuk, et al., and despite more than 100 years of study, hip osteoarthritis remains a highly painful disabling disease that strongly impacts the life quality and independence of many older adults and is challenging to treat, hence efforts to understand its possible origins other than age and injury continue to be highly salient [12]. This is increasingly so in light of data that reveal surgery, the most common form of hip osteoarthritis treatment, is often not without risk, and may yet be delayed in the post COVID-19 period due to health resource shortages and immense wait lists. Drugs too, may not be beneficial and lead to narcotic addictions and cartilage destruction.

Search Results

Among the literature retrieved from the aforementioned data bases, a thematic analysis of all previously published clinical articles revealed a limited albeit diverse body of approximately 100 works referring to muscle attributes that began in the 1950s, and only truly increased substantively in publication numbers as of 2020, but yet remains limited in scope and importance when

considering the term 'osteoarthritis' alone current yields 110,508 PUBMED listings that began in 1885 with 1683 of these alone in 2023. Of the 265 hip osteoarthritis listings highlighted between Jan 1 and February 27, 2023, only 19 refer specifically to muscle attributes. These as well as those published over time are highly diverse however, and include, but are not limited to studies examining aspects of muscle measurements, muscular strength deficits, aberrant movement patterns, muscle fat infiltration, and intervention studies. Various studies discussing muscle atrophy in the context of one or more hip muscles and that may have implications for hip osteoarthritis prevention and intervention do however prevail, for example studies that discuss atrophy of the iliopsoas muscle or the hip abductor muscles in hip osteoarthritis realms [14-17].

The discussions here do not focus on the nature of the research design to any degree, but rather focus on either what appears to be the impact of hip osteoarthritis on one or more surrounding muscles as manifest by changes in muscle size and composition, or the less well accepted idea of muscle mass and its properties as a causative or hip osteoarthritis mediating factor [15,18]. Although increasing numbers of articles have discussed other aspects of muscle rather than muscle mass or size, it was assumed muscle atrophy and/or its association with joint instability, muscle fibrosis and fat infiltration may be an understudied albeit clinically important contributing factors to hip osteoarthritis pathology that may also be expected to produce disturbances of cartilage metabolism and consequent manifestations of joint degeneration [19-26].

As per Godzink, et al., one aspect that is additionally pertinent in this context is that muscle mass alterations, are relevant to study because these muscle mass losses are commonly associated with aging, a well-established probable correlate of disabling hip joint osteoarthritis [13]. In this regard, recent data does specifically show the presence of a sarcopenic obesity phenotype, characterized by high adiposity and low skeletal muscle mass is associated with higher infection rates, poorer function, and slower recovery after surgery in various populations, as well as possibly in adults with hip osteoarthritis and low skeletal muscle mass. Although not well studied, de Ceuninck, et al., argue that altered joint stability, maladapted postures and defective neuromuscular features in hip osteoarthritis for example, that may ensue if muscle force capacity is reduced when needed, could impact cartilage cells at the cellular and molecular levels negatively and significantly [24]. A parallel idea supported by Wu, et al., is that as far as senescent skeletal cells and synovial cells are concerned, they are able to communicate and may well be influenced by the muscle quality and prevailing degree of muscle protection [26].

Current work by Iwasa, et al., who did find fatty degeneration of the hamstrings, iliopsoas, and hip adductor muscles to be significantly related to health quality of life in patients with hip osteoarthritis tended to support a role for muscle fat infiltration in the osteoarthritis pain and disability cycle [27]. They also implied these muscles should be targeted accordingly during conservative rehabilitation for hip osteoarthritis as well as in the context of rehabilitation efforts following hip joint surgical replacement. In support of this idea, Peires, et al., found a greater cross-sectional area of hip adductors was associated with better function and quality of life in individuals with mild-to-moderate hip osteoarthritis [28]. In addition, it appeared a greater cross-sectional area of hip flexors might be associated with better quality of life. To the contrary, a further recent report has shown hip abductor muscle fat is associated with markers of cartilage degeneration [29].

Kivle, et al., who examined data from patients with end-stage osteoarthritis and matched healthy controls found the gluteus minimus muscle was significantly smaller in the osteoarthritis-group compared to the control group [30]. Higher levels of fatty infiltration were identified in the anterior segment of the muscle along with the anterior and middle segments of the gluteus medius muscle in the osteoarthritis group. Overall, their findings showed end stage osteoarthritis was associated with significant atrophy of the gluteus minimus and higher levels of fatty infiltration, particularly in the anterior segments of both the gluteus minimus and medius. As well, even though minor fatty infiltration of the anterior segment of gluteus minimus appeared to be a normal part of ageing, a different pattern of atrophy and fatty infiltration was observed between patients with end-stage osteoarthritis and their healthy matched peers.

Several other studies conducted since 2000 have concluded the presence of muscle atrophy in one or more of the muscles surrounding the hip may not only contribute to the magnitude and progression of hip osteoarthritis disability, but may persist in the operated limb of some patients with advanced or complicated hip osteoarthritis manifestations even after surgical reconstruction [20,31]. Mak, et al., who strove to determine if there is significant psoas muscle atrophy as indicated by muscle cross-sectional area and a high degree of fat infiltration in post-unilateral hip arthroplasty cases over an eight-year period found

the cross-sectional area of the psoas muscle on the implant side was significantly smaller than that on the non-operative side [32]. There was also an association between the cross-sectional area differences and the fat grade of the implant side, results that aligned somewhat with those of Lee, et al., [16].

Loureiro, et al., concluded that atrophic, bilateral hip and knee muscle weakness is a feature of individuals with mild-to-moderate hip osteoarthritis and that early interventions to target muscle weakness and prevent the development of strength asymmetries that are characteristic of advanced hip osteoarthritis appear warranted [33]. This idea, while far from robust, does seem to have considerable support despite the fact one review tended to discount any muscle mass involvement in the realm of hip osteoarthritis, despite obvious strength declines [34].

However, Marshall, et al., did not specifically assess muscle size issues to any degree, and an earlier study of men with hip osteoarthritis did show a significantly lower abduction, adduction, and flexion set of muscle strength measures than those of controls [34]. The finding of a decrease of muscle size in those cases with severe signs of hip osteoarthritis and hip pain may contribute to the overall decrease of muscle strength in those with hip osteoarthritis as time progresses, although other possible underlying causes of the muscle weakness need to be studied.

As per Lawrenson, et al., while there is some low-quality evidence that specific hip muscles are smaller in unilateral hip osteoarthritis, variation in the magnitude of differences indicate the observed changes in size that have been noted are not necessarily uniform across all muscles or stages of pathology [35]. As noted by Grimaldi, et al., what is demonstrated may depend on what is measured and how and at what disease stage, thus definitive conclusions must await further study [36]. Uemura, et al., agree that hip osteoarthritis can however lead to atrophy of the gluteal and thigh muscles, even though they report the muscle volume of these can recover even though this may take for up to two years after hip joint replacement surgery [37]. However, their 2016 data may not reflect that occurring currently in 2023, for example in cases who are non-Japanese, in higher age ranges, and with multiple chronic health challenges that limit activity, including long COVID-19 syndrome, unanticipated cuts in health services, and persistent severe obesity.

Other researchers indicate cases in the older age ranges with signs of hip osteoarthritis may well exhibit age associated general muscle mass declines, and possible joint instability and with this, pain that if untreated at its source may further muscle mass losses, as well as increase the risk for poor walking ability and possible falls injuries [21,38,39]. Studies in larger cohorts investigating muscle size and composition across the spectrum of articular pathologies and age ranges are however clearly required here to arrive at a consensus about a role for hip muscle mass, and possible decreases in their contractile tissue features that affect muscle function [40].

As well, while the current research appears promising for helping to reduce the suffering attributable to hip osteoarthritis, the use of single rather than multiple measures or theorizing about-but not measuring muscle mass attributes in any way- may fail to clearly depict the presence and implications of any actual degree of excess muscle atrophy as far as this could markedly impact muscle function and possible joint physiology [41,42]. Moreover, while Vadala, et al., indicate a possible negative role for muscle intrinsic structures and fatty infiltration of the muscle in the context of hip osteoarthritis, what the role of muscles other than those studied may play in this regard or whether the observed tendon pathology that was observed was the cause or consequence of the severe hip joint damage that emerged over time could not be discerned from the limited muscle analysis [43]. In addition, the role of pain, as well as measurement approach, injury and age could not be discerned among the study findings and that could possibly explain the observed fatty infiltration of the contra-lateral pre- and post-operative muscles as well as the post-operative operated muscle. Other data show that older females with lower than desired muscle mass tend to have a greater chance of having hip joint osteoarthritis that may reflect a deficit in joint protection ability of the remaining muscle mass, regardless of whether this effect is a cause or an outcome of the disease, but why not all older people sustain the disease, or why the disease is often more evident on one side of the body or affects one muscle but not others remain unclear [44].

Thus, while findings from most published data on this topic imply a mediating or moderating or causative role of persistent muscle atrophy at the hip in the progression of unilateral hip osteoarthritis that warrants attention, it appears prudent to await further insightful well-designed research that can yield more solid conclusions. In the interim, it seems clear that surgical hip

replacement does not always attenuate muscle atrophy in any immediate manner in all cases, suggesting either a preexistent problem or one invoked by surgery or both. Indeed, cases exhibiting the presence of poor hand grip strength before hip surgery, which may denote, muscle mass or general sarcopenic deficiencies, have been found to have poorer than anticipated post-surgery outcomes than stronger cases with similar pathology [37,45,46]. More recently, the high percentage of fat observed in various hip joint muscles in cases undergoing surgery could explain the observed link between obesity and hip joint osteoarthritis, while a deficient pre surgical or post-surgical muscle strength loss could be expected to induce more adverse outcomes than not, including higher levels of obesity if activity is suboptimal [47]. In addition, the presence of a type II muscle fiber deficit that impacts joint loading mechanisms detrimentally, as well as weightbearing endurance may be an additional pathogenic factor that additively fosters cartilage injury or additional trauma to the already affected joint[s] if untreated because muscle response times may be duly delayed or slowed due to this particular selective muscle fiber loss [41,48].

Discussion

Osteoarthritis, a widespread joint disorder resulting in dysfunction of one or more freely moving joints such as the hip joint, is presently not reversible, and invariably leads to various degrees of intractable pain, disability and loss of independence in the older population in particular. As with aging, the disease may be accompanied by various degrees of muscle mass declines, sarcopenia, and muscle fiber atrophy. Moreover, in the absence of any explanation for the disease and its origins, muscle wasting has been suggested to be a probable factor affecting and being influenced by the stability of the joints such as the hip, wherein a loss of mobility, or impaired mobility patterns, can lead to gradual articular cartilage degeneration [49].

This brief examined this hypothetical unproven scenario in the belief that if it is a possible pathogenic factor, early identification of hip muscle atrophy may help clinicians to develop effective exercise programs to counter hip osteoarthritis onset and progression or at least its severity. Although negative findings may not be published, or may emerge in the future, and only a small percentage of the many muscles at the hip have been studied, most prevailing observations indicate that where present, atrophy of one or more key hip muscles, which are important in hip joint and pelvic stability, may alter hip joint function quite measurably during gait and functional tasks, even if not commonly detected in substantive numbers of hip osteoarthritis cases [50,51]. Clinicians treating patients with intra-articular hip joint pathology may hence need to assess their clients very thoroughly and if indicated prescribe exercises targeting the specific muscles that may be vulnerable to atrophy or are already atrophic carefully and using best practices. The finding that muscle mass deficits even if not present before surgery, may prevail for extended periods after successful reconstruction surgery of the osteoarthritis hip joint implies a further need for vigilance both before and after hip joint arthroplasty surgery [52]. Observations that fatty atrophy of one or more hip muscles may increase the extent of hip osteoarthritis pathology, further suggests efforts to control weight may be a highly salient management approach in those with hip pain, given that preoperatively, muscular atrophy and fatty infiltration has been demonstrated in the diseased hip [53]. A role for suboptimal muscle volumes, which appear associated with the extent of pathology and walking ability in cases complaining of hip pain has also been put forth [54] and may well foster or exacerbate any inadvertent surgically induced neuromuscular adaptations such as marked muscle wasting, weakness, and deterioration in muscle grade or preexisting gluteal muscle weakness and a limping type gait that may impact post-surgical outcomes adversely and negatively [55-59].

Momose, et al., further conclude that an increase in muscle volume, plus allowing for the recovery of any muscle degeneration, may help to improve limping in patients with hip osteoarthritis [59]. Additionally, appropriate exercise may help as well if applied early on and pursued because as indicated by Zacharius, et al., a higher degree of gluteal muscles atrophy along with fatty infiltration of these muscles and others is quite strongly associated with hip osteoarthritis clinical severity [53,60]. In addition, if this possible linkage between muscle size and composition and hip osteoarthritis joint changes can be further supported in the future, it would, at least, provide a possible avenue for supporting more targeted intervention opportunities to reduce muscle atrophy due to hip osteoarthritis. This in turn might mitigate or reduce any fatty infiltration of one or more hip muscles more effectively than not, thus potentially yielding more optimal surgical outcomes as well as possible non-surgical functional outcomes for adults with osteoarthritis of the hip than is presently observed [65,66]. Indeed, as outlined by de Ceuninck, et al., the consistent body of evidence showing that a decline in lower limb muscle strength is associated with hip osteoarthritis may lead one to conclude that chondrocytes and myoblasts may indeed share common pathological targets and pathways and that the potential advantage of understanding this association has far reaching clinical implications [23]. Muscles may also undergo property changes that induce muscle atrophy due to hip surgical approaches that should not be ignored and thus unless

this is acknowledged, it may be difficult to improve function capacity and ease, in general, even after corrective joint surgery [61].

In the interim, it appears due consideration of all possible pathogenic factors including the structure and composition of muscles surrounding the hip joints may well help to eliminate or minimize much suffering, and/or delay the need for harmful drugs and surgery. Cases assessed accordingly after surgery may likewise benefit [24]. Since different muscles or patterns of muscle mass loss may yield different pre-or post-surgical hip osteoarthritis functional responses and pathomechanics, researchers can potentially help to vastly advance this field by conducting more elaborate and carefully construed multipronged studies. Clinicians in the interim are urged to assess their clients muscle status periodically and carefully so as to apply appropriately tailored treatment approaches rather than generic approaches that can impact muscle size and fiber type composition and its shock absorbing properties favorably, while helping to control or eliminate biomechanical disruptions of the joint due to excess fat infiltration and associated age and other factors that impact skeletal muscle mass, composition, and elasticity [62-64,67-69]. Surgeons too can continue to examine how various forms of surgery impact muscle degeneration or recovery at the osteoarthritic hip and report these potentially revealing data using advanced and multiple functional measures [70].

Concluding Remarks

In summarizing what we know about the possible interaction of hip muscle atrophy and its presence or impact in the realm of hip joint osteoarthritis in the older adult population, and while this work is possibly not all inclusive or reflective of emerging data, it appears safe to say:

1. Hip osteoarthritis a widespread health condition that continues to induce high degrees of disablement among many older adults in all parts of the world may be impacted negatively by various biomechanical influences attributable to declines in muscle size, among other muscle correlates
2. The disease, while largely impervious to the sole application of various drugs, may yet be impacted favorably by concomitant or independent efforts of clinicians to carefully examine the status of the muscles at the hip joint for any signs of muscle mass or volume losses and to intervene accordingly to improve any atrophic presence that is evidenced through well accepted conservative approaches, regardless of surgery
3. Researchers can greatly help to advance this line of inquiry urgently needed to allay suffering and health costs by concerted efforts to study the nature and role of muscle atrophy and fatty infiltration of those muscles at the hip that may lead to muscle weakness in the context of the vulnerable older individual in a careful comprehensive manner over time
4. How muscle mass at the hip varies or manifests when using advanced technology and radionuclide scintigraphy to examine any parallel bone and synovium changes, along with biochemical markers of cartilage destruction is especially indicated
5. Until more research is forthcoming, the application of carefully construed muscle-oriented approaches should still become more mainstream especially for safely modifying pain, instability, stiffness, function, but must be carefully tailored and insightfully targeted, rather than attempting to rely solely on aggregated approaches and understandings from systematic reviews
6. Older overweight adults should be encouraged to lose weight so as to avert any damaging muscle fat infiltration build up

Conflict of Interest

The authors have no conflict of interest to declare.

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