

DIFFERENTIAL EFFECTS OF UNILATERAL VERSUS BILATERAL KNEE
OSTEOARTHRITIS ON GAIT

BY

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ABSTRACT

PURPOSE: The purpose of this investigation was to compare the gait kinematics and kinetics of older adults with radiographic unilateral knee osteoarthritis and those with radiographic bilateral knee osteoarthritis with accompanying pain in one or both knees. We hypothesized that the gait of those with unilateral disease would be significantly different than those with bilateral disease, and therefore, should be analyzed independent of those with bilateral disease in future research studies.

METHODS: Baseline data from a subset of participants (136 out of 454) enrolled in the Intensive Diet and Exercise for Arthritis (IDEA) study were used for this study. 68 participants were identified as having unilateral radiographic OA and were matched to 68 participants with bilateral OA based on age, gender, BMI, race, walking speed, WOMAC pain score, and WOMAC function score. Internal joint moments and knee joint reaction forces were calculated using a 3-D gait analysis with a 37-reflective marker set arranged in a Cleveland Clinic full-body configuration, a 6-Camera Motion Analysis System set to sample data at 60 Hz, and a torque-driven musculoskeletal model (DeVita and Hortobagyi). Means for peak values of percent of the gait cycle spent in the stance phase, joint ROM at the hip, knee, and ankle, frontal plane joint moments at the hip, knee, and ankle, sagittal plane moments at the knee, vertical GRF, bone-on-bone forces at the knee, and power generated at the hip, knee and ankle were calculated for each individual. Comparisons were made between the most affected leg in the bilateral cohort (self-selected by the participant) to the affected leg in the unilateral population, and between the least affected leg in the bilateral cohort to the healthy leg of the unilateral cohort.

Peak mean values were then used to calculate symmetry between limbs in each individual using the symmetry index (SI), and mean values for each group were compared.

RESULTS: After controlling for age, gender, K/L grade, BMI, presence of hip OA, and WOMAC pain score, significant differences were found in between groups in hip flexion moment ($p=0.01$) and the ankle plantar flexion moment ($p=0.04$) in the comparison of the healthy limb in the unilateral cohort to the least affected limb of the bilateral cohort, and in the ankle plantar flexion moment ($p=0.04$) in the comparison of the most affected limb in both cohorts. Significant asymmetry (greater than 10% difference between limbs) was seen in knee power in the unilateral cohort ($SI = 18\%$, $p=0.03$). Significant differences were seen in SI between unilateral and bilateral groups in hip flexion moment ($p=0.03$) and hip extension moment ($p=0.01$).

CONCLUSIONS: Distinctions are not always made in the classification of unilateral or bilateral disease in those with osteoarthritic knee joints in statistical analysis. While it is known that OA gait differs from the gait of healthy, asymptomatic controls, it cannot be assumed that the gait of painful unilateral knee OA and painful bilateral knee OA patients are similar. Our results indicate few differences when comparing the gait mechanics of those with unilateral OA to those with bilateral OA with accompanying mild to moderate pain. Overall, only 6 variables were found to be significantly different in 59 comparisons. This amount of variance may be expected due to chance alone. Due to this lack of statistical differences between groups in multiple comparisons, unilateral and bilateral OA can be analyzed collectively as one OA group, and that results discovered in one population may be generalized to both unilateral and bilateral OA.

INTRODUCTION

Arthritis is the leading cause of disability in adults over the age of 55 years. Over 40 million Americans have been diagnosed with arthritis, and this figure is projected to increase to 60 million by 2020. Among over 100 different types of arthritis, osteoarthritis (OA) is the most common, accounting for over 27 million cases¹. The knee is the most common weight-bearing joint affected by OA, and knee OA is a primary cause of disability in those over the age of 65 years².

A primary symptom of knee OA is chronic pain. There is a wide range of treatments, including pharmaceuticals, exercise, weight loss, and surgery. Compensatory gait mechanics are often a result of pain and are used to reduce loads on the knee. It is well documented that the gait of individuals with OA differs from that of healthy adults³⁻¹⁹. Alterations in gait are usually an attempt to alleviate symptoms of knee OA through the use of compensatory strategies. In order to decrease pain and overcome limitations in ROM, common gait alterations include decreasing walking velocity¹³, decreased flexion at the knee throughout the gait cycle^{16,19}, decreased total knee ROM, decreased stride length¹⁹, increased cadence, and alterations in both joint range of motion and joint moments at both the ankle and hip^{8,15,19}.

OA can be unilateral, which affects just one knee joint in an individual, or bilateral, which affects both knees in the same individual. Reported prevalence of unilateral OA has ranged from 12.6 – 34.1% individuals with OA^{20,21}. In OA studies, participants are often categorized as either diseased or healthy. Few studies categorize those persons with OA as having either unilateral or bilateral disease; rather, they are

commonly grouped together as an OA population and analyzed as one group. Studies have been conducted to assess gait in both unilateral and bilateral OA, comparing both to the healthy population, the assumption being that compensatory gait mechanics will be utilized in both populations. Individuals with bilateral OA can be expected to apply gait compensations to both lower limbs. Those with unilateral OA are only affected by disease on one side; therefore, it cannot be assumed that the same compensations applied to the diseased joint are also applied to the non-diseased joint. However, very few studies directly compare the gait mechanics used by patients with unilateral knee OA and patients with bilateral knee OA, and those that have found conflicting results. One study found an increased stance phase time in those with unilateral OA compared to those with bilateral disease, but no difference in walking velocity⁸. A second study found no differences in limb strength, power, or physical function²².

Recently, some studies have examined the symmetry between limbs in unilateral disease and again found conflicting results. Studies have reported no difference in ground reaction force²³ and knee adduction moment^{24,25}. However, in another study, knee adduction moment was found to be different between limbs²³. Asymmetries have also been found in the peak knee flexion angle, peak hip flexion angle, and peak hip flexion moment²⁴. Creaby et al²⁵ (2012) compared the symmetry between those with unilateral and bilateral knee OA incorporating both symptomatic and radiographic OA into analysis groups. They found asymmetries in peak knee flexion angle and peak knee flexion moment. More clarity is needed to determine if unilateral and bilateral disease can be assessed as one diseased group, or if they should be stratified into unilateral and bilateral diseased groups for analysis.

Therefore, the purpose of this study is to compare the gait kinematics and kinetics of older adults with radiographic unilateral knee OA and those with radiographic bilateral knee OA with accompanying pain in one or both knees. We hypothesized that the gait of those with unilateral disease would be significantly different than those with bilateral disease, and therefore, should be analyzed independent of those with bilateral disease in future research studies.

REVIEW OF THE LITERATURE

Epidemiology

Disability is most often caused by arthritic disorders in the adult population ²⁶. The World Health Organization estimates that 10% of all people over the age of 60 years suffer from OA and 80% of those with OA have movement limitations as a result. Arthritic and other rheumatoid conditions cost the United States an estimated 128 billion dollars, attributed to medical expenditures and lost earnings ²⁷. The knee is the most commonly affected weight-bearing joint. In 2005, Lawrence et al¹ (2008) reported that 27 million Americans were diagnosed by a physician as having tibiofemoral OA. The relationship between OA and disability has been attributed to pain and loss of joint motion associated with muscle weakness ¹². McAlindon et al²⁸ (1994) found that pain due to OA was a predictor of disability, stating that those with unilateral pain increase the risk of disability by 1.72 and those with bilateral pain increase this risk by 3.41.

Prevalence of OA is affected by age and gender. As age increases, prevalence of OA also increases. Over 50% of men and 70% of women over the age of 80 years showed evidence of radiographic knee OA²⁹. Reported prevalence of unilateral versus bilateral knee OA has ranged across studies. Lethbridge-Cejku et al²⁹ (1994) found that of individuals over the age of 80 years with knee OA, 45.7% of men and 51.5% of women had bilateral radiographic involvement. Men under the age of 60 years were more likely to have unilateral involvement when compared to men over the age of 60 years. In total, 54.3% of men and 48.5% of women had unilateral knee OA. Günther et al²¹ (1998) reported 87.4% of participants had bilateral radiographic OA and only 12.6% had

radiographic unilateral OA. Forestier et al²⁰ (2011) more recently reported a higher prevalence of radiographic unilateral OA, the sample was 65.9% bilateral OA and 34.1% unilateral OA. It has also been shown that those with unilateral knee OA increase their risk of developing OA in the contralateral limb. In a two year follow up of women with unilateral knee OA, 34% developed OA in the contralateral limb³⁰.

Incident OA can be classified as either idiopathic (primary) OA or secondary OA. Idiopathic OA is of unknown cause. This type is more common among adults over the age of 40 years³¹. Idiopathic OA is associated with systemic risk factors or those that place a greater load on the knee over a longer period of time (i.e. obesity)³². Secondary OA can be traced back to a known cause. This type develops following a traumatic injury³¹ or surgery³², or is the result of a disease, a significant joint deformity, or infection resulting in an inflammatory response³¹.

Pathology

The precise pathology of OA remains unclear. OA is associated with the degradation of articular cartilage within the joint, narrowing of the joint space width, an increase in inflammation, and changes to the subchondral bone^{2,33}. It is very difficult to determine the exact cause of OA, as symptoms typically do not appear in the early stages of radiographic changes. Therefore, OA often remains undiagnosed until radiographic changes become more severe. Furthermore, radiographic changes may never be accompanied by symptoms, or persons with symptoms may not have significant radiographic changes^{1,34,35}.

Some theories have been proposed as to the cause of OA. Currently, a biomechanical hypothesis is the most prevalent, which states that acute trauma, overuse, or altered mechanics contribute to the destruction of chondrocytes and the extracellular matrix. This breakdown causes the depletion of proteoglycans, which are cells necessary for load-bearing aspects of cartilage³⁶. Matrix damage also results in the breaking of interfibrillar cross-links within the articular cartilage. Breaking of the cross-links may result in the loss of proteoglycans out of the extracellular matrix. Alterations in mechanical loading across the articular cartilage may initiate matrix breakdown, and thus lead to eventual development of OA³⁷.

A second hypothesis states that microfractures in subchondral bone caused by trauma and excessive loading are repaired with fracture callous. This causes the trabeculae to both thicken and stiffen resulting in less ability to absorb shock which leads to additional cartilage stress and degradation³⁶. Burr suggests that subchondral tissue is associated with OA. In response to mechanical and pharmaceutical stimuli, the processes of modeling, remodeling, and growth result in an increase in the thickness calcified cartilage³⁸.

Yet another hypothesis points to excessive inflammation as it contributes to the breakdown of the extracellular matrix. Increased levels of inflammatory cytokines, such as interleukin-1(IL-6) and tumor necrosis factor α (TNF- α), cause the release of enzymes that breakdown the extracellular matrix. The cartilage breakdown encourages the inflammatory process, leading to more cartilage destruction²⁶. Ultimately, this increased presence of inflammatory cytokines and proteases results in an imbalance in tissue breakdown and repair, resulting in the gradual loss of cartilage in the joint³⁹.

Risk Factors

Risk factors for OA have been categorized into systemic and local factors. Systemic risk factors are generally non-modifiable and increase the vulnerability of the joint to OA development⁴⁰. Local factors, however, are generally modifiable and an individual can undergo intervention to reduce disease risk. Systemic factors are said to predispose a joint to OA development, but it is thought that when local factors act on this predisposed joint that OA develops or progresses further⁴¹ (Figure 1).

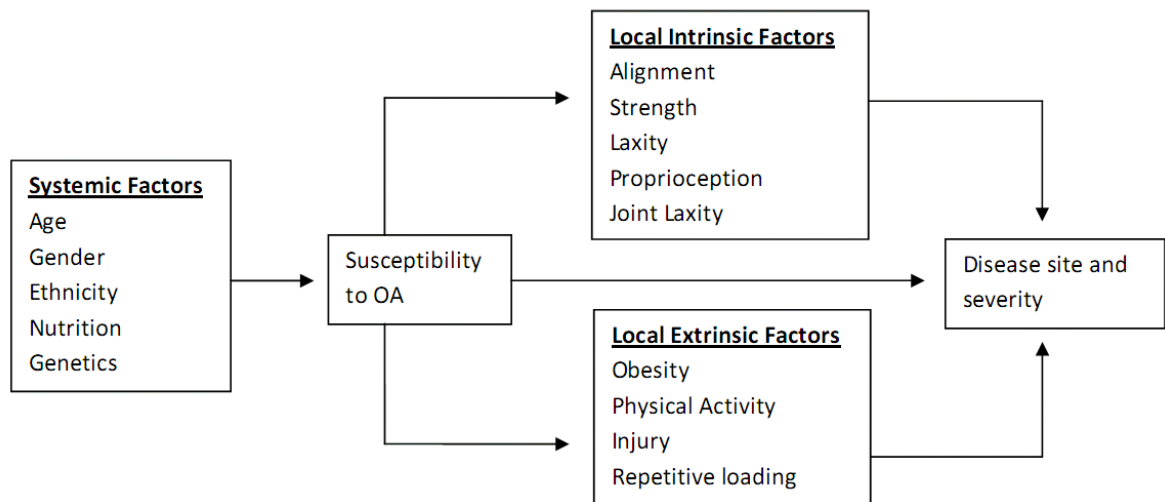


Figure 1: Interaction of systemic, intrinsic, and extrinsic risk factors and the development of OA⁴⁰⁻⁴²

Age has been identified as one of the most important risk factors for osteoarthritis. OA is much more prevalent in those with advanced age, increasing from just 5% in persons 15-44 years of age to more than 60% in persons over the age of 65 years³¹. Davis et al⁴³ (1989) found that the odds of developing bilateral OA increased significantly from 55-64 years (OR=2.37, 95%CI=1.45, 3.88) to 65-74 years (OR=8.77,

95% CI=5.77, 13.33). Similarly, odds of developing OA in the right knee only increased from 1.7 (95% CI 0.68, 4.27) in those 55-64 years to 5.4 (95%CI=2.47, 11.81) in those 65-74 years. Development of OA in only the left knee increased from 1.62 (95%CI=0.63, 4.17) in those 55-64 years to 3.06 (95%CI=1.35-6.92) in those 65-74 years⁴³. Age alone does not cause this increased risk. Instead, age-related changes in articular cartilage such as the degradation of the cartilage matrix, in conjunction with other risk factors may facilitate the development of OA.

Gender is also a factor making the knee more susceptible to OA. Studies have indicated a higher incidence and prevalence of knee OA in women compared to men⁴⁴. Estimates for prevalence in women based on radiographic changes are between 29.8 - 42.1% of all women compared to 22.9 - 31.2% of all men⁴⁵⁻⁴⁷. The reason for this higher prevalence in women is unclear, but may relate to differences in hormone levels⁴¹.

Local risk factors can be further broken down into extrinsic and intrinsic factors. Extrinsic factors are those factors or events that originate outside of the joint or its immediate environment such as physical activity, injury⁴², and obesity⁴¹. Physical activity includes activities of repetitive joint loading over a long period of time, such as occupational activity. To date, there is no definite link between recreational physical activity and the incidence of OA⁴². Injury to the knee joint causes alterations within the knee joint and is more associated with the development of unilateral osteoarthritis. Davis et al⁴³ (1989) found that injury to the right knee increased the odds of radiographic OA in that knee by 16.30 (CI=6.50-40.89) and injury to the left knee increased odds of OA in that same knee by 10.90 (CI=3.72-31.93). Obesity is also a well-documented risk factor for the development and progression OA. Excess weight causes excessive loading at the

joint. Messier et al³ (1994) found that for every 1 pound of weight lost, the load at the knee is decreased by 4 pounds per step. After following disease free individuals for 22 years, Toivanen et al (2010)⁴⁸ found that those with a BMI of 25-29.9 classifying them as overweight had increased the odds of radiographic OA development (OR= 1.8, 95%CI 1.1, 3.1), and these odds were increased to an even greater extent in those with a BMI $\geq$ 30 (OR= 7.1, 95% CI 3.6, 13.7).

Local intrinsic factors are those that may change the shape or environment of the joint. Alignment is thought to play a mediating role in the progression of OA. Sharma et al⁴⁹ (2000) found that varus alignment of the hip, knee, and ankle (“bow-leggedness”) was significantly correlated with BMI as well as with medial compartment joint space narrowing. This indicates a higher risk of OA development or progression if malalignment and obesity are both present. Malalignment also changes load distribution in the joint. Varus alignment is associated with loading in the medial compartment of the knee and valgus alignment is associated with lateral compartment loading. Over 18 months, those with varus alignment exhibited a fourfold increase in the odds of developing medial compartment OA and similarly, those with valgus alignment exhibited the same increase in odds of developing OA in the lateral compartment⁵⁰.

Diagnosis

Knee OA can be diagnosed symptomatically and/or radiographically. There is a range of symptoms associated with OA. Most often, there is pain in the affected joint, which is felt most days of the past month². This pain can be described as an ache deep within the joint and can flare up with activity or sudden changes in weather and may be

alleviated by rest ^{26,31}. Also common is limited range of motion of the affected joint ³¹. Many will experience crepitus especially in passive range of motion, stiffness that is exaggerated in the morning, tenderness at the joint line, and joint effusion. As disease progresses, deformity may occur as a result of osteophyte formation³¹. Diagnosis of symptomatic knee OA typically requires the individual to experience pain at the knee joint most days of the last month. This joint pain is typically supported with radiographic changes to that joint ⁵¹.

Radiographic changes may occur without accompanying symptoms ¹. Radiographic knee OA is determined using the Kellegren-Lawrence (K-L) grading system. Anteroposterior radiographs are examined for osteophytes ^{1,52}, narrowed joint space indicative of cartilage loss ^{52,53}, changes in the shape of the ends of bones, and sclerosis of the bone ⁵². Severity and disease progression are determined using a 0-4 grading scale (4 being the most severe) based on the criteria as follows:

Grade 0, no evidence of joint space narrowing or osteophytes

grade 1, Doubtful joint space narrowing, possible osteophyte formation

grade 2, Osteophytes with possible joint space narrowing

grade 3, Definite joint space narrowing, multiple osteophytes, and sclerosis

grade 4, Severe Joint space narrowing, large osteophytes, severe sclerosis and
definite deformity ^{28,54}

Although the K-L scale is the most widely used and accepted method for classification of OA, it is limited. Measurement of joint space width indirectly measures

cartilage loss. This measure is also subject to interpretation. Due to inter-rater variability, there is the potential to inflate the prevalence of OA by up to 3 times the actual prevalence⁵². Radiographs are also by nature 2-dimensional pictures of a 3-dimensional joint which could lead to discrepancies in diagnosis⁵⁵.

Healthy Gait

Gait Cycle

A gait cycle follows one leg from heel strike to the subsequent heel strike with the same foot. This cycle can be broken down into two distinct phases, stance and swing. Stance refers to the time during which the foot is in contact with the ground. Stance can be further divided into single limb support and double limb support. Each stance phase begins with a period of double limb support (also termed initial double stance), followed by a period of single limb stance, and completed with a second period of double limb support (also known as terminal double limb stance)⁵⁶. Stance accounts for 60% of the total gait cycle, but is divided into approximately 10% for each double leg support and approximately 40% for single leg support (Figure 2). The swing phase accounts for the remaining 40% of the gait cycle^{56,57}.

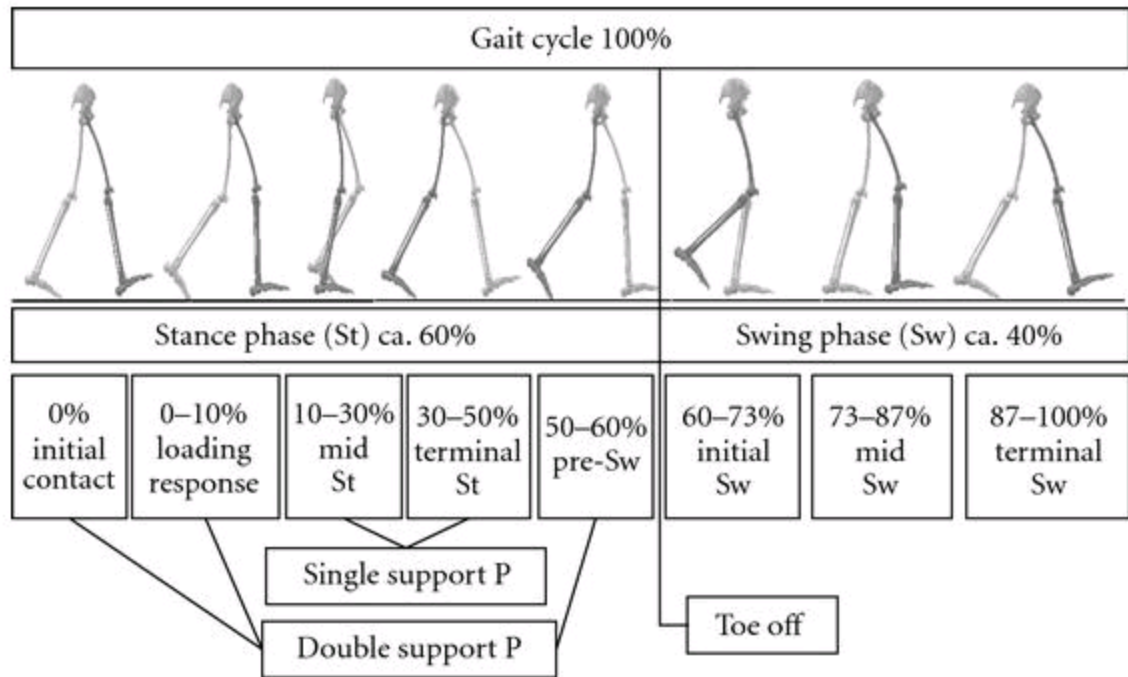


Fig 2: Phases of gait⁵⁸

The gait cycle has eight sub-phases which assist in determining overall coordination of the limbs, and assessing functional tasks associated with walking.

The first phase is weight acceptance and begins with initial contact (phase 1). During the first 2% of the gait cycle, the foot is positioned to initiate shock absorption through the heel rocker mechanism, in which the foot rotates around the calcaneus once it is planted on the ground and rolls the foot into plantar flexion with the assistance of the pretibial muscles to slow foot drop movements⁵⁶. As the limb strikes the ground and begins to accept the load, loading response (phase 2) begins, and continues through the period of double limb support. During this phase, the limb attempts to absorb shock caused by ground reaction forces, stabilize the limb to bear the weight of the body, and continue forward progression⁵⁶.

The second task completed in the gait cycle is single limb support. This includes midstance (phase 3) and terminal stance (phase 4). Both of these phases continue forward progression of the body: first over the supporting limb (midstance), and then beyond (terminal stance) until the second foot initiates heel strike⁵⁶.

The final task is limb advancement. This task encompasses the entire swing phase, and is broken down into pre-swing (phase 5), initial swing (phase 6), mid swing (phase 7) and terminal swing (phase 8). Pre-swing completes the stance phase of gait and prepares the limb for the swing phase. The next 3 phases serve to pick up the foot enough to clear the floor and then advance the limb forward in preparation for initial contact⁵⁶.

Kinematics

The gait of healthy individuals has been examined to determine normal values and can be used in comparison with diseased populations. The kinematics of walking describes movement from a spatial and temporal perspective. Kinematics includes joint positions, velocities, movement sequences, and accelerations.

Stride length is defined as the distance from one heel strike to the following heel strike with the same foot⁴. The mean stride length for healthy individuals is 1.4 meters for men and 1.3 meters for women⁵⁶. Stride length is related to limb length; a longer leg would result in a longer stride. Therefore stride length is often expressed as a ratio of stride length to limb length⁴. Stride length is used to determine walking speed, as walking speed is a function of stride length and stride frequency (cadence) through the equation: $\text{Velocity} = \text{stride length} \times 0.5(\text{cadence})$. Cadence is defined as the number of steps taken in a given amount of time. Increasing walking velocity is accomplished

through increasing stride length and/or increasing cadence⁵⁹. When asked to walk at a self-selected speed, walking velocity averages around 80 m/min, or 1.3 m/s, in normal, healthy adults⁵⁶.

Knee Range of Motion (ROM)

The full range of sagittal plane motion at the knee is approximately 70° in healthy individuals. At initial contact, the knee is flexed to 5°. At the onset of the stance phase, the limb must load the weight of the body. To do so, the knee flexes rapidly, reaching approximately 18° of flexion. In midstance, the knee extends until it reaches 3° of flexion by midway through terminal stance. The knee then flexes a second time to 7° of flexion, and then proceeds to flex rapidly as the foot prepares for the swing phase, reaching 40° of flexion at pre-swing and 60° of flexion in initial swing. The knee then extends rapidly throughout mid- and terminal swing until peak extension is reached just before initial contact. Peak extension can range from 3° of hyperextension to 5° of flexion. Knee ROM is affected by gait velocity. As an individual increases walking speed, he or she also increases maximum flexion. A typical knee ROM curve is shown in Figure 3.

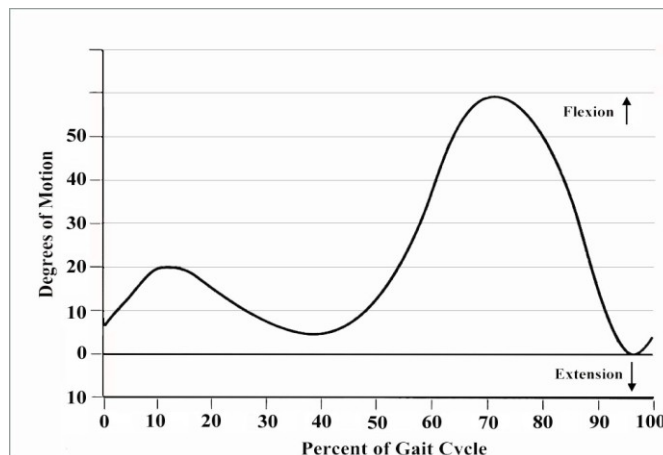


Figure 3: Knee flexion and extension during healthy gait (adapted from Perry, 1992⁵⁶)

Hip ROM

The hip normally passes through 40° of motion in the sagittal plane throughout a gait cycle. The hip begins by extending through the stance phase and flexing through the swing phase. Using 0 as a reference point of hip extension during standing, the hip reaches up to 10° of extension and 30° of flexion. When the hip initially strikes the ground, it is at 20° of flexion. Once the leg reached midstance, the hip extends until it reaches 10° of extension right before toe-off. Maximum flexion is reached during the swing phase to ensure limb advancement and toe clearance ⁵⁶. A typical hip ROM curve is shown in Figure 4.

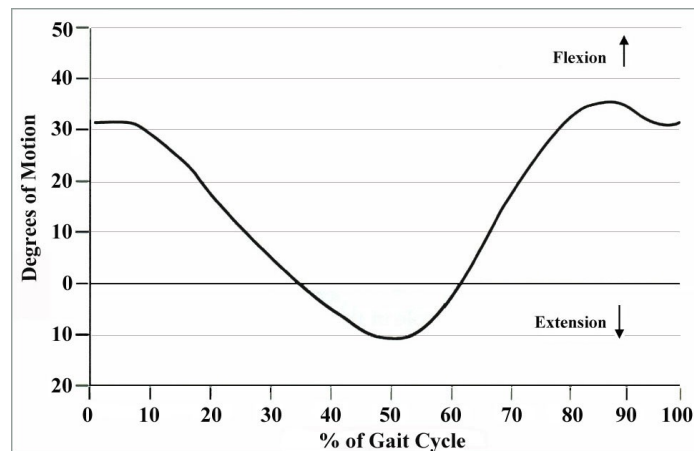


Figure 4: Hip flexion and extension during healthy gait (adapted from Perry, 1992⁵⁶)

Ankle ROM

The mean ankle ROM throughout the gait cycle is 30°. At initial contact, the ankle is in a neutral position. The first motion after heel strike is rapid plantarflexion to approximately 7°. As the body advances over the stationary foot, the ankle dorsiflexes reaching a neutral position at 20% of the gait cycle and continuing to approximately 10°

of dorsiflexion by the time the second half of terminal stance is reached. At the point of heel strike on the contralateral limb, plantar flexion begins once again, reaching maximum plantar flexion of approximately 20° at the time of toe-off. The foot returns to a neutral position for the remainder of the swing phase. A typical ankle ROM curve is shown in Figure 5.

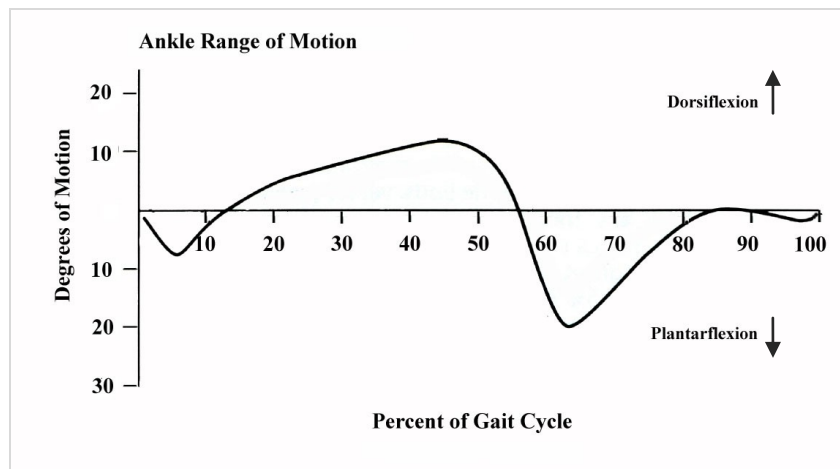


Figure 5: Ankle plantar flexion and dorsiflexion during healthy gait (adapted from Perry, 1992⁵⁶)

Kinetics

Joint kinetics describes the forces acting on a system in an attempt to define the forces that cause movement. External moments are created by the body coming in contact with the environment, thus creating a vector. In walking, the ground reaction force creates a vertical vector which creates a torque about the hip, knee, and ankle joints. To counteract this external moment, the muscles respond by creating an internal torque which is equal and opposite of the external torque⁶⁰. A net joint moment indicates the total

force acting on the joint, which includes the external torque and the counteracting internal torque, and indicates which torque is greater.

Vertical Ground Reaction Force (GRF)

The typical graph of the vertical ground reaction force presents with 2 peaks, each peak reaching a range of 1.3-1.5 x body weight and are separated by a trough (see Figure 6). The first peak (F_1) is typically reached during loading response and the second peak (F_3) is reached at push off^{56,59}. The valley (F_2) typically occurs as the body moves over the limb in stance, and the contralateral limb swing⁵⁶. The magnitude of the peaks is not statistically different⁵⁷. In normal subjects, gender seems to cause variation from the normal magnitude, but not in the shape of the graph⁵⁷. The magnitude of the vertical reaction force is affected by speed. Increased gait speed shows higher peaks⁵⁶.

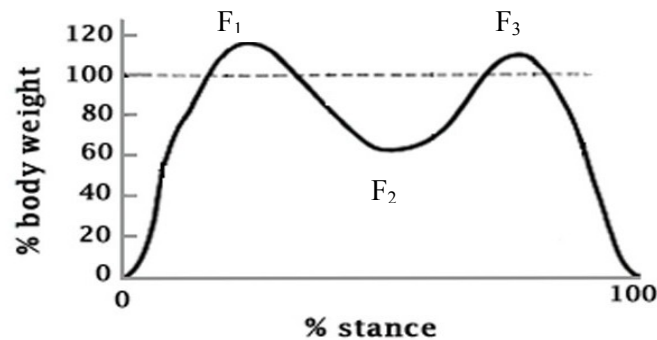


Figure 6: Vertical ground reaction force through stance phase during healthy gait⁶¹

The loading rate is the time it takes from initial contact to the first peak of the vertical ground reaction force. This loading rate may vary from men to women, with reported normal values ranging from 18-24% of stance for men and 19-27% of stance for women in two different studies^{4,57}. Increase in loading rate is associated with increased forces experienced at the knee⁶².

Joint Moments at the Knee

There are typically 4 peaks in the sagittal plane representing flexion and extension moments about the knee. At heel strike, a vertical ground reaction force vector is positioned anterior to the knee, causing an extensor torque that may reach a magnitude of up to an absolute value of 25 N*m. As the limb is loaded, the vector shifts towards the joint center and continues to move posteriorly throughout stance. A flexor torque is created at approximately 3% of the stance cycle which can reach a magnitude of 51 N*m. At the start of single limb support, the body shifts over the stance limb, gradually shifting the vector back to the knee joint center through decreasing flexion until neutral alignment is reached in midstance. The continuation of the body weight forward moves the vector anterior to the knee, creating a second extensor torque that reaches a peak of 30 N*m. At the onset of the second double limb support, neutral alignment is restored. A flexor torque is created in pre-swing to prepare the limb for the swing phase^{56,59}. Typical joint moments about the knee can be seen in figure 7.

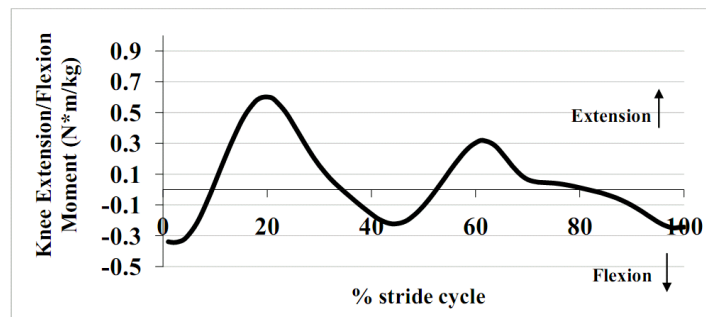


Figure 7: Knee joint flexion/extension moment during healthy gait (adapted from Perry, 1992⁵⁶)

Joint Moments at the Hip

During loading response, there is an immediate peak hip flexor torque due to the large GRF vector and the distance of the vector anterior to the center of the hip. This peak reaches 35 N*m. As body weight advances forward, the vector rapidly becomes aligned with the hip joint center. As the body weight continues to progress forwards, the vertical GRF vector moves posterior to the hip joint and an extensor torque is generated by the end of loading response (7% of gait cycle). Peak extensor torque is reached at the onset of the second double limb support phase. As the leg then prepares for swing, the hip is returned to neutral alignment through decreasing the extensor torque^{56,59}. Typical joint moments about the ankle can be seen in figure 8.

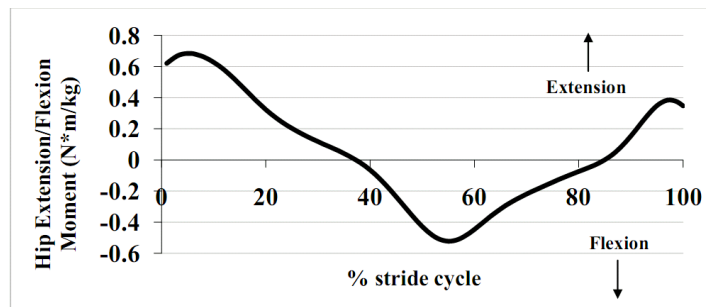


Figure 8: Hip joint flexion/extension moment during normal gait (adapted from Perry, 1992⁵⁶)

Joint Moments at the Ankle

At initial contact, a small plantar flexor torque is created as the VGRF vector passes anterior to the joint center. The peak plantar flexor torque occurs in the initial 2% of the gait cycle. As the body weight shifts forward, neutral alignment with a net torque of zero is reached at 5% of the gait cycle. A dorsiflexor torque is then generated

throughout the remainder of the stance phase. Peak dorsiflexor torque is reached in late terminal stance^{56,59}. Typical joint moments about the ankle can be seen in figure 9.

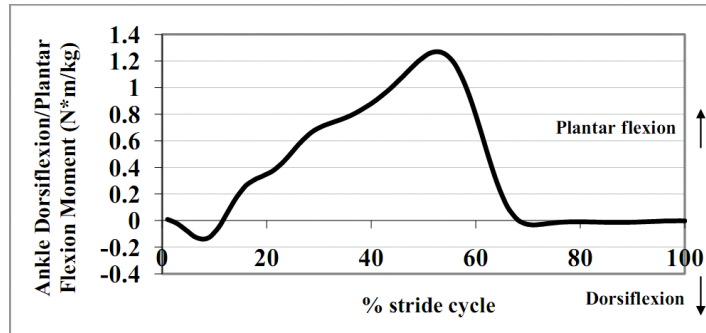


Figure 9: Ankle joint plantar flexion/dorsiflexion moment during healthy gait (adapted from Perry⁵⁶)

Power

Power can be defined as the force applied to an object and the distance that object moves (work) per unit of time. The faster an object moves, the more powerful that individual moving that object is⁵⁶. In gait analysis, power increases with increasing walking velocity. An increase in power is associated with more forceful muscle contractions about a joint⁵⁹. When looking at human movement, positive power results when there is a concentric muscle contraction that results in movement in the same direction of the contraction. For example, positive work about the knee will result if the quadriceps contract concentrically and the resultant movement is knee extension⁶³. This is also described as power generation. Conversely, when there is negative power when the muscle is eccentrically contracting, it is called power absorption^{59,63}. Power can be measured about each joint throughout the entire gait cycle. Typical peak power generation reported for healthy elderly subjects for the knee, hip, and ankle are 0.037

J/kg, 0.098 J/kg, and 0.0029 J/kg⁶⁴. At the ankle, the plantar flexors are the major power generating muscle. When the ankle dorsiflexes, the plantar flexors are absorbing power. When the foot plantar flexes at pushoff, the plantar flexors are generating power. At the knee, the quadriceps are active in power absorption when the knee is flexed and the quadriceps are eccentrically contracting. When the knee extends, which happens at midstance and during the second half the swing phase, the quadriceps are generating power to advance the limb forward⁶⁵. Throughout much of the stance phase, the quadriceps are predominately contracting eccentrically, resulting in power absorption at the knee. Prior to heel strike, the hamstrings contract concentrically to prepare the limb for impact and flex the knee, also resulting in power production at the knee. At the hip, the hip extensor muscles are actively generating power as the hip is being extended to bring the body over the limb. At terminal stance, hip extension is slowed, followed by the contraction of the hip flexors to generate power throughout the swing phase⁵⁹. Mean joint powers for healthy individuals at the hip, knee, and ankle are presented in figure 10. Power is graphed as a product of the knee moment and angular velocity and is expressed in Watts (W)⁶⁵. Power is affected by walking velocity. As walking velocity increases, the power that must be generated and absorbed by the muscles around each joint also increases at each peak⁶⁵.

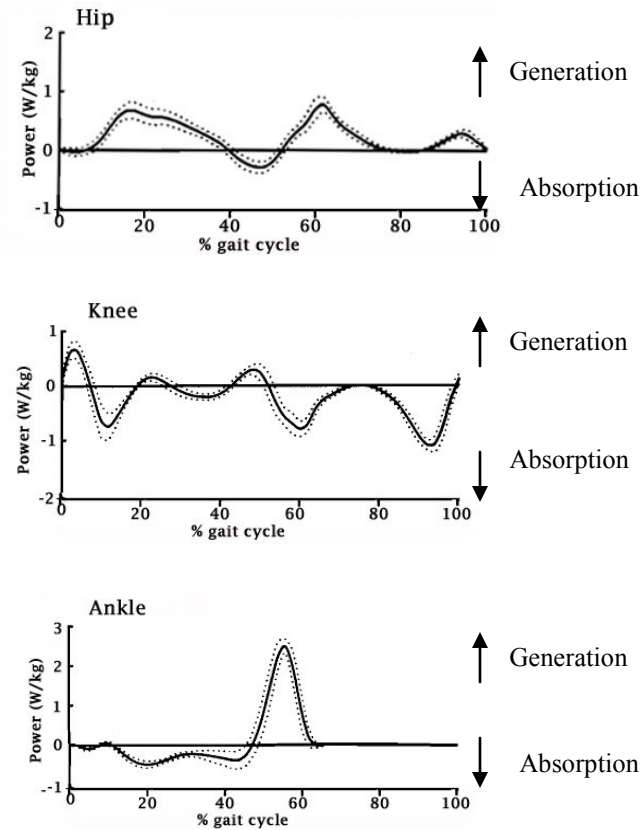


Figure 10: Power output at the hip, knee, and ankle during healthy gait⁶⁴.

Muscle Modeling

Direct measurement of muscle forces would offer the most accurate assessment of muscle forces and subsequent calculation of forces acting on the joint. In vivo measurements have been conducted, but have been limited to superficial tendons, such as the Achilles tendon. Komi et al⁶⁶ (1987) implanted a buckle force transducer on the Achilles tendon to measure forces across the tendon during running, walking, stretching, and jumping. Due to the impracticality and invasiveness of in vivo measurement of muscle forces, biomechanical models have been developed to estimate muscle forces, joint forces, and joint loads non-invasively. The most common models used are forward

dynamics and inverse dynamics. While both models generate similar resultant muscle force, they come to these results by different means ⁶⁷.

Forward dynamics, also called dynamic optimization ⁶⁷, calculates movements and external forces based on known internal forces. This method is used when muscle forces and joint torques are available to calculate the corresponding body movements. First, a neural command is measured using an EMG or a mathematical model. This neural command is then transformed into muscle forces using values known for the musculotendon length, shortening velocity, and muscular activation. These muscle forces are then used to calculate corresponding body movements through equations of skeletal dynamics. Forward dynamics solves one optimization problem for each complete cycle of movement, which is one of the biggest differences between the two methods. These calculations make forward dynamics computationally expensive and involve complex and nonlinear relationships ⁶⁸. Other limitations include variability in tendons and muscles between individuals. This makes it difficult to calculate the exact moment arms of the muscles and tendons and limits accuracy in the estimation of force production of each individual muscle. In addition, converting from muscle activation to muscle forces is difficult to measure and is not entirely understood. ^{67,68}.

The second model is inverse dynamics, or static optimization ⁶⁷ in which external moments are used to calculate internal forces. In this “torque-driven” model, a different optimization problem is solved at each instant during the movement. Inverse dynamics used position, acceleration, and velocity data of each segment gathered through a gait analysis as well as external forces obtained from a force plate are applied to linear and

angular Newtonian equations to calculate joint torques and resulting muscle forces^{67,69} using the general equation:

$$\text{TMT} = \text{M}(\dot{q}) \ddot{q} + \text{G}(q) + \text{E}$$

where TMT are the muscle joint torques, q and $\dot{q}$ are vectors of the generalized coordinates, velocities and accelerations respectively, $\text{G}(q)$ is the gravitational loading, and E is the external force. Once joint torques are calculated, the individual muscle forces responding to the joint torque can be calculated. There are some limitations accompanying the use of inverse dynamics. First, the validity of the calculated joint torque depends on the accuracy of the kinematic data gathered through the gait analysis. If there were errors in collecting the positions, velocities, or accelerations of the body segments, calculated values of the net joint torques and the subsequent muscle force calculations are also subject to error. It is also difficult to include muscle physiology into an inverse dynamic model due to estimates of muscle length and contraction velocity also being dependent on the accuracy of the positions and velocities of the body segments⁶⁷.

DeVita and Hortobagyi⁷⁰ (2001) developed a model that used inverse dynamics to determine the forces of the quadriceps, hamstrings, and gastrocnemius, which were then used to calculate compressive and anteroposterior (AP) shear forces at the knee. This model has been compared and validated against other inverse dynamics models and has been found to produce similar results⁷¹. A primary limitation of this model is that it is a lumped muscle model. Muscles are grouped as quadriceps, hamstrings, and gastrocnemius, and does not account for individual muscle units⁷¹. It also does not account for co-contraction of the quadriceps, specifically the rectus femoris⁷², at the hip

during initial stance. While the quadriceps activity is small, this may result in an underestimation of calculated muscle forces of the hamstring and quadriceps and subsequent calculation in knee forces which can reach an error of 5% ⁷¹. Co-contraction is accounted for at the knee and ankle joints. Additionally, most knee ligaments are absent from the model, ligaments that may contribute to forces at the knee⁷¹. The lateral collateral ligament is the only one accounted for. While this model has been used in healthy⁷⁰ and OA¹⁸ populations, the cross sectional area of the muscles used in this model is based on healthy participants. Those with OA may have altered muscle cross sectional area and using numbers based on healthy participants may lead to error in force calculation.

Osteoarthritic Gait

It is well established that there are differences in gait characteristics when subjects with OA are compared to normal or healthy individuals. There are different theories as to why compensatory gait mechanics are used among those with OA. One theory suggests those with OA work to provide stability for an unstable knee joint even when increasing stability at times may decrease mechanical efficiency ¹⁵. Gyory et al⁴ (1976) found an increase in stride length in subjects with medial knee instability. Other mechanisms include increased knee extension to the point of hyperextension at the knee to counteract weak quadriceps, increasing step width, and increasing double support time ¹⁸.

The most commonly accepted theory is that those with OA alter gait mechanics to reduce pain at the knee joint. Hurwitz et al⁵ (2000) used pharmaceuticals to relieve pain in osteoarthritic knee joints. This masked the protective mechanism pain has on a

diseased joint. When pain was reduced, the gait of persons with knee OA was similar to the pain free population. The main effect of pain relief was an increase in walking velocity which, in turn, resulted in larger moments at the knee. Henriksen et al⁷ (2006) conducted a similar study, relieving pain through the use of lidocaine injections. Although there was an overall decrease in compressive forces at the knee, the authors concluded that a decrease in pain resulted in an increase in joint loads, potentially contributing to disease progression.

Without pharmaceutical pain relief, those with knee OA attempt to reduce pain by mechanical means. This includes reducing dynamic joint loading and compressive forces at the knee through reduced extensor moment ⁹, decreased range of knee flexion ⁸, decreased external flexion moments, and decreased knee adduction moment ⁵. Mechanical compensations are often manifested in changes in the kinematics of walking. OA patients tend to walk with altered range of motion at the knee which then may be compensated for in the hip and ankle. Other alterations include changes in gait phases, walking velocity, and stride length.

Gait Cycle

The percent of the gait cycle spent in the stance phase is significantly increased in patients with OA, with the largest difference seen in the increase in double support time ^{4,8,10,11,18}. The increase in stance time served to allow a reduction of compressive forces on both knees by reducing the load on a single knee as it is during single limb support ⁸. Increased support time was accompanied by a decreased swing time, again in an attempt to reduce the load on a single knee ¹⁸. Percent stance was affected by disease severity.

Significant correlations were found between joint space narrowing, irregularity, and cyst formation, where increases in these indicators of disease resulted in increased stance phase time ⁴.

Walking Velocity

A well-documented consequence of osteoarthritis in the knee is reduced walking speed^{8,9,11–13,18}. Stauffer et al¹¹ (1977) found that those with osteoarthritic knees walked on average 55% slower than healthy controls. Freely chosen walking speeds tended to be slower in diseased populations when compared to healthy populations. Slower walking speed was a result of longer stance times and lower cadence in the osteoarthritic groups when compared to both a young healthy group and an older healthy group, although the older control group walked considerably slower than the young control group. When walking speed was paced, those with OA walked significantly slower than a control group of healthy elders ¹². Changes in walking speed may serve to reduce forces experienced throughout the gait cycle. When asked to walk at a controlled speed of 1.0 m/s, loading rate was significantly lower in the group with moderate OA as compared to severe OA and healthy controls. Diseased subjects also reduced vertical ground reaction force ¹³.

Stride Length

Stride length (also expressed as the ratio of stride length to lower extremity length) is markedly reduced among individuals with OA^{18,19} and may be related to disease severity. As disability due to progressing disease increased, stride length was further reduced⁴. As previously suggested, stride length is related to walking velocity

indicating that a decreased walking velocity may be due to a decrease in stride length. However, when walking velocities in OA groups did not significantly differ from controls, a decreased mean stride length still remained ¹⁴.

Joint ROM

Most studies show that those with OA tend to decrease the total range of motion of the knee (expressed as maximum flexion – maximum extension)¹⁴. Dynamic knee flexion was also reduced when diseased joints were compared to controls ⁸ with reports of up to 6° less flexion of the knee in age and gender matched groups ¹⁶. Differences were also seen when comparing affected versus unaffected legs in the same sample ¹⁹. Several studies have found those with OA initiate heel strike with the knee hyperextended (see figure 11)^{15,18}. In one study, this hyperextension averaged 5.3° more extension than controls and became more distinct as disease severity increased ¹⁵. One study of unilateral OA participants found that those with affected knees landed in a flexed position compared to controls¹⁶.

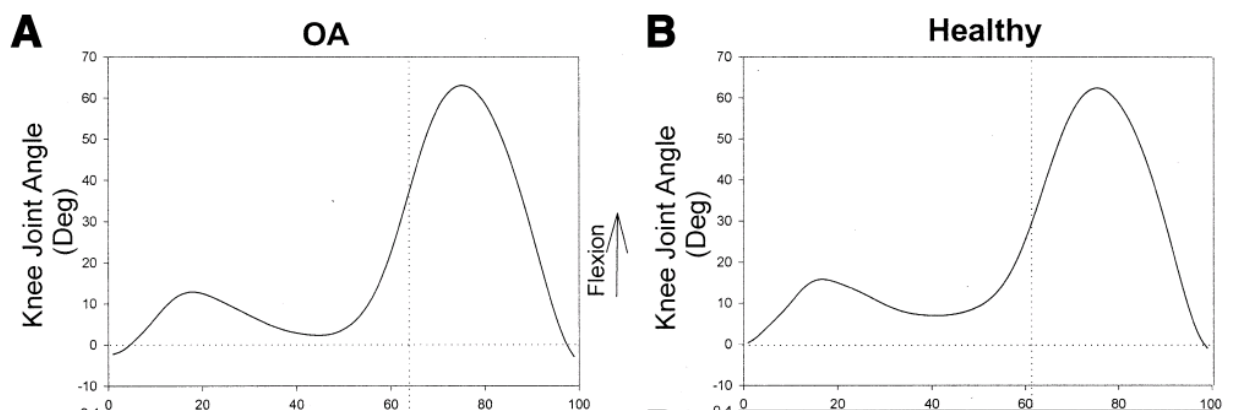


Fig 11: Knee joint ROM in those with osteoarthritis knee and healthy controls¹⁸

There are varied findings for the changes in hip and ankle range of motion during osteoarthritic gait. Some studies have found no significant differences in hip or ankle range of motion when comparing OA to healthy individuals ¹⁵. Other studies show a decrease in hip ROM during the stance phase of gait. The authors suggest that it is likely due to a decrease in stride length, making normal hip extension unnecessary ¹².

Vertical Ground Reaction Force

Changes in GRF can be used to detect abnormal loading of a joint. When compared to a normal curve, those with OA decrease the peak vertical GRF ^{4,11,13,19} and also may smooth out a normally 'M' shaped curve, so that the second peak is markedly reduced ^{8,11}. Peak vertical GRF has been reported as approximately 9% lower than normal, meaning that the force did not reach 100% of the subject's body weight ¹¹. This peak decreased further in increasing disability suggesting a relationship between disease severity and peak vertical GRF⁴. The second peak of the GRF was also reduced in the OA group compared to normal. This suggests a reluctance to push off at the end of the stance phase ^{4,11}. A larger than normal trough has also been noted in OA subjects⁸, indicating that the knee is experiencing greater than normal force during midstance¹¹. After controlling for possible effects of a decreased walking velocity among OA patients, significant differences in peak forces still remained, indicating abnormal joint loading regardless of changes in walking speed¹³.

Individuals with OA extend the loading rate, resulting in a longer time to the first peak of the vertical GRF ^{8,11}. One theory is that this elongated time is an attempt to smooth out vertical accelerations and decelerations that may produce excess stress on the

supporting knee joint ¹¹. One study, however, showed an increase in loading rate when the individuals with OA were compared to controls. Although, when the unaffected side was compared with the affect side within the OA population, the affected leg showed a reduced loading rate ¹⁹.

Joint Moments at the Knee

In both the frontal and sagittal planes there have been differences between those with OA and healthy controls. Kaufman et al⁹ (2001) found an external knee extensor moment in those with radiographic disease accompanied by pain, instead of a more normal flexion moment in stance. This was supported by the work of Baliunas et al¹⁴ (2002) who suggest that an extensor moment during the stance phase is indicative of quadriceps avoidance gait. Landry et al¹⁰ (2007) showed a decreased flexion moment in early stance, again potentially a mechanism of reducing pain associated with the use of the quadriceps. There may also be a link between magnitude of the decreased flexor moment and disease severity¹³.

The external adduction moment at the knee has been used as surrogate measure of medial compartment loading in the knee joint, as in vivo testing is not feasible in an osteoarthritic population⁵. In those with OA, the peak external adduction moment was significantly larger than control subjects^{6,10,23,73}. Landry et al ¹⁰(2007) specified between the peak adduction moment, which was not affected by OA, and the overall adduction moment, which was affected by OA. This overall increase in adduction moment over a longer period of time would result in greater cumulative loading at the knee joint. Peak adduction moment was affected by speed, further explaining decreased walking velocity

in this population. The magnitude of the external adduction moment is associated with disease severity. Sharma et al¹⁷ (1998) found a correlation between joint space width, K/L grade, and magnitude of the external adduction moment. This was supported by Zeni et al¹³ (2009) who found no significant difference in knee adduction moment but did find a similar correlation. This suggests that the medial compartment accepts more of the load as disease progresses.

After adjusting for walking speed, Messier et al¹⁸ (2005) found that compressive forces were 3.7 x BW. Compressive forces at the knee may be increased through a variety of mechanisms. One theory is that it is a result of reduced knee flexion moment in combination with muscle co-activation during the loading response phase of the gait cycle. It also might be the result of reduced surface area over which the load is accepted, concentrating the compressive forces to just one area¹⁶.

Joint Moments at the Hip and Ankle

The hip extension moment has been reported as 9% greater in the OA group compared to healthy individuals. Hip flexion was also reduced⁷³. Mundermann et al¹⁵ (2005) found 18.1% larger hip flexion moments in those with radiographic and symptomatic OA, but hip extension moments were similar between groups. Ankle dorsiflexion moments were affected in the OA groups. Reduced peak dorsiflexion moment has been seen in both moderate OA⁷³ and severe OA groups¹³.

Power

Those with knee OA have markedly decreased strength when compared to the healthy population, especially in the quadriceps muscles. Measurements of quadriceps

strength in OA populations have been shown to be up to 55% of normal subjects^{8,19,74}, and those with more advanced disease have weaker muscles than those with earlier stages of OA⁷⁴. This decrease in muscle strength may manifest in less power production, much lower than that of the normal population⁷⁵. McGibbon and Krebs found that participants with unilateral knee OA had decreased positive peak power at the ankle and loss of a second peak of power generation at the knee when walking at a preferred speed. Hip power was similar to the healthy population¹². This decrease in power also means a decrease in mechanical work⁶⁴.

Unilateral Compensation

One compensation unique to unilateral OA is the development of an antalgic limp. The goal is to use the unaffected knee to reduce the load on the affected knee. The individual will shorten the single limb support phase on the affected side⁸ and shift his or her body weight to the unaffected limb¹⁸. This limp may cause the contralateral limb to experience excess stress, potentially leading to the development of OA in that limb as well¹².

Comparison of Bilateral OA to Unilateral OA

Few studies have reported both unilateral and bilateral gait mechanics. Stauffer et al⁸ (1976) compared several gait parameters between normal healthy individuals and diseased individuals. A diseased joint could refer to either OA or rheumatoid arthritis. Results were further dissected to examine unilateral and bilateral disease, as well as potential differences between genders. The results indicated no significant difference in walking velocity or sagittal plane motion between unilaterally and bilaterally affected

knees. There was a significant difference in stance time between unilateral and bilateral diseased joints where individuals with unilaterally affected knees walked with an antalgic limp. Bilaterally affected knees increased total double support during stance in order to reduce the load on both knees. In all analyses it is not specified if the diseased knees are affected with OA or rheumatoid arthritis. Segal et al²² (2009) assessed functional limitations, power, and isometric lower limb strength in those with radiographic OA and found that there were no significant differences in those with unilateral or bilateral OA although there were small differences in muscular strength and physical function, where those with bilateral OA had reduced muscle strength and physical function.

Studies have compared affected and unaffected limbs in unilateral populations. The affected limb typically is defined as a limb with radiographic changes indicative of knee OA with accompanying pain. Hunt et al²³ (2006) found that the magnitude of GRF was greater in the unaffected limb compared to the affected limb. This measurement changed depending on time of the measurement. In midstance, there was no difference in GRF between limbs. Knee adduction moment was greater in the affected limb. Differences here were attributed to moment arm differences. Briem and Snyder-Mackler²⁴ (2009) also looked at asymmetries in the unilateral OA population. Asymmetries were found in the peak knee flexion angle, peak hip flexion angle, and peak hip adduction moment. In all comparisons, the involved limb was slightly less than the uninvolved limb. In both previous studies, there was no bilateral group to compare to. There was also limited generalizability to older OA populations as these populations were younger and more active than those typically affected by OA.

More recently, Creaby et al²⁵ (2012) compared knee adduction moment, knee flexion moment, knee flexion angle, toe-out angle, varus knee angle, and gait symmetry between those with unilateral and bilateral knee OA. Subjects were grouped by both pain and x-ray status so that there were 4 groups: bilateral pain with bilateral radiographic OA (BB), unilateral pain with bilateral radiographic OA (UB), unilateral pain with unilateral radiographic OA (UU) and healthy controls (C). Asymmetries were found for peak knee flexion angle in those with UB, indicating less knee flexion in the more painful knee. In addition, asymmetries were found in knee flexion moment, showing decreased flexion moment in the more painful limb in the UU and UB groups.

Symmetry Index

One way to determine abnormalities in gait patterns is through the use of the symmetry index. This is a comparison of a variable between limbs of the same individual. Gait asymmetry is calculated using the equation:

$$SI = \frac{x_a - x_u}{1.5(x_a + x_u)} < 0.0$$

where x_a = affected leg and x_u = unaffected leg. Perfect symmetry is indicated when $SI = 0$ ^{76,77}. Acceptable symmetry is indicated by an $SI \leq 10\%$, meaning an $SI > 10\%$ would be classified as asymmetric⁷⁸. A negative SI indicates a higher magnitude in the unaffected leg whereas a positive SI indicates a greater magnitude in the affected leg. In young healthy adults, gait has been shown to be symmetrical. In a study of men and women with the average age of 23 years, Burnett et al⁷⁹ found that there were no significant differences between the dominant and non-dominant lower limb when comparing VGRF,

stance time, and muscle activity. The only asymmetries reported were in rectus femoris and hamstring activity. Teichtahl et al⁸⁰ (2009) found symmetry in knee adduction moments throughout stance between dominant and non-dominant limbs in healthy subjects.

Gait symmetry has been assessed in patients who have undergone surgery to one limb. James et al⁸¹ (1994) assessed limb symmetry in persons with unilateral hip OA scheduled to undergo unilateral total hip replacement. Preoperatively, participants showed the greatest asymmetry in loading and unloading rate of the VGRF when compared to post-surgery at 2 month, 6 month, and 5 year follow ups. McCrory et al⁷⁷ (2001) found that those who had undergone unilateral hip arthroplasty but were pain free at the time of the study exhibited higher magnitude of VGRF variables on the unaffected side. These variables included first peak force, time to first peak force, time to second peak force, loading rate, push off rate, impulse, and stance time.

When investigating symmetry, specifically in those with knee OA, there is conflicting literature. In those with asymptomatic knee OA, no significant asymmetry was found in any loading rate, push off rate, step frequency, or forces of ground reaction forces during level walking⁸². Creaby et al²⁵ (2012) found symmetry in some measures of gait regardless of unilateral or bilateral OA status including knee adduction moment, but symmetry depended on unilateral or bilateral OA in others. Asymmetries were found in knee flexion moment and varus/valgus angle. Bilateral OA, which was defined as radiographic bilateral OA and bilateral pain, was symmetrical in all moment and angle measures, similar to the healthy control population. Bilateral radiographic OA

accompanied by unilateral pain as well as unilateral radiographic OA accompanied by unilateral pain varied in symmetry among measures.

Populations in which differences between unilateral and bilateral health status affect gait

Although few comparisons have been made between those with unilateral and bilateral knee OA, research has been conducted on other populations in which unilateral and bilateral differences have different effects on gait. Looking into other populations in which unilateral and bilateral may have different effects on gait may shed some light into expected differences in gait characteristics of unilateral and bilateral OA.

McGibbon and Krebs¹² (2002) utilized patients that had undergone unilateral joint replacement surgery. There were no significant differences between these former bilateral patients and the unilateral patients in regards to gait speed, step length, and mechanical energy expenditures. This sample size was small (four with unilateral replacement). It also was not a truly bilateral sample.

There are marked differences in gait between those with bilateral trans-tibial amputations and the normal healthy population. Differences include shorter step length, lower cadence, and slower walking velocity in bilateral trans-tibial amputees when compared to healthy controls⁸³. Similar differences are seen in the osteoarthritic population when compared to healthy controls.

It has been suggested that those with unilateral trans-tibial amputations compensate for alterations in gait caused by the prosthetic limb. Royer and Wasilewski⁸⁴ (2004) found longer swing time, shorter stance time, and smaller push off force with the prosthetic limb when compared to the intact limb in the same individual theoretically due

to pain, instability and/or muscle weakness on the affected side. This placed a higher demand on the intact limb to compensate for pain and limited range of motion due to the prosthetic limb. The intact limb also experienced abnormal loading as shown through a peak internal knee abduction moment that was 46% greater than the prosthetic limb. This value was also compared to a reported normal peak knee adduction moment and was found to be 17% greater than normal. This abnormal loading may increase susceptibility of the intact limb to the development of OA. Similarly, when looking at a population of those who had undergone unilateral total knee arthroplasty, Milner and O'Bryan⁸⁵(2008) found a greater first peak external knee adduction moment in the non-operated knee than in the operated knee. This suggests that the non-operated knee is at a higher risk of developing OA than healthy controls.

Based on these conclusions, it is reasonable to suggest that those with unilateral radiographic and painful OA walk with an asymmetric gait pattern, causing the healthy limb to experience higher joint loads and leaving this limb more susceptible to development of radiographic OA. However, those with bilateral disease may not exhibit this same asymmetry. It is unclear if the gait of those with unilateral disease significantly differs from those with bilateral disease. Though many gait studies have been conducted in an osteoarthritic population, there has yet to be a direct comparison between those with unilateral and those with bilateral OA. Matching participants on age will allow for control of age-related decline in function and strength, gender will account for the greater prevalence in females, BMI will control for obesity related increases in forces or gait mechanics, footwear will allow the same shoe characteristic between participants, WOMAC pain and functions scores will decrease the likelihood of greater compensations

due to more pain, K/L grade to control for disease severity, and the presence of OA in the hip will help to eliminate other unknown compensations related to the hip.

METHODS

The Intensive Diet and Exercise for Arthritis (IDEA) study investigated weight loss and its effects on inflammatory biomarkers and knee joint loads in overweight and obese adults with knee OA. Weight loss was achieved through 3 different interventions: intensive dietary restriction-plus-exercise, intensive dietary restriction-only, and exercise-only. This study utilized data collected at baseline from a subset of participants including those with unilateral knee OA and a set of matched participants with bilateral disease.

Study Sample

Participants were ambulatory, community-dwelling persons who were 55 years of age or older with: 1) mild to moderate radiographic knee osteoarthritis with Kellgren and Lawrence grade 2 – 3 in one or both knees; 2) $27 \leq \text{BMI} \leq 41 \text{ kg/m}^2$; and 3) current sedentary lifestyle defined as less than 30 minutes of formal exercise per week within the past 6 months.

Exclusion criteria included: 1) significant co-morbidity that may pose a safety threat or impair the ability to participate such as symptomatic or severe coronary artery disease, severe hypertension, active cancer (excluding skin cancer), anemia, dementia, liver disease, COPD, peripheral artery disease, or inability to walk without an assistive device; 2) unwillingness or the inability to change eating or physical activity habits; 3) cannot speak or read English; 4) excess alcohol use defined as 21 drinks or more per week; 5) lives more than 50 miles from the intervention site or is planning to leave the area for 3 months or more during the 18 month intervention period; 6) condition that would prohibit an MRI such as a pacemaker, severe claustrophobia, or a defibrillator; 7)

significant cognitive impairment or depression defined as a diagnosis of dementia or a Modified Mini-Mental State exam (3MSE) score <70 or CES-D score >17 ; 8) knee OA other than tibiofemoral or previous acute knee injury; 9) failure to complete a graded exercise test or a positive test; and 10) knee surgery in the past 6 months or injection in the knee within the past month.

Those who met all eligibility criteria were randomized into one of the three intervention groups. Recruiting took place over a 37 month period (November 2006 to December 2009) via mass mailings, newspaper advertisements, and presentations at local aging service networks, senior centers, and churches. Other methods included the use of a large database of older adults who consented to be contacted for research and select local physicians who were asked to encourage those who met initial eligibility to participate.

For our subsample, unilateral subject selection was based on radiographic evidence with painful unilateral OA defined as a K/L grade of 0-1 in one knee and a K/L grade of ≥ 2 in the contralateral knee. Subjects with painful bilateral OA, defined as a K/L grade of ≥ 2 in both knees, were then matched to those with unilateral OA on (1) age, (2) gender, (3) K/L grade, (4) WOMAC pain score, (5) presence of OA in other joints, and (6) BMI.

Screening Visits

Data presented here was collected at baseline through a series of screening visits conducted at the Clinical Research Center at Wake Forest University, JB Snow Biomechanics Lab at Wake Forest University, and at Wake Forest University Baptist Medical Center.

Prescreening Visit (PSV)

Once an individual expressed interest in the study through contact with the recruiting office, he or she participated in a telephone interview to determine initial eligibility based on major eligibility criteria and willingness to participate. If eligibility criteria were met, a screening visit was scheduled and each participant received a medical history form and a medication form by mail for them to complete.

Screening Visit 1 (SV1)

Screening visit 1 was conducted at the Clinical Research Center at Wake Forest University. Prior to testing, an explanation of the study was given and informed consent was obtained. Assessments at SV1 included medical history and medication forms (previously mailed), height and weight for BMI calculations, and a brief physical exam which included a knee exam to identify the source of pain and exclude participants who presented with pes anserine and iliotibial band friction syndrome. The 3MSE was given and those with a score <70 were excluded.

Those determined to be eligible continued with an interview to ensure ability and willingness to comply throughout the intervention. Individuals then underwent a treadmill graded exercise test (GXT) using a modified Naughton treadmill protocol, which is explained in detail elsewhere⁸⁶. Those with positive tests, defined as: 1) ≥ 2 mm ST depression at ≤ 4 METS; 2) hypotension; or 3) complex arrhythmias, were excluded.

SV1 was concluded at Outpatient Radiology at Wake Forest University Baptist Medical Center. Eligible individuals underwent both bilateral standing and sunrise view

knee x-rays. Subjects were then scheduled for a second screening visit and given a 3 day food diary after meeting with a nutritionist.

Height, Weight, and Body Mass Index (BMI)

Height was measured with a vertically-mounted stadiometer and was recorded to the nearest 0.1 cm. Participants were instructed to stand erect with feet together without shoes. Weight was measured on a standard calibrated scale. Participants were weighed with no shoe or heavy clothing. Body mass index (BMI) was calculated as mass in kg divided by height in meters squared.

Knee X-rays

To assess disease severity and location, x-rays were taken on all participants. Bilateral weight bearing anteroposterior (AP) x-rays were used to identify tibiofemoral OA, and sunrise views were used to identify patellofemoral OA. Disease level was identified using the Kellgren and Lawrence (K/L) score, grading each knee on a scale of 0 to 4 based on the following criteria: grade 0) absence of x-ray changes, grade 1) doubtful x-ray changes, grade 2) minimal severity of x-ray changes, grade 3) moderate x-ray changes, grade 4) severe x-ray changes. X-ray changes that were considered radiographic evidence of osteoarthritis included the formation of osteophytes on the joint margins, narrowing of the joint space width, sclerosis of subchondral bone, pseudocystic areas with sclerotic walls in the subchondral bone, periarticular ossicles, and deformation of the bone ends. Radiographs were read by a single physician who was blinded to the subject's treatment group.

A single AP view of each knee was obtained with the subject standing facing the Bucky or cassette holder. The anterior wall of a positioning guide was in contact with the Bucky. The subject stood on the positioning guide with the index foot aligned with the foot angulation support and the great toe of each foot in contact with the front edge of the positioning guide. The index foot was fixed at 10 degrees of external rotation against the v-shaped support on the base of the Plexiglas frame.

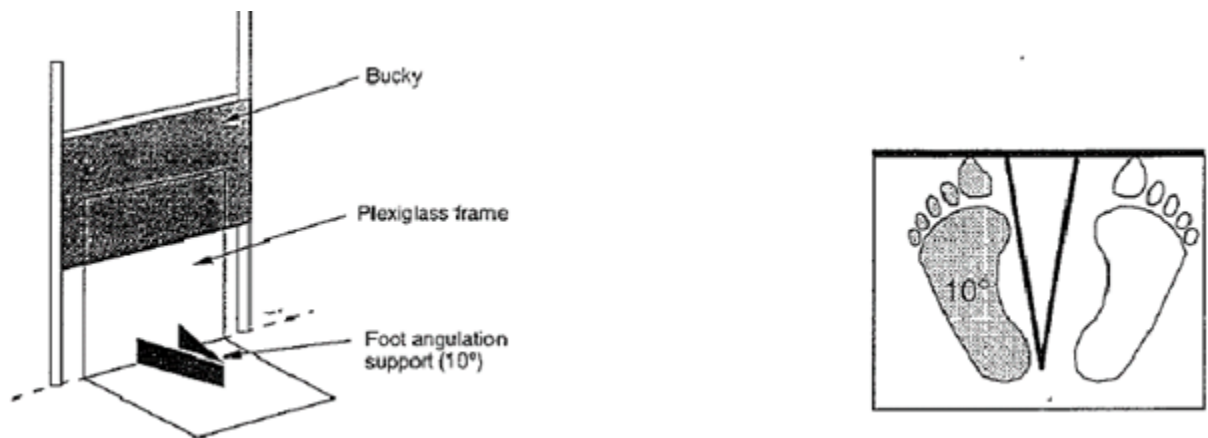


Figure 12: Plexiglass frame for foot fixation and rotation

The knee of interest was centered on the film. The subject stood with body weight evenly distributed between both legs and with both knees flexed to approximately 15° (until the knees and the anterior thigh contacted the Bucky). The focus-to-film distance was 100 cm and was constant throughout the study. The x-ray beam was centered on the joint space of the knee of interest posteriorly and was angulated caudally to 10°.

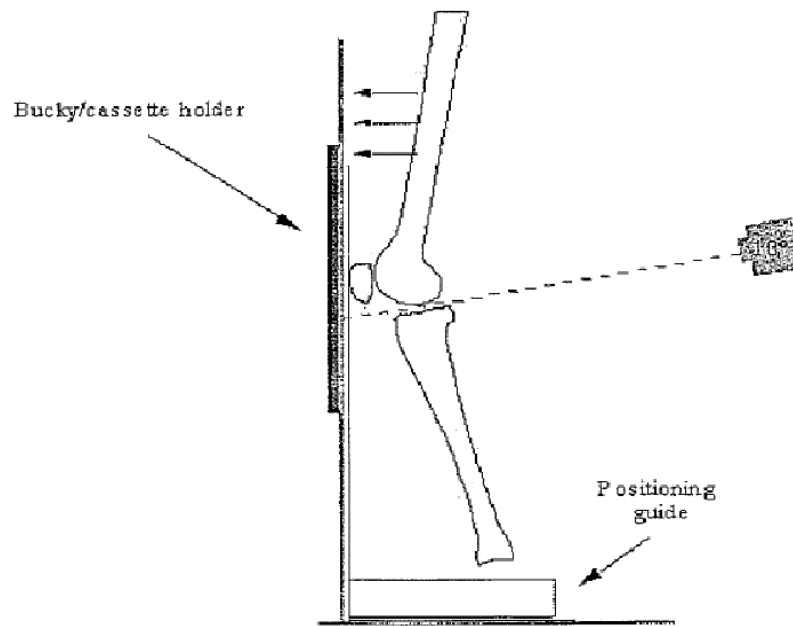


Figure 13: Proper patient positioning for A-P x-ray of the knee

Each knee met the following criteria for image evaluation: 1) each knee was radiographed separately on a film, 2) the joint spaces of the knee were radiographically open, 3) the long axis of the tibia was vertical, 4) there was no rotation of the knee, 5) all joints including the osteophytes appeared on the film, 6) left or right markers were on each film, 7) film was not over or under exposed, 8) the metallic beads on the center of the positioning guide were visualized on the radiograph.

Screening Visit 2 (SV2)

SV2 took place at Reynolds Gymnasium at Wake Forest University. Assessments included height and weight, demographic information, a variety of questionnaires including the Western Ontario McMasters Universities Osteoarthritis Index (WOMAC), and gait analysis. Other questionnaires and function measures were conducted at the SV2 but were not used in this analysis. Descriptions of tests are recorded elsewhere ⁸⁷.

WOMAC

The LK version of the WOMAC was used to determine function and pain due to knee OA. Subjects were asked to indicate the degree of difficulty experienced during various activities in the last 48 hours due to knee OA based on a scale of 0 (none) to 4 (extreme). This function subscale was made up of 17 items. Individual scores for each question were totaled to a summary score ranging from 0-68, with higher scores indicating poorer function. Participants were then asked to use the same scale to indicate levels of pain during different activities. This subscale consisted of 5 items and could generate a summary score ranging from 0-20, with higher scores indicating more pain.

Gait Analysis

3-D temporal/spatial and kinematic gait data were collected using a 37-reflective marker set. Markers were arranged in a Cleveland Clinic full-body configuration. Data were recorded using 6 Camera Motion Analysis system set to sample data at 60 Hz. Kinematic data were collected, tracked, edited, and smoothed using EVaRT 4.4 software and a Butterworth low-pass filter set with a cut-off frequency of 6 Hz. Once processed, data were compiled using OrthoTrak 6.0 β 4 clinical gait analysis software that generated kinetic and kinematic data of the lower extremity. A 6 channel AMTI force platform (Advanced Mechanical Technologies, Inc., Newton, MA) integrated with the motion capture system allowed for simultaneous collection of kinetic data at 400 Hz. Each subject completed 6 successful trials, defined as a trial in which the participant's entire foot contacted the surface of the force platform and the participant maintained a walking speed within 3.5% of his or her freely chosen speed. Of the successful trials, 3 were

chosen for additional analysis. Smoothed data were used to inform an inverse dynamics model to calculate 3 dimensional hip, knee, and ankle moments.

Biomechanical Model of the Knee

Compressive and shear knee joint forces were calculated using the musculoskeletal model developed by DeVita and Hortobagyi⁷⁰. This model consists of two basic components. The first uses a three dimensional inverse dynamics analysis of the lower extremity to determine joint torques and reaction forces at the hip, knee, and ankle. The second component uses these calculations along with the individual subject's kinematic and anthropometric data to determine the forces of the muscles, lateral collateral ligament, and joint forces during walking. In this model, the lower extremity is modeled as a system made up of three rigid segments⁷⁰. Segmental masses, moments of inertia, and locations of the mass centers of each joint were estimated based on the mathematical model previously published by Hanavan⁸⁸ (1964), segmental masses reported by Dempster⁸⁹ (1959), and the individual's anthropometric data⁷⁰.

Analysis included applying the ground reaction forces to the foot at the center of pressure, calculating the vertical and horizontal joint reaction forces at the ankle, and calculating the joint torque at the ankle using linear and angular Newtonian equations listed below.

$$F_z = ma_z + mg - GRF_z \quad (1)$$

$$F_y = ma_y - GRF_y \quad (2)$$

$$T = I\alpha - GRF_z(d1) - GRF_y(d2) - F_z(d3) - F_y(d4) \quad (3)$$

Forces in the vertical and horizontal directions (F_z and F_y , respectively) are derived from ankle GRF, m is the mass of the foot, I is the moment of inertia of the foot, a is the linear acceleration of the foot, α is the angular acceleration of the foot, and d_1 , d_2 , d_3 , and d_4 are the lever arms between the ankle and the vertical GRF and the center of mass of the foot. Ankle joint torque (T) is expressed as a reaction torque to the calculated external torque, giving an internal moment reflective of force produced by muscles and tissues across the joint. From these equations, the torque is reversed and then applied to the leg. The same equations are used to calculate forces and torques at the knee. These knee values are then used to calculate the hip forces and torques.

The second component of the model calculated the forces of the three largest muscles in the lower extremity and the force of the lateral support structure, namely the lateral collateral ligament, which, in combination with knee joint reaction forces, allows for calculation of bone-on-bone forces at the knee. The three basic steps of this process are 1) calculate the forces of the gastrocnemius, hamstrings, and quadriceps muscles and the lateral support tissues of the knee, 2) apply these muscle forces along with the joint reaction forces to the tibia, and 3) determine knee joint forces.

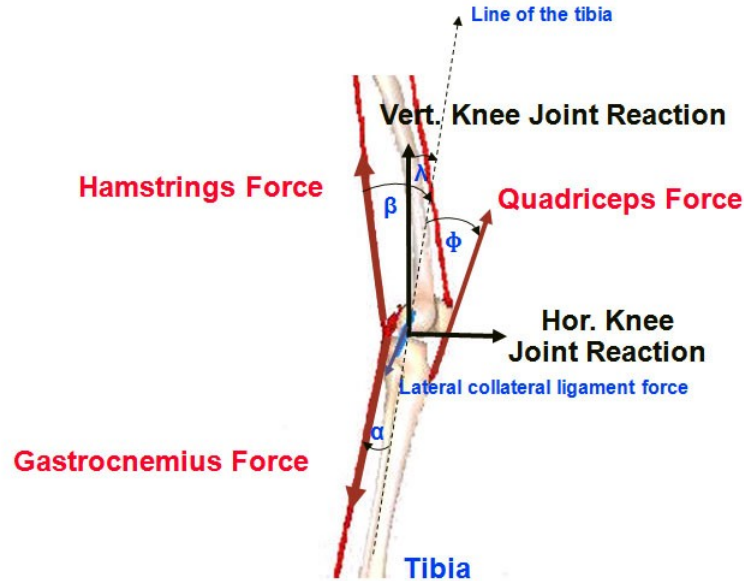


Figure 14: Schematic representation of the biomechanical model used to calculate forces at the knee ⁷¹

Following torque calculation, muscle forces are determined as the total proportion to torque produced about a joint. Gastrocnemius force was determined using the plantar flexor moment at the ankle joint. It is assumed that the plantar flexor torque is produced by the triceps surae (gastrocnemius and soleus) and that the tibialis anterior is co-active during the first 25% of stance which increases gastrocnemius force by 10% in the initial part of stance ¹⁶. The force of the triceps surae was calculated as the quotient of the ankle joint torque and the moment arm (obtained from the literature^{90,91}) for the triceps surae at the observed angular position of the ankle joint plus the 10% increase in force due to co-activation of the tibialis anterior. The gastrocnemius was then portioned out of the triceps surae force using the proportion of the total physiological cross sectional area of the tricep surae which is 0.319⁹².

The direction of the gastrocnemius force is based on the marker positions of the heel and knee marker expressed as α (the angle between the gastrocnemius force and the tibia). The heel marker represents the distal end of the muscle and the proximal end is located 0.020 m superior and 0.023 m posterior to the knee joint, along the line of the femur⁹³, a position which accounts for the gastrocnemius wrapping around the femoral condyles. Average resultant direction of the gastrocnemius force is at $\alpha=3^\circ$ from parallel with the leg, resulting in a large compressive force but small shear load on the knee.

Hamstring force is calculated from the hip extensor torque typically observed during the first half of stance, where there is a strong association between EMG hamstring activity and hip extensor torque^{93,94}. Hip extensor torque is assumed to be produced by the hamstrings and gluteus maximus with no co-contraction of the hip flexors during the first half of stance⁹⁵, however there may be some co-contraction. First, the hip extensor torque is calculated accounting for both the hamstring and gluteus maximus physiological cross-sectional area and moment arms (equation 4), then the hamstrings force is portioned out based on their proportion of the hip extensors (equation 5). Values for hamstring and gluteus maximus physiological cross-sectional area and moment arms as well as the hamstrings proportion of the hip extensors (Hp) were obtained from the literature⁹⁶ and are as follows: Ham PCA = 42.4 mm²; GM PCA = 17.39 mm²; Hd = 0.042 m; GMd = 0.047 m; and Hp = 0.63. Hamstring force is assumed to be parallel with the femur and creates angle β with the tibia.

$$H_p = [\text{Ham PCA} / (\text{Ham PCA} + \text{GM PCA})] (H_d \times \text{GMd}) \quad (4)$$

$$H = H_p (H_{et}) / H_d \quad (5)$$

Quadriceps force was calculated from the observed knee joint torque that was determined by inverse dynamics and the calculated hamstrings and gastrocnemius forces.

Knee torque (K_t) is calculated using the equation:

$$K_t = Q(Q_d) - H(H_d) - G(G_d) \quad (6)$$

where K_t is the knee torque from inverse dynamics, Q and Q_d are the quadriceps force and moment arm, respectively, H and H_d are the hamstring force and moment arm respectively, and G and G_d are the gastrocnemius force and moment arm respectively. Values for the moment arms throughout knee ROM were obtained by averaging the values from several studies^{90,92,97-99}. Those values are as follows: $Q_d = 0.035$ m, $H_d = 0.032$ m, and $G_d = 0.018$ m. This equation does account for the co-contraction of the knee flexors, as it accounts for all muscles crossing over the joint. The direction for Q is determined based on the literature and created angle ϕ with the tibia⁹².

In order to determine frontal plane loads, specifically the force of the lateral collateral ligament, the methods of Schipplein et al¹⁰⁰ (1991) were used. The external loads place an adductor torque on the knee that is resisted by an abductor torque from the quadriceps in combination with the lateral structures. During walking, the quadriceps exert a small to moderate abductor torque due to the location of the center of pressure between the tibia and femur, which is located on the medial tibial plateau, medial to the center of the patellar tendon. The moment arms for the quadriceps and lateral support structures are estimated for each individual based on x-ray film, but these distances average between 2.5 cm and 7 cm respectively. The quadriceps abductor torque is calculated as the product of the quadriceps force and its frontal plane lever arm, and is

then subtracted from the observed net internal abductor torque determined through inverse dynamics. This leaves the remaining torque distributed over the lateral support structures. Force from these structures is then calculated as the quotient of this torque and the lever arm, and is assumed to act parallel to the line of the tibia.

The final calculation is that of the knee joint forces. After all muscle forces, forces and in the lateral support structures, and joint reaction forces are obtained, they are partitioned into their compressive (K_c) and anteroposterior shear (K_s) components and summed using the equations below. Compressive components are those parallel with the tibia and AP shear are those perpendicular to the frontal plane.

$$K_c = G \sin \alpha - H \sin \beta + Q \sin \phi - K_z \sin \lambda + K_y \cos \lambda \quad (7)$$

$$K_s = G \cos \alpha - H \cos \beta + Q \cos \phi - K_z \cos \lambda + K_y \sin \lambda + L_{ss} \quad (8)$$

where K_z and K_y are the vertical and horizontal knee joint reaction forces and L_{ss} is the force exerted by the lateral support structures. A positive K_s indicates an anterior load to the tibia. A positive K_c indicates a compressive force pushing into the tibia.

Statistical Analysis

All statistical analyses were conducted using SAS software (version 19). We conducted a one-way, 2 level ANCOVA to compare the means of all variables between the unilateral and bilateral cohorts. Covariates included age, gender, K/L grade, BMI, presence of hip OA, and WOMAC pain score. Significance is reported at the level $p = 0.05$. Comparisons were made first between the most affected leg in the bilateral cohort (self-selected by the participant) to the affected leg in the unilateral population. The

second comparison was between the least affected leg in the bilateral cohort to the healthy leg of the unilateral cohort. Lastly, symmetry between sides in each population based on SI was calculated and compared between groups.

RESULTS

The purpose of this study was to compare the gait mechanics of older adults with unilateral knee OA to those with bilateral knee OA. A total of 136 participants from the IDEA cohort (N=454) were analyzed, 68 with unilateral OA and 68 with bilateral OA. Mean values and standard deviations for selected descriptive characteristics for the unilateral and bilateral cohorts as well as the total IDEA sample are summarized in Table I. Subjects were matched on age, gender, race, BMI, average walking speed, WOMAC pain score, and WOMAC function score.

Table I: Descriptive statistics (Mean $\pm$ SD) of unilateral and bilateral cohorts, and the entire IDEA population.

	Unilateral	Bilateral	All IDEA
Age (years)	64.01 $\pm$ 5.56	64.33 $\pm$ 5.29	65.58 $\pm$ 6.19
Gender (% female)	82%	82%	71.59%
Race (% white)	85%	85%	83.04%
BMI	33.26 $\pm$ 3.75	33.20 $\pm$ 3.65	33.59 $\pm$ 3.71
Weight (kg)	89.5 $\pm$ 14.4	91.7 $\pm$ 14.6	92.9 $\pm$ 14.7
Average walking speed (m/s)	1.22 $\pm$ 0.18	1.20 $\pm$ 0.21	1.20 $\pm$ 0.18
WOMAC pain	6.69 $\pm$ 3.53	6.84 $\pm$ 3.26	6.45 $\pm$ 3.11
WOMAC function	24.50 $\pm$ 12.38	25.65 $\pm$ 10.78	24.18 $\pm$ 10.85

Kinematic, temporal, and kinetic variables were compared between 1) the most affected leg in the bilateral OA group and the affected leg in the unilateral OA group, and 2) the least affected leg in the bilateral OA group to the unaffected leg in the unilateral group. Kinematic variables included total ROM at the knee, hip, and ankle, and are summarized in Table II along with temporal variables including percent of the gait cycle

in stance, percent of the gait cycle in initial double support phase, and step length. These same measures comparing the least affected leg in the bilateral cohort to the unaffected leg in the unilateral cohort are summarized in Table III. Our results showed that there were no differences in any kinematic measures between the unilateral OA and bilateral OA cohorts in either comparison.

Table II: Comparison of kinematic and temporal variables in the affected leg in the unilateral OA group and the most affected leg of the bilateral OA group*.

	Unilateral OA	Bilateral OA	P value for group significance
Range of Motion			
Hip (°)	41.5 (1.44)	41.6 (1.31)	0.915
Knee (°)	62.4 (2.10)	62.6 (1.92)	0.926
Ankle (°)	22.9 (0.98)	23.6 (0.89)	0.302
Temporal			
Step length (cm)	125.3 (3.76)	126.3 (3.44)	0.692
Support time (%)	65.7 (0.48)	65.9 (0.43)	0.557
Initial double support (%)	14.7 (0.75)	14.5 (0.69)	0.774

* Presented as mean (SE)

Data for the kinetic variables in the most affected leg are presented in Table IV. Peak moments were defined as the largest magnitude throughout the stance phase. Peak power was the point at which the power generated (positive) or absorbed (negative) at that joint was the largest. With the exception of the peak plantar flexion moment, there were no significant differences in any kinetic variables. The peak plantar flexion moment was lower in the unilateral OA group compared to the bilateral OA group, 114.70 ± 3.67 N*m/kg vs. 108.90 ± 4.02 N*m/kg ($p=0.031$).

Table III: Comparison of kinematic and temporal variables in the unaffected leg in the unilateral OA group to the least affected leg in the bilateral OA group*.

	Unilateral OA	Bilateral OA	P value for group significance
Range of Motion			
Hip (°)	41.4 (1.48)	41.2 (1.35)	0.851
Knee (°)	68.6 (1.55)	66.9 (1.42)	0.124
Ankle (°)	24.1 (0.98)	24.1 (0.90)	0.951
Temporal			
Step length (cm)	126.0 (3.86)	127.0 (3.53)	0.707
Support time (%)	67.7 (0.69)	67.9 (0.63)	0.653
Initial double support (%)	12.9 (0.65)	13.2 (0.59)	0.619

* Presented as mean (SE)

Similar results were apparent in the analysis of the kinetic variables in the least affected/unaffected leg, which are summarized in Table V. With the exception of peak plantar flexion moment and peak hip flexion moment, no kinetic variables were significantly different between groups. The bilateral cohort had a significantly larger peak hip flexion moment, $-70.86 \text{ Nm} \pm 7.11$ vs. $-57.40 \text{ Nm} \pm 7.77$ ($p = 0.014$). As with the most affected leg, those with unilateral OA had a lower peak plantar flexion moment than those with bilateral OA, where those with unilateral OA averaged $106.16 \text{ Nm} \pm 3.93$ and those with bilateral OA averaged 111.75 ± 3.59 ($p = 0.042$).

Table IV: Comparison of kinetic variables in the affected leg in the unilateral OA group to the most affected leg in the bilateral OA group*.

	Unilateral OA	Bilateral OA	P value for group significance
Joint Moments			
Hip flexion (Nm)	-58.8 (7.61)	-64.7 (6.96)	0.262
Hip extension (Nm)	77.2 (7.01)	84.1 (6.41)	0.155
Knee flexion (Nm)	-29.5 (2.59)	-30.7 (2.37)	0.484
Knee extension (Nm)	77.3 (6.70)	81.2 (6.13)	0.444
Knee abduction (Nm)	32.3 (3.47)	34.7 (3.18)	0.315
Dorsiflexion (Nm)	-11.5 (1.57)	-11.3 (1.43)	0.317
Plantar flexion (Nm)	108 (4.02)	115 (3.67)	0.039 [‡]
Ground reaction force			
VGRF (N)	1.07 (0.02)	1.06 (0.05)	0.339
Power			
Knee (W/kg)	0.55 (0.07)	0.56 (0.06)	0.804
Hip (W/kg)	1.24 (0.12)	1.38 (0.11)	0.076
Ankle (W/kg)	1.64 (0.12)	1.70 (0.11)	0.501

* Presented as mean (SE), [‡]p < 0.05

There were no differences in any knee variables. The knee abduction moment (Figure 15) did not differ significantly in either the most affected knee (p = 0.315) or in the least affected knee (p = 0.435). Maximum ROM, knee flexion/extension moment, and power are shown in the most affected leg (Figure 16, left) and in the least affected leg (Figure 16, right). Knee joint bone-on-bone forces are summarized in Table VI. There were no differences between groups in knee compressive and A-P shear forces for either the most affected leg (p = 0.528 and p = 0.481 for compressive and A-P shear forces respectively) or the least affected leg (p = 0.827 and p = 0.938 for compressive and A-P shear forces respectively).

Table V: Comparison of kinetic variables in the unaffected leg in the unilateral OA group to the least affected leg in the bilateral OA group*.

	Unilateral OA	Bilateral OA	P value for group significance
Joint Moments			
Hip flexion (Nm)	-57.4 (7.77)	-70.9 (7.11)	0.014 [‡]
Hip extension (Nm)	77.9 (6.95)	78.2 (6.36)	0.944
Knee flexion (Nm)	-27.2 (2.48)	-28.4 (2.26)	0.481
Knee extension (Nm)	42.3 (4.50)	42.1 (4.12)	0.945
Knee abduction (Nm)	32.3 (3.36)	34.0 (3.07)	0.435
Dorsiflexion (Nm)	-14.4 (1.47)	-13.5 (1.35)	0.385
Plantar flexion (Nm)	106 (3.93)	112(3.59)	0.042 [‡]
Ground reaction force			
VGRF (N)	1.09 (0.02)	1.08 (0.02)	0.582
Power			
Knee (W/kg)	0.67 (0.07)	0.60 (0.06)	0.162
Hip (W/kg)	1.29 (0.12)	1.32 (0.11)	0.713
Ankle (W/kg)	1.66 (0.12)	1.71 (0.11)	0.559

* Presented as mean (SE), [‡]p < 0.05

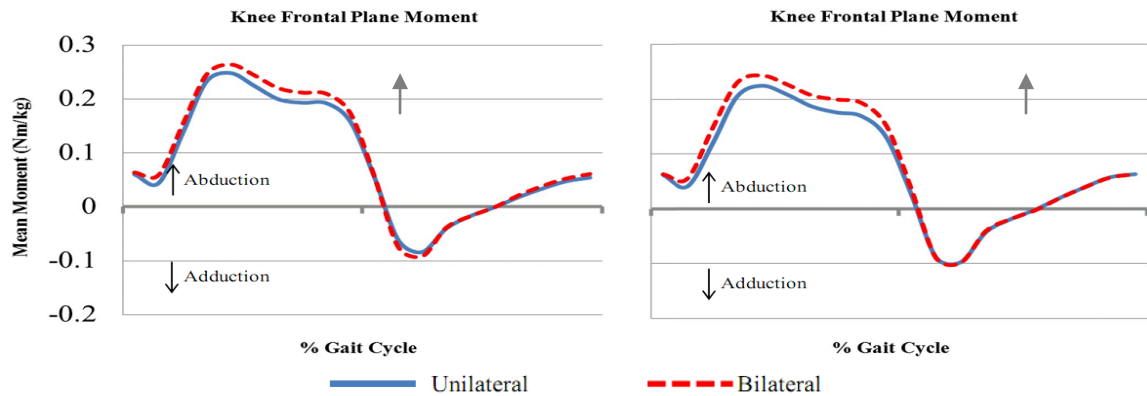


Figure 15: Knee abduction moment in the most affected leg (left in figure) and the least affected leg (right in figure)

Table VI: Loads at the knee for the affected leg in the unilateral OA group vs the most affected leg in the bilateral group and for the unaffected leg in the unilateral OA group vs the least affected leg in the bilateral OA group*.

	Unilateral OA	Bilateral OA	P value for group significance
Most Affected Leg			
Knee compressive force (N)	3002.15 (188.47)	3084.00 (172.37)	0.528
Knee A-P shear force (N)	385.50 (32.63)	369.66 (29.84)	0.481
Least Affected Leg			
Knee compressive force (N)	3044.66 (179.61)	3017.74 (164.27)	0.827
Knee A-P shear force (N)	420.57 (32.84)	422.31 (30.03)	0.938

* Presented as mean (SE)

Gait symmetry was calculated to determine side to side differences in all kinetic and kinematic variables. Mean gait symmetry for each of the groups is reported for each variable using the symmetry index (SI) and is presented in Table VII. Perfect symmetry is indicated by a score of 0 and acceptable symmetry is $\leq 10\%$ ⁷⁸. A negative SI indicates the greater value was seen in the least affected or healthy leg compared to the most affected leg. A positive SI indicates a greater value in the most affected leg when compared to the least affected or healthy leg. Our results show an SI < 10% in ~83% of the variables indicating acceptable symmetry between legs for both cohorts. The exception was knee power which reached a value indicative of asymmetry in the unilateral cohort (SI = -18.35 ± 5.06). The hip was asymmetric in peak flexion and extension moments in the side that indicated greater values, however SI values were similar.

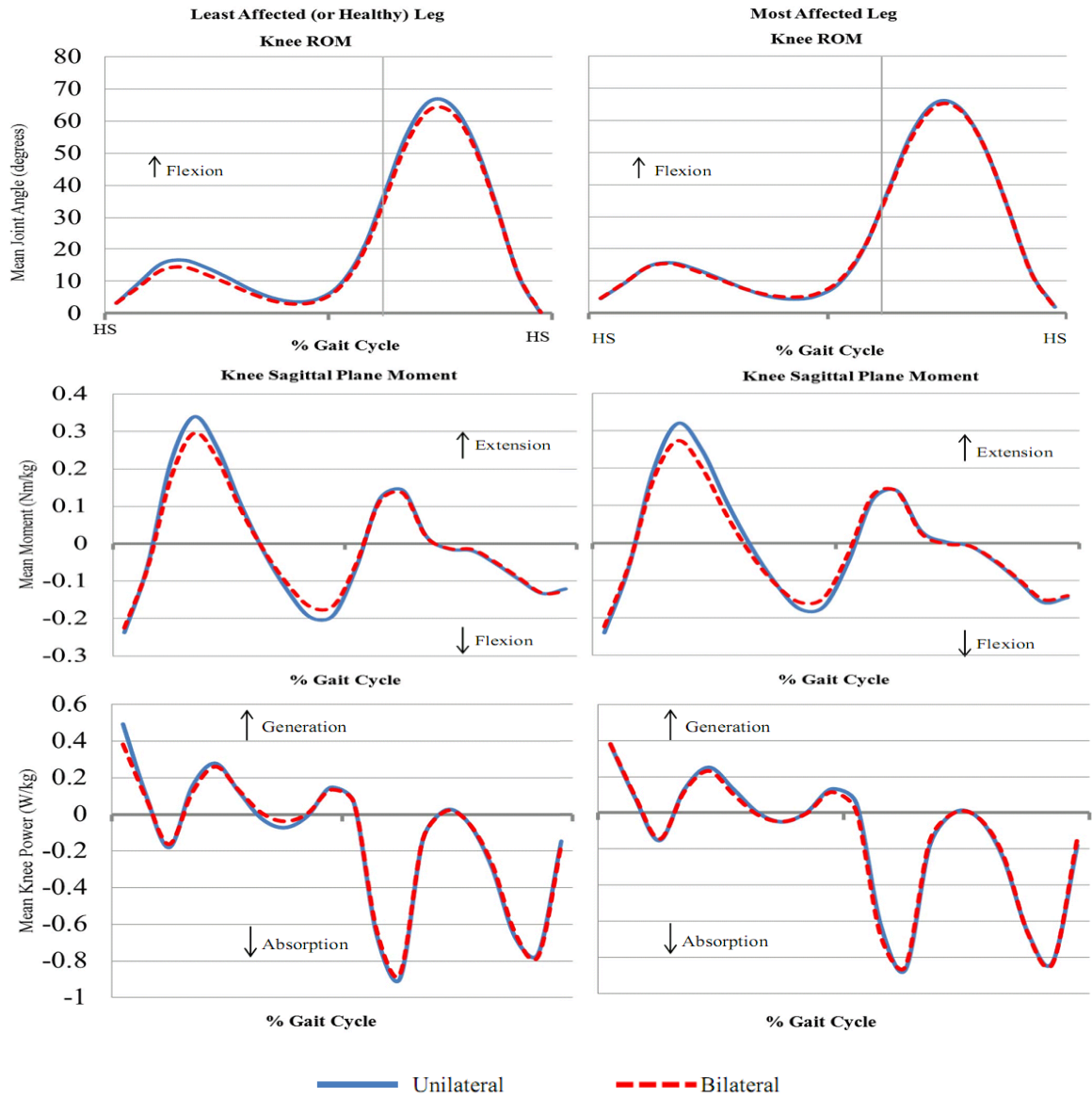


Figure 16: Knee angular position, flexion/extension moment, and power for the most affected leg (left in figure) and the least affected leg (right in figure).

Table VII: Gait symmetry in unilateral OA and bilateral OA*.

	Unilateral OA	Bilateral OA	P-value
Temporal			
Support time	-0.411 (0.27)	-0.910 (0.38)	0.291
Initial double support	1.54 (0.99)	-0.25 (1.26)	0.267
Range of Motion			
Hip	1.43 (1.52)	0.41 (1.35)	0.619
Knee	-4.27 (0.93)	-3.90 (2.36)	0.884
Ankle	-0.97 (1.68)	0.62 (1.60)	0.497
Knee Joint Forces			
Compressive force	-2.65 (2.26)	-0.13 (2.44)	0.452
AP shear force	-1.79 (3.72)	-9.49 (3.63)	0.144
Joint Moments			
Hip flexion	4.32 (3.46)	-6.48 (3.43)	0.030 [‡]
Hip extension	-4.65 (2.89)	4.8 (2.15)	0.011 [‡]
Knee flexion	-2.53 (3.69)	3.57 (3.92)	0.261
Knee extension	-5.26 (4.89)	-7.76 (5.24)	0.728
Knee abduction	-6.26 (5.48)	0.88 (5.61)	0.366
Dorsiflexion	-2.82 (4.18)	-3.42 (4.70)	0.924
Plantar flexion	-0.69 (1.06)	0.74 (1.19)	0.494
Vertical GRF	-0.687 (0.463)	-1.34 (0.515)	0.353
Power			
Knee	-18.35 (5.06)	-3.3 (4.53)	0.031 [‡]
Hip	-2.06 (3.34)	3.90 (2.57)	0.172
Ankle	-0.03 (2.36)	-1.48 (3.11)	0.712

* Presented as mean (SE), [‡]p < 0.05

DISCUSSION

It is well established that the gait of older adults with knee OA differs from the gait of the healthy population⁴⁻¹⁹. Efforts are made through gait modifications to reduce the loads on diseased knees. What was previously unknown was if there is a different effect on gait having just one versus two osteoarthritic knees. This is of critical importance to recruitment for clinical trials. In trials that include gait mechanics as outcomes, should patients with unilateral disease be included in the same cohort as bilateral diseased patients. After controlling for confounding variables including age, gender, K/L grade, BMI, race, average walking speed, WOMAC pain and function scores, and the presence of hip OA, we found little to no difference in kinetic and kinematic measures of gait mechanics between adults with unilateral and bilateral knee OA.

Kinematic analysis

When comparing the most affected leg in those with bilateral OA to the affected leg in those with unilateral OA, no differences were found in any kinematic variables. Our values were reasonable compared to previous reports of kinematic variables^{19,101}. This was not surprising as both knees analyzed were diseased. It is reasonable to assume that the same compensatory mechanics would be applied in both populations.

We hypothesized that differences would be present between the least affected leg of the bilateral OA group and the healthy leg in the unilateral OA group as this analysis included one diseased knee and one healthy knee. However, our results indicate no significant difference between groups in all kinematic variables. Based on previous

reports of gait symmetry, it was reasonable to expect differences between the two lower limbs of the same individual in the unilateral group^{24,25}. Creaby et al²⁵ (2012) found that those with unilateral symptomatic OA accompanied by bilateral structural OA (N=22) had asymmetric knee flexion, where the painful knee moved through a decreased peak flexion angle than the non-painful knee. Similarly, Briem and Snyder-Mackler²⁴ (2009) found a decreased peak knee flexion angle and decreased total knee joint excursion in the frontal plane in the involved limb of individuals with unilateral knee OA (N=32). Similar results were seen at the hip, with the hip on the side of the involved knee has significantly smaller flexion than the uninvolved knee. We found acceptable asymmetry in knee range of motion and hip range of motion in both the unilateral and bilateral diseased populations. In both of these studies, symmetry was analyzed using 95% confidence intervals. In the current study, symmetry was analyzed with a symmetry index^{76,77}.

Our results indicated acceptable symmetry ($SI < 10\%$ ⁷⁸) in all kinematic variables for both the unilateral OA group and the bilateral OA group. We also found no differences in symmetry between the unilateral and bilateral cohorts. This conflicts with the previous reports of asymmetry of knee and hip ROM in the literature. Our population had a larger sample size and may have been able to account for more variance in the variables. There were also differences in the populations of both studies. Our study was generally older and included more females. Unilateral disease is more prevalent in younger populations and may be more prevalent in younger males²⁹. Bilateral disease is more prevalent in the older population. It is possible that as those with unilateral disease may begin to develop radiographic disease in the contralateral limb, as contralateral

disease increases the odds of disease development in the healthy knee^{30,42}. This may account for differences in our results.

Temporal analysis

No differences were found when comparing the most affected limb in the bilateral OA group to the affected limb in the unilateral OA group or in comparing the least affected limb in the bilateral OA group to the healthy limb of the unilateral OA group. Our analysis of stance phase time, initial double support time, and stride length are similar to those previously reported for an osteoarthritic population^{4,11,18}. Symmetry in all tempo-spatial variables was acceptable (less than 10% difference between sides) and did not differ in unilateral vs bilateral cohorts. Particularly surprising was the absence of an antalgic limp, which is reported characteristic of unilateral disease^{8,12}. Stauffer et al⁸ (1977) found that those with unilateral knee disease decreased the time in which the affected leg was in the stance phase, resulting in a limp. Our results indicate relative symmetry in gait parameters and the unilateral cohort showed no preference to the healthy limb. Temporal gait symmetry has not been adequately discussed in the literature as a variable assessed with a symmetry index. The differences noted between OA and healthy knees might be apparent in those with unilateral disease as they have one diseased knee and one healthy knee^{8,12}. This would result in the previously mentioned antalgic limp. However, our result showed that those with unilateral disease apply similar gait mechanics to both the diseased limb and the healthy limb.

Kinetic analysis

When comparing the most affected leg in the bilateral OA group to the affected leg in the unilateral OA group, peak moments at the hip and knee, peak dorsiflexion moment, hip and ankle power, and compressive and shear forces at the knee were not significantly different. Mean peak plantar flexion moment, however was significantly less in the affected leg of the unilateral cohort compared to the most affected leg of the bilateral cohort as well as in the comparison of the healthy leg in the unilateral cohort versus the least affected leg in the bilateral cohort. Our values were similar to those previously reported in the literature^{16,73}.

Peak plantar flexion moment typically occurs during terminal stance, as it did in our population. In our population, the peak plantar flexion moment was 5% greater in the more affected knee of the bilateral OA group compared to the affected leg in the unilateral cohort. This indicates that those with bilateral OA required more muscle force from the gastrocnemius and soleus to control and advance their upper body weight over their lower limb during stance than was needed for the same task in those in the unilateral OA group⁵⁶. Comparing the least affected leg in the bilateral OA cohort to the healthy knee of the unilateral OA cohort, the bilateral group had a 5% greater peak plantar flexion moment, again indicating the need for more gastrocnemius muscle force to control upper body movement.

Generally, those with OA have reduced plantar flexion moments. Zeni and Higginson¹³ (2009) found that as disease severity increased, there was a decrease in external ankle dorsiflexion moment (which is opposed by the internal plantar flexion

moment observed in our study). One might expect that in the unilateral population, the more diseased limb would have decreased plantar flexion moments compared to the healthy limb. This would lead to asymmetries in unilateral OA ankle plantar flexion moments. However, our results indicated that there was relative symmetry in the mean plantar flexion moment for the unilateral cohort, suggesting that the magnitude of the ankle plantar flexion moment in knee OA may not be affected by disease alone as the same alteration was made in the leg with no disease.

The hip flexion moment was also significantly lower in the healthy limb of the unilateral population when compared to the least affected limb of the bilateral population. Mundermann et al¹⁵ (2005) found that those with OA walked with 18.1% higher peak hip flexion moments compared to healthy controls. In the current study, the bilateral group's least affected leg had 19% higher peak hip flexion moments than the healthy leg of the unilateral cohort. The increased hip flexion moment suggests that this compensation at the hip is only needed in the diseased limbs and not in the healthy limbs. In a previous study, Briem and Snyder-Mackler²⁴ (2009) suggested that there is more hip extension throughout the stance phase in a limb affected with knee OA compared to one that is not. Greater hip extension would require a greater internal hip flexion moment indicative of hip flexor muscle action (primarily the adductor longus and rectus femoris)⁵⁶. In our study, there were no differences in hip ROM that would have attributed to increases in hip flexion moments.

As with the plantar flexion moment, peak hip flexion moment occurred during terminal stance. Increased hip flexion moments (and therefore increased hip flexor muscle activity) in the least diseased limb of the bilateral group work to assist the plantar

flexors in controlling upper body weight in the upright, erect position as the individual prepares to shift their weight to the other leg. The hip flexors also work to stop hip extension, and then work to initiate limb advancement at the onset of the swing phase. Increased hip flexor moments may indicate that the hip flexors have to work harder to initiate limb advancement⁵⁶. It is important to note that changes in both moments between groups were small. The plantar flexion moment was 5% less in the ankle of the most affected limb of the bilateral OA group than the affected limb for the unilateral group for the ankle of both the least affected or healthy and most affected limb and 19% difference in the hip flexor moments. Considering we did not control for multiple comparisons, the significant p values were modest, with a range of 0.01 to 0.04. Finally, the number of significant differences in symmetry comparisons (n =1, n=2 for the comparison of the least affected limbs and most affected limbs, respectively) compared to the total number of comparisons made (n = 20 per side) was small, suggesting these could have been significant by chance alone.

Symmetry was acceptable (SI < 10%) in all kinetic variables in both the unilateral and bilateral cohorts with the exception of knee power in the unilateral cohort, which slightly favored the healthy limb, with a ~18% (SE = 5.06%) difference between legs. On average, those with unilateral OA favored the healthy limb in most variables, though these values did not reach significance. Again, variability between subjects in both groups was extremely large. Symmetry was expected in the diseased knees, as conceivably, those with bilateral disease would adopt bilateral gait compensations. Our data provide support for gait symmetry in bilateral OA, as reported by Creaby et al²⁵ (2012). Our results are in agreement with previous data that suggest there is no difference

in internal knee abduction moment regardless of disease status in the contralateral limb^{24,25}. However, our results are in conflict with previous reports of unequal limb loading in the sagittal plane^{12,24,25}. Our study included older adults compared to the younger populations of previous studies. Our study also did not assess pain in the contralateral limb, and pain may have been similar between limbs, resulting in similar mechanics applied to each limb.

Recently, Creaby et al²⁵ incorporated pain into a direct comparison of unilateral OA versus bilateral OA analysis for gait mechanics. They found symmetric gait in asymptomatic controls and in those with bilateral pain with bilateral radiographic OA, though OA mechanics differed significantly from the controls, indicating altered gait applied equally in both limbs. However, those with bilateral radiographic OA but unilateral pain, as well as those with unilateral pain with unilateral radiographic OA, had asymmetries between limbs. Lower knee flexion moment and lower peak flexion angle were observed only in the painful limb, supporting the notion that compensatory gait mechanics are associated with the presence of pain regardless of the level of structural disease. In the current study, pain was only assessed in the self-selected, most painful limb. Pain in the contralateral limb was not assessed. Radiographic OA and symptoms do not always progress simultaneously^{1,34,35}, making it possible that pain was equal in both limbs. Radiographic OA is not necessarily indicative of pain level, meaning a knee with a lower K/L grade may be more painful. Our study classified OA based on radiographic OA with pain present in one limb, but pain in the other limb could also affect walking mechanics.

In the previous study by Creaby et al²⁵, BMI differed between groups and those with unilateral pain and unilateral radiographic OA had a significantly higher BMI than all other groups. This difference was not explicitly controlled in the analysis. Obesity does affect joint loading and gait mechanics in the OA population^{49,73}. In examining trunk lean, they identified greater trunk lean towards the affected limb. Excess weight alone contributes to higher joint loads and having this weight favoring one limb may further increase loads in that limb. Subjects in this study also walked barefoot. Footwear does affect walking mechanics and more specifically frontal plane knee moments with OA¹⁰³. Kemp et al¹⁰² found increases in knee joint loads in those wearing shoes to those walking barefoot. Alterations may have been made in gait due to lack of footwear, and may not be representative of normal walking in this population. All participants in the current analysis walked wearing the same model of neutral walking shoe.

The total sample size of Creaby et al²⁵ (2012) was large, the individual sample sizes of the groups were fairly small, especially in the unilateral pain with unilateral structural OA (N=11). This small sample leaves open the possibility that power was not sufficient to account for large variability in measures. Our study included a much larger sample size (N=68) in this population. The current study attempted a direct comparison of gait mechanics in both the most affected and the least affected limb which included both the most affected limb in the bilateral OA group compared to the effected leg of the unilateral OA group (as defined by K/L grade as well as the presence symptoms) and the least affected limb (fewer symptoms but maintained K/L grade 2-3) compared to the healthy limb in the unilateral OA group. Our study controlled for BMI to minimize the

effect of excess body weight on the magnitude of joint moments and alterations in gait attributed to obesity regardless of disease state¹⁰⁴.

Briem and Snyder-Mackler²⁴ (2009) also investigated gait symmetry in the unilateral OA population. This study included 32 subjects with unilateral OA (K/L grade 2-3) and compared the healthy limb to the disease limb in each individual. Relevant to the current study, they found significant asymmetries in peak flexion angles in the hip and knee. This is in contrast to our findings of relative symmetry in hip and knee ROM. However, no significant asymmetry was found in the knee external adduction moments in side-to-side comparison, supporting our results indicating relative symmetry in frontal plane knee moments. The sample in this study was generally younger than the current analysis. Unilateral disease as a result of or surgery is generally more common in younger populations²⁹, however our study focused on older adults with knee OA, a population in which knee OA is more prevalent²⁹, making our results more generalizable to the population most affected by OA. The younger sample was also generally active and mostly men, which is not representative of the mostly female overall OA population. The group analyzed by Briem and Snyder-Mackler²⁴ was not compared to any other group (either a bilateral OA group or a control group), but was only compared between limbs, including one healthy and one diseased knee.

Hunt et al²³(2006) also examined gait symmetry in 100 men and women with moderate to severe knee OA. They found increased knee adduction moments in the affected limb which may have been influenced by alignment. Alignment was not investigated in the current analysis. Unlike our analysis, which found no significant asymmetry in the VGRF, there was a decreased GRF in the affected limb, which was

attributed to the magnitude of the frontal plane lever arm (the distance between the resultant GRF vector and the knee joint center of rotation). Again, the magnitude of this variable was not measured in the current analysis. Another factor that could have contributed to differences in GRF was that the participants completed the gait analysis barefoot, which as may have affected walking mechanics (as described above).

Different from the current analysis, participants in the two previous studies^{23,24} walked at self-selected walking speeds that were not matched between groups, and was not explicitly controlled for during analysis. Decreased walking velocities are associated with decreased joint moments¹⁸. When participants were walking at the same speed, this compensation could, in part, account for similar joint moments and knee joint forces at the knee. In the current study, participants walked at a self-selected speed, but this speed was matched between participants to control for the effect of decreased walking speed on gait kinetics and kinematics. The current analysis also accounted for many compensations not previously examined. To our knowledge, this was the first study to investigate symmetry in multiple variables of the hip and ankle joints as potential compensations for diseased knee joints. To our knowledge, we are also the first to examine symmetries in power variables in those with knee OA.

When comparing gait symmetry of the unilateral cohort to the bilateral cohort, symmetry differed in only 3 variables out of 20, knee power, hip flexion moment, and hip extension moment. Peak knee power production in the healthy limb was 18% greater than that of the affected knee of the unilateral OA group. In the bilateral group, this difference was much less (3% greater in the least affected leg). This resulted in a greater asymmetry in the unilateral cohort than in the bilateral cohort ($p=0.031$). Time of peak power

generation coincides with the flexion of the knee prior heel strike, Knee flexion is achieved through concentric contraction of the hamstring muscles, producing positive power through the muscle contraction⁶³. Knee ROM in the unaffected leg moved through a slightly larger range of motion than all of the other limbs analyzed, although this did not reach significance. Osteoarthritic knees tend to limit range of motion, truncating peak flexion^{9,18,24,25}. Though we did not find a significant increase in knee ROM, the slight increase could have resulted in a slightly more power production in the knee flexors (i.e. hamstrings) in the healthy limb than in the diseased limbs, as the knee could be more flexed.

Differences were also seen in the symmetry of both the hip flexion moment and the hip extension moment between the unilateral and bilateral cohorts. The magnitude of the symmetry index was similar between groups, however the difference was seen in which leg experiences a greater moment (the most affected or the least affected leg). In the unilateral cohort, a greater peak hip flexion moment was reached in the affected leg whereas in the bilateral cohort, a higher hip flexion moment was reached in the least affected leg. Conversely, greater hip extension moments were seen in the unaffected leg in the unilateral cohort but in the more affected limb in the bilateral cohort. Previous reports of sagittal plane hip moments for those with knee OA indicate that the OA population may not differ significantly from the healthy population. Zeni and Higginson¹³ (2009) found no difference in hip moments when participants with both moderate and severe knee OA walked at a self-selected walking speed and were compared to healthy controls. This may partially explain why the diseased limb had higher values in one

cohort but the least affected limb had higher values in the other. Hip flexion and extension moments may not be affected by disease state or severity.

Limitations

There have been other variables shown to affect gait that we did not control. Malalignment focuses compressive knee forces in a specific compartment of the knee. In varus alignment, load is more focused on the medial compartment^{17,50,105}. Among those with knee OA, varus alignment is common¹⁵, but an individual may not have the same alignment in both limbs. Briem and Snyder-Mackler²⁴ (2009) found that the mechanical axis of the involved limbs was significantly more varus than the uninvolved limbs. In involved limbs, there was an association between varus alignment and knee flexion that was not present in uninvolved limbs. Differences in alignment in our population may then have different effects on those with unilateral and bilateral OA. A second limitation linked to load dispersion across the knee is the inability of the knee model to determine the exact area within the knee joint (medial or lateral tibial plateau) on which the knee load is focused.

OA was defined as a K/L grade of 2-3 indicating mild to moderate knee OA. A K/L grade of 1 was not included as OA. However, there are radiographic changes associated with K/L grade 1. There is the beginning of osteophyte formation and doubtful joint space narrowing⁵². These early indicators of OA may present symptoms of OA, including pain. An individual with this K/L grade in one leg but a K/L grade of 2 or 3 in the contralateral leg would be classified as having unilateral OA. This may be a misclassification as symptoms do not always progress at the same rate as radiographic

changes. It is possible to have more pain in the knee that has been given a lower K/L grade²⁵. This may lead to compensatory gait mechanics applied to a knee that in the analysis was considered healthy.

As previously discussed, limitations in the biomechanical model may lead to an underestimation of total knee forces. Finally, there may have been errors in the placement of the reflective markers for gait analysis. Excess adipose tissue as seen in our obese participants makes it difficult to locate the correct bony landmarks for marker placement. Should the marker placement be incorrect, the joint center calculations would also be incorrect.

Future research is needed to further investigate compensations that may differ between those with unilateral OA and bilateral OA. Many studies focus on symptoms in the most affected limb. However, symptoms in the contralateral limb may affect gait mechanics. More research is needed which incorporates the symptoms in the least affected limb. Future studies might also make adjustments to the biomechanical knee model to incorporate the cross-sectional area of muscles in those with OA instead of using the cross sectional area of muscles in healthy subjects. Our analysis included large standard error of the mean, indicating a large amount of variance, particularly in the analysis of gait symmetry. One way to reduce this error is to increase sample size in future studies. A final suggestion would be to investigate the symmetry of variables that contribute to power, such as strength and muscle activity, between limbs in unilateral and bilateral OA.

Conclusions

Distinctions are not always made in the classification of unilateral or bilateral disease in those with osteoarthritic knee joints in statistical analysis. While it is known that OA gait differs from the gait of healthy, asymptomatic controls, it cannot be assumed that the gait of painful unilateral knee OA and painful bilateral knee OA patients are similar. Our results indicate few differences when comparing the gait mechanics of those with unilateral OA to those with bilateral OA with accompanying mild to moderate pain. With the exception of reduced plantar flexion moments in both the most affected leg and the least affected leg in the unilateral compared to the bilateral cohort and reduced hip flexion moment in the healthy limb of the unilateral cohort, no other variable was found to be significantly different between the cohorts. There were also acceptable levels of gait symmetry in all variables (excluding knee power in the unilateral cohort) for both the unilateral and bilateral cohorts, indicating that the unilateral cohort did not favor their less affected limb. Overall, only 6 variables were found to be significantly different in 59 comparisons. This amount of variance may be expected due to chance alone. Due to this lack of statistical differences between groups in multiple comparisons, we suggest that these two groups can be analyzed collectively as an OA population, and that results discovered in one population may be generalized to both unilateral and bilateral OA.

REFERENCES

1. 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.
2. Creamer P, Lethbridge-Cejku M, Hochberg MC. Factors associated with functional impairment in symptomatic knee osteoarthritis. *Rheumatology (Oxford)*. 2000;39(5):490–496.
3. Messier SP. Osteoarthritis of the knee and associated factors of age and obesity: effects on gait. *Med Sci Sports Exerc.* 1994;26(12):1446–1452.
4. Györy AN, Chao EY, Stauffer RN. Functional evaluation of normal and pathologic knees during gait. *Arch Phys Med Rehabil.* 1976;57(12):571–577.
5. Hurwitz DE, Ryals AR, Block JA, et al. Knee pain and joint loading in subjects with osteoarthritis of the knee. *J. Orthop. Res.* 2000;18(4):572–579.
6. Hurwitz DE, Ryals AB, Case JP, Block JA, Andriacchi TP. The knee adduction moment during gait in subjects with knee osteoarthritis is more closely correlated with static alignment than radiographic disease severity, toe out angle and pain. *J. Orthop. Res.* 2002;20(1):101–107.
7. Henriksen M, Simonsen EB, Alkjaer T, et al. Increased joint loads during walking--a consequence of pain relief in knee osteoarthritis. *Knee.* 2006;13(6):445–450.
8. Stauffer RN, Chao EY, Györy AN. Biomechanical gait analysis of the diseased knee joint. *Clin. Orthop. Relat. Res.* 1977;(126):246–255.
9. Kaufman KR, Hughes C, Morrey BF, Morrey M, An K-N. Gait characteristics of patients with knee osteoarthritis. *Journal of Biomechanics.* 2001;34(7):907–915.
10. Landry SC, McKean KA, Hubley-Kozey CL, Stanish WD, Deluzio KJ. Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed. *J Biomech.* 2007;40(8):1754–1761.
11. Chen CPC, Chen MJL, Pei Y-C, et al. Sagittal plane loading response during gait in different age groups and in people with knee osteoarthritis. *Am J Phys Med Rehabil.* 2003;82(4):307–312.
12. McGibbon CA, Krebs DE. Compensatory gait mechanics in patients with unilateral knee arthritis. *J. Rheumatol.* 2002;29(11):2410–2419.
13. Zeni JA Jr, Higginson JS. Differences in gait parameters between healthy subjects and persons with moderate and severe knee osteoarthritis: a result of altered walking speed? *Clin Biomech (Bristol, Avon)*. 2009;24(4):372–378.

14. Baliunas AJ, Hurwitz DE, Ryals AB, et al. Increased knee joint loads during walking are present in subjects with knee osteoarthritis. *Osteoarthr. Cartil.* 2002;10(7):573–579.
15. 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):2835–2844.
16. Childs JD, Sparto PJ, Fitzgerald GK, Bizzini M, Irrgang JJ. Alterations in lower extremity movement and muscle activation patterns in individuals with knee osteoarthritis. *Clin Biomech (Bristol, Avon).* 2004;19(1):44–49.
17. Sharma L, Hurwitz DE, Thonar EJ, et al. Knee adduction moment, serum hyaluronan level, and disease severity in medial tibiofemoral osteoarthritis. *Arthritis Rheum.* 1998;41(7):1233–1240.
18. Messier S, Devita P, Cowan R, et al. Do older adults with knee osteoarthritis place greater loads on the knee during gait? A preliminary study. *Archives of Physical Medicine and Rehabilitation.* 2005;86:703–709.
19. Messier SP, Loeser RF, Hoover JL, Semble EL, Wise CM. Osteoarthritis of the knee: effects on gait, strength, and flexibility. *Arch Phys Med Rehabil.* 1992;73(1):29–36.
20. Forestier R, Francon A, Briole V, et al. Prevalence of generalized osteoarthritis in a population with knee osteoarthritis. *Joint Bone Spine.* 2011;78(3):275–278.
21. Günther KP, Stürmer T, Sauerland S, et al. Prevalence of generalised osteoarthritis in patients with advanced hip and knee osteoarthritis: the Ulm Osteoarthritis Study. *Ann. Rheum. Dis.* 1998;57(12):717–723.
22. Segal NA, Yack HJ, Brubaker M, Torner JC, Wallace R. Association of Dynamic Joint Power With Functional Limitations in Older Adults With Symptomatic Knee Osteoarthritis. *Archives of Physical Medicine and Rehabilitation.* 2009;90(11):1821–1828.
23. Hunt MA, Birmingham TB, Giffin JR, Jenkyn TR. Associations among knee adduction moment, frontal plane ground reaction force, and lever arm during walking in patients with knee osteoarthritis. *Journal of Biomechanics.* 2006;39(12):2213–2220.
24. Briem K, Snyder-Mackler L. Proximal gait adaptations in medial knee OA. *J. Orthop. Res.* 2009;27(1):78–83.
25. Creaby MW, Bennell KL, Hunt MA. Gait Differs Between Unilateral and Bilateral Knee Osteoarthritis. *Archives of Physical Medicine and Rehabilitation.* 2012. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/22385873>. Accessed April 5, 2012.
26. Glass GG. Osteoarthritis. *Dis Mon.* 2006;52(9):343–362.

27. Theis KA, Helmick CG, Hootman JM. Arthritis burden and impact are greater among U.S. women than men: intervention opportunities. *J Womens Health (Larchmt)*. 2007;16(4):441–453.
28. McAlindon TE, Wilson PW, Aliabadi P, Weissman B, Felson DT. Level of physical activity and the risk of radiographic and symptomatic knee osteoarthritis in the elderly: the Framingham study. *Am. J. Med.* 1999;106(2):151–157.
29. Lethbridge-Cejku M, Tobin JD, Scott WW Jr, et al. The relationship of age and gender to prevalence and pattern of radiographic changes of osteoarthritis of the knee: data from Caucasian participants in the Baltimore Longitudinal Study of Aging. *Aging (Milano)*. 1994;6(5):353–357.
30. Spector TD, Hart DJ, Doyle DV. Incidence and progression of osteoarthritis in women with unilateral knee disease in the general population: the effect of obesity. *Ann Rheum Dis*. 1994;53(9):565–568.
31. Buckwalter JA, Martin JA. Osteoarthritis. *Advanced Drug Delivery Reviews*. 2006;58(2):150–167.
32. Moskowitz RW, Kelly MA, Lewallen DG. Understanding osteoarthritis of the knee--causes and effects. *Am J. Orthop.* 2004;33(2 Suppl):5–9.
33. Brandt KD, Dieppe P, Radin E. Etiopathogenesis of osteoarthritis. *Med. Clin. North Am.* 2009;93(1):1–24, xv.
34. Peat G, McCarney R, Croft P. Knee pain and osteoarthritis in older adults: a review of community burden and current use of primary health care. *Ann. Rheum. Dis.* 2001;60(2):91–97.
35. Peat G, Thomas E, Duncan R, et al. Clinical classification criteria for knee osteoarthritis: performance in the general population and primary care. *Ann. Rheum. Dis.* 2006;65(10):1363–1367.
36. Wilson DR, McWalter EJ, Johnston JD. The measurement of joint mechanics and their role in osteoarthritis genesis and progression. *Med. Clin. North Am.* 2009;93(1):67–82, x.
37. Radin EL, Burr DB, Caterson B, et al. Mechanical determinants of osteoarthritis. *Semin. Arthritis Rheum.* 1991;21(3 Suppl 2):12–21.
38. Burr DB. Anatomy and physiology of the mineralized tissues: role in the pathogenesis of osteoarthritis. *Osteoarthr. Cartil.* 2004;12 Suppl A:S20–30.
39. Hunter DJ. Osteoarthritis. *Best Pract Res Clin Rheumatol.* 2011;25(6):801–814.
40. Chaganti R, Lane N. Risk factors for incident osteoarthritis of the hip and knee. *Current Reviews in Musculoskeletal Medicine.* 2011;4(3):99–104.

41. Felson DT. An update on the pathogenesis and epidemiology of osteoarthritis. *Radiologic Clinics of North America*. 2004;42(1):1–9.
42. Sharma L. Local factors in osteoarthritis. *Curr Opin Rheumatol*. 2001;13(5):441–446.
43. Davis MA, Ettinger WH, Neuhaus JM, Cho SA, Hauck WW. The association of knee injury and obesity with unilateral and bilateral osteoarthritis of the knee. *Am. J. Epidemiol*. 1989;130(2):278–288.
44. Srikanth VK, Fryer JL, Zhai G, et al. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. *Osteoarthritis and Cartilage*. 2005;13(9):769–781.
45. Felson DT, Naimark A, Anderson J, et al. The prevalence of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. *Arthritis Rheum*. 1987;30(8):914–918.
46. 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):2271–2279.
47. Jordan JM, Helmick CG, Renner JB, et al. Prevalence of Knee Symptoms and Radiographic and Symptomatic Knee Osteoarthritis in African Americans and Caucasians: The Johnston County Osteoarthritis Project. *J Rheumatol*. 2007;34(1):172–180.
48. Toivanen AT, Heliövaara M, Impivaara O, et al. Obesity, physically demanding work and traumatic knee injury are major risk factors for knee osteoarthritis--a population-based study with a follow-up of 22 years. *Rheumatology (Oxford)*. 2010;49(2):308–314.
49. Sharma L, Lou C, Cahue S, Dunlop DD. The mechanism of the effect of obesity in knee osteoarthritis: the mediating role of malalignment. *Arthritis Rheum*. 2000;43(3):568–575.
50. Sharma L, Song J, Felson DT, et al. The role of knee alignment in disease progression and functional decline in knee osteoarthritis. *JAMA*. 2001;286(2):188–195.
51. Felson DT. Osteoarthritis of the Knee. *New England Journal of Medicine*. 2006;354(8):841–848.
52. Kellgren JH, Lawrence JS. Radiological Assessment of Osteo-Arthrosis. *Ann Rheum Dis*. 1957;16(4):494–502.
53. Neumann G, Hunter D, Nevitt M, et al. Location specific radiographic joint space width for osteoarthritis progression. *Osteoarthr. Cartil*. 2009;17(6):761–765.
54. Hunter DJ, Le Graverand M-PH, Eckstein F. Radiologic markers of osteoarthritis progression. *Curr Opin Rheumatol*. 2009;21(2):110–117.

55. Guermazi A, Eckstein F, Hellio Le Graverand-Gastineau M-P, et al. Osteoarthritis: current role of imaging. *Med. Clin. North Am.* 2009;93(1):101–126, xi.
56. Perry J. *Gait analysis: normal and pathological function*. SLACK; 1992.
57. Chao EY, Laughman RK, Schneider E, Stauffer RN. Normative data of knee joint motion and ground reaction forces in adult level walking. *J Biomech.* 1983;16(3):219–233.
58. Hartmann M, Kreuzpointner F, Haefner R, et al. Effects of juvenile idiopathic arthritis on kinematics and kinetics of the lower extremities call for consequences in physical activities recommendations. *Int J Pediatr.* 2010;2010. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/20862334>. Accessed March 3, 2012.
59. Ounpuu S. The biomechanics of walking and running. *Clin Sports Med.* 1994;13(4):843–863.
60. Miller DI, Nelson RC. *Biomechanics of sport: a research approach*. Lea & Febiger; 1973.
61. Inman VT. Human locomotion. *Can Med Assoc J.* 1966;94(20):1047–1054.
62. Radin EL, Paul IL, Rose RM. Role of mechanical factors in pathogenesis of primary osteoarthritis. *Lancet.* 1972;1(7749):519–522.
63. Hamill J, Knutzen KM. *Biomechanical Basis Of Human Movement*. Lippincott Williams & Wilkins; 2006.
64. Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical walking pattern changes in the fit and healthy elderly. *Phys Ther.* 1990;70(6):340–347.
65. Winter DA. Energy generation and absorption at the ankle and knee during fast, natural, and slow cadences. *Clin. Orthop. Relat. Res.* 1983;(175):147–154.
66. Komi PV, Salonen M, Järvinen M, Kokko O. In vivo registration of Achilles tendon forces in man. I. Methodological development. *Int J Sports Med.* 1987;8 Suppl 1:3–8.
67. Pandy MG. Computer modeling and simulation of human movement. *Annu Rev Biomed Eng.* 2001;3:245–273.
68. Buchanan TS, Lloyd DG, Manal K, Besier TF. Estimation of muscle forces and joint moments using a forward-inverse dynamics model. *Med Sci Sports Exerc.* 2005;37(11):1911–1916.
69. Erdemir A, McLean S, Herzog W, van den Bogert AJ. Model-based estimation of muscle forces exerted during movements. *Clin Biomech (Bristol, Avon).* 2007;22(2):131–154.

70. DeVita P, Hortobagyi T. Functional knee brace alters predicted knee muscle and joint forces in people with ACL reconstruction during walking. *Journal of Applied Biomechanics*. 2001;17(4):297–311.
71. 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? *Osteoarthr. Cartil.* 2011;19(3):272–280.
72. Nene A, Byrne C, Hermens H. Is rectus femoris really a part of quadriceps?: Assessment of rectus femoris function during gait in able-bodied adults. *Gait & Posture*. 2004;20(1):1–13.
73. Messier SP, Gutekunst DJ, Davis C, DeVita P. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. *Arthritis & Rheumatism*. 2005;52(7):2026–2032.
74. Diraçoğlu D, Baskent A, Yagci I, Özçakar L, Aydın R. Isokinetic strength measurements in early knee osteoarthritis. *Acta Reumatol Port.* 2009;34(1):72–77.
75. Barker K, Lamb SE, Toye F, Jackson S, Barrington S. Association between radiographic joint space narrowing, function, pain and muscle power in severe osteoarthritis of the knee. *Clin Rehabil.* 2004;18(7):793–800.
76. Herzog W, Nigg BM, Read LJ, Olsson E. Asymmetries in ground reaction force patterns in normal human gait. *Med Sci Sports Exerc.* 1989;21(1):110–114.
77. McCrory JL, White SC, Lifeso RM. Vertical ground reaction forces: objective measures of gait following hip arthroplasty. *Gait Posture*. 2001;14(2):104–109.
78. Giakas G, Baltzopoulos V, Dangerfield PH, Dorgan JC, Dalmira S. Comparison of gait patterns between healthy and scoliotic patients using time and frequency domain analysis of ground reaction forces. *Spine*. 1996;21(19):2235–2242.
79. Burnett DR, Campbell-Kyureghyan NH, Cerrito PB, Quesada PM. Symmetry of ground reaction forces and muscle activity in asymptomatic subjects during walking, sit-to-stand, and stand-to-sit tasks. *J. Electromyogr. Kinesiol.* 2011;21(4):610–615.
80. Teichtahl AJ, Wluka AE, Morris ME, Davis SR, Cicuttini FM. The Associations Between the Dominant and Nondominant Peak External Knee Adductor Moments During Gait in Healthy Subjects: Evidence for Symmetry. *Archives of Physical Medicine and Rehabilitation*. 2009;90(2):320–324.
81. James PJ, Nicol AC, Hamblen DL. A comparison of gait symmetry and hip movements in the assessment of patients with monarticular hip arthritis. *Clinical Biomechanics*. 1994;9(3):162–166.
82. Liikavainio T, Isolehto J, Helminen HJ, et al. Loading and gait symmetry during level and stair walking in asymptomatic subjects with knee osteoarthritis: Importance of

quadriceps femoris in reducing impact force during heel strike? *The Knee*. 2007;14(3):231–238.

83. Su P-F, Gard SA, Lipschutz RD, Kuiken TA. Gait characteristics of persons with bilateral transtibial amputations. *J Rehabil Res Dev*. 2007;44(4):491–501.

84. Royer TD, Wasilewski CA. Hip and knee frontal plane moments in persons with unilateral, trans-tibial amputation. *Gait Posture*. 2006;23(3):303–306.

85. Milner CE, O'Bryan ME. Bilateral frontal plane mechanics after unilateral total knee arthroplasty. *Arch Phys Med Rehabil*. 2008;89(10):1965–1969.

86. Thompson WR, Medicine AC of S, Gordon NF, Pescatello LS. *ACSM's Guidelines for Exercise Testing and Prescription*. Lippincott Williams & Wilkins; 2009.

87. Messier S, Legault C, Mihalko S, et al. The Intensive Diet and Exercise for Arthritis (IDEA) trial: design and rationale. *BMC Musculoskeletal Disorders*. 2009;10(1):93.

88. Hanavan EP. *A mathematical model of the human body*. Aerospace Medical Research Laboratories, Aerospace Medical Division, Air Force Systems Command; 1964.

89. Dempster WT. *Space requirements of the seated operator: geometrical, kinematic, and mechanical aspects of the body with special reference to the limbs*. Wright Air Development Center, Air Research and Development Command, U.S. Air Force; 1955.

90. Spoor CW, van Leeuwen JL. Knee muscle moment arms from MRI and from tendon travel. *J Biomech*. 1992;25(2):201–206.

91. Rugg SG, Gregor RJ, Mandelbaum BR, Chiu L. In vivo moment arm calculations at the ankle using magnetic resonance imaging (MRI). *J Biomech*. 1990;23(5):495–501.

92. Yamaguchi G, Sawa A, Moran D, Fessler M, Winters J. A survey of human musculotendon parameters. pp. 717-773. In: *Multiple Muscle Systems: Biomechanics and Movement Organization*. Winters J, Woo SL-Y (eds), Springer-Verlag, 1990. In: *Multiple Muscle Systems, edited by J.M. Winters*. New York: Springer-Verlag; 1990:717 – 773.

93. Winter DA, Yack HJ. EMG profiles during normal human walking: stride-to-stride and inter-subject variability. *Electroencephalogr Clin Neurophysiol*. 1987;67(5):402–411.

94. Simonsen EB, Dyhre-Poulsen P, Voigt M, Aagaard P, Fallentin N. Mechanisms contributing to different joint moments observed during human walking. *Scand J Med Sci Sports*. 1997;7(1):1–13.

95. Andersson EA, Nilsson J, Thorstensson A. Intramuscular EMG from the hip flexor muscles during human locomotion. *Acta Physiol. Scand*. 1997;161(3):361–370.

96. Duda GN, Brand D, Freitag S, Lierse W, Schneider E. Variability of femoral muscle attachments. *J Biomech.* 1996;29(9):1185–1190.
97. Herzog W, Read LJ. Lines of action and moment arms of the major force-carrying structures crossing the human knee joint. *J. Anat.* 1993;182 (Pt 2):213–230.
98. Nisell R. Mechanics of the knee. A study of joint and muscle load with clinical applications. *Acta Orthop Scand Suppl.* 1985;216:1–42.
99. Visser JJ, Hoogkamer JE, Bobbert MF, Huijing PA. Length and moment arm of human leg muscles as a function of knee and hip-joint angles. *Eur J Appl Physiol Occup Physiol.* 1990;61(5-6):453–460.
100. Schipplein OD, Andriacchi TP. Interaction between active and passive knee stabilizers during level walking. *J. Orthop. Res.* 1991;9(1):113–119.
101. Astephen Wilson JL, Deluzio KJ, Dunbar MJ, Caldwell GE, Hubley-Kozey CL. The association between knee joint biomechanics and neuromuscular control and moderate knee osteoarthritis radiographic and pain severity. *Osteoarthr. Cartil.* 2011;19(2):186–193.
102. Kemp G, Crossley KM, Wrigley TV, Metcalf BR, Hinman RS. Reducing joint loading in medial knee osteoarthritis: shoes and canes. *Arthritis Rheum.* 2008;59(5):609–614.
103. Shakoor N, Sengupta M, Foucher KC, et al. Effects of common footwear on joint loading in osteoarthritis of the knee. *Arthritis Care Res (Hoboken).* 2010;62(7):917–923.
104. Messier S, Ettinger W, Doyle T, et al. Obesity: Effects on gait in an osteoarthritic population. *J. Appl. Biomech.* 1996;12(2):161–172.
105. Miyazaki T, Wada M, Kawahara H, et al. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Ann. Rheum. Dis.* 2002;61(7):617–622.

CURRICULUM VITAE

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Education

- M.S. Health and Exercise Science, 2012
Wake Forest University, Winston Salem, NC
Thesis: *Differential effects of unilateral and bilateral knee osteoarthritis on gait*
- B.A. Physical Education –Exercise Science Emphasis with Honors, 2010
Calvin College, Grand Rapids, MI

Research Experience

- 2010 – present Research Assistant
J.B Snow Biomechanics Laboratory
Department of Health and Exercise Science, Wake Forest University
The Runners And Injury Longitudinal Study (TRAILS)
PI: Dr. Stephen Messier, Ph. D.
- Laboratory experience: Obtained informed consent, administered questionnaires, collected anthropometric data including flexibility, arch height, and Q-angle, collected strength data using isokinetic dynamometer, applied marker set for gait analysis
 - Administrative experience: entered data, scheduled appointments
- Intensive Diet and Exercise for Arthritis (IDEA) Study*
PI: Dr. Stephen Messier, Ph.D.
- Assisted with exercise intervention,
- Strength Training for Arthritis Trial (START)*
PI: Dr. Stephen Messier, Ph.D.
- Assisted exercise intervention
 - Conducted one repetition maximum tests for older adults on strength training machines
- 2010 *Energy Expenditure and Cardiopulmonary Response to WiiFit Hula Hoop versus Treadmill Running in College Females*
Calvin College, Grand Rapids, MI
- Collected expired gases for analysis during exercise

Professional Experience

- 2010 – 2011 Exercise Leader/Lab Technician, Healthy Exercise and Lifestyle ProgramS (HELPS)
Wake Forest University
- Lead group exercise for cardiac patients
 - Monitored heart rhythm, blood pressure, and blood glucose
 - Conducted GXTs for chronic disease populations
- 2010 Personal Trainer, Healthy Habits
Calvin College, Grand Rapids, MI

Teaching Experience

- 2010 – 2012 Graduate Teaching Assistant, Wake Forest University
HES 101: Exercise for Health
- 2011 Laboratory Assistant, Wake Forest University
HES 370: Biomechanics of Human Movement
- 2010 Student Assistant, Calvin College
PER 107: Strength and Conditioning

Professional Societies

- 2010 – present Southeast Chapter of American College of Sports Medicine
(SEACSM)

Certifications

- 2010 – present Basic Life Support (BLS), CPR, and AED – American Heart Association