IJSPT
INTERNATIONAL JOURNAL OF SPORTS PHYSICAL THERAPY

Duke MPR, Jones A, Takla A, Johnson MI. The Relationship Between Tensor Fascia Latae and Gluteus Maximus Has the Potential to Indicate Early Intra-articular and Degenerative Pathologies of the Femoral-Acetabular Joint: A Narrative Review. *IJSPT*. 2025;20(3):476-484. doi:10.26603/001c.129974

Narrative Review

The Relationship Between Tensor Fascia Latae and Gluteus Maximus Has the Potential to Indicate Early Intra-articular and Degenerative Pathologies of the Femoral-Acetabular Joint: A Narrative Review.

Matt P.R. Duke[1a], Ashley Jones[1], Amir Takla[2], Mark I. Johnson[1]

[1] School of Health, Leeds Beckett University, [2] University of Melbourne

Keywords: Hip, Tensor Fascia Latae, Gluteus Maximus, Femoroacetabular impingement, Total hip replacement, Hip Osteoarthritis

https://doi.org/10.26603/001c.129974

International Journal of Sports Physical Therapy

Vol. 20, Issue 3, 2025

Intra-articular and degenerative hip pathologies have become common place with the number of total hip replacements rising year on year in the United Kingdom (UK). Pathology is identified by clinicians using special tests which are researched maneuvers used by clinicians to rule in or rule out specific musculoskeletal pathologies. Special tests used for hip pathology usually have high specificity to exclude degenerative and intraarticular pathology but vary in sensitivity. These special tests are usually only conducted when a person is symptomatic and typically require radiological confirmation to diagnose. The aim of this review was to appraise research to determine whether functional changes in the TFL and UGM muscle complex could indicate degenerative and/or intra articular pathology, with a specific focus on the utility of the ratio in strength of TFL and UGM to assist clinical diagnosis. The hypothesis was that the ratio of the strength of Tensor Fascia Latae (TFL) and the upper fibres of Gluteus Maximus (UGM) could suggest early intra-articular hip pathology, and that changes to this ratio could indicate deterioration of the hip joint before symptoms present/progress.

Level of Evidence

5

INTRODUCTION

The Acetabular-femoral joint (hip) is a multiaxial load-bearing complex joint with three degrees of freedom.[1] The hip joint can move through a sagittal, frontal, and transverse plane and must be able to withstand three times body weight during walking and 5.5-8.4 times body weight during a vertical jump/ running action.[2,3] In the United Kingdom (UK), Total Hip Replacement (THR) surgeries have increased from 63,625 in 2009/10 (among a 62.26 million population) to 76,401 in 2019/20 (among a 66.8 million population).[4] Data reported by the National Joint registry in 2021 suggest that surgical hip procedures have increased from 42,578 in 2014 to 98,649 in 2019 and that this is most likely due to increased hip preservation awareness, improved surgical techniques and increased clinical diagnostic skills by practitioners.[5] In 2020/21, the National Health Service (NHS) THR's were performed on a broad spectrum of patients from the ages of 10 years old (Males: 0.2 per 100,000; Females: 0.2 per 100,000) to 85+ years old (Males: 94.9 per 100,000; Females: 108.7 per 100,000).[6] Females were reported to have surgery more often than males in all age brackets except for three age brackets: 10-14 years (Males: 0.2 per 100,000; Females: 0.2 per 100,000), 20-24years (Males: 2 per 100,000; Females: 0.8 per 100,000) and 30-34years (Males:4.4 per 100,000; Females 4 per 100,000). Thes results suggest that gender differences occur above the age of 34.[6] The average total waiting time for THR surgeries has also increased from 87.5 days to 105.2 due to increased number of patients who require surgical intervention.[7] Recent data from England reported that 24,202 THR surgeries were performed after an 18week+ waiting period and 1,093 after a one year waiting period.[6]

a Corresponding Author:
Matt Duke, Leeds Beckett University, City Campus, Leeds, Ls1 3HE
Tel : +447851228572
Email : M.PR.Duke@Leedsbeckett.ac.uk

With THR surgery increasing and some surgeries now being performed on patients from the age of 10 upwards, it is important for clinicians to identify hip pathology as early as possible to assist early intervention to preserve hip health and improve patient's long-term prognosis.

THE CONCEPT

The presence of hip pathology in humans may be reflected in muscular changes of the Tensor Fascia Late (TFL), Gluteus Medius (GMed), Minimus (GMin)and Maximus (GMax) muscles evident on magnetic resonance imaging (MRI), via strength assessments, and in displayed functional deficits.[8] More specifically, the strength and recruitment of the TFL compared to the upper fibers of Gluteus Maximus (UGMax) may be indicative of hip pathology; hence, the ratio of strength of TFL and UGMax could provide an early indicator of hip pathology. UGMax refers to the upper fibers that produce abduction forces of the GMax muscle.

The aim of this review is to appraise research to determine whether functional changes in the TFL and UGMax muscle complex could indicate degenerative and/or intra articular pathology, with a specific focus on the utility of the ratio in strength of TFL and UGMax to assist clinical diagnosis.

ANATOMY OF THE LATERAL MUSCULATURE: DIAGNOSTIC UTILITY?

The musculature around the lateral aspect of the hip includes the superficial layer consisting of TFL, GMax, GMed, and GMin along with the deep musculature (Piriformis, Quadratus Femoris, Obturator Internus, Gemellus Superior, Gemellus Inferior and Obturator Externus).[9]

Internal rotation (IR) ROM and extension and abduction strength are commonly affected in the presence of hip pathology.[9-11] It is interesting that the lateral musculature (TFL and GMax) is a complex that provides the same movements that are affected by hip pathology (flexion, extension, abduction, external rotation, and internal rotation) because the TFL and GMax have a common attachment point as they both insert into the iliotibial band, therefore perform all the movements of the hip when working together and individually.[9] Hence, could the strength of the TFL and UGMax be related to intra-articular pathology and could the ratio of strength between the two muscles be of diagnostic utility?

TENSOR FASCIA LATAE (TFL)

The TFL is a muscle that is located on the anterolateral aspect of the hip joint and attaches to the anterolateral aspect of the iliac crest and into the iliotibial band around the greater trochanter and is innovated by the superior gluteal nerve L4, L5, S1.[11] The TFL has anteromedial (AM) fibers and posterolateral (PL) fibres. The AM fibers flex the hip in open chain kinematics such as hip flexion during the swing phase of gait[12] which has been confirmed by electromyography (EMG).[13] TFL has been shown on EMG to be silent during the heel strike which suggests the TFL is inactive during hip extension and is most active during the acceleration phase of running, supporting the role of TFL as a hip flexor.[12,13] Interestingly, this research showed that in isolated movements, the TFL was most active in hip flexion and abduction and was silent if the hip was externally rotated. The PM fibers are active during the stance phase of gait, suggesting TFL is also a major hip stabilizer.[12,13] In isolated open kinetic chain movements, the PL fibres were active in all hip internal rotation movements and in abduction movements.[12,13] Like the AM fibers, the PL fibers stay silent if the hip is abducting whilst in external rotation.[9]

Previous research has investigated the biomechanics of gait post hip arthroplasty and found that hip flexion and extension peak angles were lower (Flexion -5.5 degrees, extension -5 degrees) for a total of 11 degrees reduction on an operated side (up to 15 months post operation) compared to the non-operated side and a healthy control group during gait.[10] This suggests that flexion and extension ROM are reduced during gait post-surgery. This could be due to the trauma associated with surgical intervention but also may indicate that the relationship between TFL and UGMax and hip pathology may exist and requires further exploration.

GLUTEUS MAXIMUS

GMax is a strong and powerful muscle located on the posterior aspect of the body.[9] The muscle moves in three degrees of freedom, meaning that it moves through a sagittal, frontal, and transverse plane providing hip extension, abduction and external rotation movements as well as providing stability to the knee via the iliotibial band, and stability to the Sacroiliac Joint (SIJ) and hip.[9] It is innovated by the inferior gluteal nerve L5, S1, S2 therefore having a different nerve supply to the TFL.[9]

GMax has two parts, the UGMax attaches to the lateral aspect of the hip inserting directly into the iliotibial tract, and the Lower Gluteus Maximus (LGMax) attaching posteriorly on the gluteal tuberosity of the hip.[9] Despite, the UGMax and LGMax having the same nerve supply, these segments work in different planes of movement; the UGMax works in the frontal plane and the LGMax works in the sagittal and transverse planes.[9]

The lateral attachment of UGMax into the IT band helps to stabilize the hip and knee joints and, hence, hip abduction occurs when co-contracting with the TFL.[9] Activation of the UGMax during a glute bridge exercise with 30 degrees of hip abduction maintained by a resistance band is 21.1% greater than the same exercise without resistance against abduction.[14] This suggests that when the hip is in extension and resistance applied to challenge abduction of the hip (30 degrees of abduction) there is further activation of UGMax, supporting UGMax contributions as a hip abductor.[15] EMG studies have demonstrated that the UGMax is active during the walking phase of gait as GMax decelerates the thigh going into flexion, providing an eccentric contraction prior to heel strike during running.[9,12,14]

The TFL and UGMax have a common insertional attachment point and are active in both hip stabilization and contributing to gait.[15] Hence, hip degenerative and intra-articular pathologies that impair gait through muscle weakness or inhibition of muscle contraction may be reflected in alterations in the relative activities of TFL and UGMax.

TFL AND UGMAX DURING GAIT

The TFL and UGMax during gait help with stabilizing the hip joint during single leg stance and help to move the hip joint during the swing phase of gait, suggesting that both muscles have specific roles depending on whether the leg is weight bearing or unloaded. The insertion of the UGMax and TFL into the IT band provides the stabilization of the hip joint movements when weight bearing, whereas individually, the muscles assist in moving the hip joint when unloaded.[12,13]

During gait, the average hip angle during locomotion in a control population was shown to be 33.9 degrees of flexion when heel strike occurs, 15.1 degrees of hip extension during toe off and that the abduction angle was 0.9 degrees during gait.[10] These numbers show the peak angle of the hip joint when force is applied during gait and therefore and future testing would need to address these angles. Therefore, evaluating the lateral musculature in these hip angle positions could help identify changes in the TFL and UGMax. As these hip angles are associated with gait and a common symptom of hip pathology is altered gait, changes can potentially identify underlying pathology.

ARTHROGENIC NEUROMUSCULAR INHIBITION

Arthrogenic neuromuscular inhibition (ANI) is a concept that is linked to joint pathology. ANI is defined as 'A continued reflex inhibition of musculature surrounding a joint following injury or joint effusion.[16] Previous research has tested the hip joint for inhibition of the lateral musculature and found that surface EMG values showed reduced muscle activation for GMax (165 to 118 %MVC) and GMed (92 to 48%MVC) in the presence of intra-articular administration of fluid, suggesting that any injury or swelling affecting the hip joint could provide neural inhibition to GMax and GMed.[17] This means that injury could reduce the amount muscle activation of the gluteal muscles and therefore could show a reduction in muscle recruitment when comparing GMax to the TFL.

THE RELATIONSHIP BETWEEN TFL AND UGMAX FOR DIAGNOSTIC UTILITY.

Previous research has used Magnetic Resonance Imaging (MRI) to measure TFL and UGMax muscle volume in healthy subjects and participants with unilateral osteoarthritis and found that GMax fibers were smaller on the affected side (5.8%) with mild degenerative OA changes, and was found to be statistically significant.[8] However, in advanced OA, there was atrophy in GMax of the affected hip when compared with the healthy hip, with atrophy more notable in upper fibers compared to lower fibers (UGMax; 21% difference, LGMax; 19.7% difference). Despite having the same neural innervation, differences in biomechanics between hips with OA symptoms and/or pathology compared with asymptomatic individuals, notably differences in hip extension and abduction, may provide clinical value when diagnosing hip pathology. Interestingly, the TFL was on average 10.5% larger on the affected side in mild cases of OA.

The finding that UGMax exhibits muscle atrophy in severe cases of OA and TFL exhibits muscle hypertrophy in mild OA supports the idea that imbalances in strength between TFL (hypertrophic in early pathology) and UGMax (atrophic in progressing pathology) is an indicator of articular hip pathology. Hypertrophic changes to the TFL in the early stage of OA could demonstrate potential increased TFL workload, potentially increasing TFL strength and UGMax atrophy potentially demonstrates decrease in strength. Therefore, this would suggest a strength change has the potential to indicate early identification of hip pathology.

GMed is the bodies main hip abductor and has been shown to atrophy in patients with hip pathology, therefore suggesting that TFL and UGMax must assist stability more during locomotion.[18] During the heel strike action in running, GMax eccentrically contracts to maintain the body's center of gravity. This is then altered to a concentric contraction when the body enters a neutral position to continue maintaining hip stability and center of gravity and prevent transverse and frontal plain excursion.[18] This suggests that due to GMed and GMin atrophying in the presence of degenerative hip pathology, TFL and GMax must assist hip abduction. With GMax also atrophying in the presence of progressive hip pathology, this could explain the reliance, activation, and hypertrophic changes of the TFL and therefore why this muscle could be a key indicator for identifying degenerative hip pathology.

There has been no research that directly compares the strength of the TFL and UGMax despite these muscles being anatomically attached to each other and seeming intrinsically linked to each other in the presence of hip pathology. This could be because by time a person is symptomatic, changes to the TFL and UGMax have already occurred and therefore the relationship is overlooked or clinicians' anatomical knowledge of the hip means that because both muscles present differently during hip pathology, they are treated separately, and the common attachment is not considered. This muscular complex produces the movements that are affected in the presences of intraarticular hip pathology. Therefore, the strength ratio between these two muscles needs to be considered, as it could help early identification of hip pathology.

ARTHROLOGY AND PATHOLOGY AT THE HIP JOINT: WHAT IS OCCURRING AT THE JOINT?

There are several joint pathologies that occur at the hip joint:

- Femoral-acetabular Impingement (FAI) presenting in 30% of the population, and higher in an athletic population.[19]
- Osteoarthritis (OA) affects 3.2 million UK citizens, with 8% of the UK population seeking treatment.[20,21]
- Hip dysplasia (DDH) accounted for 5-30/1000 births in the UK in 2016 compared to 3.6/1000 in 2011 means DDH is increasing.[22,23]
- Chondrolabral pathology (CLP) was found under ultrasound in 96.1% of people with FAIS[24] and in 22-55% of patients with hip or groin pain.[25]

These conditions can be present both singularly and in combination with each other in a patient.

FEMOROACETABULAR IMPINGEMENT SYNDROME (FAIS)

FAIS is the "abnormal contact that may arise as a result of either abnormal morphological features...or as a result of subjecting the hip to excessive and supraphysiological range of motion."[23,26] The Warrick agreement, a consensus statement from international expert practitioners, has built upon this with the diagnosis of FAIS as "a triad of symptoms, clinical signs and relevant imaging findings must all be present for diagnosis.[26]

Research investigated muscle forces of the hip in patients with FAIS matched to a healthy control group during gait and found that the correlation coefficients for the hip osteoarthritis outcome score (HOOS) for function of the GMin (r = 0.03) and GMed (r = 0.02) were lower in patients with FAIS.[27] There were reductions in strength for Sartorius (p=0.006) and function for Iliopsoas (r =0.01) in patients with more severe joint pain and dysfunction. This demonstrated that function and strength impairment is associated with underlying hip FAIS. Research has also found that the hip flexors, specifically the Sartorius and Iliopsoas, had reduced peak forces during gait in patients with FAIS.[27] Interestingly, the TFL was not tested in the study, despite the TFL being important for both hip stability during gait when load bearing and for locomotion of the leg when unloaded.[13]

Participants with FAIS had a significantly higher osteoarthritis outcome scores (HOOS) for pain and decrease in function on the side with FAI, with further research supporting that flexion was reduced on the affected hip compared with the unaffected hip, with a mean reduction of 9° on the affected hip compared to the unaffected hip with mean abduction (4°) adduction (3°), IR (4°), ER (3°).[27,28] Therefore, with a reduction in flexion ROM, weakness in GMax and GMin, yet no data on TFL (flexor and abductor capacities) reported, TFL strength warrants investigation into its potential to indicate pathology.

HIP DYSPLASIA (DDS)

Hip dysplasia is a congenital condition where the acetabulum of the hip joint is shallow, particularly anteriorly and superior-laterally leaving the hip joint less congruent.[29] Patients with diagnosed DDS present with apprehensive gait patterns and avoidance of extension due to the shallow anterior and superior-lateral acetabulum. Research has reported that TFL becomes hypertrophic in patients with DDS as it needs to assist stabilization the hip due to atrophy of the gluteal muscles.[30,31] Therefore, because of the anterolateral location of TFL on the hip and that patients have shallow anterolateral acetabulum in DDS cases, the TFL may become a key stabilizer. Hip dysplasia patients presented with significantly greater internal rotation in 90°of hip flexion (mean SD = $33^{o}\pm16^{o}$) and abduction ($46^{o}\pm11^{o}$) compared to FAI patients ($38^{o}\pm10^{o}$ and $21^{o}\pm12^{o}$ respectively). Therefore, the presence of a hypertrophic TFL in DDS patients may be due to having more available hip flexion and IR movement capabilities. A systematic review found that hip dysplasia was more prevalent in left hips with 64% of DDS diagnosis being found in the left hip.[23] Therefore, with increased internal rotation and flexion (movements that the TFL produce and assist) and avoidance of extension (stretching TFL) along with most individuals with DDS having a shallow acetabulum located anteriorly and superior-lateral (position of the TFL anatomically) the TFL could become hypertrophic/ overused compared to a non DDS joint and therefore could help to indicate the presence of hip dysplasia.

HIP OSTEOARTHRITIS (OA)

Hip osteoarthritis is a degenerative condition that causes deterioration of the articular cartilage within the hip joint.[32] Radiographically, OA is defined by the presence of osteophytes, sclerosis of subchondral tissue, cysts, joint space narrowing and bone contour abnormalities.[33] Degeneration of the cartilage leads to decreased range of movement, pain, and inflammation.[32] ROM in an arthritic hip becomes limited, with loss of strength of flexion (22%), abduction (31%) and adduction (25%), but in particular IR, ER and abduction ROMs are reduced the higher the grade of severity the OA hip joint (Control group 13-52% more flexible though IR, ER and Abduction p=<.0001-.001) when using the Kellgren-Lawrence grading system.[34] This suggests that the reduction of strength could be the reason for loss of hip ROM. Previous research found functional movements such as marching on the spot, ascending/descending stairs, 25m walking and flexion-extension and abduction-adduction movements were significantly better in a control group vs group with diagnosed hip OA.[34] The research found a reduction in hip abductor strength (31%) and flexor strength (18-22%) in subjects with hip OA compared to age matched controls.[35] The research also found that the affected side was 13-22% weaker in flexion and abduction than the subject's asymptomatic side, suggesting that flexion and abduction are affected in patients with hip OA.[35] Hip OA usually occurs as a secondary pathology in the latter phases of previous joint pathologies such as DDS and FAIS.[33,36-41] Once hip OA has been diagnosed via radiographs, daily function is impaired, and conservative interventions have been exhausted, a total hip replacement (THR) is usually the next phase in treatment. Therefore,

Table 1. Normative range of movement values and direction of range of movement in the presence of intra-articular pathology between 25- and 74-year-old.[45]

	CI ROM	FAIS	DDS	OA	CLP
Flexion	120-121°	Reduced	Reduced	Reduced	Reduced
Extension	19-20°	Reduced	Reduced	Reduced	-
Abduction	42-43°	Reduced	Reduced	Reduced	-
Adduction	30°-32°	-	-	Reduced	Reduced
External Rotation	31-32°	Reduced	-	Reduced	-
Internal rotation	32-33°	Reduced	Increased	Reduced	Reduced

CI = Confidence interval.

with OA affecting all joint movements and causing a reduction in strength of the abductors and flexors (TFL provides flexion and abduction) along with OA affecting gait, the TFL will be affected due to its role in gait and role in producing force in the movements that are limited.

CHONDROLABRAL PATHOLOGY

The labrum of the hip is defined as "a fibrocartilage tissue that plays important roles in proprioception, nociception, synovial seal effect and static and dynamic joint stability and as a shock absorber".[42] Labral pathology (tears and degeneration) of the hip is associated with increased risk of hip OA and has seen a rise in surgical treatment, usually arthroscopically.[43] Patients who do not rectify any muscular strength or ROM deficits found in chondrolabral pathology increase the likelihood of progression to hip OA. 73% (273) of patients with labral lesions had evidence of chondral damage in the acetabulum.[41] Research compared arthroscopically treated patients with chondrolabral pathologies and healthy participants who were asymptomatic and had no previous surgery.[44] The labral pathology group followed the same pattern as hip OA and FAIS, with hip chondrolabral pathology participants having differences in reduced ROM and strength at the hip joint. Patients with chondrolabral pathology presented with reduced IR (p=0.004) and increased extension (p= 0.019). Interestingly, GMax strength was reduced (p=0.033) in the pathology group, supporting the premise that chondrolabral pathology is affecting the gluteus musculature the same as FAIS and DDS.[44] Labral pathology displays the same characteristics as FAIS and DDS pathologies, notably abnormal IR ROM (hypo and hyper), and both abduction and gluteal strength issues. Hence, could the TFL-UGMax complex, specifically the strength ratio between them, help identify the presence or intraarticular and degenerative hip pathology?

COMPARISON OF PATHOLOGIES

Table 1 shows that flexion and internal rotation deficits are present across all pathologies, abduction deficits across three pathologies which are TFL movements. Extension is also reduced across three pathologies, with reduction in ER and abduction which are all Gluteus Maximus movements. Fine wire EMG during gait and found that TFL worked during the early stance phase and during mid stance and that greater hip abduction force was required during external hip adduction moment during the stance phase.[46] This suggests that TFL is directly involved in the gait cycle. The UGMax was found to be active during weight acceptance of the hip; thus, both the TFL and UGMax were working to stabilize the lateral hip, thus showing potential diagnostic utility.

DIAGNOSING DEGENERATIVE AND INTRA-ARTICULAR HIP PATHOLOGY

Currently, the accepted method of diagnosing hip pathology, requires three criteria to be present to confirm diagnosis:

- A patient has to be symptomatic (presence of pain and/or loss of function, etc)
- Have positive results on special tests conducted by a health professional (Doctor, Physiotherapist, etc).
- Radiographic confirmation (using X-ray, MRI etc).[26]

Special tests, commonly used when hip pain is reported, are the FADIR (Flexion-Adduction-external rotation) and FABER's (Flexion-Abduction-External Rotation) tests.[46] A systematic review found that even with x-ray, MRI and CT scans used to confirm diagnosis, the FADIR test still had a wide range of variation with sensitivity findings of 0.08 to 1, 0.33 to 1 and 0.9 respectively (1 being 100% accurate, 0.0 being 0% accurate) and specificity findings of 0.11-1 for both x-ray and MRI respectively.[47] However, the FADIR and FABER's tests were found to have a higher specificity score than most studies used in the review. This suggests that these two tests are more specific at identifying patients without intraarticular pathology but cannot categorically confirm the presence of intraarticular pathology. This was supported by another systematic review that included 21 studies describing 18 clinical tests for FAIS and labral pathology which concluded that no combination of physical diagnostic tests by themselves can reliably diagnose FAIS or labral pathology due to the discrepancies between test executions.[48] This has led to the Warrick agreement to advise the procedure of special tests, imaging and a symptomatic patient to diagnose

With the TFL and UGMax complex correlating with degenerative/intraarticular pathology affecting loss of function, coupled with accepted rehabilitation including addressing muscle weakness of the Gluteus muscles and hip flexors, it stands to reason that the TFL and UGMax complex could provide indication to intra articular hip pathology.[26] Therefore, this would allow clinicians to suspect and address potential pathology before it has had opportunity to progress to full hip OA.

LIMITATIONS AND THE FUTURE DIRECTION OF RESEARCH

Research surrounding the lateral hip complex has investigated the size of muscles, strength of muscles and activation of the musculature around the hip in relation to hip pathology. However, there is a paucity of research on the impact of the relationships between the muscles except for which is the best gluteal exercise to inhibit the TFL as it is accepted that rehabilitation requires GMax strengthening and TFL relaxation.

Research into the strength of the TFL and UGMax is needed to observe any strength differences between TFL and UGMax between affected hip joints compared to healthy hip joints. Normative data on the strength of TFL and UGMax is required. By evaluating the strength on the TFL and UGMax within normal gait angles and gaining a ratio of their individual roles within abduction, this would enable a comparison between suspected pathology and baseline values to indicate the potential for intraarticular pathology. This then needs to translate into clinical diagnosis so an inter-rater reliability study would also be useful and research into the best way to reverse a ratio change.

CONCLUSION

The lateral muscular complex of the TFL and the UGMax could offer valuable information relating to the assessment of individuals with intra-articular and degenerative hip pathologies. The arthrogenic neuromuscular inhibition evident with injury at the hip, trophic changes to TFL and UGMax, changes in ROM at the hip specifically in IR, extension and abduction with the presence of pathology, and the role of TFL and UGMax within gait all indicate that this complex must be researched, and its potential role in identifying hip pathology.

Therefore, at this stage, it is plausible to suggest that the strength ratio of the TFL compared to the UGMax should be examined as it may identify individuals with degenerative and/or intra articular hip pathology.

DISCLOSURE

In the previous 5 years, one authors employer has received income for expert consultancy activities that lie outside of the submitted work. The author declares book royalties from Oxford University Press. All other authors declare no conflicts of interests.

ABBREVIATIONS

TFL, Tensor Fascia Latae; GMax, Gluteus Maximus; UGMax, Upper Gluteus Maximus; LGMax, Lower Gluteus Maximus; GMed, Gluteus Medius: GMin, Gluteus Minimus; FAIS, Femoroacetabular impingement syndrome; DDS, Dysplasia; OA, Osteoarthritis; MRI, magnetic resonance imaging; ANI, Arthrogenic neuromuscular inhibition; UK, United Kingdom; THR, Total hip replacement; NHS, National Health service; IR, Internal rotation; AM, anteromedial; PL, posterolateral; EMG, Electromyography; SIJ, Sacroiliac joint; LGM, Lower gluteus maximus; IT, iliotibial; CLP, Chondrolabral pathology; HOOS, hip osteoarthritis outcome score; SD, standard deviation; ROM, Range of movement; FADIR, flexion-adduction-internal rotation; FABER, Flexion-abduction-external rotation;

Submitted: October 23, 2024 CST. Accepted: February 04, 2025 CST. Published: March 01, 2025 CST.

REFERENCES

1. Sato T, Sato N. Clinical relevance of the hip joint: Part 1 – Review of the anatomy of the hip joint. *Int Muscul Med*. 2015;37:141-145. doi:10.1179/1753615415Y.0000000005

2. Layton R, Messenger N, Stewart T. Characteristics of hip joint forces during range of activities. *Med Eng and Phys*. 2022;108. doi:10.1016/j.medengphy.2022.103894

3. Cleather DJ, Goodwin JE, Bull AMJ. Hip and knee joint loading during vertical jumping and push jerking. *Clin Biomech*. 2013;28:98-103. doi:10.1016/j.clinbiomech.2012.10.006

4. NHS Digital. Annual number of hip replacement procedures in England from 2009 to 2021. Statista. 2022. Accessed March 16, 2023. https://www-statista-com.leedsbeckett.idm.oclc.org/statistics/1099162/hip-replacement-procedures-in-england/?locale=en

5. National Joint Registry. Number of hip replacement procedures in England, Wales, and Northern Ireland from 2004 to 2020. Statista. 2021. Accessed March 16, 2023. https://www-statista-com.leedsbeckett.idm.oclc.org/statistics/1060563/hip-replacements-in-eng-wales-ni-since-2004/

6. NHS Digital. Rate of hip replacements in England 2020/21, by age and gender (per 100,000 population). Statista.

7. Patients Association. Average number of days waiting for hip surgery on the National Health Service in the United Kingdom (UK) from 2010 to 2015. Statista. 2016. Accessed March 14, 2023. https://www-statista-com.leedsbeckett.idm.oclc.org/statistics/488135/hip-surgery-waiting-time-elective-procedure-united-kingdom/?locale=en

8. Grimaldi A, Richardson C, Durbridge G, Donnelly W, Darnell R, Hides J. The association between degenerative hip joint pathology and size of the gluteus maximus and tensor fascia lata muscles. *Man Ther*. 2009;14(6):611-617. doi:10.1016/j.math.2008.11.002

9. Flack NAMS, Nicholson HD, Woodley SJ. A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus Minimus, and tensor fascia Lata. *Clin Anat*. 2012;25:697-708. doi:10.1002/ca.22004

10. Beaulieu ML, Lamotagne M, Beaule PE. Lower limb biomechanics during gait do not return to normal following total hip arthroplasty. *Gait Posture*. 2010;32:269-273. doi:10.1016/j.gaitpost.2010.05.007

11. Trammell AP, Nahian A, Pilson H. *Anatomy, Bony Pelvis, and Lower Limb: Tensor Fascia Latae*. StatPearls; 2022.

12. Pare EB, Stern JT Jr, Schwartz JM. Functional differentiation within the tensor fascia latae. A telemetered electromyographic analysis of its locomotor roles. *J Bone Joint Surg*. 1981;63:1457-1471. doi:10.2106/00004623-198163090-00013

13. Besomi M, Maclachlan L, Mellor R. Tensor Fascia Latae muscle structure and activation in individuals with lower limb musculoskeletal conditions: a systematic review and meta-analysis. *Sports Med*. 2020;50:965-985.

14. Novacheck TF. The biomechanics of running. *Gait Posture*. 1998;7:77-95. doi:10.1016/S0966-6362(97)00038-6

15. Choi SA, Cynn HS, Yi CH, et al. Isometric hip abduction using a Thera-Band alters gluteus maximus muscle activity and the anterior pelvic tilt angle during bridging exercise. *J Electromyogr Kinesiol*. 2015;25(2):310-315. doi:10.1016/j.jelekin.2014.09.005

16. Hopkins J, Ingersoll C. Arthrogenic muscle inhibition: a limiting factor in joint rehabilitation. *J Sport Rehab*. 2000;9:135-159. doi:10.1123/jsr.9.2.135

17. Freeman S, Mascia A, Mcgill S. Arthrogenic neuromusculature inhibition: A foundational investigation of existence in the hip joint. *Clin Biomech*. 2013;28:171-177. doi:10.1016/j.clinbiomech.2012.11.014

18. Siverling S, O'Sullivan E, Garofalo M, Moley P. Hip Osteoarthritis, and the active patient: will I run again? *Cur Rev in Musculo Med*. 2012;5:24-31.

19. Wall PDH, Dickenson EJ, Robinson D, et al. Personalised Hip Therapy: development of a non-operative protocol to treat Femoroacetabular impingement syndrome in the FASHIoN randomised controlled trial. *Br J of Sport Med*. 2016;50:1217-1223. doi:10.1136/bjsports-2016-096368

20. Versus Arthritis. *The State of Musculoskeletal Health 2023. Arthritis and Other Musculoskeletal Conditions in Numbers*. Versus Arthritis; 2023. http://www.versuarthritis.org

21. Arthritis Research UK. Oseteoarthritis in general practice. Arthritis Research UK. 2013. https://www.versusarthritis.org

22. Woodacre T, Ball T, Cox P. Epidemiology of developmental dysplasia of the hip within the UK: refining the risk factors. *J of Child Ortho*. 2016;10:633-642. doi:10.1007/s11832-016-0798-5

23. Loder RT, Skopelja EN. The epidemiology and demographics of hip dysplasia. *ISRN Orthopaedics*. 2011;10. doi:10.5402/2011/238607

24. Shamberger CT, Tuffs C, Suda AJ, Grossner T, Schmidmaier G, Stein S. Association of Chondrolabral lesions with untrasound-gudied detection of pathological head-neck contour. *Diagnostics*. 2023;13:3334. doi:10.3390/diagnostics13213334

25. Lewis CL, Sahrmann SA. Acetabular labral tears. *Physical Thera*. 2006;86:110-121. doi:10.1093/ptj/86.1.110

26. Griffin DR, Dickenson EJ, O'Donnell J, et al. The Warick Agreement on Femoroacetabular impingement syndrome (FAI syndrome): an international consensus statement. *Br J of Sport Med*. 2016;50:1169-1176. doi:10.1136/bjsports-2016-096743

27. Saaman MA, Zhang AL, Popovic T, Pedoia V, Majumdar S, Souza RB. Hip joint muscle forces during gait in patients with Femoroacetabular impingement syndrome are associated with the patient reported outcomes and cartilage composition. *J of Biomech*. 2019;84:138-146. doi:10.1016/j.jbiomech.2018.12.026

28. Philippon MJ, Maxwell RB, Johnston TL, Schenker M, Briggs KK. Clinical presentation of Femoroacetabular impingement. *Knee Surgery, Sport Trauma, Arthro*. 2007;15:1041-1047. doi:10.1007/s00167-007-0348-2

29. Kraeutler MJ, Garabekyan T, Pascual-Garrido C, Mei-Dan O. Hip instability: a review of hip dysplasia and other contributing factors. *Musc Lig and Tend J*. 2016;6:343-353.

30. Mathews B, Courtney CA, Fernandez – de-la-Penas C. *Hip and Knee Disorders: Integrating Manual Therapy and Exercise*. Handspring; 2022.

31. Pereira C, Toukouki A, Kurth W. Unilateral Hypertrophy of the Tensor Fascia Lata. *J of the Belgian Soc of Rad*. 2024;108:32. doi:10.5334/jbsr.3462

32. Aresti N, Kassam J, Nicholas N, Achan P. Hip Osteoarthritis. *Br Med J*. 2016;354:3405. doi:10.1136/bmj.i3405

33. Arden N, Nevitt CM. Osteoarthritis: epidemiology. *Best Prac and Res: Clin Rheuma*. 2006;20:3-25. doi:10.1016/j.berh.2005.09.007

34. Arokoski MH, Haara M, Helminen HJ, Arokoski JP. Physical function in men with and without hip osteoarthritis. *Arch of Phys Med and Rehab*. 2004;85:574-581. doi:10.1016/j.apmr.2003.07.011

35. Arokoski MH, Arokoski JPA, Haara M, et al. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. *J of Rheuma*. 2002;29:2185-2195.

36. Ganz R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Ortho and Rel Res*. 2003;417:112-120. doi:10.1097/01.blo.0000096804.78689.c2

37. Tonnis D, Heinecke A. Acetabular and femoral anteversion: Relationship with osteoarthritis of the hip. *J of None and Joint Surg*. 1999;81:1747-1770.

38. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage. *J of Bone Joint Surg*. 2005;87:1012-1018. doi:10.1302/0301-620X.87B7.15203

39. Boykin RE, Anz AW, Bushnell BD, Kocher MS, Stubbs AJ, Philippon MJ. Hip instability. *J of the Amer Acad of Ortho Surg*. 2011;6:340-309. doi:10.5435/00124635-201106000-00004

40. Ganz R, Leunig M, Leunig-Ganz K, Harris WH. The etiology of osteoarthritis of the hip. *Clin Ortho and Rel Res*. 2008;466:264-272. doi:10.1007/s11999-007-0060-z

41. McCarthy JC, Noble PC, Schuck MR, Wright J, Lee J. The Role of Labral Lesions to Development of Early Degenerative Hip Disease. *Clin Ortho and Rel Res*. 2001;39:325-337. doi:10.1097/00003086-200112000-00004

42. Nakashima H, Tsukamoto M, Ohnishi Y, et al. Clinical and radiographic predictors for unsalvageable labral tear at the time of initial hip athroscopic management of Femoroacetabular impingement. *Amer J of Sport Med*. 2019;47:2029-2037. doi:10.1177/0363546519856018

43. Mughal A, Vukanic D, Murphy EP, Crozier-shaw G, Mahapatra A. Labral tear is associated with increased rish of osteoarthritis – A cross-sectional study. *J of Ortho Reports*. 2023;2:1-5.

44. Kemp JL, Makdissi M, Schache AG, Pritchard MG, Pollard TCB, Crossley KM. Hip chondropathy at arthroscopy: prevalence and relationship to labral pathology, Femoroacetabular impingement and patient-reported outcomes. *Br J of Sport Med*. 2014;48:1102-1107. doi:10.1136/bjsports-2013-093312

45. Roach KE, Miles TP. Normal hip, and knee active range of motion: the relationship to age. *Phys Ther*. 1991;71:656-665. doi:10.1093/ptj/71.9.656

46. Trevethan R. Sensitivity, specificity, and predictive values: foundations, pliability's and pitfall in research and practice. *Front in pub health*. 2017;5. doi:10.3389/fpubh.2017.00307

47. Shanmugaraj A, Shell JR, Horner NS, et al. How useful is the flexion-adduction-internal rotation test for diagnosing Femoroacetabular impingement: A systematic review. *J of Clin Sport Med*. 2020;30:76-82. doi:10.1097/JSM.0000000000000575

48. Tijssen PT, Van Cengel R, Williemsen L, deVisser E. Diagnostics of Femoroacetabular Impingement and Labral Pathology of the Hip: A Systematic Review of the Accuracy and Validity of Physical Tests. *Arthroscopy*. 2012;28:860-871. doi:10.1016/j.arthro.2011.12.004