MUSCLE ACTIVATION DURING HIGH AND LOW INTENSITY STRENGTH TRAINING IN OLDER ADULTS WITH KNEE OSTEOARTHRITIS

BY

ALICIA N SABATINO

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Approved By:

Stephen P. Messier, Ph.D., Advisor

Daniel Beavers, Ph.D., Chair

Paul DeVita, Ph.D.

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Abstract

Introduction: Muscle weakness is a common impairment in the knee osteoarthritis (OA) population that is partially due to an impairment in the central nervous systems ability to activate a muscle fully. The effect of resistance training intensities on muscle activation in the knee OA population has not been studied. Therefore the purpose of this study was to compare muscle activation of older adults with knee OA during low intensity versus high intensity resistance training programs over an 18 week period.

Methods: Participants (N=25) were recruited from the Strength Training for Arthritis Trial (START). Participants were randomized into low intensity (30% 1-RM) or high intensity (75% 1-RM) strength training groups. EMG of the vastus lateralis, biceps femoris and gluteus medius were analyzed at baseline, 9 weeks and 18 weeks following training.

Results: Mean activation of the gluteus medius was significantly greater (P=0.028) in the high intensity group at 18 weeks compared to the low intensity group. Mean activation for the biceps femoris was significantly greater (P=0.024) in the high intensity group at 9 weeks compared to the low intensity group. No significance was found for the vastus lateralis.

Conclusion: Rehabilitation efforts should utilize high intensity resistance training to maximize muscle adaptations in the knee OA population.

Introduction

The most common cause of disability among the United States population of older adults is arthritis[1]. Osteoarthritis (OA) is the most prevalent form[2] and the knee is the most affected weight bearing joint, affecting 250 million people worldwide[3]. Knee OA is a progressive, degenerative disease which affects the entire joint. Breakdown of the joint tissues can lead to pain, stiffness, functional limitation, disability, and ultimately a poor quality of life.

Osteoarthritis is currently a disease that has no cure. Pain is the most debilitating symptom and is the focus of current treatment. Exercise is a common treatment for patients with knee OA due to its potential to reduce pain, increase muscle strength, aid in weight loss, improve mobility and enhance health related quality of life. Individuals with knee OA commonly exhibit quadriceps muscle weakness which is inversely related to knee pain[4]. Resistance training is commonly prescribed as a form of treatment; it decreases pain significantly while increasing functional ability [5–10].

The gradual decline in quadriceps strength associated with knee OA is attributed, in part, to impairment in the central nervous system's ability to activate the muscle fully. This is termed central activation failure or arthrogenous muscle inhibition (AMI)[11–13]. It is theorized that AMI can result from progressive joint degeneration resulting in abnormal articular afferent information being sent to the motor neurons that reduces activation[14]. Increases in strength during the early stages of a resistance training program are mainly due to neural adaptations that

elevate motor unit recruitment. According to the Henneman's size principle of recruitment[15], muscle fibers are recruited progressively so that smaller motor units are recruited first followed by the larger motor units. Since substantial force production is required when lifting heavy loads such as 75% of 1 repetition max (RM), to meet the force demands both small and larger motor units must be recruited. Slow twitch muscle fibers are recruited first followed by fast twitch muscles fibers. This is in contrast to lifting lighter loads such as 30% of 1RM which has lower force demands therefore requiring less motor unit recruitment and consequently less muscle activation[16].

Patients with knee OA have significant lower extremity muscle weakness as well as lower muscle activation compared to the healthy population. To maximize strength gains in this population it is important to increase muscle activation which leads to an increase in lean mass. Since muscle activation may be a predictor of strength gains in the OA population[17], determining the resistance training intensity that elicits a greater activation response may shed light on the optimal resistance training load prescription for treatment of knee OA. To initiate an adaptive response to strength training, a muscle fiber must be recruited, which makes high load training logical to maximize muscle adaptation. The effect of exercise intensities on muscle activation in the knee OA population has not been studied. Therefore the purpose of this study was to compare muscle activation of older adults with knee OA during low intensity versus high intensity strength training programs over an 18 week period.

Review of the Literature

Epidemiology of Osteoarthritis

The most common cause of disability among older adults is arthritis[18]. Approximately 52.5 million Americans have been diagnosed with some form of arthritis[1]. Osteoarthritis (OA) is the most prevalent[2] with approximately 27 million Americans affected [19]. OA is a progressive, degenerative disease that effects the entire joint. Breakdown of the joint tissues can lead to relative degrees of pain and stiffness leading to functional limitation and disability, and reduced quality of life.

In the United States 13.9% of adults aged 25 or older and 33.6% of those 65 years and older are affected by OA[2]. The joints of the hand are most commonly affected by OA,[2] with knee OA being the second most prevalent form.[20] Approximately 6% of all adults are affected by knee OA[21]. The Framingham Osteoarthritis Study[22] examined the prevalence of knee OA in the older population and found that it was high and that it increased with age. There were 1,418 participants in the study of which 33% had radiographic knee OA and 9.5% had symptomatic knee OA. The data indicated 27% of those aged 65-69 had evidence of radiographic OA that increased to 51% over the age of 85 years. Furthermore, women had a slightly higher prevalence compared to men. Interestingly, although there was a correlation between increased severity of radiographic knee OA and symptoms, only 40% of those with Kellgren-Lawrence grade 3 or 4 (moderate-severe) radiographic changes had symptoms. The Johnston County Osteoarthritis Project[23] used a sample of both African American

and Caucasian participants. They found that African Americans had a higher prevalence of OA compared to Caucasians. Similar to the Framingham study, this study also found that prevalence among women was higher, and increased with age.

The overall incidence of knee OA is 240 per 100,000 person years[24], it increases with age, especially after 50 years, with the highest incidence in the age range of 70-79 years for both men (839/100,000 person years) and women (1,082/100,000 person years). According to Murphy and colleagues,[25] the lifetime risk of OA (developing OA by the age of 85) is 44.7%. Obese individuals had significantly higher lifetime risk at 60.5% compared to 30.2% and 46.9% among normal weight and overweight respectively. The incidence rates are highest among those with the following baseline characteristics: age greater than 75 years, obese, history of knee injury, or an annual household income less than $15,000. Grotle and colleagues found that individuals with a high BMI ($\geq$ 30) were 2.8 times more likely to have knee OA than those of normal weight (OR 2.81; 95%CI 1.32-5.96)[26]. Pain and stiffness have the greatest effect on functional limitation and disability,[27–29] suggesting that efforts to reduce pain will have beneficial effects on function and quality of life.

Pathology/ Development and Progression of OA

OA is complex in that there are multiple disease pathways.[30]. There is no single pathogenesis to the onset and progression of knee OA but rather a combination of factors that coalesce. The development of knee OA is attributed

to factors including the breakdown of the articular cartilage on the femur and tibia as well as alterations to the subchondral bone[31]. The disease affects components of the entire joint including bone, cartilage, muscles, ligaments, the synovial membrane, and the joint capsule. There is joint space narrowing with an increase in joint inflammation. The causes of degenerative changes are complex and involve interrelated biological, mechanical and structural changes[30,32–46].

The mechanical component refers to any signal that delivers a mechanical stimulus to the body and spans all the way to the mechanical cell environment[30]. Mechanical factors can include acute trauma, muscle weakness, joint deformity and repetitive use[47] which destroy chondrocytes and disrupt the extracellular matrix[48]. This results in proteoglycan depletion which is essential to maintaining the load bearing role of cartilage. Studies have shown that the external adduction moment during walking plays a role in the progression of knee OA. A high adduction moment can accelerate the rate of disease progression. Subjects with lower adduction moments had substantially better clinical outcomes than those with higher values[49,50,51]. Trauma such as ACL or meniscal injury is a factor in the onset and progression of knee OA due to kinematic changes[52–55] that can shift the load bearing areas of the knee away from normal contact locations. If the cartilage in the new load regions cannot adapt, degeneration occurs[55].

The biological component includes the factors that influence cell metabolism, levels of systemic inflammation and genetic etiologies[30]. Although OA has been widely recognized as a non-inflammatory disease, more recent

studies have suggested inflammation is detrimental to the clinical outcome[36,42,43,56]. Evidence shows presence of chronic synovitis is common among individuals with OA, even in the absence of acute inflammatory flares[36]. Pro-inflammatory cytokines such as Interleukin-6 (IL-6) are up-regulated during synovial inflammation that can augment inflammatory angiogenesis[36,42,43]. Angiogenesis is the formation of new capillary blood vessels that contribute to inflammation, osteophyte formation and increased pain[36]. New blood vessels have increased permeability to macromolecules that facilitate edema formation and can transport inflammatory cells, nutrients and oxygen to the site of inflammation. Angiogenesis can facilitate pain through structural reorganization of the joint and growth of fine sensory nerves[36]. Elevated serum levels of C-reactive protein (CRP) are present in patients with OA [57] and could be attributed to increased levels of IL-6 stimulating CRP production[39]. Higher levels of CRP have been associated with progression of radiologic disease, especially cartilage loss[57]. In addition, those with rapidly progressive OA have higher levels of CRP compared to those with slower or non-progressive OA, suggesting the importance of inflammatory markers to the progression of OA[36,57].

Diagnosis

Knee OA can be diagnosed radiographically and symptomatically. Radiographic knee OA is most commonly diagnosed using the Kellgren-Lawrence[58] grading scale. This scale ranges from 0 to 4 and is based on joint space narrowing and the formation of osteophytes and bone deformity. A grade

of 0 is an absence of any evidence of joint space narrowing or osteophyte formation, grade 1 indicates doubtful narrowing of the joint space with possible osteophyte formation, grade 2 displays definite joint space narrowing with definite osteophytes formed, grade 3 expresses moderate multiple osteophytes with definite joint space narrowing, and grade 4 displays large osteophytes, substantial joint space narrowing and definite bone deformity.

Symptomatic knee OA is an expression of the severity of pain associated with the knee. Generally pain is evaluated using questionnaires that inquire about an individual's presence of pain, stiffness, and/or joint swelling. Diagnosis of knee OA requires a combination of approaches due to the weak association between symptoms of knee OA and radiographic findings[59]. According to the American College of Rheumatology, clinical diagnosis of OA is defined as knee pain plus at least 3 of the following 6 criteria: age > 50 years, stiffness < 30 minutes, crepitus, bony tenderness, boney enlargement, and no palpable warmth[60]. These clinical criteria are often used in combination with radiographic and laboratory findings. Studies have shown that individuals who show low levels of radiographic knee OA could have severe pain levels while in other incidences, individuals with high levels of radiographic knee OA may have low pain levels[61]. Since symptomatic and radiographic knee OA are poorly correlated, for clinical purposes, the goal of treatment is pain reduction.

Risk Factors

The etiology of knee OA is due to either systemic or local/mechanical risk factors that interplay resulting in the onset and progression of OA. Local/mechanical factors are further divided into extrinsic and intrinsic factors. Systemic factors such as age and ethnicity may predispose and individual to OA, while local factors such as abnormal joint mechanics due to injury may be what initiates the cascade of changes to the joint that can result in OA[62]. Systemic factors such as age and sex are considered non-modifiable, while local factors such as BMI, muscle weakness and level of physical activity are considered modifiable. These are generally the main focus for risk reduction and intervention.

Increasing age is associated with an increased incidence and prevalence of both radiographic and symptomatic knee OA[22,24,62]. Risk of OA increases drastically at ages above 50 years[24]. Data from the Framingham Osteoarthritis Study showed 27% of those aged 65-69 had evidence of radiographic OA that increased to 51% over the age of 85 years[22]. Furthermore, a health maintenance based survey showed a ten-fold increase in OA between the ages of 30-65 years[24]. The high risk due to an increase in age can be due to various factors such as changes in articular cartilage with possible joint space narrowing, increased joint laxity, past injury with resultant biomechanical changes or other age related factors[62].

Sex is another risk factor associated with increased incidence and prevalence of knee OA. Data from the Framingham Knee Osteoarthritis Study reported a 1.7 times higher incidence of osteoarthritis of the knee in women than in men (95%CI: 1.5–2.7). Similarly, women were estimated to have a lifetime increased risk of symptomatic knee OA of 46.8% (95%CI: 41.2–52.5)[22]. A possible explanation for the differences seen between males and females could be due to hormonal changes. There is a significant increase in the incidence of OA in women above the age of 50[24] that coincides with the onset of menopause. However, results from both observational studies and larger randomized trials on the association of estrogen and osteoarthritis have been mixed[62].

Racial and ethnic differences may impact the prevalence of knee OA in certain populations. Data from the Johnson County Osteoarthritis Project showed a 6% higher prevalence of radiographic knee OA among African-Americans than Caucasians [32.4%(95%CI:29.8–35.1) v. 26.8%(25.3–28.4)][23]. This was supported by the NHANES-III data that also reported a higher incidence of knee OA in African Americans than Caucasians [OR = 1.65, 95%CI: 1.17–2.37], as well as more symptomatic knee OA in African-Americans than Caucasians: [OR = 1.52, 95%CI: 1.06–2.19][63].

Local factors that affect the risk of knee OA are thought to work through abnormal joint loading that alters the load across the joint[62]. These local factors include obesity, malalignment, muscle weakness, previous trauma/injury, sports participation, or occupation. Intrinsic local factors include muscle weakness, joint

malalignment, and joint laxity. Extrinsic include factors such as obesity, physical activity, acute injury, and repetitive joint loading.

Numerous studies have demonstrated that overweight and obese individuals have an increased risk for developing knee OA. A 2015 meta-analysis of the risk factors of OA found 25 studies that reported overweight or obese as a significant risk for developing knee OA[64]. Of the 25 studies used in the meta-analysis there was a pooled OR of 2.10 (CI 1.82-2.42) showing an increased risk of knee OA in those overweight or obese[64]. There is a progressive increase in shear and compressive forces with each increase in BMI category[65]. These increased forces are associated with alterations in gait that contributes to knee OA risk compared to those who are healthy weight[66]. There is also a significant association between weight loss and decreased compressive loads. Messier et al. demonstrated a direct relationship between weight reduction and decreased tibiofemoral joint contact forces. With each pound of weight loss there is a subsequent 4 pound reduction of joint load[67]. Furthermore Aaboe et al. showed that with each 1N of weight loss, compressive loading was reduced by 2.2 N[68]. This demonstrates that BMI as a risk factor is modifiable and should be considered when treating knee OA. The prevalence of knee OA is projected to increase dramatically in future years due to the increasing age of the population and ongoing obesity epidemic[69]

Although varus or valgus alignment is considered a risk factor for incident knee OA, the current research has shown conflicting results. Data from the

Rotterdam study found that varus alignment was associated with a 2-fold increased risk for knee OA (OR 2.06, 95% CI 1.28-3.32) as well as an increased risk of OA progression compared to the group with normal alignment (OR 2.90, 95% CI 1.07-7.88)[70]. Data from the Framingham Osteoarthritis Study showed different results with no significant increase in incident OA in the highest quartile compared with the lowest quartile category for any of the alignment measures. It was suggested that malalignment be used as a marker of disease severity and/ or progression.[71].

Knees with existing OA have weaker quadriceps than healthy knees however, whether greater muscle strength surrounding the knee can protect against further progression is uncertain. Greater muscle contraction increases joint loads during activity[72], whether this is a factor in progression of OA is questionable. Some studies suggest that uadriceps and hamstring co-contraction during gait work to attenuate loads in the knee, which would suggest that greater strength in the muscles surrounding the knee would act as a protective mechanism against the development and progression of knee OA[72]. Segal[73] and Hootman[74] conducted studies supporting this idea and found that quadriceps strength significantly reduced the risk for knee OA. On the other hand, there are data that imply that greater quadriceps muscle strength may be associated with increased risk of OA in malaligned[72] and lax knees[75]. Omori and colleagues observed a tendency of the quadriceps muscle strength level to decrease with progression of knee OA grade[76]. Individuals with knee OA often use a pain reduction technique called quadriceps avoidance in which muscle

atrophy is likely to occur. During gait individuals reduce knee flexion therefor reducing articular forces in an attempt to decrease knee joint pain[77]. This is a likely link between muscle weakness and knee OA.

Knee injury is also a significant risk factor in the development of knee OA. Injury to the joint can lead to altered mechanics which in turn can lead to the development of knee OA. Multiple studies have examined the impact of sustaining an ACL injury on the prevalence of knee OA. Two studies in particular demonstrated an increased prevalence of OA in females 12 years after injury and in males 14 years after injury[78,79]. Meniscal resection has also been found to increase the risk of incident knee OA (RR=2.6, 95% CI: 1.3-6.1), with total meniscectomy showing an even stronger risk up to 15-22 years after the procedure[80]. Total meniscectomy has been associated with a 6-fold increased relative risk for the development of knee OA[80].

The association of physical activity and knee OA is somewhat conflicting. Engaging in physical activity results in repetitive loading at the knee joint that could potentially increase the risk for developing OA. However, physical activity can yield many benefits such as joint lubrication and strengthening of the muscles surrounding the knee. High level participation in sports has been associated with an increased risk of knee OA[81]. In the Framingham study, individuals who participated in 4 or more hours a day of leisure time physical activity were at a 3-fold increased risk for developing radiographic knee OA compared to a sedentary group[22]. However, studies examining the association of

running with knee OA found that neither heavy mileage nor the number of years running were contributory to the future development of OA[82,83]. Data are clearer when examining occupational physical activity and its association with knee OA. Jobs which require prolonged kneeling or squatting/bending result in an elevated risk for development of knee OA compared to jobs that don't require these actions[84,85].

Treatment Options

OA is currently a disease that has no treatment to completely cure arthritis. Pain is the most debilitating symptom of OA and is different for each person. For this reason treatments focus on pain reduction to improve function and quality of life. There are various ways to attempt to reduce pain including exercise, patient education, medication, surgical procedures and alternative medicine options such as Tai Chi, meditation or yoga.

Exercise is commonly prescribed for knee OA because of its potential to reduce pain, increase muscle strength, aid in weight loss, improve mobility and enhance overall well-being. Currently the research regarding which form of exercise (resistance or aerobic) is most effective is unclear, however there have been various studies reporting both resistance and/or aerobic exercise can be successful. The Fitness Arthritis and Seniors Trail (FAST)[86] compared aerobic exercise to resistance training in community dwelling subjects with knee OA. Walking three times per week for 18 months reduced pain levels by 12% while

resistance exercises reduced pain by 8%, suggesting that both exercise modalities can be successful.

Patient education is also effective in reducing pain due to OA. Mazzuca and colleagues[87] analyzed the effects of self-care education on the health status of patients with knee OA. The education group received 30-60 minute individualized educational intervention that emphasized non-pharmacologic management of joint pain, preservation of function by problem solving and principals of joint protection. Data showed the education group had significantly lower scores for disability and resting knee pain.

Pharmacological intervention is another treatment for the reduction and management of knee OA pain. Common pharmacological interventions include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, and intra articular corticosteroids[88,89]. A 2015 systematic review comparing the effectiveness of pharmacological interventions for knee OA found acetaminophen the least effective treatment for pain compared to all other treatments under review[90]. They also found intra-articular treatments to be more effective than oral treatments. NSAIDs have been proven effective with decreases in WOMAC pain by 17.6 points and are most commonly prescribed[91]. NSAIDs and opioids have high toxicity and are of greater concern in persons with multiple co-morbidities. Up to 40% of the knee OA population are affected by co-morbidities that increase the toxicity of these drugs[91] . Major toxicities of concern include gastrointestinal, cardiovascular and fracture events[88,90,91]. Due to the risk of toxicity the

Osteoarthritis Research Society International (OARSI) guidelines have classified the use of NSAIDs and opioids for the treatment of knee OA as “uncertain”[88]. Despite the risk of toxicity the use of these drugs in the OA population has continued to increase[92].

When knee OA becomes so severe that other treatment options do not help, total joint replacement may be recommended. A randomized controlled trial comparing total knee replacement (TKR) to non-surgical treatment over a 12 month period found that those who underwent the surgical procedure had greater pain relief and functional improvement compared to the non-surgical group. The surgical procedure however, does come with more risks, as the study did mention the surgical group had 24 serious adverse events compared to only 6 in the non-surgical group[93]. Another study reported 60% of patients who received TKR improved >20 points (range 0-100) on WOMAC pain score 6 months post-surgery[94]. A retrospective study found >70% of the TKA patients had no pain at 1, 3, 5 and 10 years post operation[95]. Post-operative pain did not differ between patients age >80 years and <80 years, however, a greater complication rate was present in the group >80 years (19%) compared to the younger group (15%), suggesting the elderly are more prone to complication. Complications include acute renal failure, cerebrovascular accident, deep infection, deep venous thrombosis, implant loosening, myocardial infarction, periprosthetic fracture, prolonged wound drainage, pulmonary embolism, superficial infection and tibiofemoral dislocation.

Effects of Strength Training on OA

OA patients suffer limitations that lead to physical disability that can have a direct impact on other aspects of life such as social interactions, mental functioning and sleep quality. Health related quality of life (HRQoL) measures are important to help quantify the physical, social and emotional impact of knee OA[96]. Knee OA patients report lower scores on every HRQoL parameter compared to age-matched controls[97]. Resistance training is an essential part of the management of OA due to its effectiveness in reducing pain, improving function and overall quality of life.

The most significant clinical improvements seen from strength training in knee OA patients is reduced pain and increased function[6–10,98,99]. Resistance training programs ranged from home based training, isokinetic, dynamic and isometric techniques. Topp and colleagues[10] evaluated the effect of dynamic vs. isometric resistance training on four functional tasks and found that both methods reduced time to complete the tasks, including ascending and descending stairs, with no significant difference between treatment groups. They also found that knee pain while performing the functional tasks decreased by 28%-58%. Furthermore, a study done by Gur et al. compared concentric to combined concentric-eccentric isokinetic training and found both methods were effective in reducing pain scores and functional capacity[9]. Two studies evaluating the effectiveness of home based strength training on patients with knee OA reported decreased pain and increased function based on the Western Ontario and

McMaster Universities Osteoarthritis (WOMAC) index. Results exhibited a reduction of pain by 22.5%[98] and 36%[7] and increases in physical function by 17.4%[98] and 38%[7] in the exercise groups compared to the controls. Home based exercise makes treatment more accessible to OA patients which could therefore improve long term adherence.

Resistance training improves various measures of muscular function such as strength, range of motion, walking speed and endurance, as well as stair climb. These functional measures are important for improved quality of life as well as to decrease the risk for OA development and progression[100]. Multiple studies have demonstrated that older adults with knee OA are able to significantly increase their muscular strength in the muscles surrounding the knee joint following a resistance training program[8–10,86,98,99]. A review done by Bennell et al. found strength increases in knee OA by 5%-71% depending on various components such as intensity, compliance and specificity[101]. The most beneficial intensity of training is currently unclear. A meta-analysis examining high intensity strength training compared to other intensities on the lower limb in older adults found that high intensity training does have some advantages over lower intensities. The analysis however did find that when participants performed a similar volume of training, strength gains were the same regardless of intensity. They also found that shorter high intensity strength training programs of 12 weeks demonstrated significantly greater gains in outcomes such as strength and endurance compared to lower intensities of training[102]. This might be beneficial to those with time constraints. With an increase in muscle mass, knee

OA patients have improved metabolic profiles due to an increase in total energy expenditure (TEE) and decreased respiratory exchange ratio (RER) that increases lipid oxidation[103]. The increased TEE was due to an increase in physical activity (controlling for resistance training) and resting energy expenditure. This is consistent with the findings of Farr and colleagues who found that participants in a resistance training group increased and sustained their moderate to vigorous physical activity levels by 10% over 9 months compared to a self-management group who did not sustain increased PA levels[6].

The focus of strength training for knee OA patients should be to target the muscles of the lower extremity, especially those surrounding the knee joint. Patients with knee OA are 20-40% weaker in relative quadriceps strength compared to healthy controls[101]. The ROAD study[4] was a prospective study that found an increase in quadriceps muscle strength. Those with quadriceps muscle strength of < 10kgf had a 53.9% prevalence of knee pain while those with quadriceps muscle strength $\geq$ 40kgf had a 9.8% prevalence of knee pain. This demonstrates the importance of focusing resistance training on the quadriceps muscles, however it is important to balance the strength of the agonist and antagonist muscles, therefore strengthening of the hamstring muscles is also important. Hip abductor strength such as that of the gluteus medius is associated with functional performance measures such as stair climb in patients with knee OA[104]. Hinman and colleagues reported strength deficits in OA patients of 16% (hip extensors) to 27% (hip external rotators) compared to controls[105]. All of the above mentioned muscle groups are important during phases of gait, increasing

muscle strength in these muscle groups can provide an increased level of stabilization at the joint. Overall muscular strength surrounding the osteoarthritic joint is important for joint protection by providing added support.

Various factors contribute to an increase in muscle strength following a resistance training program. Factors include increased muscle cross sectional area (CSA), decrease in antagonist muscle co-activation, and improved neural drive. Ferreri and colleagues examined the effects of strength training in older adults and found that increases in torque and power were partly due to significant increases in CSA (.7%) of the knee extensors. They suggested an increase in neural drive also contributed to the increases in strength, however, did not see significant changes in EMG activity of the vastus lateralis[106]. Another study compared high and low intensity resistance training in older women and found that type I fiber CSA increased significantly in both groups. Type II fiber CSA increased but not significantly[107]. CSA and muscle activation increased in a study done on both elderly males and females during 12 weeks of high intensity resistance training[108]. Another study that examined high intensity strength training in frail elderly over 90 years old resulted in a 9% increase in midthigh muscle area[109]. Walker and Hakkinen conducted a 10 week strength training study on young and older adults and found that while both groups significantly increased strength the dominant mechanism to the strength gains were different between groups. Older adults significantly increased neural drive based on an increase in EMG amplitude while the young group did not. The young group increased strength through an increase in lean mass, however both young and

older adults significantly increased CSA of the vastus lateralis[110]. Co-contraction of the vastus lateralis and biceps femoris muscles decreases with the implementation of an exercise program in patients with knee OA. This decrease in co-contraction lead to decreased pain and increased strength[111].

Although the various studies ranged in resistance training techniques, it is clear that regardless of the method, resistance training is an effective treatment for OA patients. It is important to realize the various components contributing to increased strength to maximize training. With a range of techniques to choose from patients can choose the option that best fits their interests to make the treatment more individualized which is important for success.

Surface Electromyography

The electromyographic signal (EMG) is the electrical manifestation of the neuromuscular activation associated with a contracting muscle[112]. Surface electromyography (sEMG) uses electrodes placed on the skins surface to detect the average activity of superficial muscles. There are both square and circular electrodes that have no functional difference in recording characteristics, as long as the surface area is the same[113]. Electrodes are made of conductive materials that can range from plated metals to stainless steel. Electrodes convert the electric potential generated by the muscle into an electric signal that is conducted to an amplifier. The amplifier detects the small currents from the electrodes and increases their magnitude to a large enough size that can be recorded[114]. The muscle action potential (m.a.p) generates extracellular currents that extend from

the muscle membrane to the electrode at the skins surface. The electrode will record the algebraic sum of all of the m.a.p's being transmitted along the muscle fibers at that point in time. The EMG signal is measured in millivolts.

There are two types of electrodes. The first is a passive electrode where the metal recording surface is recessed within a plastic casing. Electrode gel is used to form a conduction path from the skin to the metal surface. The second type of electrode is an active electrode that incorporates a preamplifier within the small case that surrounds the metal recording surface. The metal makes direct contact with the skin and does not require electrolyte gel as long as the skin is thoroughly cleaned so the natural electrolytes present in the derma can conduct the signal[114]. The advantage to using active electrodes is that the signal strength is large in comparison to the surrounding noise[115]. In general, surface electrodes are advantageous compared to needle electrodes in that it is noninvasive and easy to apply.

Noise

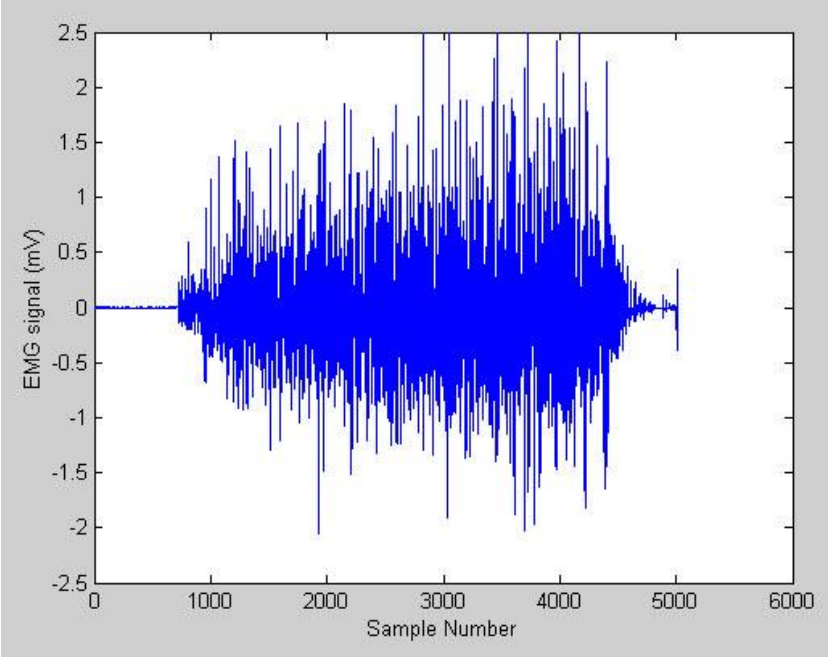
"Noise" is unwanted signal from the surrounding electrical fields and atmosphere that is acquired while traveling through tissue. There are a multitude of influences that can induce noise in an EMG signal. It can be categorized into the following types[116,117]: (1) inherent noise in electronics equipment which is described by Winters[118] as a hum which cannot be eliminated but can only be reduced; (2) ambient noise which refers to the electromagnetic radiation that the surface of our body is constantly inundated with and can't be avoided; (3) motion

artifact that can come from either the electrode interface or electrode cable; (4) cross talk that is an undesired signal from a neighboring muscle group. The best way to avoid this is with careful placement of the electrode on the belly of the muscle being analyzed and, (5) inherent instability of the signal that refers to the fact that the amplitude of the signal is random in nature due to the different firing rates of motor units. The frequency components between 0 and 20 Hz are considered unwanted noise. The amplitude and frequency of the signal will also vary depending on the electrode orientation with respect to the muscle fibers due to muscle tissue being anisotropic[119]. The goal during acquisition of the EMG signal is to reduce the amount of noise that may alter the signal. The two most common filters applied to EMG to filter noise are the Butterworth and Bessel filters[114].

EMG Signal Amplitude

The amplitude of the EMG signal is used as a measure of motor unit recruitment. The peak to peak amplitude of a waveform is proportional to the number of motor units recruited during electrical stimulation, however during voluntary contraction the relationship is influenced by wave cancellation. Wave cancellation can cause an underestimation of motor unit firing due to an overlapping of positive and negative phases of m.a.p's which cancel each other out[120]. Figure 1 shows a raw EMG signal with both positive and negative phases.

Figure 1: Raw EMG signal



Subcutaneous fat can also cause variations in the EMG signal amplitude. EMG amplitude decreases with increasing muscle-electrode distance during both maximal and submaximal contractions[121]. One study found that fat layers of 3, 9, and 18 mm decreased the amplitude of the EMG by 31.3%, 80.2% and 90.0% respectively. This same study also found that reducing the fat layer from 9mm to 3mm caused the EMG amplitude to increase by 241% and decrease cross-talk by 68%[122]. This is an important consideration when analyzing individuals with knee OA due to the high prevalence of overweight and obese individuals in this population.

Treatment of EMG Data

The raw EMG signal contains valuable information that can only be used if it can be quantified. Various processing techniques are applied to the raw EMG to reduce noise and to extract useful information from the signal. The EMG signal provides information on amplitude, frequency and timing. The most common amplitude demodulation technique is linear envelope detection. This process involves full wave rectification that takes the absolute value of the raw signal so the signal has a positive polarity. The high frequency of the signal is then removed by low-pass filtering. Cutoff frequencies can range from 3-60 Hz, however most EMG researchers have used cutoff frequencies below 20 Hz[114].

There are two EMG detectors that involve the band passed EMG signal (meaning the signal was high and low pass filtered) which requires little or no additional processing. The average rectified value (AVR) is a linear detector that calculates only the absolute value of each EMG over a specific interval. The root mean squares (RMS) technique is a nonlinear method that does not need rectification before calculation. The amplitude of the RMS signal is considered superior to the ARV method because it is not influenced by wave cancellation of the positive and negative phases of m.a.p's[114]. Farfán and colleagues[123] evaluated processing techniques for static and dynamic contractions. They used EMG during abduction/adduction movement of the arm in the scapular plain on the middle deltoid muscle. They found that the best processing technique for static and dynamic contractions was RMS. This was assessed based on the

amount of information (the relation degree between stimulus and response) each processing technique evaluated as well as its linear relationship to the abduction/adduction position of the arm. To determine the onset of muscle activation manual inspection of the EMG signal remains the gold standard[124].

EMG Reliability

EMG is a reliable measure of muscle activation during dynamic movements such as that done in resistance training as well as isometric contractions in both the healthy and OA populations[125–129]. Zech and colleagues[126] tested the reliability of EMG on highly skilled athletes, sports students and untrained subjects. A high reliability was shown with maximal voluntary muscle contractions (MVC) between session 1 vs. 2 and 2 vs. 3 (ICC= 0.92 and .97). In an OA population, Staehli et al. found an ICC of 0.94 for quadriceps muscle function using EMG on the Vastus Lateralis.[129] Test re-test reliability of EMG recordings during walking in individuals with knee OA separated by one month had ICC values greater than 0.81[128]. The various studies conducted have shown EMG to be a reliable measure of muscle activation for various populations including those with knee OA.

Muscle Activation

Skeletal muscle is composed of fine muscle fibers that upon stimulation will shorten, thereby causing a muscular contraction. Each muscle fiber is innervated by nerve fibers or axons which originate from the anterior horn of the

spinal grey matter. The nerve cell body, long axon of the motor nerve, and all of the muscle fibers supplied by the nerve are as one known as a motor unit [112]. The activation of individual muscle fibers in a motor unit produces a summed muscle action potential (m.a.p)[118,114]. For a given muscle there is a variable number of motor units, each controlled by a motor neuron through synaptic junctions called motor end plates. An action potential transmitted down the motor neuron will arrive at the motor end plate and consequently trigger a sequence of electrochemical events leading to a muscle contraction[118]. The depolarization of the transverse tubular system and the sarcoplasmic reticulum results in a depolarization "wave" along the direction of the muscle fibers. It is the depolarization wavefront and the subsequent repolarization wave that are sensed by the recording electrodes of an EMG system[118].

Motor units are recruited during a contraction in sequence of increasing size. The smallest motor units are recruited first with the larger motor units recruited as the force demand increases. This is referred to as the Henneman size principle[15,114]. The number of motor units recruited has a large impact on the EMG, with an increase in motor unit recruitment there is an increase in the EMG amplitude. Amplitude of the EMG is also directly affected by the firing rate of the motor neurons.

Age Differences

Declines in muscle size and strength are often reported as a consequence of aging, however this may not be true in relation to age and muscle activation.

Several studies have demonstrated that an older healthy population is able to produce the same muscle activation compared to a young healthy population[130–132]. However one study found that although the best effort of maximal voluntary muscle activation was not different between young and old men, the old men needed more attempts to reach maximal activation[131]. There is evidence to suggest that body fat percentage (BF %) can affect muscle activation differently based on age. Tomlinson[133] found that the young group with ≥ 40% BF had significantly lower activation compared to young with <40% BF. Interestingly however, the old group did not exhibit this BF effect.

Muscle Activation and Knee OA

The gradual decline in quadriceps strength associated with knee OA is attributed, in part, to impairment in the central nervous system's ability to fully activate the muscle. This is termed central activation failure or arthrogenous muscle inhibition (AMI)[11–13]. It is theorized that AMI can result from progressive joint degeneration like that seen in OA patients that results in abnormal articular afferent information being sent to the motor neurons which consequently reduces activation[14]. Because of this decrease in activation there is some question as to whether or not physical rehabilitation can be effective in restoring function and reversing muscle weakness. Hurley and colleagues analyzed OA participants who at baseline could not fully activate their quadriceps femoris. These participants were able to increase strength and activation after an 8 week

rehabilitation program[12]. This demonstrates the plausibility of a strength training regime for OA patients.

Effects of Strength Training on Muscle Activation

Increases in strength during the early stages of a resistance training program are mainly due to neural adaptations which elevate motor unit recruitment. Neural adaptations play a particularly important role in the strength improvements of the elderly with resistance training[134]. Cannon et al. conducted a study in which both young (20-30 years) and older (64-78 years) women completed a 10 week resistance training program at 75% 1RM. Muscle activation was collected using surface EMG of the vastus lateralis and vastus medialis muscles. EMG amplitude was then averaged between the vastii muscles to provide an overall indication of total motor unit activity of the knee extensors. They found that both young and old groups had similar increases in muscle activation (19% and 21% respectively)[130]. In contrast, a study analyzing the effects of single vs. multiple-set short-term strength training in elderly women did not find a significant increase in muscle activation of the knee extensors[135]. It is possible that the dose was not great enough to elicit a significant increase in activation since they only trained twice a week for six weeks. There is also evidence to suggest that there may be more variability in how older adults respond to training. After a four week resistance training program for the elbow flexors, Barry et al. reported that both young (21-35 years) and old (60-79 years) participants increased muscle activation for the brachialis and brachioradialis

muscles, however the old group did not have significant increases in muscle activation for the biceps brachii or the pronator teres muscle while the young did[136]. Walker and Hakkinen found that after 10 weeks of resistance training older adults significantly increased muscle activation while the young group did not[110]. Other modes of resistance training have been effective; isometric resistance training increased muscle activation not only at the angle at which the subjects trained, but also at angles through a full range of motion[137].

Since substantial force production is required when lifting heavy loads such as 75% of 1 repetition max (RM), to meet the force demands both small and larger motor units are recruited. This is in contrast to lifting lighter loads such as 30% of 1-RM which has lower force demands therefore requires less motor unit recruitment and consequently less muscle activation[16]. Three studies have been conducted that collected data on muscle activation during low load vs. high load resistance training[16,138,139]. Each study found that high loads (70%[138,139] or 75%[16] 1RM) resulted in higher EMG muscle activation during resistance exercise to volitional fatigue compared to low loads (20%[139], 30%[16], and 50%[138] 1RM).

Pietrosimone and Saliba[17] investigated whether changes in quadriceps activation would predict changes in quadriceps strength in patients with knee OA. They found that changes in activation predicted 47% of the variance in the change in quadriceps strength following a 4 week exercise program. These data emphasize the importance of focusing rehabilitation efforts on muscle activation to improve strength in this population.

Conclusion

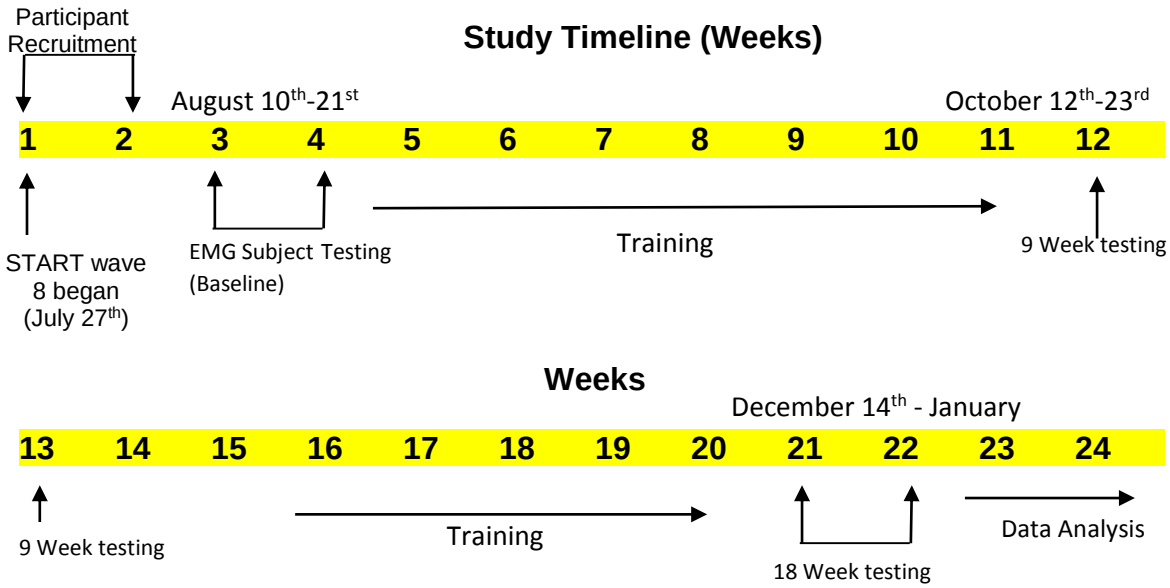
Older adults with knee OA have significant lower extremity muscle weakness as well as lower muscle activation compared to the healthy population. To maximize strength gains in this population it is important to increase lean muscle mass as well as enhance muscle activation. Since muscle activation may be a predictor of strength gains in the OA population, determining the resistance training intensity that elicits a greater activation response may shed light on the optimal resistance training load prescription for treatment of knee OA. To initiate an adaptive response to strength training, a muscle fiber must be recruited, which makes high load training logical to maximize muscle adaptation. No study has analyzed the effect of exercise intensities on muscle activation in the knee OA population. Therefore, the purpose of this study was to compare muscle activation of the knee OA population during a low intensity versus high intensity strength training program over an 18 week period.

Materials and Methods

Subjects

Participants consisted of 25 older adults (6 Male, 19 Female) recruited from the current Strength Training for Arthritis Trial (START). The participants were either in a low intensity (LI) strength training group (N=11, 30% 1-RM) or a high intensity (HI) strength training group (N=14, 75% 1-RM). All participants had clinical and radiographic mild to moderate medial tibiofemoral osteoarthritis and were ≥ 50 years old. All participants provided written informed consent prior to participation. Participants were in the study for 18 weeks with 3 testing periods (Baseline, 9 week follow up, and 18 week follow up). A study timeline is represented in figure 2. The research protocol was approved by the institutional review board of Wake Forest Health Sciences.

Figure 2: Study timeline



Procedures

Each START participant randomized to an exercise group went through 9 week training cycles. The high intensity group trained at 75% 1-RM for weeks 1-2, 80% 1-RM for weeks 3-4, 85% 1-RM for weeks 5-6 and 90% 1-RM for weeks 7-8. The low intensity group trained at 30% 1-RM for weeks 1-2, 35% 1-RM for weeks 3-4, 40% 1-RM for weeks 5-6 and 35% 1-RM for weeks 7-8. During the 9th week participants performed one repetition max (1-RM) testing. The new 1-RM on each machine was then used to adjust resistance to continue strength progression. EMG acquisition occurred at baseline before subjects began training, at the completion of 9 weeks of training, and after 18 weeks of training. Details about the START protocol are reported elsewhere[140].

Participants were scheduled on one of their regular days of training (M/W/F or T/TH/S). The participants were prepped for EMG electrodes by shaving the desired area of skin, followed by scrubbing the area with a textured alcohol pad, and lastly swiped with a smooth alcohol pad. This procedure enhanced electrode to skin contact and reduced signal noise. After preparation, wireless Trigno surface electrodes with a contact surface of 5 x 1 mm were attached to the skin parallel to the fiber direction of the muscle belly of the vastus lateralis, biceps femoris and gluteus medius Electrode placement was done on the most affected leg defined as the leg with the most knee pain.

The methods for surface electrode placement were based on the recommendations of surface EMG for non-invasive assessment of muscles (SENIAM)[141] . In conjunction with these recommendations, the muscle was

palpated to ensure accurate placement on the muscle belly. Subsequently, baseline measures were taken. Each participant lay supine on a patient table and was asked to completely relax for three minutes. Using the Delsys EMG Acquisition system, baseline measures were checked to ensure minimal noise. Measures under 20 μV were accepted. If measures were above 20 μV the participant was re-prepped until optimal baseline measures were attained. A validation study was previously conducted to ensure the reliability and validity of the investigators technique of surface EMG placement. Test re-test data resulted in an ICC of 0.83.

Instrumentation

Raw EMG signals were collected at 2,000 Hz with a Delsys Trigno Wireless EMG system, and filtered by a fourth order Butterworth band-pass filter with cut off frequencies of 20- 400 Hz. Data were sent in real time to a computer and analyzed in Delsys EMGworks Analysis software. Visual inspection of the EMG signal was used to determine onset of muscle activation. Signals were rectified and smoothed using a root mean squares (RMS) technique. Figures 3 and 4 demonstrate the transformation of raw EMG to rectified EMG with RMS.

Figure 3: Raw EMG MVC of the vastus lateralis

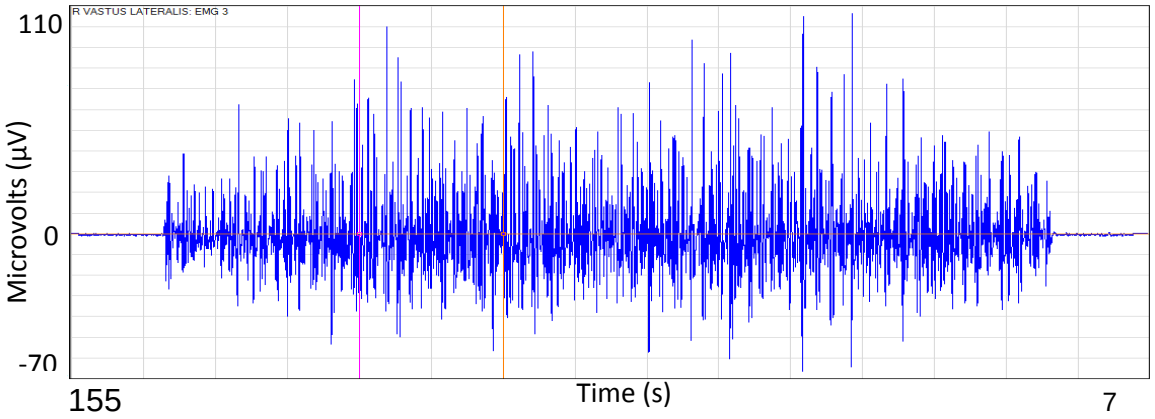
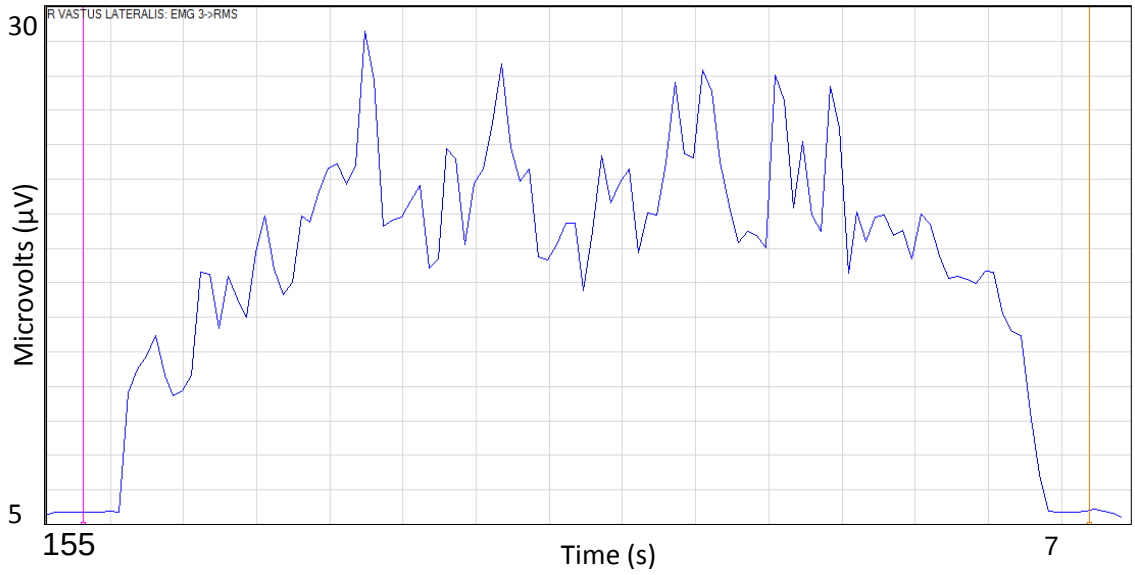


Figure 4: EMG signal of the vastus lateralis after RMS

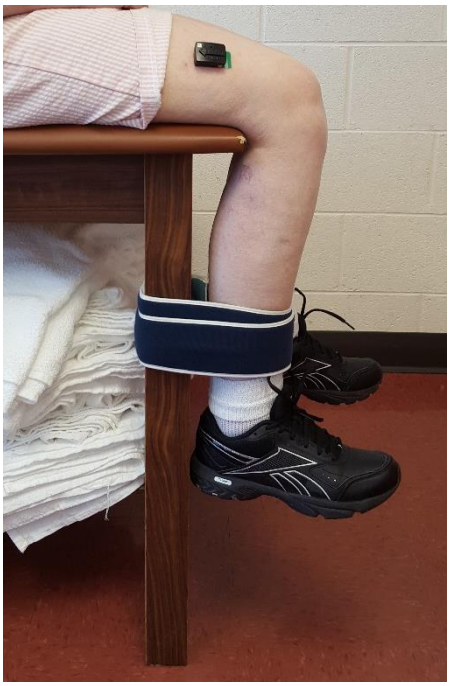


Maximal Voluntary Isometric Contractions

Maximal voluntary isometric contractions (MVC) were used for EMG signal normalization. MVCs were collected for each of the three muscles being analyzed starting with the vastus lateralis. Participants were seated on the edge

of a patient table with knees flexed at 90 degrees[11,142,16]. The leg being analyzed was strapped to the leg of the patient table just above the ankle to provide resistance[11,16]. The participant was instructed to hold the edges of the table for stabilization and to breathe out while pushing against the strap. One practice round at about 50% of maximum strength was performed to accommodate to the testing environment. The participant was subsequently instructed to extend the leg straight out as hard as they could as if they were trying to break through the strap. Figure 5 illustrates the leg position and strap placement for the vastus lateralis MVC.

Figure 5: Vastus lateralis MVC



MVC of the biceps femoris was done by instructing participants to lie prone on the patient table. Five warm ups were conducted that consisted of the participant flexing their knee and pulling against a manual resistance at about

50% of maximal effort. Subsequently, the participants leg was positioned to 55 degrees of knee flexion[143] using a goniometer as illustrated in figure 6. Manual resistance was applied to the posterior leg just above the ankle[16]. Participants were instructed to hold the table for stabilization and to breathe while flexing the knee. Participants were instructed to pull their leg as hard as they could as if they were trying to kick their backside.

Figure 6: Biceps femoris MVC

MVC for the gluteus medius was conducted while the participant was lying on their side with the leg under analysis facing up to perform a side lying hip abduction isometric contraction[144,145]. Participants were told to hold the edge of the table for stabilization and breathe while lifting their leg. A warm up was first conducted during which participants lifted their leg straight up and down five times. After the warm up, manual resistance[146,145] was applied to the lateral side of the leg with one hand placed below the knee and the other above the knee.

The position of the participant is illustrated in figure 7. Participants were instructed to lift their leg as hard as they could.

Figure 7: Gluteus medius MVC

Three trials of each MVC were performed against manual resistance, each held for five seconds. Manual resistance was chosen based on previous research[11,16,145,146] as well as its practical value. Raw EMG peak to peak measures were reviewed between each MVC to make sure the difference between trials was not too large. If the difference was greater than 100 μV another trial was performed to ensure an accurate average of three trials. Every participant was given verbal encouragement during each trial and at least one minute rest between trials. The order of MVCs was consistent for every participant and each trial (baseline, 9 week and 18 week).

Submaximal Machine Lifts

Participants performed submaximal lifts on Nautilus machines (shown in figure 9) after all of their MVCs were completed with the EMG sensors still attached. Each participant was randomly assigned either sequence A (Leg Extension, Hip Abduction, Leg Curl) or sequence B (Leg Curl, Leg Extension, Hip Abduction) to determine the order of exercises. The EMG sensor on the vastus lateralis needed to be removed before participants performed the hip abduction exercise. If the sensor was left on, due to the position of the sensor it would press into the pad of the resistance machine and cause discomfort to the participant and risk the sensor falling off due to friction. For this reason, the leg extension exercise had to precede the hip abduction exercise which is why there were two sequences assigned. Each participant retained the same sequence for each testing period. Participants in the LI group performed 15 repetitions at 30% of their 1-RM while the HI group performed 8 repetitions at 75% of their 1-RM. The participant first performed one set on each leg at their own pace. The second and third set were paced using a metronome fixed at 60 bpm so that both concentric and eccentric actions were performed at a count of 1 s each. In time with the metronome the participants were given verbal cues to follow the correct cadence, for example on the leg extension machine the investigator would say “up, down, pause”. EMG signals were recorded during the third set.

Figure 8: Images of the Nautilus resistance machines used for submaximal machine lifts

Hip Abduction Leg Extension Leg Curl

Treatment of the Data

Mean MVCs were determined by taking the RMS of each participants MVC and averaging the output data points of the activation. Then each participants mean MVCs were averaged together within their respective intensity group. Peak MVC data were determined by taking the highest activation data point of the RMS output for each participant. For each intensity group, the participants peak MVCs were averaged to get mean peak MVC for each group.

Submaximal activation was recorded using the final 3 repetitions of the last set performed while on the resistance machines. Each repetition forms a burst of activation seen in the EMG signal. Each burst was analyzed separately and then averaged together to determine a mean and peak submaximal activation for each participant. Each participant's activation was normalized to their MVC to determine percent activation. This was done by dividing the

participant's submaximal activation by their MVC and then multiplying by 100 to get a percent. This is demonstrated by the following equation:

$$\left(\frac{Submaximal\ activation\ (\mu V)}{MVC\ (\mu V)}\right) \times 100 = \%\ Activation$$

Mean and peak percent activation for each group was calculated by averaging each participant's percent activation. Mean percent activation was calculated using mean submaximal activation and mean MVC. Peak percent activation was calculated using peak submaximal activation and peak MVC.

Statistical Analysis

Baseline characteristics were summarized using descriptive means and standard deviations for continuous variables and counts and percentages for discrete variables. Treatment effects for each of the outcomes were estimated at 9 and 18 weeks using a repeated measures analysis of co-variance using treatment (low intensity vs high intensity), follow-up visit (9 vs. 18 weeks), the treatment by visit interaction, gender, and baseline values of the outcome. Comparisons of treatments at follow-up visits and estimates of mean differences were generated using contrasts from each respective outcome model, and significance was set at the 0.05 level. Because this is a pilot study, we did not plan to adjust for multiple comparisons. All analyses were conducted using SAS v9.4 (SAS Institute, Cary, NC)

Results

Baseline Characteristics

Baseline subject demographics are found in Table I. Of the 29 participants initially enrolled in the study, 25 completed 18 weeks of testing (N=14 HI, N=11 LI). The 4 participants who did not complete the study did not continue coming to the START exercise intervention hence no follow up data were obtained. The average age of the participants was 63 years and did not differ between groups. Average BMI was 32.6 and was even across groups. The average weight was 92.2 ± 21.6 kg. 76% of the participants were white and 76% were female.

Table I: Subject demographics

Variable	All, Mean ± SD	High Intensity, Mean ± SD	Low Intensity, Mean ± SD
Age (years)	63.3 ± 9.6	64.3 ± 10.8	61.9 ± 7.8
BMI (kg/m^2)	32.6 ± 6.3	32.4 ± 6.5	33.0 ± 6.4
Female	19 (76.0%)	11 (78.6%)	8 (72.7%)
Weight (kg)	92.2 ± 21.6	89.2 ± 18.5	96.1 ± 25.5
White (%)	75.8%	76.9%	83.3%

MVCs

The MVCs tended to be steady across time and were similar between groups. MVC data are shown in table II.

Table II: Mean and peak MVCs at baseline, 9 and 18 weeks (95% CI) for the high and low intensity groups

	Baseline			Week 9			Week 18		
Variable	**All, Mean (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**	**Difference (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**	**Difference (95% CI)**
Vastus lateralis mean MVC (µV)	40.8 (32.4, 49.2)	40.8 (27.3, 54.3)	40.8 (29.6, 52.1)	49.7 (42.5, 56.9)	37.9 (29.8, 45.9)	11.8 (1.0, 22.7)	51.9 (45.6, 58.3)	45.8 (38.4, 53.2)	6.1 (-3.7, 15.9)
Vastus lateralis peak MVC (µV)	62.4 (50.0, 74.7)	63.0 (43.6, 82.5)	61.5 (43.9, 79.1)	71.0 (60.1, 81.8)	58.0 (46.3, 69.8)	12.9 (-3.1, 28.9)	73.8 (66.0, 81.5)	68.8 (59.7, 77.8)	5.0 (-7.0, 17.0)
Biceps femoris mean MVC (µV)	52.2 (43.2, 61.3)	52.9 (41.3, 64.5)	51.4 (34.6, 68.2)	53.2 (47.5, 58.9)	57.3 (50.7, 63.9)	-4.1 (-12.9, 4.7)	53.5 (45.1, 61.9)	53.7 (43.8, 63.7)	-0.2 (-13.3, 12.8)
Biceps femoris peak MVC (µV)	70.4 (56.4, 84.5)	72.8 (53.9, 91.8)	67.3 (42.6, 92.1)	70.6 (63.7, 77.6)	80.2 (72.0, 88.3)	-9.5 (-20.3, 1.3)	73.2 (62.0, 84.5)	73.6 (60.2, 87.0)	-0.4 (-18.0, 17.2)
Gluteus medius mean MVC (µV)	24.8 (21.1, 28.4)	24.9 (19.3, 30.6)	24.6 (19.3, 29.9)	25.7 (21.5, 29.9)	24.8 (20.2, 29.4)	0.9 (-5.3, 7.1)	23.9 (20.7, 27.1)	22.6 (18.9, 26.3)	1.3 (-3.6, 6.2)
Gluteus medius peak MVC (µV)	35.5 (29.8, 41.2)	35.9 (27.1, 44.8)	34.9 (26.7, 43.2)	36.1 (30.1, 42.2)	33.5 (26.9, 40.0)	2.7 (-6.2, 11.6)	33.7 (29.2, 38.1)	30.7 (25.5, 35.9)	3.0 (-3.9, 9.8)

Means estimated from a mixed linear model tested at 9 and 18 weeks adjusting for gender and baseline values of the outcome.

Submaximal Activation

Submaximal activation was based on the activation of the final 3 repetitions on each machine and can be found in table III. The mean submaximal activation on the leg extension machine for the vastus lateralis was 49.1 µV and the mean peak activation was 65.5 µV. The mean submaximal activation on the leg curl machine for the biceps femoris was 41.1 µV and the mean peak activation was 49.2 µV. The mean submaximal activation on the hip abduction machine for the gluteus medius was 15.9 µV and the mean peak activation was 18.9 µV.

Percent Activation

Table IV shows mean activation of the vastus lateralis as a percent of MVC at baseline, 9 and 18 week follow-up. At baseline mean percent activation was 117%. Week 9 activation for the HI group was 129% and 114% for the LI group. Week 18 resulted in activation levels of 127% and 114% for the HI and LI respectively. There were no statistical differences for the vastus lateralis, however there was a 15% and 13% difference at 9 and 18 weeks respectively with the trend being in favor of the HI group. Graphical representation of the vastus lateralis mean percent activation can be found in Figure 9.

Table III: Non- normalized mean and peak activation of the submaximal machine lifts at baseline, 9 and 18 weeks. These values were subsequently normalized to MVC and used in the analyses

	Baseline			Week 9			Week 18		
Variable	**All, Mean (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**	**All, Mean (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**	**All, Mean (95% CI)**	**High Intensity, Mean (95% CI)**	**Low Intensity, Mean (95% CI)**
Vastus lateralis mean activation (μV)	49.1 (37.3, 60.9)	54.9 (34.8, 74.9)	41.8 (30.5, 53.1)	61.5 (53.4, 69.6)	44.4 (35.0, 53.8)	17.1 (4.4, 29.8)	62.5 (52.3, 72.6)	55.3 (43.3, 67.3)	7.2 (-8.8, 23.2)
Vastus lateralis peak activation (μV)	65.5 (49.7, 81.3)	71.9 (45.0, 98.8)	57.3 (41.3, 73.3)	79.4 (69.2, 89.7)	56.2 (44.3, 68.1)	23.2 (7.2, 39.2)	78.6 (65.2, 92.1)	69.8 (53.9, 85.7)	8.9 (-12.2, 29.9)
Biceps femoris mean activation (μV)	41.1 (30.9, 51.3)	52.0 (35.7, 68.4)	27.2 (22.4, 31.9)	52.6 (42.6, 62.6)	43.6 (32.3, 54.8)	9.0 (-7.1, 25.2)	46.9 (37.9, 56.0)	44.5 (33.7, 55.2)	2.5 (-12.9, 17.8)
Biceps femoris peak activation (μV)	49.2 (34.5, 64.0)	64.4 (40.6, 88.2)	30.0 (22.9, 37.1)	64.4 (51.6, 77.2)	51.6 (37.1, 66.1)	12.8 (-8.0, 33.6)	57.9 (46.0, 69.7)	54.7 (40.6, 68.7)	3.2 (-16.8, 23.3)
Gluteus medius mean activation (μV)	15.9 (13.1, 18.7)	16.6 (12.6, 20.5)	15.0 (10.5, 19.6)	19.6 (16.5, 22.7)	16.5 (13.1, 19.9)	3.1 (-1.5, 7.7)	19.0 (16.3, 21.6)	14.9 (11.8, 18.0)	4.1 (-0.1, 8.2)
Gluteus medius peak activation (μV)	18.9 (15.3, 22.4)	19.4 (14.9, 23.9)	18.2 (11.5, 24.8)	23.7 (20.2, 27.2)	18.4 (14.5, 22.3)	5.3 (0.1, 10.6)	23.1 (19.9, 26.2)	16.6 (12.9, 20.3)	6.4 (1.5, 11.3)

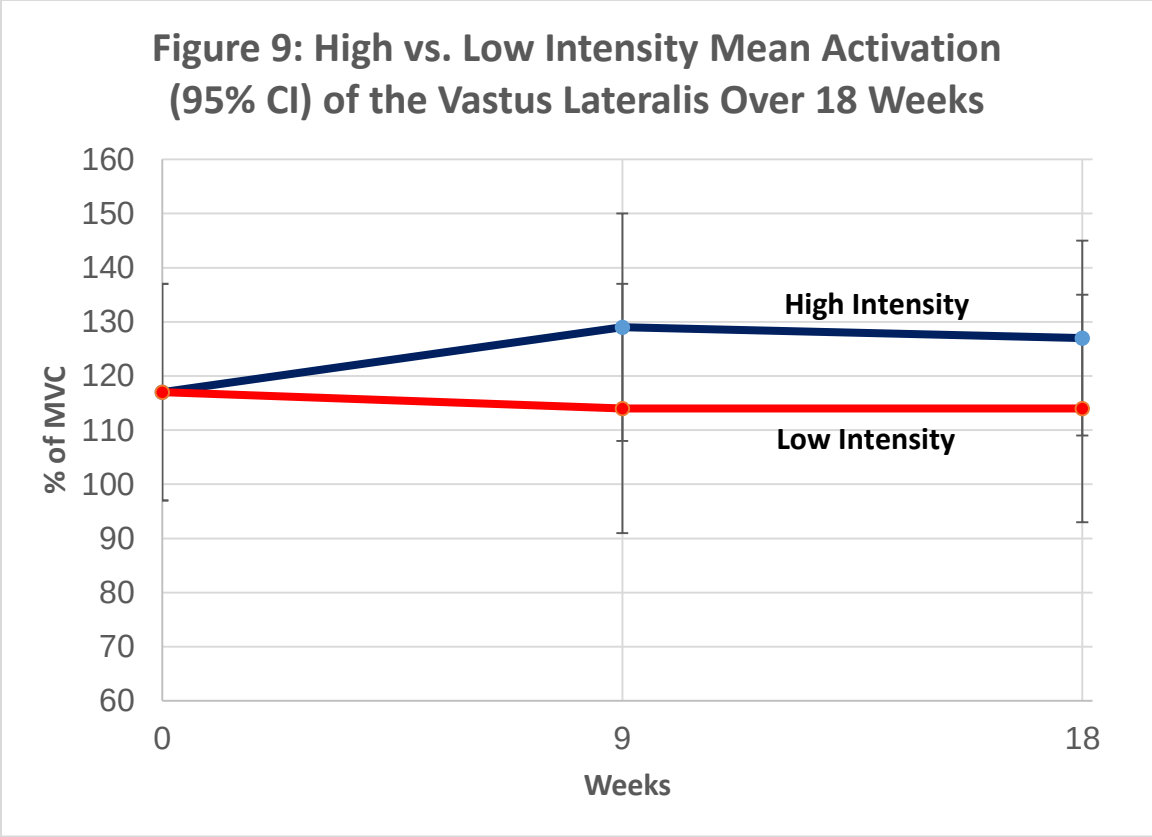
Mean and peak activation are based on the final 3 repetitions of participant's submaximal machine lifts

Table IV: Mean percent activation of the vastus lateralis at baseline, 9 and 18 weeks

Leg Extension Exercise											
	Baseline			Week 9				Week 18			
Variable	All, Mean (95% CI)	High Mean (95% CI)	Low Mean (95% CI)	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value
Vastus lateralis mean activation (% MVC)**	117 (0.97, 1.36)	132 (1.03, 1.61)	97 (0.74, 1.21)	129 (1.08, 1.50)	114 (0.91, 1.37)	15 (-0.17, 0.47)	0.351	127 (1.09, 1.45)	114 (0.93, 1.35)	13 (-0.15, 0.42)	0.339

P-value is between groups at follow-up with means estimated from a mixed linear model tested at 9 and 18 weeks adjusting for gender and baseline values of the outcome.

**(μV (submaximal leg extension represented as the mean of the final 3 repetitions) / μV (MVC)) x 100 = %MVC



% activation was normalized to MVCs

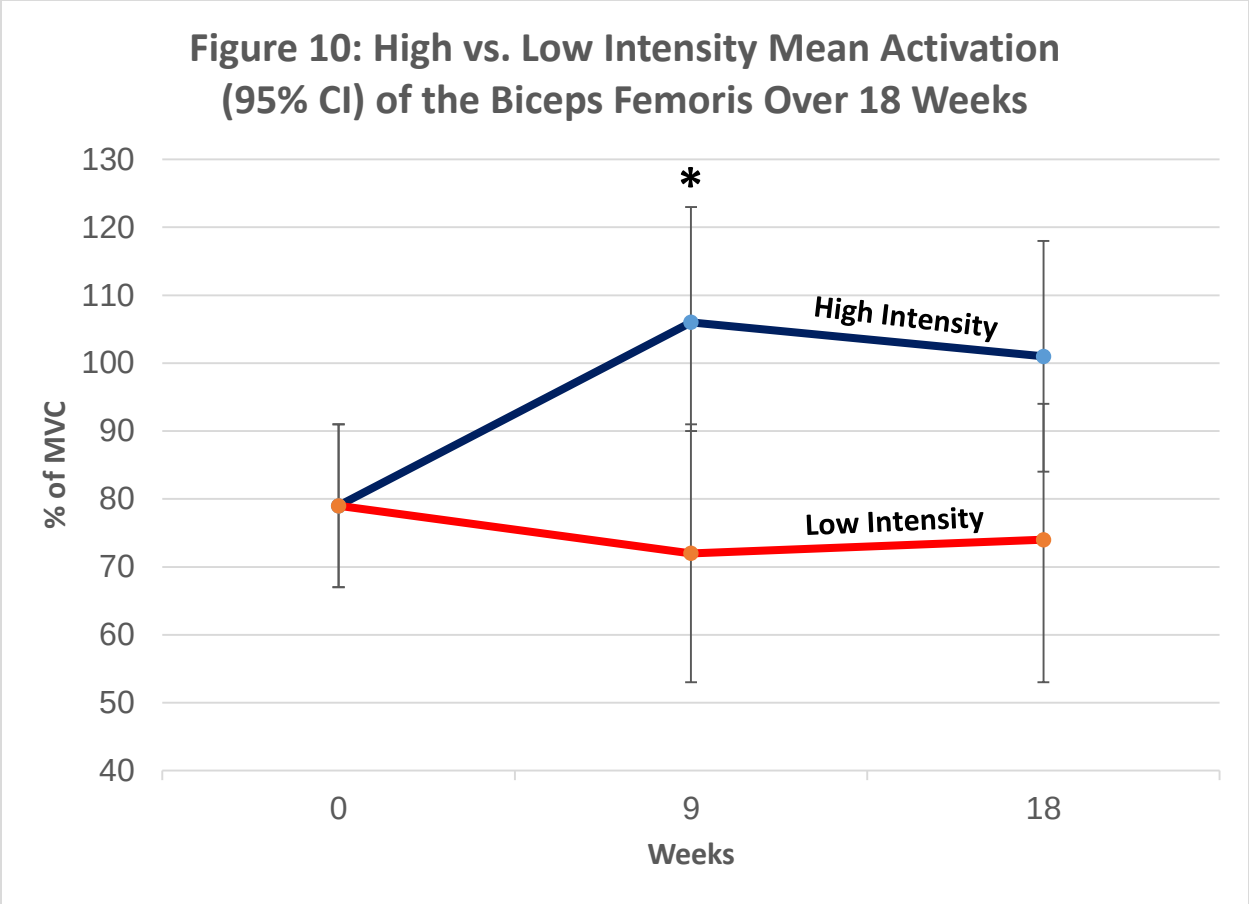
Table V represents mean activation of the biceps femoris as a percent of MVC at baseline, 9 and 18 weeks. There was a significant difference between the HI and LI training groups. Mean activation was significantly (P= 0.024) greater in the HI group at 9 weeks with 106% activation (95% CI= 0.89, 1.22) compared to the LI with 72% (95% CI= 0.53, 0.91). There were no significant between group differences at 18 week follow-up. Graphical representation of the biceps femoris mean percent activation can be found in Figure 10.

Table V: Mean percent activation of the biceps femoris at baseline, 9 and 18 weeks

Leg Curl Exercise											
	Baseline			Week 9				Week 18			
Variable	All, Mean (95% CI)	High Mean (95% CI	Low Mean (95% CI)	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value
Biceps femoris mean activation (% MVC)**	79 (0.67, 0.92)	98 (0.83, 1.13)	56 (0.46, 0.66)	106 (0.89, 1.22)	72 (0.53, 0.91)	34 (0.05, 0.62)	0.024*	101 (0.84, 1.18)	74 (0.54, 0.95)	27 (-0.04, 0.57)	0.084

P-value is between groups at follow-up with means estimated from a mixed linear model tested at 9 and 18 weeks adjusting for gender and baseline values of the outcome. * represents significance ($p \leq 0.05$)

**(μV (submaximal leg extension) / μV (MVC)) x 100 = %MVC



*represents significance (P≤ 0.05). % activation was normalized to MVCs

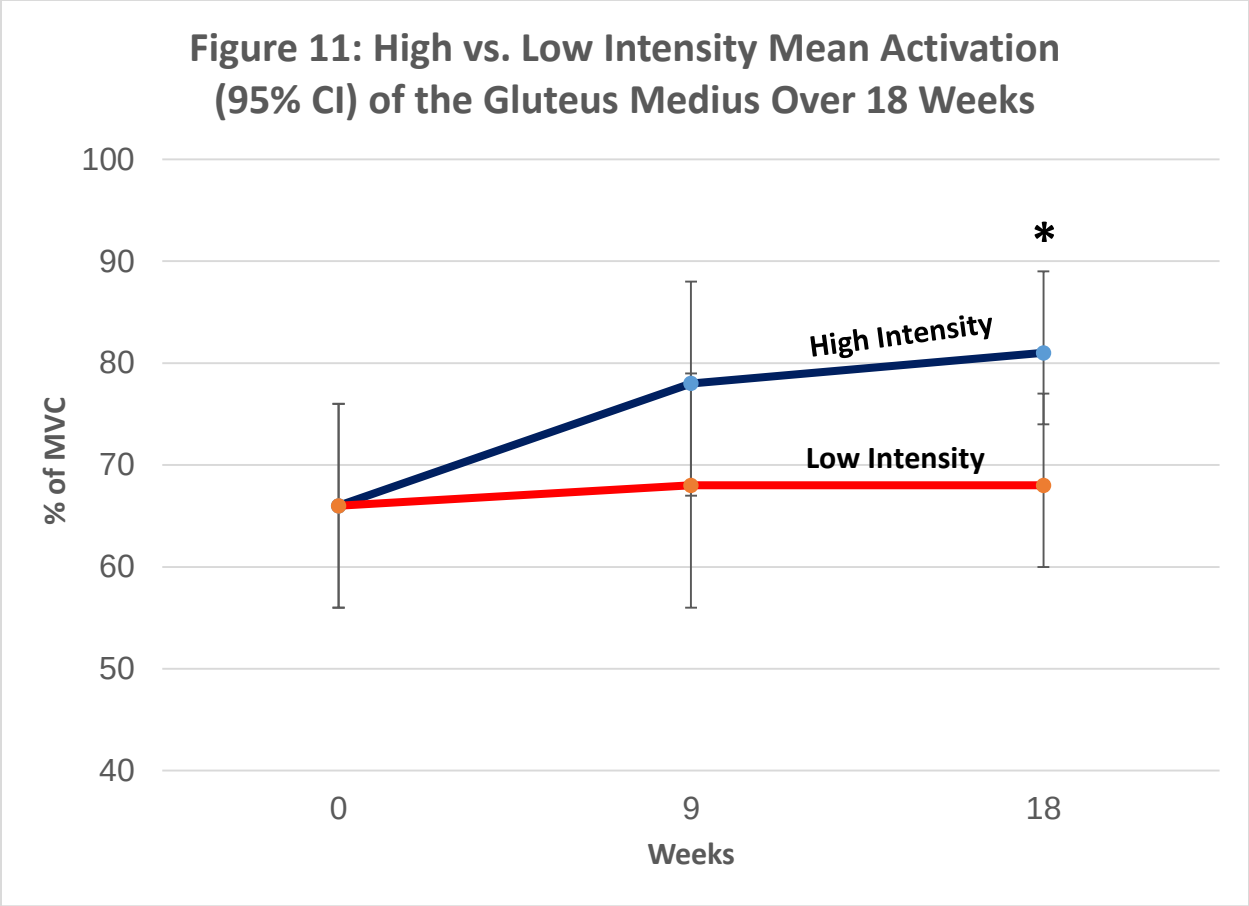
Table VI represents mean activation of the gluteus medius as a percent of MVC at baseline, 9 and 18 weeks. The mean activation in the HI group was significantly (P = 0.028) greater than in the LI group at 18 weeks with 81% activation (95% CI=0.73, 0.88) compared to 68%, (95% CI= 0.59, 0.76). Graphical representation of the Gluteus medius mean percent activation can be found in Figure 11.

Table VI: Mean percent activation of the gluteus medius at baseline, 9 and 18 week follow-up

Hip Abduction Exercise											
	Baseline			9 Week – Training Intensity				18 Week – Training Intensity			
Variable	All, Mean (95% CI)	High Mean (95% CI)	Low Mean (95% CI)	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value	High Mean (95% CI)	Low Mean (95% CI)	Difference (95% CI)	P-value
Gluteus medius mean % activation	66 (0.56, 0.76)	69 (0.54, 0.84)	62 (0.48, 0.76)	78 (0.68, 0.89)	68 (0.57, 0.80)	10 (-0.06, 0.26)	0.195	81 (0.73, 0.88)	68 (0.59, 0.76)	13 (0.02, 0.24)	0.028*

P-value is between groups at follow-up with means estimated from a mixed linear model tested at 9 and 18 weeks adjusting for gender and baseline values of the outcome. * represents significance ($p \leq 0.05$)

**(µV (submaximal leg extension) / µV (MVC)) x 100 = %MVC



*represents significance (P≤ 0.05). % activation was normalized to MVCs

Discussion

The purpose of this study was to determine whether differences in muscle activation existed during 18 weeks of either low intensity or high intensity strength training in older adults with knee OA. This study produced several important findings. First, both mean and peak activation were significantly greater in the high intensity training group compared to the low intensity group for the biceps femoris and gluteus medius muscles. These findings suggest that training at 30% of 1-RM does not maximize activation of the full motor unit pool as compared to training at 75% of 1-RM. This is in agreement with previous studies that have found significantly greater activation when using high intensity loads equating to 70 to 75% of 1-RM versus lower intensity loads ranging from 20 to 50% of 1-RM[138,139,147] The present findings have several important implications for the resistance training load prescriptions with respect to muscular adaptations for the knee OA population.

There was a significant difference in muscle activation of the gluteus medius between groups. Gluteus medius weakness has been implicated in the progression of knee OA. More specifically, Hinman and colleagues found that participants with knee OA had 24% lower hip abduction strength compared to healthy controls with a correlation r= -0.29, P = 0.007 between hip abduction muscle strength and radiographic disease. Weaker hip abduction was associated with more severe disease [105]. Hip abduction muscle weakness during gait causes excessive pelvic drop in the contralateral swing limb. The pelvic drop shifts the body's center of mass toward the swing limb, thus increasing forces across the

medial tibiofemoral cartilage of the stance limb[148]. Chang et al.[149] tested whether greater internal hip abduction moments during gait could prevent excessive medial compartment loading in participants with knee OA to potentially protect against OA progression. They found that the odds of medial tibiofemoral OA progression were reduced by 43% with an additional 1 unit of hip abduction moment during the 18 months following baseline. Increasing the activation of the gluteus medius could help to increase hip abduction moments and therefore help protect against further OA progression. Although this ancillary study did not examine gait, those data are being collected as part of the START parent trial. We speculate that increasing activation during training will result in strength gains that will facilitate positive changes at the hip and knee during gait. These data will not be available until the START trial is completed.

The gradual decline in muscle strength associated with knee OA is attributed, in part, to impairment in the central nervous system's ability to activate the muscle fully. This is termed arthrogenous muscle inhibition (AMI)[12,14,13]. Due to AMI, efforts to rehabilitate knees affected by OA may not be effective in restoring function and reversing muscle weakness[12]. Hurley and colleagues, however, demonstrated that knee OA participants who could not fully activate their quadriceps muscle at baseline were able to increase activation after 8 weeks of rehabilitation. The deficits between the affected and unaffected leg however remained[150]. The present study builds on this previous work showing that not only can knee OA participants increase activation, but training at 75% of 1-RM elicits higher activation compared to 30% of 1-RM. Perhaps high intensity

training could bridge the gap between strength and activation deficits seen between affected and unaffected legs and combat the effects of AMI.

Biceps femoris activation was significant between groups at week 9 follow up but not at 18 week follow-up. This finding is comparable to Ferri et al[106] who showed older adults had the largest improvement in muscle activation after 4 weeks and was reduced after 16 weeks.

There were no significant differences in activation between the LI and HI training groups for the vastus lateralis at 9 and 18 weeks. This lack of significance is disappointing, since quadriceps muscle weakness is strongly implicated in knee OA pathogenesis[74,76,151,152]. It is possible, however, that the quadriceps muscle weakness associated with knee OA is so pronounced that it requires a longer training period before differences in neural adaptations are present. Alternatively, a larger sample size would have increased our power to potentially detect a difference.

It was interesting that submaximal activation for the vastus lateralis and biceps femoris resulted in activation greater than the MVCs. Possible explanations for this include using the technique of manual resistance for collection of MVCs. This could have induced greater variability compared to using an isokinetic dynamometer, although this technique has been used successfully in previous studies[11,16,145,146]. Ferri et al.[106] found that 1-RM strength increases were significantly greater than isometric strength after training. Furthermore, Frontera et al.[153] found that despite increases in muscle cross sectional area after training the knee extensors failed to increase isometric

strength. This was also reported by Brown et al.[154] who attributed the difference to neural adaptations specific to training. This could partly explain why activation on the resistance machines in some cases was greater than the MVC activation, since training was performed concentrically and eccentrically and MVCs were done isometrically. It is also possible that participants weren't able to achieve a truly maximal activation. Jakobi and Rice[131] found that older adults were able to activate their muscle fully but needed more trials to achieve maximum activation compared to young healthy controls. Perhaps 3-5 trials was not sufficient for them to achieve maximum activation. Another mechanism could be co-contraction of the antagonist muscle. Contraction of the antagonist can impair by reciprocal inhibition, reducing the ability to fully activate the agonist muscle as indicated by reduced EMG activity. This is a protective mechanism in activities such as weight lifting, hence when participants are introduced to a new strength task such as that done during MVCs, excessive co-contraction may limit full motor unit activation[155].

Participants in the present study, although training at different intensities, were relatively equal in training volume. A systematic review examining differences in HI and LI resistance training of the lower limb in older adults found that when participants performed similar volumes of training strength gains were the same regardless of intensity[102]. The present study however suggests that because there are greater increases in muscle activation in the high intensity group, perhaps there will also be greater strength gains. Strength data will not be published until the current START trial concludes.

Pietrosimone and Saliba[17] investigated whether changes in quadriceps activation would predict changes in quadriceps strength in patients with knee OA. They found that changes in activation predicted 47% of the variance in the change in quadriceps strength following a 4 week exercise program. These data emphasize the importance of focusing rehabilitation efforts on muscle activation to improve strength in this population.

This study is not without limitations. First, the study consisted of mostly white, female participants, limiting the generalizability of the findings. The sample size was relatively small. It is possible that a larger sample size would increase the power to detect a between group difference. It is also possible that following the subjects through the entire 18 months of training would elicit a more significant difference between groups and provide more insight to changes in activation over time. Using manual resistance to collect MVCs could have induced some variability compared to using an isokinetic dynamometer.

Future research should aim to analyze the effects of high intensity resistance training and its effects on muscle activation during gait in the OA population. It is important to see if changes in activation during strength training are also seen during gait. Doing this on a larger scale for longer duration might also provide better insight to the effects of long term strength training and its effects on muscle activation in this population. Lastly it would be interesting to compare the activation of the affected leg to unaffected leg to see if the increases in activation of the affected leg reach the levels of activation in the non-affected leg. This of course would have to be done on participants who did not have

bilateral knee OA.

In conclusion, this study demonstrated that high intensity training at 75% of 1-RM increases muscle activation more than low intensity training at 30% of 1-RM, particularly in the gluteus medius. This is important given that patients with knee OA have significant lower extremity muscle weakness as well as impaired muscle activation compared to the healthy population. To maximize strength gains in this population it is important to increase lean muscle mass as well as activation of the muscles. Since increases in muscle activation are a large contributor to increased strength following a resistance training program, based on the results of this study rehabilitation efforts for the knee OA population should utilize a high intensity training program to maximize muscle adaptations.

References

1. Kamil E. Barbour, PhD, Charles G. Helmick, MD KATM. Prevalence and most common causes of disability among adults--United States, 2005. MMWR Morb Mortal Wkly Rep 2009;58(16):421–6.

2. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum 2008;58(1):26–35.

3. Vos T, Flaxman AD, Naghavi M, et al. Years lived with disability for 1160 sequelae of 289 diseases and injuries 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380(9859):2163–96.

4. Muraki S, Akune T, Teraguchi M, et al. Quadriceps muscle strength, radiographic knee osteoarthritis and knee pain: the ROAD study. BMC Musculoskelet Disord 2015;16(1):305.

5. O'Reilly SC, Muir KR, Doherty M. Effectiveness of home exercise on pain and disability from osteoarthritis of the knee: a randomised controlled trial. Ann Rheum Dis 1999;58(1):15–9.

6. Farr JN, Going SB, McKnight PE, Kasle S, Cussler EC, Cornett M. Progressive resistance training improves overall physical activity levels in patients with early osteoarthritis of the knee: a randomized controlled trial. Phys Ther 2010;90(3):356–66.

7. Baker KR, Nelson ME, Felson DT, Layne JE, Sarno R, Roubenoff R. The efficacy of home based progressive strength training in older adults with knee osteoarthritis: A randomized controlled trial. J Rheumatol 2001;28(7):1655–65.

8. Thomas KS, Muir KR, Doherty M, Jones AC, O'Reilly SC, Bassey EJ. Home based exercise programme for knee pain and knee osteoarthritis: randomised controlled trial. BMJ 2002;325(7367):752.

9. Gür H, Çakın N, Akova B, Okay E, Küçükoğlu S. Concentric versus combined concentric-eccentric isokinetic training: Effects on functional capacity and symptoms in patients with osteoarthrosis of the knee. Arch Phys Med Rehabil 2002;83(3):308–16.

10. Topp R, Woolley S, Hornyak J, Khuder S, Kahaleh B. The effect of dynamic versus isometric resistance training on pain and functioning among adults with osteoarthritis of the knee. Arch Phys Med Rehabil 2002;83(9):1187–95.

11. Hassan BS, Mockett S, Doherty M. Static postural sway, proprioception, and maximal voluntary quadriceps contraction in patients with knee osteoarthritis and normal control subjects. Ann Rheum Dis 2001;60(6):612–8.

12. Hurley M V. The effects of joint damage on muscle function, proprioception and rehabilitation. Man Ther 1997;2(1):11–7.

13. O'Reilly SC, Jones A, Muir KR, Doherty M. Quadriceps weakness in knee osteoarthritis: the effect on pain and disability. Ann Rheum Dis 1998;57(10):588–94.

14. Hurley M V, Scott DL, Rees J, Newham DJ. Sensorimotor changes and functional performance in patients with knee osteoarthritis. Ann Rheum Dis 1997;56(11):641–8.

15. Henneman E, Somjen G, Carpenter DO. EXCITABILITY AND INHIBITIBILITY OF MOTONEURONS OF DIFFERENT SIZES. J Neurophysiol 1965;28(3):599–620.

16. Schoenfeld BJ, Contreras B, Willardson JM, Fontana F, Tiryaki-Sonmez G. Muscle activation during low- versus high-load resistance training in well-trained men. Eur J Appl Physiol 2014;114(12):1–7.

17. Pietrosimone BG, Saliba SA. Changes in voluntary quadriceps activation predict changes in quadriceps strength after therapeutic exercise in patients with knee osteoarthritis. Knee 2012;19(6):939–43.

18. Prevalence and Most Common Causes of Disability Among Adults --- United States, 2005 [Internet]. [cited 2015 Jun 29];Available from: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5816a2.htm

19. Sacks JJ, Luo Y-H, Helmick CG. Prevalence of specific types of arthritis and other rheumatic conditions in the ambulatory health care system in the United States, 2001-2005. Arthritis Care Res (Hoboken) 2010;62(4):460–4.

20. Guccione AA, Felson DT, Anderson JJ, et al. The effects of specific medical conditions on the functional limitations of elders in the Framingham Study. Am J Public Health 1994;84(3):351–8.

21. Michael JW-P, Schlüter-Brust KU, Eysel P. The epidemiology, etiology, diagnosis, and treatment of osteoarthritis of the knee. Dtsch Arztebl Int 2010;107(9):152–62.

22. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W MR. The prevalence of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. Arthritis Rheum 1987;30(8):914–8.

23. Project O, Jordan JM, Helmick CG, et al. Prevalence of knee symptoms and radiographic and symptomatic knee osteoarthritis in African Americans and Caucasians : the Johnston County Prevalence of Knee Symptoms and Radiographic and Symptomatic Knee Osteoarthritis in African Americans and Caucasians. J Rheumatol 2007;34(1).

24. Oliveria SA, Felson DT, Reed JI, Cirillo PA WA. Incidence of symptomatic hand, hip, and knee osteoarthritis among patients in a health maintenance organization. Arthritis Rheum 1995;38(8):1134–41.

25. Murphy L, Schwartz TA, Helmick CG, et al. Lifetime risk of symptomatic knee osteoarthritis. Arthritis Rheum 2008;59(9):1207–13.

26. Grotle M, Hagen KB, Natvig B, Dahl FA, Kvien TK. Obesity and osteoarthritis in knee, hip and/or hand: An epidemiological study in the general population with 10 years follow-up. BMC Musculoskelet Disord 2008;9(1):132.

27. Aghdam A, Kolahi S, Hasankhani H, Behshid M. The relationship between pain and physical function in adults with Knee Osteoarthritis. Int Res J Appl Basic Sci 2013;4(5):1102–6.

28. Cubukcu D, Sarsan A, Alkan H. Relationships between Pain, Function and Radiographic Findings in Osteoarthritis of the Knee: A Cross-Sectional Study. Arthritis 2012;2012(April 2007):984060.

29. Hawker GA, Stewart L, French MR, et al. Understanding the pain experience in hip and knee osteoarthritis--an OARSI/OMERACT initiative. Osteoarthritis Cartilage 2008;16(4):415–22.

30. Andriacchi TP, Favre J, Erhart-Hledik JC, Chu CR. A Systems View of Risk Factors for Knee Osteoarthritis Reveals Insights into the Pathogenesis of the Disease. Ann Biomed Eng 2014;43(2):376–87.

31. P. Creamer ML-C and MCH. Factors Associated with functional impairment in symptomatic knee osteoarthritis. Rheumatology 2000;39(5):490–6.

32. Andriacchi TP, M"undermann a, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the \textit{in vivo} pathomechanics of osteoarthritis at the knee. Ann Biomed Eng 2004;32(3):447–57.

33. Andriacchi TP, Favre J. The Nature of In Vivo Mechanical Signals That Influence Cartilage Health and Progression to Knee Osteoarthritis. Curr Rheumatol Rep 2014;16(11).

34. Andriacchi TP, Mündermann A. The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. Curr Opin Rheumatol 2006;18(5):514–8.

35. Arokoski JP, Jurvelin JS, Väätäinen U, Helminen HJ. Normal and pathological adaptations of articular cartilage to joint loading. Scand J Med Sci Sports 2000;10(4):186–98.

36. Bonnet CS, Walsh D a. Osteoarthritis, angiogenesis and inflammation. Rheumatology 2005;44(1):7–16.

37. Burr DB. Anatomy and physiology of the mineralized tissues: Role in the pathogenesis of osteoarthrosis. Osteoarthr Cartil 2004;12:20–30.

38. Carter DR, Beaupré GS, Wong M, Smith RL, Andriacchi TP, Schurman DJ. The mechanobiology of articular cartilage development and degeneration. Clin Orthop Relat Res 2004;(427 Suppl):S69–77.

39. Cem Gabay, M.D., and Irving Kushner M. Acute-Phase Proteins and Other Systemic Responses to Inflammation. N Engl J Med 2005;43(10):1111.

40. Englund M. The role of biomechanics in the initiation and progression of OA of the knee. Best Pract Res Clin Rheumatol 2010;24(1):39–46.

41. Goldring SR. The role of cytokines in cartilage matrix degeneration in osteoarthritis. Clin Orthop Relat Res 4AD;427(427):S27–36.

42. Griffin TM, Guilak F. The role of mechanical loading in the onset and progression of osteoarthritis. Exerc Sport Sci Rev 2005;33(4):195–200.

43. Haywood L, McWilliams DF, Pearson CI, et al. Inflammation and angiogenesis in osteoarthritis. Arthritis Rheum 2003;48(8):2173–7.

44. S. Larsson, M. Englund, A. Stuglies SL. Interleukin-6 and tumor necrosis factor alpha in synovia fluid are associated with progression of radiographic osteoarthritis in subjects with previous menisectomy. 23(2015):50–2.

45. Sharma L, Song J, Felson DT, Cahue S, Shamiyeh E, Dunlop DD. The role of knee alignment in disease progression and functional decline in knee osteoarthritis. JAMA 2001;286(2):188–95.

46. Sharma L, Cahue S, Song J, Hayes K, Pai Y-C, Dunlop D. Physical functioning over three years in knee osteoarthritis: role of psychosocial, local mechanical, and neuromuscular factors. Arthritis Rheum 2003;48(12):3359–70.

47. Seedhom BB. Conditioning of cartilage during normal activities is an important factor in the development of osteoarthritis. Rheumatology 2006;45(2):146–9.

48. Wilson DR, McWalter EJ, Johnston JD. The Measurement of Joint Mechanics and Their Role in Osteoarthritis Genesis and Progression. Rheum Dis Clin North Am 2013;39(1):21–44.

49. Mündermann A, Dyrby CO, Hurwitz DE, Sharma L, Andriacchi TP. Potential Strategies to Reduce Medial Compartment Loading in Patients With Knee Osteoarthritis of Varying Severity: Reduced Walking Speed. Arthritis Rheum 2004;50(4):1172–8.

50. Mündermann A, Dyrby CO, Andriacchi TP. Secondary gait changes in patients with medial compartment knee osteoarthritis: Increased load at the ankle, knee, and hip during walking. Arthritis Rheum 2005;52(9):2835–44.

51. T Miyazaki, M Wada, H Kawahara, M Sato, H Baba SS. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. Annnals Rhuematic Dis 2002;61(7):617–22.

52. Andriacchi TP, Dyrby CO. Interactions between kinematics and loading during walking for the normal and ACL deficient knee. J Biomech 2005;38(2):293–8.

53. Barrance PJ, Williams GN, Snyder-Mackler L, Buchanan TS. Altered knee kinematics in ACL-deficient non-copers: A comparison using dynamic MRI. J Orthop Res 2006;24(2):132–40.

54. Edd SN, Netravali N a., Favre J, Giori NJ, Andriacchi TP. Alterations in Knee Kinematics After Partial Medial Meniscectomy Are Activity Dependent. Am J Sports Med 2015;

55. M. Titchenal, C.R Chu TPA. Prospective changes in the knee joint center of rotation relative to the contralateral knee and over time provide a comprehensive view of kinematic changes following anterior cruciate ligament reconstruction. Osteoarthr Cartil 2015;23(2015).

56. Ashraf S, Mapp PI, Walsh D a. Contributions of angiogenesis to inflammation, joint damage, and pain in a rat model of osteoarthritis. Arthritis Rheum 2011;63(9):2700–10.

57. T.D. Spector, D.J Hart, D. Nandra, D.V. Doyle, N. Mackillop, J.R. Gallimore and MBP. Low-Level Increases in Serum C-Reactive Protein are Present in Early Osteoarthritis of the Knee and Predict Progressive Disease. Am Coll Rhematology 1997;40(4):723–7.

58. Kellgren JH, Lawrence JS. Radiological Assessment of Osteo-Atrhosis. 1957;(16):494–503.

59. Heidari B. Knee osteoarthritis prevalence, risk factors, pathogenesis and features: Part I. Casp J Intern Med 2011;2(2):205–12.

60. R. Altman, E. Asch, D. Bloch, G. Bole, D. Borenstein, K. Brandt, W. Christy, T.D. Cooke, R. Greenwald, M. Hoachberg, D. Howell, D. Kaplan, W. Koopman, S. Longely, III, H. Mankin, D.J. McShane, T. Medsger, Jr., R. Meenan, W. Mikkelsen, R. Moskowitz, W. Mur and FW. Development of Criteria for the Classification and reporting of Osteoarthritis. Arthritis and rhematism 1986;29(8):1039–49.

61. Finan PH, Buenaver LF, Bounds SC, et al. Discordance between pain and radiographic severity in knee osteoarthritis: findings from quantitative sensory testing of central sensitization. Arthritis Rheum 2013;65(2):363–72.

62. Chaganti RK, Lane NE. Risk factors for incident osteoarthritis of the hip and knee. Curr Rev Musculoskelet Med 2011;4(3):99–104.

63. Dillon CF, Rasch EK, Gu Q, Hirsch R. Prevalence of knee osteoarthritis in the United States: arthritis data from the Third National Health and Nutrition Examination Survey 1991-94. J Rheumatol 2006;33(11):2271–9.

64. Silverwood V, Blagojevic-Bucknall M, Jinks C, Jordan JL, Protheroe J, Jordan KP. Current evidence on risk factors for knee osteoarthritis in older adults: a systematic review and meta-analysis. Osteoarthritis Cartilage 2014;23(4):507–15.

65. Graeme Harding, Michael Dunbar, Cheryl Hubley-Kozey, William Stanish JLAW. Obesity is Associated with Higher Absolute Tibiofemoral Contact and Muscle Forces during Gait with and without Knee Osteoarthritis. Clin Biomech 2015;

66. DeVita P, Hortobágyi T. Obesity is not associated with increased knee joint torque and power during level walking. J Biomech 2003;36(9):1355–62.

67. Messier SP, Legault C, Loeser RF, et al. Does high weight loss in older adults with knee osteoarthritis affect bone-on-bone joint loads and muscle forces during walking? Osteoarthritis Cartilage 2011;19(3):272–80.

68. Aaboe J, Bliddal H, Messier SP, Alkjær T, Henriksen M. Effects of an intensive weight loss program on knee joint loading in obese adults with knee osteoarthritis. Osteoarthr Cartil 2011;19(7):822–8.

69. Nguyen U-SDT, Zhang Y, Zhu Y, Niu J, Zhang B, Felson DT. Increasing prevalence of knee pain and symptomatic knee osteoarthritis: survey and cohort data. Ann Intern Med 2011;155(11):725–32.

70. Brouwer GM, Tol AW Van, Bergink AP, et al. Association between valgus and varus alignment and the development and progression of radiographic osteoarthritis of the knee. Arthritis Rheum 2007;56(4):1204–11.

71. Hunter DJ, Niu J, Felson DT, et al. Knee alignment does not predict incident osteoarthritis: The Framingham osteoarthritis study. Arthritis Rheum 2007;56(4):1212–8.

72. ARDEN N, NEVITT M. Osteoarthritis: Epidemiology. Best Pract Res Clin Rheumatol 2006;20(1):3–25.

73. Segal NA, Glass NA, Felson DT, et al. Effect of quadriceps strength and proprioception on risk for knee osteoarthritis. Med Sci Sports Exerc 2010;42(11):2081–8.

74. Hootman JM, Fitzgerald S, Macera C a, Blair SN. Lower Extremity Muscle Strength and Risk of Self-Reported Hip or Knee Osteoarthritis. North 2004;1(4):321–30.

75. Sharma L, Dunlop DD, Cahue S, Song J, Hayes KW. Quadriceps Strength and Osteoarthritis Progression in Malaligned and Lax Knees. Ann Intern Med 2003;(138):613–9.

76. Omori G, Koga Y, Tanaka M, et al. Quadriceps muscle strength and its relationship to radiographic knee osteoarthritis in Japanese elderly. J Orthop Sci 2013;18(4):536–42.

77. Clark DA, Simpson DL, Eldridge J, Colborne GR. Damian A. Clark.

78. Lohmander LS, Ostenberg A, Englund M, Roos H. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. Arthritis Rheum 2004;50(10):3145–52.

79. von Porat A, Roos EM, Roos H. High prevalence of osteoarthritis 14 years after an anterior cruciate ligament tear in male soccer players: a study of radiographic and patient relevant outcomes. Ann Rheum Dis 2004;63(3):269–73.

80. Englund M, Lohmander LS. Risk factors for symptomatic knee osteoarthritis fifteen to twenty-two years after meniscectomy. Arthritis Rheum 2004;50(9):2811–9.

81. Spector TD, Harris PA, Hart DJ, et al. Risk of osteoarthritis associated with long-term weight-bearing sports: a radiologic survey of the hips and knees in female ex-athletes and population controls. Arthritis Rheum 1996;39(6):988–95.

82. Sohn RS, Micheli LJ. The effect of running on the pathogenesis of osteoarthritis of the hips and knees. Clin Orthop Relat Res 1985;(198):106–9.

83. Panush RS, Hanson CS, Caldwell JR, Longley S, Stork J, Thoburn R. Is Running Associated with Osteoarthritis? An Eight-Year Follow-up Study. J Clin Rheumatol 1995;1(1):35–9.

84. Felson DT, Hannan MT, Naimark A, et al. Occupational physical demands, knee bending, and knee osteoarthritis: results from the Framingham Study. J Rheumatol 1991;18(10):1587–92.

85. Coggon D, Croft P, Kellingray S, Barrett D, McLaren M, Cooper C. Occupational physical activities and osteoarthritis of the knee. Arthritis Rheum 2000;43(7):1443–9.

86. Walter H. Ettinger, Jr, MD; Robert Burns, MD; Stephen P. Messier, PhD; Wiliam Applegate, MD; Jack Rejeski, PhD; Timothy Morgan, PhD; Sally Shumaker, PhD; Michael Berry, PhD; Mary O'Toole, PhD, Johnny Monu, MD; Timothy Craven M. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: The Fitness Arthritis and Seniors Trial (FAST). JAMA 1997;25–31.

87. Mazzuca SA, Brandt KD, Katz BP, Chambers M, Byrd D, Hanna M. Effects of self-care education on the health status of inner-city patients with osteoarthritis of the knee. Arthritis Rheum 1997;40(8):1466–74.

88. McAlindon TE, Bannuru RR, Sullivan MC, et al. OARSI guidelines for the non-surgical management of knee osteoarthritis. Osteoarthritis Cartilage 2014;22(3):363–88.

89. Creamer P. Osteoarthritis pain and its treatment. Curr Opin Rheumatol 2000;12(5):450–5.

90. Bannuru RR, Schmid CH, Kent DM, Vaysbrot EE, Wong JB, McAlindon TE. Comparative Effectiveness of Pharmacologic Interventions for Knee Osteoarthritis. Ann Intern Med 2015;162(1):46.

91. Katz JN, Smith SR, Collins JE, et al. Cost-effectiveness of nonsteroidal anti-inflammatory drugs and opioids in the treatment of knee osteoarthritis in older patients with multiple comorbidities. Osteoarthritis Cartilage 2015;24(3):409–18.

92. Wright EA, Katz JN, Abrams S, Solomon DH, Losina E. Trends in prescription of opioids from 2003-2009 in persons with knee osteoarthritis. Arthritis Care Res (Hoboken) 2014;66(10):1489–95.

93. Skou ST, Roos EM, Laursen MB, et al. A Randomized, Controlled Trial of Total Knee Replacement. N Engl J Med 2015;373(17):1597–606.

94. Waimann CA, Fernandez-Mazarambroz RJ, Cantor SB, et al. Cost-effectiveness of total knee replacement: a prospective cohort study. Arthritis Care Res (Hoboken) 2014;66(4):592–9.

95. Kennedy JW, Johnston L, Cochrane L, Boscainos PJ. Total knee arthroplasty in the elderly: does age affect pain, function or complications? Clin Orthop Relat Res 2013;471(6):1964–9.

96. Farr Ii J, Miller LE, Block JE. Quality of life in patients with knee osteoarthritis: a commentary on nonsurgical and surgical treatments. Open Orthop J 2013;7:619–23.

97. Picavet HSJ, Hoeymans N. Health related quality of life in multiple musculoskeletal diseases: SF-36 and EQ-5D in the DMC3 study. Ann Rheum Dis 2004;63(6):723–9.

98. S.C. O, K.R. M, M. D. Effectiveness of home exercise on pain and disability from osteoarthritis of the knee: A randomised controlled trial. Ann Rheum Dis 1999;58(1):15–9.

99. Maurer BT, Stern AG, Kinossian B, Cook KD, Schumacher HR. Osteoarthritis of the knee: Isokinetic quadriceps exercise versus an educational intervention. Arch Phys Med Rehabil 1999;80(10):1293–9.

100. Lange AK, Vanwanseele B, Fiatarone singh MA. Strength training for treatment of osteoarthritis of the knee: A systematic review. Arthritis Rheum 2008;59(10):1488–94.

101. Bennell KL, Hunt MA, Wrigley T V., Lim BW, Hinman RS. Muscle and Exercise in the Prevention and Management of Knee Osteoarthritis: an Internal Medicine Specialist's Guide. Med Clin North Am 2009;93(1):161–77.

102. Raymond MJ, Bramley-Tzerefos RE, Jeffs KJ, Winter A, Holland AE. Systematic review of high-intensity progressive resistance strength training of the lower limb compared with other intensities of strength training in older adults. Arch Phys Med Rehabil 2013;94(8):1458–72.

103. Hunter GR, Wetzstein CJ, Fields DA, Brown A, Bamman MM. Resistance training increases total energy expenditure and free-living physical activity in older adults. J Appl Physiol 2000;89(3):977–84.

104. Tevald MA, Murray A, Luc BA, Lai K, Sohn D, Pietrosimone B. Hip abductor strength in people with knee osteoarthritis: A cross-sectional study of reliability and association with function. Knee 2015;

105. Hinman RS, Hunt MA, Creaby MW, Wrigley T V, McManus FJ, Bennell KL. Hip muscle weakness in individuals with medial knee osteoarthritis. Arthritis Care Res (Hoboken) 2010;62(8):1190–3.

106. Ferri A, Scaglioni G, Pousson M, Capodaglio P, Van Hoecke J, Narici M V. Strength and power changes of the human plantar flexors and knee extensors in response to resistance training in old age. Acta Physiol Scand 2003;177(1):69–78.

107. Taaffe D, Pruitt L, Pyka G, Guido D. Comparative effects of high- and low-intensity resistance training on thigh muscle strength, fiber area, and tissue composition in elderly women. Clin Physiol 1996;16(4):381–92.

108. Häkkinen K, Häkkinen A. Neuromuscular adaptations during intensive strength training in middle-aged and elderly males and females. Electromyogr Clin Neurophysiol 1995;35(3):137–47.

109. Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA EW. High-Intensity Strength Training in Nonagenarians: Effects on Skeletal Muscle. JAMA 1990;263(22):3029–34.

110. Simon Walker KH. Similar Increases in Strength After Short-Term Resistance Training Due to Different Neuromuscular Adaptations in Young and Older Men. J Strength Cond Res 2014;28(11):3041–8.

111. Al-Khlaifat L, Herrington LC, Hammond A, Tyson SF, Jones RK. The effectiveness of an exercise programme on knee loading, muscle co-contraction, and pain in patients with medial knee osteoarthritis: A pilot study. Knee 2016;23(1):63–9.

112. Basmajian JV. Muscles Alive. 4th ed. Baltimore: The Williams & Wilkins Company; 1979.

113. Jonas D. Influence of different types of surface electrodes on amplitude, area and duration of the compound muscle action potential. Clin Neurophysiol 1999;110(12):2171–5.

114. Gary Kamen DAG. Essentials of Electromyography. Champaign, IL: Human Kinetics; 2010.

115. Johnson SW, Lynn PA, Miller JSG, Reed GAL. Miniature skin-mounted preamplifier for measurement of surface electromyographic potentials. Med Biol Eng Comput 1977;15(6):710–1.

116. Raez MBI, Hussain MS, Mohd-Yasin F. Techniques of EMG signal analysis: detection, processing, classification and applications. Biol Proced Online 2006;8:11–35.

117. Chowdhury R, Reaz M, Ali M, Bakar A, Chellappan K, Chang T. Surface Electromyography Signal Processing and Classification Techniques. Sensors 2013;13(9):12431–66.

118. Winter DA. Biomechanics of Human Movement. Waterloo: Wiley-Interscience; 1979.

119. Baker LAG and LE. The scientific resistance of biological material- a compendium of data for the biomedical engineer and physiologist. Med Biol Eng Comput 1967;5:271–93.

120. Keenan KG, Farina D, Maluf KS, Merletti R, Enoka RM. Influence of amplitude cancellation on the simulated surface electromyogram. J Appl Physiol 2005;98(1):120–31.

121. Nordander C, Willner J, Hansson G-�., et al. Influence of the subcutaneous fat layer, as measured by ultrasound, skinfold calipers and BMI, on the EMG amplitude. Eur J Appl Physiol 2003;89(6):514–9.

122. Kuiken T a, Lowery MM, Stoykov NS. The effect of subcutaneous fat on myoelectric signal amplitude and cross-talk. Prosthet Orthot Int 2003;27(1):48–54.

123. F.D. F, J.C. P, C.J. F, Farfan FD, Politti JC, Felice CJ. Evaluation of EMG processing techniques using Information Theory. Biomed Eng Online 2010;9:72.

124. Hodges PW, Bui BH. A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. Electroencephalogr Clin Neurophysiol Mot Control 1996;101(6):511–9.

125. Brandon R, Howatson G, Hunter A. Reliability of a combined biomechanical and surface electromyographical analysis system during dynamic barbell squat exercise. J Sports Sci 2011;29(13):1389–97.

126. Zech a., Witte K, Pfeifer K. Reliability and performance-dependent variations of muscle function variables during isometric knee extension. J Electromyogr Kinesiol 2008;18:262–9.

127. Bolgla L a., Malone TR, Umberger BR, Uhl TL. Reliability of electromyographic methods used for assessing hip and knee neuromuscular activity in females diagnosed with patellofemoral pain syndrome. J Electromyogr Kinesiol 2010;20(1):142–7.

128. Hubley-Kozey CL, Robbins SM, Rutherford DJ, Stanish WD. Reliability of surface electromyographic recordings during walking in individuals with knee osteoarthritis. J Electromyogr Kinesiol 2013;23(2):334–41.

129. Staehli S, Glatthorn JF, Casartelli N, Maffiuletti N a. Test-retest reliability of quadriceps muscle function outcomes in patients with knee osteoarthritis. J Electromyogr Kinesiol 2010;20(6):1058–65.

130. Cannon JDKMMFE. Comparative effects of resistance training on peak isometric torque, muscle hypertrophy, voluntary activation and surface EMG between young and elderly women.

131. Jakobi JM, Rice CL. Voluntary muscle activation varies with age and muscle group. J Appl Physiol 2002;93(2):457–62.

132. Kent-Braun JA, Ng A V. Specific strength and voluntary muscle activation in young and elderly women and men. J Appl Physiol 1999;87(1):22–9.

133. Tomlinson DJ, Erskine RM, Morse CI, Winwood K, Onambélé-Pearson GL. Combined effects of body composition and ageing on joint torque, muscle activation and co-contraction in sedentary women. Age (Dordr) 2014;36(3):9652.

134. William D. McArdle, Frank I. Katch VLK. Exercise Physiology. 2010.

135. Radaelli R, Wilhelm EN, Botton CE, et al. Effects of single vs. multiple-set short-term strength training in elderly women. Age (Dordr) 2014;36(6):9720.

136. Barry BK, Warman GE, Carson RG. Age-related differences in rapid muscle activation after rate of force development training of the elbow flexors. Exp brain Res 2005;162(1):122–32.

137. Hanten WDB and WP. Changes in Torque and Electromyographic Activity

of the Quadriceps Femoris Muscles Following Isometric Training. J Am Phys Ther Assoc 1993;73:455–65.

138. Akima H, Saito A. Activation of quadriceps femoris including vastus intermedius during fatiguing dynamic knee extensions. Eur J Appl Physiol 2013;113(11):2829–40.

139. COOK SB, MURPHY BG, LABARBERA KE. Neuromuscular Function after a Bout of Low-Load Blood Flow–Restricted Exercise. Med Sci Sport Exerc 2013;45(1):67–74.

140. Messier SP, Mihalko SL, Beavers DP, et al. Strength Training for Arthritis Trial (START): design and rationale. BMC Musculoskelet Disord 2013;14(1):208.

141. SENIAM Project [Internet]. 2005;Available from: http://www.seniam.org/sensor_location.htm

142. Lewek MD, Rudolph KS, Snyder-Mackler L. Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis. J Orthop Res 2004;22(1):110–5.

143. Rutherford DJ, Hubley-Kozey CL, Stanish WD. Maximal voluntary isometric contraction exercises: A methodological investigation in moderate knee osteoarthritis. J Electromyogr Kinesiol 2011;21(1):154–60.

144. Ayotte NW, Stetts DM, Keenan G, Greenway EH. Electromyographical analysis of selected lower extremity muscles during 5 unilateral weight-bearing exercises. J Orthop Sports Phys Ther 2007;37(2):48–55.

145. Boren K, Conrey C, Le Coguic J, Paprocki L, Voight M, Robinson TK. Electromyographic analysis of gluteus medius and gluteus maximus during rehabilitation exercises. Int J Sports Phys Ther 2011;6(3):206–23.

146. Ekstrom R a, Donatelli R a, Carp KC. Electromyographic analysis of core trunk, hip, and thigh muscles during 9 rehabilitation exercises. J Orthop Sports Phys Ther 2007;37(12):754–62.

147. Schoenfeld BJ, Contreras B, Willardson JM, Fontana F, Tiryaki-Sonmez G.

Muscle activation during low- versus high-load resistance training in well-trained men. Eur J Appl Physiol 2014;2491–7.

148. MacKinnon CD, Winter DA. Control of whole body balance in the frontal plane during human walking. J Biomech 1993;26(6):633–44.

149. Chang A, Hayes K, Dunlop D, et al. Hip abduction moment and protection against medial tibiofemoral osteoarthritis progression. Arthritis Rheum 2005;52(11):3515–9.

150. HURLEY M, NEWHAM D. THE INFLUENCE OF ARTHROGENOUS MUSCLE INHIBITION ON QUADRICEPS REHABILITATION OF PATIENTS WITH EARLY, UNILATERAL OSTEOARTHRITIC KNEES. Br J Rheumatol 1993;32(2):127–31.

151. Segal N a., Glass N a., Torner J, et al. Quadriceps weakness predicts risk for knee joint space narrowing in women in the MOST cohort. Osteoarthr Cartil 2010;18(6):769–75.

152. Øiestad BE, Juhl CB, Eitzen I, Thorlund JB. Knee extensor muscle weakness is a risk factor for development of knee osteoarthritis. A systematic review and meta-analysis. Osteoarthritis Cartilage 2015;23(2):171–7.

153. Frontera WR, Meredith CN, O'Reilly KP, Knuttgen HG, Evans WJ. Strength conditioning in older men: skeletal muscle hypertrophy and improved function. J Appl Physiol 1988;64:1038–44.

154. Brown a B, McCartney N, Sale DG. Positive adaptations to weight-lifting training in the elderly. J Appl Physiol 1990;69(5):1725–33.

155. Sale DG. Neural adaptations to resistance training. Med Sci Sport Exerc 1988;20(5):S135–45.

Curriculum Vitae

Alicia Sabatino

(336)-758-3762

Sabaan14@wfu.edu

EDUCATION

August 2014 - May 2016, Wake Forest University, NC

- Master of Science, Health and Exercise Science (M.S)

August 2010 - May 2014, East Stroudsburg University of Pennsylvania, PA

- Bachelor of Science, Exercise Science - concentration in Clinical Exercise Physiology (B.S)

EMPLOYMENT HISTORY

March 2016 – Present, Wake Forest University, NC

Research Assistant/Clinical Exercise Physiologist, Department of Health and Exercise Science

- Weight Loss and Exercise for Communities with Arthritis in North Carolina (WE-CAN)

August 2014 – Present, Wake Forest University, NC

- Fitness Instructor, Healthy Exercise Lifestyle Programs (HELPS)
- Teaching Assistant, Department of Health and Exercise Science
    - Health and Exercise 101

    - Biomechanics lab

- Graduate Student Intern, Strength Training for Arthritis Trial (START)

CERTIFICATIONS

- American College of Sports Medicine, Clinical Exercise Physiologist

MEMBERSHIPS

- 2010- present, American College of Sports Medicine