THE INFLUENCE OF OBESITY AND ALIGNMENT ON KNEE JOINT LOADS
DURING OSTEOARTHRITIC GAIT

BY

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ABSTRACT

PURPOSE: The purpose of this cross-sectional investigation was to determine the influences of alignment and obesity on the knee joint loads of overweight and obese older adults with knee osteoarthritis. Our primary hypothesis was that there would be a significant interaction between knee alignment and Body Mass Index (BMI) on the knee joint load, independent of gender and walking speed.

METHODS: Baseline data from a subset of participants (157 out of 454) enrolled in the Intensive Diet and Exercise for Arthritis (IDEA) clinical trial were utilized for this study. Knee alignment was quantified on all subjects (N = 157) using a full length anteroposterior (AP) radiograph. Varus alignment was defined as an angle greater than 2°, neutral was 0-2°, and valgus was less than 0°. BMI was quantified in all subjects using each participant’s height and weight. Internal knee moments, knee joint forces, and quadriceps, hamstrings, and gastrocnemius forces were calculated using a 3-D gait analysis with a 37-reflective maker 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. Linear regression models were fitted for each kinetic outcome controlling for gender and walking speed.

RESULTS: After adjusting for walking speed and gender, there was not a significant interaction between BMI and knee alignment on the knee joint forces, or on the internal knee abduction moment. However, alignment had a significant (p < 0.0001) association with the internal knee abduction moment, independent of BMI. BMI had significant associations with the peak knee compressive and shear forces and knee muscle forces, independent of alignment.
CONCLUSION: A higher BMI was associated with greater peak knee compressive, shear, and muscle forces, regardless of alignment, and alignment was associated with the internal knee abductor moment, independent of BMI. Hence, BMI and alignment influence different joint loading measures that have both been linked to disease progression.
REVIEW OF LITERATURE

Introduction

Osteoarthritis (OA) is the most common form of arthritis, affecting nearly 27 million Americans \(^1\). Knee OA specifically is the leading cause of chronic disability and affects 15\% of the US population over 65 years old \(^2\,^3\). Although the etiology of knee OA is unclear, several risk factors are known. Among these, knee malalignment and obesity play important roles by increasing the knee joint loads and further damaging the articular cartilage \(^4\,^9\).

Obesity is considered the most modifiable risk factor for knee OA. Both Felson et al. \(^10\) and Reijman et al. \(^6\) found that excessive mechanical joint stress from increasing BMI is a possible pathway for the incidence and progression of knee OA. Excessive joint stress may initiate cartilage breakdown and lead to progression of knee joint destruction \(^10\). In fact, results from Messier et al. \(^11\) showed that for each pound lost, there was a resultant 4-fold reduction in the amount of load exerted on the knee per step as well as a 1.4\% reduction in the internal knee abduction moment (a surrogate measure of knee joint loading), illustrating the contribution body mass has on knee joint loading.

Knee malalignment may play a part in the strong association between obesity and OA progression. Malalignment results in abnormal loading of the joint. When joint loads are not well distributed across the articular surfaces, local stresses are increased, leading to the local breakdown of articular cartilage \(^4\). Varus malalignment is strongly associated with medial compartment disease progression while valgus malalignment is associated with lateral compartment progression \(^5\,^9\,^12\).
Moyer et al.\textsuperscript{13} examined the interaction of malalignment and body mass on the knee abduction moment in 487 patients with knee OA and found that those with the greatest body mass had a 3.2 Nm increase in knee loads with every one degree increase in varus malalignment. In the highest weight category, those with the least amount of malalignment had a mean peak adduction moment of 37 Nm, compared to 72 Nm in those with the severe varus malalignment. Further, they found that those with the greatest varus malalignment had a .4 Nm increase in knee load for every 1 kg increase in mass. In the most varus malaligned, mean peak adduction moments went from 50 Nm in the lowest weight category to 72 Nm in the greatest weight category.

Other studies have examined the effect of alignment on knee joint loading across normal and obese adults. Felson et al.\textsuperscript{4} found that knees with neutral alignment (0–2°) or severe malalignment (>7°) and an increase in BMI had no effect on the risk of disease progression; however, moderate malalignment was associated with disease progression, illustrating that there may be an interaction effect. Niu et al.\textsuperscript{8} reported that excess load produced by varus malalignment may be sufficient to cause progression independent of obesity. Having excess body weight may strengthen the association between alignment and load on the medial compartment of the tibiofemoral joint. In a cross-sectional study by Sharma et al.\textsuperscript{7}, they concluded that the effect of BMI on OA severity was explained by varus malalignment acting as a mediator.

To our knowledge, only Moyer et al.\textsuperscript{13} has examined the contributions of both alignment and obesity on knee joint loading; however, they used the knee abduction moment as a surrogate measure of joint loading of the medial compartment. This measure is less sensitive when alignment is not controlled. We suggest that using the bone-on-
bone forces derived from musculoskeletal modeling will provide a more accurate measure of joint loading independent of alignment. The purpose of this cross-sectional study is to examine the interaction of knee alignment and obesity and their effects on knee joint loads in older adults with knee OA. We hypothesize that those with a greater degree of knee malalignment will have increased knee joints loads, and that these loads will be exacerbated by the level of obesity. Further, individuals having varus or valgus malalignment will have greater knee joint loads with increasing obesity when compared to those with neutral alignment.

Osteoarthritis of the Knee

Epidemiology of OA

Osteoarthritis (OA) is the most common cause of disability in older adults and affects more than 1/3 of adults over the age of 65 yrs.¹ ¹⁴. Radiographic changes in at least one joint are seen in 80% of individuals over the age of 80 yrs.¹⁵, and as many as 40% of individuals over 65 yrs. report symptoms due to OA¹⁰. The tibiofemoral joint of the knee is the most commonly affected weight bearing joint and in 2005, 27 million Americans had physician diagnosed knee OA, an increase from 21 million in 1995. Since OA is strongly associated with aging, its prevalence will continue to increase as our population ages¹. Annually, OA costs the US economy over $60 billion dollars and is associated with over 700,000 hospitalizations¹⁶, stressing the importance of limiting disease incidence and progression.
**Diagnosis of Knee OA**

The diagnosis of knee OA is based on a combination of symptoms and radiographic evidence and is often a clinical call since radiographic findings do not always correlate with symptoms \cite{17}. The most common symptoms are joint pain, tenderness, limitation of movement, crepitus, and inflammation without systemic effects \cite{18}.

The Kellgren-Lawrence scale is a grading scale of radiographic OA severity and is the most common tool used to measure OA incidence and progression \cite{19}. This scale classifies knees from grades 0-4 (with 4 being the most severe) according to the following features seen on the radiograph \cite{19}:

- **Grade 0**: no joint space narrowing
- **Grade 1**: doubtful narrowing of joint space and possible osteophytic lipping
- **Grade 2**: definite osteophytes and possible narrowing of the joint space
- **Grade 3**: moderate multiple osteophytes, definite narrowing of joint space and some sclerosis and possible deformity of bone ends
- **Grade 4**: large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformity of bone ends

The K-L scale, however, does not come without its objective limitations. Thus, current practice defines the radiographic criteria for OA as asymmetrical joint space narrowing, formation of osteophytes on the joint margin, subchondral bone sclerosis, subchondral cyst formation, and in severe cases, deformity of the joint ends \cite{18}. However,
many individuals show radiographic evidence for OA without having any signs or symptoms and several interventions have shown reductions in pain without showing any improvements in radiographic evidence\textsuperscript{18, 20}.

Pathogenesis of Knee OA

OA is associated with degradation of the articular cartilage accompanied by substantial subchondral bone changes, narrowing of the joint space, and inflammation\textsuperscript{18}. How these changes lead to the actual development of OA is unknown. There are several possible mechanisms including biomechanical, biochemical, and inflammatory processes.

There is an inflammatory mechanism present in some stage of the disease process\textsuperscript{18}. Patients with OA often have elevated systemic markers of inflammation; including C-reactive protein, clear synovial hyperplasia, and a dense mononuclear cell infiltrate\textsuperscript{21, 22}. The synovium adjacent to the articular cartilage is commonly inflamed (similar to rheumatoid arthritis) giving reason to believe that the inflammatory component could be related to articular cartilage damage\textsuperscript{23}. Elevated levels of inflammatory cytokines mediate the development and progression of OA, including interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-\alpha)\textsuperscript{22, 24, 25}. Both IL-1 and TNF-\alpha are produced by joint tissue and can cause an increase in the production of enzymes responsible for degradation of the articular cartilage, representing a link between inflammation and OA\textsuperscript{26}.

Development and progression of knee OA can also be described by biomechanical changes in the articular cartilage. Biomechanical factors such as obesity\textsuperscript{10}, repetitive joint loading\textsuperscript{4, 27}, previous injury\textsuperscript{4, 28}, knee malalignment\textsuperscript{5, 29}, and joint deformity\textsuperscript{4} are all associated with knee OA. The mechanical stress applied from these
risk factors to the articular cartilage in the presence of inflammation can exacerbate inflammation and lead to more cartilage breakdown.

It is unknown, however, whether the initial abnormality leading to the onset of OA occurs in the articular cartilage or in the subchondral bone. Creamer views OA as a failure to maintain a homeostatic balance of cartilage matrix synthesis and degradation.

The role of the articular cartilage is to absorb stress, providing a smooth load bearing surface to permit low-friction movement of the joint. Radin proposed that repeated impulsive loading of the joint leads to small microfractures in the subchondral bone and that the remodeling of the bone causes the subchondral bone to thicken via the addition of new trabeculae. In addition, there is also a thinning of the overlying cartilage that increases the shear stresses on the knee and accelerates the degradation of the articular cartilage. This increased force transmission would accelerate the process of OA.

Risk Factors for Knee OA

While the precise etiology of knee OA is unknown, numerous risk factors have been identified that contribute to its incidence and progression. These include age, gender, obesity, and previous knee injury/joint trauma. Other risk factors such as malalignment, genetics, nutrition, and occupation have been identified as well. Felson et al. suggested that risk factors for OA are best described as either being impairments of joint protectors that increase joint vulnerability, or excessively load the joint. Factors increasing joint vulnerability include malalignment, muscle weakness, genetic and ethnic
predispositions, and aging. Factors causing excessive loading consist of obesity and certain physical activities.

**Figure 1:** Pathogenesis of OA with putative risk factors (Figure adapted from Felson et al. 2000) \(^3^2\)

Age is the strongest determinant of OA with prevalence rates for all joints increasing with rising age \(^1^8\). While age has been strongly associated with joint damage and degradation, age does not alone cause disease. Rather, other joint vulnerabilities that occur as a part of the aging process make it more susceptible to disease \(^4\). With aging, ligaments begin to loosen and thin, increasing the shear stress on the joint which can damage the attachment between the cartilage and the base of the joint \(^3^3\).

Gender differences also have a role in the development and progression of OA. Females in general are at a higher risk for developing OA than males, even more so after menopause \(^1^8\). Several studies have shown that sex hormones, specifically estrogen, play a role in OA \(^1^8\). Nevitt et al. studied postmenopausal women and found hormone replacement therapy to have a protective effect on the incidence of hip and knee OA \(^2^6\).
The Health and Nutrition Examination Survey (HANES I) examined the associations between OA and a variety of risk factors among over 5,000 black and white participants aged 35-74 years. The authors found a significant association of knee OA with obesity, race, and occupation \(^\text{34}\).

**Obesity and OA**

Obesity is associated with numerous health risks including cardiovascular disease, insulin resistance, diabetes, and hypertension. Obesity is also the most modifiable risk factor for knee OA. Many studies have shown a strong association between obesity and knee OA, suggesting that increased loading of the knee due to the additional weight leads to subsequent joint degradation \(^\text{10, 34, 35}\). Ettinger et al. \(^\text{36}\) examined the effects of obesity on comorbidites and found that those with a BMI of over 30 kg/m\(^2\) were 4.2 times more likely to have knee OA than those under 30 kg/m\(^2\).

**Malalignment and OA**

Malalignment is an important risk factor for knee OA \(^\text{5, 37}\). Obesity increases the overall loading of the joint and malalignment concentrates that loading to a focal area by redistributing the load causing OA disease progression. Malalignment may be the most potent risk factor for structural OA progression \(^\text{5, 12, 37}\) due to the vulnerability of the knee joint by loss of intrinsic soft tissue stability that can alther the loading patterns at the knee joint \(^\text{38}\). Typically, individuals are classified as having varus or valgus malalignment. Varus malaligned individuals tend to have a “bowlegged” appearance, and those with valgus malalignment a more “knock-kneed” appearance. Sharma et al. \(^\text{5}\) found that varus and valgus malalignment increased the risk of medial and lateral knee OA, respectively.
Healthy Gait Mechanics

Each gait cycle is divided into two phases, stance and swing. Stance refers to the period of time the foot is in contact with the ground and swing the period the foot is in the air, necessary for limb advancement. One gait cycle as defined as heel strike to heel strike on the same foot. During normal walking speeds, the stance phase occupies roughly 60% of the gait cycle, while swing occupies 40%. At slower walking speeds the stance phase percentage increases.

The gait cycle can be further divided into numerous sub-phases, each working together to accomplish 3 basic tasks: weight acceptance, single limb support, and limb advancement. When the foot comes through the swing phase and prepares to make contact with the ground, the limb must prepare to accept the transfer of body weight. Initial contact only takes 2% of the entire gait cycle and is simply the moment the foot just touches the ground. The loading response begins at heel strike and continues until the other foot is lifted for the swing phase (about 10% of the gait cycle). During this time, the individual is in a period of double support, where both feet are on the ground, providing maximal stability for weight transfer. As the other foot lifts, the single limb support phase beings and continues until the other foot goes through swing and touches the ground again. During single limb support, the primary objective is forward progression over the foot making contact with the ground. The third task is limb advancement that begins as the limb prepares for the swing phase. The pre-swing phase is where the weight from that foot is released or transferred onto the opposite limb that is making initial contact and ready to accept the weight of the body. As the leg works into the swing phase, the limb
prepares for its next stance interval by ensuring the foot clears the floor and “swinging” the foot from its trailing position at toe off 40.

Figure 2: Normal phases of the gait cycle (Figure from Perry, 20-- 40).

Joint Kinetics During Healthy Gait

Joint kinetics refer to the forces that cause motion or the forces acting on a system 39, 41. These could be ground reaction forces, joint moments, or joint powers. Two types of forces are known to act on the body, external forces and internal forces. External forces are produced by ground reaction forces, gravity and inertia, while internal forces are produced by muscles and ligaments 39. Joint kinetics are calculated in gait through simultaneous collection of positional information (the joint kinematics) and the ground reaction forces that are typically measured with a force plate 41.

Ground Reaction Forces During Healthy Gait

Ground reaction forces are the forces exerted by the ground on the foot during contact. In walking, the vertical component of the ground reaction force (GRF) generally
has a maximum value between 1.0 and 1.2 times the walkers’ body weight \(^{39}\). The vertical component of the GRF has a biomodal shape in walking where both peaks are roughly equal in magnitude. The first peak is the loading response peak and occurs during the first half of stance phase. During this time there is a vertical deceleration as the weight is accepted onto the landing foot. During mid-stance, partial unloading occurs as the knee flexes and the tibia comes over the foot as the plantar flexors act eccentrically to control tibial motion, and the GRF dips below 1.0 body weight for a short time. The second peak occurs during the second half of the stance phase as the plantar flexors act concentrically to provide the propulsive force necessary to toe off and being the swing phase \(^{39,42}\).

The sagittal component of the GRF, or the anteroposterior forces have a biphasic shape during walking. The first “negative” half corresponds with a posterior-directed GRF or braking force between the foot and the ground \(^{39}\). As the foot pushes back against the ground to progress forward, an anterior-directed GRF is produced exhibiting a “positive” peak. At most, these forces reach magnitudes of 15% of body weight \(^{39}\). The final component of the GRF is the mediolateral component and typically only reaches 1% of body weight during walking. Values for this component vary depending on foot placement and pronation/supination movements \(^{39}\).

Anderson and Pandy \(^{43}\) quantified the contributions of individual muscles to the vertical component of the GRF. They concluded that in early stance, support was provided by the dorsiflexors. When the foot reached foot-flat, support was generated by the gluteus maximus, vasti, and posterior gluteus medius/minimus muscles that together served as the first peak seen in the vertical GRF curve. Throughout the middle and
majority of stance phase, the gluteus medius/minimus provided the majority of the support with a small amount of assistance from gravity. During the late part of stance, the ankle plantar flexors were responsible for the second peak of the vertical GRF curve.

**Moments During Healthy Gait**

During walking, muscle forces are activated along with ligament and friction forces producing a net moment on the joint. Since joint angles during walking do not near their extreme limits, friction forces are minimal, suggesting the majority of the net moment is due to muscle forces. Joint force is dependent on body weight, stride length, and walking speed and it tends to increase with increasing velocity of gait for both normal and abnormal gait.

The hip, knee, and ankle exhibit characteristic moment patterns in normal gait. Motion in the sagittal, frontal, and transverse planes includes flexion/extension, abduction/adduction, and medial/lateral rotations, respectively. A three-dimensional motion capture system, force platforms, and an inverse dynamics model makes it possible to calculate the joint moments and muscle forces during gait.

**Hip Moments During Healthy Gait**

During gait, the majority of hip motion occurs in the sagittal and frontal planes. A normal arch of hip motion, consisting of extension during the stance phase and flexion during the swing phase, averages 40°. The hip also has movement in the frontal plane consisting of adduction and abduction as the unloaded hip follows the leg in swing. In fact, the net internal hip moment is dominated by an abduction moment in the frontal
plane. In the transverse plane, there are also very small amounts of medial and lateral rotation throughout the gait cycle\(^4\).

In the sagittal plane, an extension moment is present during late swing to decelerate the momentum of the limb, or slow hip flexion, to prepare for weight acceptance at heel strike\(^4\). This extension moment increases as the limb enters the stance phase to control the forward momentum of the upper body so it does not flex forward about the hip, thus stabilizing the trunk\(^4\). During 15-50\% of mid-stance, the hip extension moment changes to a flexion moment to control the leg from rotating backwards. At 50\% of mid-stance, the hip flexors begin to contract concentrically to prepare for toe off and enter into the swing phase of the gait cycle. At the transition from stance to swing, the hip flexors raise the leg as weight is fully transferred to the contralateral limb. At mid-swing, the hip flexors pull the swinging limb upwards and forward until hip extensors are re-activated to prepare for the next heel strike\(^4\).

![Graph of Hip Extension/Flexion Moment](image)

**Figure 3:** Internal hip flexion/extension moment\(^4\).
In the frontal plane, the hip abductors are mainly active during the initial half of stance, working to keep the pelvis from falling towards the stance leg while the contralateral limb is in stance. Initial contact begins with a small amount of adduction and then abducts to neutral during mid and late stance until swing, when hip abduction occurs again, making an abduction/adduction curve with two distinct abductor peaks. During the first peak following heel strike, the gluteus medius and minimus muscles prevent the pelvis from falling toward the stance leg while the body’s weight is centered medial to the hip. The abduction moment then decreases slightly in mid-stance before it produces a second peak during terminal stance to slow the advancement of the pelvis thus, stabilizing the opposite limb as it accepts the body’s weight.

Figure 4: Internal hip abduction/adduction moment.

Throughout the gait cycle, there is also a small amount of internal and external rotation in the transverse plane. At heel strike, the hip has a slight internal lateral rotation moment and then a small medial rotation moment occurs during loading; this is followed by mid-stance being dominated by a lateral rotation moment. During the stance phase of
gait, while the limb is accepting the body weight, forward rotation of the pelvis occurs that is controlled by the gluteus maximus that will slow hip rotation. In late stance, however, there is medial rotation moment as the contralateral limb prepares for weight acceptance.\textsuperscript{45}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure5.png}
\caption{Internal hip medial/lateral rotation moment.\textsuperscript{40}}
\end{figure}

\textit{Knee Moments During Healthy Gait}

During gait, the majority of knee motion is in the sagittal plane, but also small amounts occur in the frontal plane. Normal knee motion during walking has a full range of 0° to 70° of flexion throughout the gait cycle. At initial contact, the knee is flexed about 5° for the first 2-4% of the gait cycle and then the knee rapidly flexes to about 20° at 15% of the gait cycle to absorb the transfer of weight onto the leg entering stance. During mid-stance, the knee gradually extends until the leg enters swing and the knee beings to flex again in order to clear the ground.\textsuperscript{40}

The sagittal knee moment enters into a small external extension moment shortly after heel contact (2-4% of the gait cycle), where the vertical GRF vector passes anterior
to the knee, thus counteracted by an internal flexor moment\textsuperscript{40, 45}. At the beginning of stance to help with weight acceptance, the vertical GRF passes through the knee posteriorly thus creating and external flexion moment under eccentric control of the knee extensors. The quadriceps group (primarily the vastus medialis, vastus lateralis, and vastus intermedius) as well as assistance from the upper gluteus maximus through its insertion on the iliotibial band then create this extensor moment to control knee flexion and assist in knee extension\textsuperscript{40, 41}. At mid-stance, as the tibia continues to move over the ankle (about 20-30\% of the gait cycle) and the knee begins to extend, the GRF moves anterior to the knee causing an internal flexor moment yet again, this time by eccentric contraction of the gastrocnemius\textsuperscript{40, 41}. In terminal stance, the GRF again moves posterior to the joint causing the knee to flex in preparation for toe off and early swing\textsuperscript{40}. The rectus femoris primarily is responsible for yielding this internal extensor moment prior to toe off. During the swing phase, the quadriceps remain active and maintain an extension moment to decelerate the swinging leg after toe off\textsuperscript{40, 45} until the hamstrings take over in late swing producing a net internal knee flexor moment to decelerate the swinging leg and slow knee extension prior to initial contact\textsuperscript{42, 45}.
Figure 6: Internal knee flexion/extension moments\textsuperscript{40}.

During most of the gait cycle, the vertical GRF vector passes medial to the knee, producing an external adduction moment in the frontal plane\textsuperscript{46}, thus creating an internal abduction moment. The large adduction moment of the knee during stance results in loading on the medial aspect of the knee\textsuperscript{45} and is commonly used as a surrogate measure of knee joint loading\textsuperscript{38,47}. The quadriceps and gastrocnemius muscles contribute to knee stability in the frontal plane, thus being responsible for the majority of the total knee abduction moment\textsuperscript{46}. The abduction moment curve has two distinct peaks; the first occurring at contralateral toe off and the second at contralateral heel strike. The first peak is due to the force developed by the quadriceps, as well as the lateral knee stabilizers (primarily the biceps femoris and tensor fascia latae)\textsuperscript{45}. The second abduction moment peak occurs at contralateral heel strike and is due primarily to the force in the lateral gastrocnemius and tensor fascia latae\textsuperscript{40,46}. As the knee extends and pre swing begins, the abduction moment decreases once again\textsuperscript{40} until the next heel strike takes place.
There is a small amount of motion in the transverse plane at the knee as well. At initial contact, the femur is in a position of slight lateral contact on the top of the tibia. As weight bearing occurs and the knee begins to flex, the knee begins to “unlock” and the tibia begins to rotate medially at a quicker rate than the femur rotates medially. At the end of weight bearing, the femur and tibia are both maximally rotated medially. As the knee begins to extend, the knee laterally rotates until the conclusion of the stance phase. Just prior to toe off and into swing, the knee begins to rotate medially again and remains this way throughout the swing phase.

**Ankle Moments During Healthy Gait**

During gait, the majority of the net ankle moment is plantar flexion. This is not surprising since the gastrocnemius muscles (or the plantar flexors) have a much greater muscle mass than the dorsiflexors. The plantar flexors are also almost always under a constant workload to overcome gravity and the passive dorsiflexion of the foot, even
during standing posture\textsuperscript{39}. During initial contact, there is a small dorsiflexor moment of the ankle as the tibialis anterior contracts eccentrically to control the lowering of the foot to the floor\textsuperscript{41}. After initial contact, the ankle plantar flexors take over and contract eccentrically to control the forward progression of the tibia over the foot as well as to support the body’s weight. Then, at the end of stance, the plantar flexors contract concentrically to assist in advancement of the stance limb into swing, or during toe off\textsuperscript{41,45}. During the swing phase, the ankle dorsiflexes to allow toe clearance as well as to prepare the foot for initial contact\textsuperscript{45}.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure8.png}
\caption{Internal ankle plantar flexion/dorsiflexion moment\textsuperscript{40}.}
\end{figure}

\textbf{Osteoarthritic Gait}

Force and moment patterns during walking are different in individuals with knee OA compared to healthy subjects. However, a debate remains whether these changes are due to OA itself, or if they precede the development of OA. Many of the alterations in
gait are an attempt to reduce pain and protect the knee from further degeneration; however, over the long term they could have detrimental effects on the knee joint.\textsuperscript{48}

**Changes in Ground Reaction Forces During Osteoarthritic Gait**

In 1976, Györy et al.\textsuperscript{49} examined the differences in gait patterns between individuals with degenerative joint disease, diagnosed rheumatoid arthritis, and those with healthy knees. They found the mean peak ground reaction forces were significantly reduced in the diseased subjects. Further, the time from heel strike to the first vertical peak of the GRF was increased in the arthritis group, representing a delay in loading. Further, the second peak occurred sooner than that of the healthy group that represented an earlier unloading of the knee during stance. However, these reductions in the peak GRFs are likely due to the changes in temporal variables. Those with OA had reductions in velocity, cadence, and stride length that would contribute to their reductions in the peak GRF. Diseased individuals also had a decrease in their sagittal plane kinematics such as flexion at the knee during the stance phase, indicating a reduced loading tolerance in those with diseased knees.\textsuperscript{49} Childs et al. (2004) performed a similar study, and also found those with knee OA to have reduced vertical GRFs relative to body weight.\textsuperscript{48} Interestingly, these subjects were asked to walk within a similar velocity range, thus controlling for walk speed. They concluded that the decrease in the GRFs were due to other alterations besides walking speed.

Messier et al. (1992) examined the gait mechanics of 15 adults with unilateral knee OA and were age-, gender-, and mass- matched to 15 control subjects walking at the same velocity.\textsuperscript{50} The groups showed no differences in the vertical GRF but did show that...
the OA group had an increased loading rate following heel strike and a decreased peak propulsive vertical force during toe off. Mündermann et al. (2005) also saw a more rapid increase in the GRF and concluded that it was reflective of a rapid shift of the body weight to the support limb.

Other studies have found difference in GRFs when comparing those with osteoarthritic knees to healthy knees. Radin et al. found no difference in walking speed, cadence, or the general shaped of the vertical GRF between those with no pain in the knees during activity and those with intermittent activity-related pain (deemed to be “preosteoarthritic”), however, there was a significant difference during in the time immediately following footstrike. More specifically, they found that in the preosteoarthritic group, the heel hit the floor with a stronger impact and had a larger pre-impact velocity of the shank and ankle as well as a higher loading rate. This “transient heel strike” was deemed to be a reversible risk factor for the development of knee OA. While Radin’s subjects were barefoot, Messier et al. hypothesized that controlling for subjects footwear would reduce the incidence of the transient heel force by allowing the force to dissipate over time.

Joint Kinetics During Osteoarthritic Gait

It has been well established that repetitive, high dynamic knee joint loads are associated with the incidence and progression of knee OA. As previously discussed, the external adduction moment (or internal abduction moment) about the knee during walking is an indirect measure of knee joint loading. Sharma et al. (1998) examined the cross-sectional relationship between the external adduction moment and
disease severity in 54 patients with medial compartment tibiofemoral OA. Their results showed that there was a significant positive correlation between the external adduction moment and Kellgren-Lawrence (KL) grade, and a significant negative correlation with medial joint space width. After adjusting for age, sex, and pain severity, these results persisted in both knees, demonstrating that the external adduction moment is related to increased loading and cartilage degradation in the medial tibiofemoral compartment.

Hunt et al. (2005) examined the external knee adduction moment, the frontal plane lever arm, and GRFs and found that affected knees had a significantly greater knee adduction moment (46.17 ± 17.18 Nm) compared to unaffected knees (38.75 ± 15.18 Nm), suggesting the majority of the load transmitted through the tibiofemoral joint was on the medial compartment. It is important to note that one of the major limitations of this study was that all subjects had unilateral knee OA and comparisons were made to the unaffected limb of the OA patient. Other studies have shown a significant increase in the external adduction moment in those with knee OA. Hurwitz et al. (2000) and Balinas et al. (2002) also reported higher knee adduction moments in patients with medial compartment OA, signifying that those with OA may alter their gait to attempt to unload the knee during walking.

Mündermann et al. (2005) examined 42 patients with bilateral medial compartment OA and 42 age-, sex-, height-, and mass-matched control subjects. They found that all patients with medial compartment OA landed with their knee in a more extended position, had a more rapid increase in the GRF, and had greater hip and knee adduction moments compared to matched controls. In fact, following heel strike the maximum adduction moments at the knee and hip were increased 93.3% and 100.7%
respectively in those with OA compared to the matched controls. This indicates that those with osteoarthritic gait have a more rapid shift in body weight from the contralateral limb to the support limb, as well as a lateral shift of the trunk during walking. They concluded that this was a gait compensation strategy for those with OA to attempt to reduce the mediolateral distance between the body’s center of mass and the knee, thus reducing the moment arm of the GRF and reducing the knee adduction moment and load on the knee later in stance. In this study, patients with knee OA and the matched control walked at similar speeds, ensuring that the changes in gait were not attributed to the speed the patient was walking.

Messier et al. (1992) conducted a study examining the differences in gait mechanics by having 15 affected and 15 control subjects walk at similar walking speeds. They concluded that those with symptomatic knee OA had significantly less knee angular velocity and less knee range of motion during gait. This demonstrates that adults with knee OA compensate during walking to potentially reduce knee pain by reducing knee angular velocity and knee range of motion. This is further supported by a study by Childs et al. (2004) who also found less excursion of the knee in the sagittal plane in those with knee OA during the loading phase of gait. They also found significant muscle co-activation in those with knee OA. Together, this may represent stiffening at the joint in attempt to reduce pain at the knee during gait.

Other studies have found conflicting results. Kaufman et al. (2001) examined the kinematics and kinetics of 139 patients with KL grade II knee OA and found no significant differences in knee motion between patients and healthy subjects. However, they still found a significantly reduced internal knee extensor moment in the OA group,
reflecting gait compensation to reduce pain. It is also important to note that for this study the control group was significantly younger and leaner and these differences were not controlled in the statistical analysis. Subjects were also not asked to walk at the same speed, so some differences in gait mechanics may have been due to differences in walking speed.

**Obese Gait**

Few studies have examined the gait characteristics of obese individuals. Preferred walking speeds are slower, step length and step frequency are reduced, and double support time is longer in obese compared to normal weight individuals, all in an effort to maintain dynamic balance.

Messier et al. (2005) examined 142 sedentary, overweight and obese older adults with knee OA who had taken part in an 18-month clinical trial of diet and exercise. Results showed there was a significant direct association between follow-up body mass and peak follow-up compressive forces, resultant forces, and internal abduction moments. Further they found that a weight reduction of 9.8 N (1 kg) was associated with a 40.6 N reduction in knee compressive forces as well as a 1.4% reduction in the knee abduction moment. These results indicate that for each pound lost, there is a 4-fold reduction in the load exerted on the knee per step.

Browning et al. (2007) examined 20 adults (10 obese and 10 normal weight) and measured GRFs and sagittal plane kinematics at six different speeds. They concluded that obese adults have greater absolute GRF and sagittal plane knee joint moments compared to normal weight adults, suggesting that obese adults walk with greater knee joint loads
than normal weight adults. Browning et al. also observed that when these subjects walked at slower speeds, peak sagittal plane knee moments were 45% less. Further, obese adults walking at 1.1 m/s had the same absolute peak knee muscle moments as normal weight subjects walking at 1.4 m/s. This suggests that obese adults reduce their walking speed as a compensatory strategy to reduce their high knee joint loads.\(^{59}\)

Spyropoulus et al. (1991) examined the kinematic components of walking in 12 obese and non-obese men walking at a self selected pace. They concluded that obese individuals displayed a walking gait that followed a normal pattern, but some of the temporal characteristics were different mainly due to the excessive adipose tissue in their thighs. Obese persons walked significantly slower than non-obese persons (1.09 m/s vs. 1.64 m/s) and had significantly shorter strides (1.25 m vs. 1.67 m). Also noted, obese persons had twice the step width of non-obese people (0.16 m vs. 0.08 m). Greater hip abduction angles during the latter half of stance were also found and attributed to larger thigh girths in obese individuals. At the ankle, there was significantly more ankle dorsiflexion and less ankle plantar flexion throughout the gait cycle, that was due to reduced hip flexion and shorter stride lengths.\(^{60}\) However, since obese people tend to walk at a slower pace, it is unclear whether their altered kinematics are due to the slower walking speed, or are unique gait characteristics of obese individuals.

Finally, DeVita et al. (2003) found no association between obesity and knee joint moments.\(^{61}\) They examined 21 obese and 18 non-obese adults walking at both self-selected and standard walking speeds. Obese adults preferred walking at slower speeds than their lean counterparts (1.29 m/s vs. 1.54 m/s) and had shorter steps, increased double support time, and reduced knee torques and power. When walking at the same
standard speed (1.54 m/s for lean adults and 1.52 m/s for the obese adults), the obese adults had similar stride characteristics to normal weight individuals, however, the relative stance duration was still slightly longer and swing time slightly shorter. Knee torque and power was similar in both the lean and obese adults at the standard speed, but were 88% and 61% higher (respectively) at the ankle in the obese adults. Obese adults also adopted a more erect posture at the standard speed by reducing the flexion at the hip and knee during stance. Also, more plantar flexion was seen at the ankle (6 degrees) compared to non-obese adults. When scaled to body weight, they found the knee joint torques were significantly lower by nearly 46% and the ankle torques significantly higher by nearly 89% compared to the normal weight subjects when walking at a standard speed. The authors concluded that obese subjects reorganize their neuromuscular function to reduce the load at the knee.

Alignment Abnormalities

In normal stance, 60-80% of the total intrinsic load is focused on the medial compartment of the knee. Thus, a varus knee will shift the load bearing axis so that the medial compartment is subjected to even greater stresses. Current research, therefore, uses the external adduction moment as a surrogate measure for the compressive forces at the knee. Numerous studies indicate that a larger adduction moment is associated with greater joint space narrowing or greater resultant loading in the medial compartment. Further, increased knee adduction moments and decreased cartilage volume are both associated with the severity of knee OA. Since an increased abduction moment is already present in individuals with knee OA and those who are obese, having additional malalignment can focus the load even more towards the medial compartment.
of the knee, leading to more detrimental cartilage breakdown in that compartment. Using the abduction moment as a surrogate measure of the compressive forces as the knee primarily focuses on the forces on the medial compartment, where the abduction moment would be mostly affected. This model may have its limitations when observing individuals who have valgus malalignment, where the load is centered on the lateral compartment, which the abduction moment is not specific to.

Measuring Knee Alignment

Several techniques have been developed to determine knee malalignment. Currently, the gold standard is using the angle formed by the intersection of the lines representing the mechanical axis of the femur and that of the tibia. It is drawn from the hip to the knee and the knee to the ankle on a full-lower extremity radiograph. This angle is commonly called the kip-knee-ankle (HKA) angle and assesses the mechanical axis of the knee. Having a positive HKA angle above 2° is indicative of having varus malalignment. Valgus malalignment is classified as anything less than 0°, and neutral malalignment is considered to be 0 - 2°. 
**Figure 9**: Example of hip-knee-ankle (HKA) angle to measure knee alignment on a radiograph (left); examples of HKA angle’s on a varus and valgus knee (left and right, respectively).

However, full-limb radiographs are cumbersome, require specialized equipment and expertise, and can be costly, especially in large epidemiologic studies. Precise quantification of this angle is also challenging due to the lack of precise definition of the measurement landmarks, leading to wide variability among different observers. Goker and Block (2007) validated a reproducible, sensitive method defining the mechanical axis of the femur to be a line from the center of the femoral head to the center point of the intercondylar notch and the tibial mechanical axis as the line connecting the midpoint of the lateral and medial margins of the tibial plateau to the center of the tibial plafond.

In clinical practice, obtaining anteroposterior knee radiographs is the most common way to evaluate knee OA radiographically due to the high cost of full-limb radiographs. On AP radiographs, the femorotibial angle can be measured which defines the anatomical axis of the knee. This has been shown to correlate moderately with the...
HKA angle ($r = 0.75$) on full-limb radiographs. Other techniques have been developed such as physical examination with a goniometer to measure the anatomic-axis angle. Here, the examiner uses a large goniometer that can visually bisect the thigh and lower leg along their lengths with the origin of the device placed on the center of the patella. This technique showed to have a moderate correlation as well ($r = 0.70$) providing alternative valid measures for obtaining knee alignment measures when full-limb radiographs are not available or are impractical.

**Muscle Modeling**

Modeling and simulation to examine the movement of the body is driven by the belief that this approach can provide information on how the nervous system and muscles interact to produce motion of body parts. Some researchers have attempted to measure muscle forces in vivo using surgical techniques to implant force transducers within muscle tendons. Specifically, Komi et al. (1987) developed a “buckle” transducer that was placed in the Achilles tendon, measuring forces when the individuals walked, ran, stretched, and jumped. Typically, direct measurement of these forces is not usually feasible due to the invasive nature of the technique. Numerous potential problems could occur including improper force transducer placement, the transducer dislodging under loads, cost, and the time consuming, invasive nature of the device.

Due to these limitations, muscle forces cannot be measured noninvasively and different muscle modeling approaches have been developed to estimate in vivo muscle and joint forces and joint loads. The two most common techniques are inverse and forward dynamics models. Anderson and Pandy (2001) found that for relatively low-
frequency movements (including walking) both methods predicted similar muscle force values \(^68\).

In the inverse dynamics approach, noninvasive measurements of body position including position, velocity, and acceleration of each body segment, as well as the external forces (obtained from a force plate) are used to calculate joint torques and muscle forces \(^66\). Inverse dynamics (or static optimization) solves a different optimization problem at each instant during movement. This torque-driven model inputs the obtained kinematic data (position, velocity and acceleration) as well as the external force data to calculate muscular joint torques \(^66,69\).

\[
T_{MT} = M(q)q_1 + G(q) + E
\]

(Equation 1)

Where, \(T_{MT}\) are muscular joint torques; \(q, q_1\) are vectors of the generalized coordinates, velocities, and accelerations, respectively; \(G(q)\) is the gravitational loading; and \(E\) represents external force. Joint torques calculated from this model are used to calculate individual muscle forces (more details on this process can be found under the Inverse Dynamics heading in the Methods section). Using inverse dynamics, however, does not come without its limitations. The primary limitation is that the validity of results depends on the accuracy of the data collected during the gait analysis, specifically the positions, velocities, and accelerations of the body segments. Errors here could affect the calculated values of the net joint torques and therefore the estimation of muscle forces. It is also difficult to include muscle physiology into the formulation because estimates of muscle length and contraction velocity also depend on the accuracy of the positions and velocities of the body segments \(^66\).
A model developed by DeVita and Hortobagyi (2001) used quadriceps, gastrocnemius, and hamstring forces from inverse dynamics equations to calculate knee compressive and AP shear forces. This model has advantages in that it is easier to validate, it can determine individual muscle groups and muscle forces, and it involves less calculation and estimation of values. A major limitation of this model is that it is a “lumped muscle model” and cannot distinguish muscle forces from smaller muscle units. Also, the model cannot separate joint forces into lateral and medial compartment loads, and would likely lead to an underestimation of the knee compressive forces. More information about this model as well as some other limitations can be found in Appendix I.

Buchanan et al. notes several limitations with this model. Primarily, joint moments and joint reaction forces are calculated as net values, arguing that the model is better described as a “lumped” muscle model as opposed to an individual muscle model. The transformation of joint moments to muscle forces yields several solutions that cannot be readily determined because of the large number of muscles involved to produce the muscle force. They also noted that currently, there is no model that will calculate muscle activation by inverse transformation from muscle forces.

The forward dynamics model uses calculations of movements and external reaction forces based on known inertial forces of body segments. Muscle excitations (or muscle activations) are used as the inputs to calculate the corresponding body motions. Different than inverse dynamics, the forward dynamics (or dynamic optimization) model solves one optimization problem for one complete cycle of movement. This is where the most differences lie between the two models, and also the reason that forward dynamics
is also more computationally expensive. First, the neural command needs to be estimated or measured. This can be done using EMG techniques or using a mathematically based optimization approach. Neural command is then transformed to muscle forces by musculotendon dynamics that involves calculation of muscle forces by the musculotendon length, musculotendon shortening velocity, and muscle activation. Musculoskeletal geometry is used to calculate joint torques from muscle forces. From there, the joint torques are applied to equations of skeletal dynamics to predict and measure body motions. These steps while being computational expensive, also involve complex and nonlinear relationships. However, this model is considered to be more powerful because the system equations are integrated forward in time. This allows the muscle physiology to be easily incorporated into the formulation.

**Summary Statement**

Numerous researchers have examined the effects of alignment and body mass on the progression and incidence of knee OA. Given that changes in the joint loading could be a possible biomechanical pathway to the progression of knee OA, some researchers have also examined the effect of BMI and knee alignment on the loading at the knee joint. These studies all use the internal abduction moment to detect the loading in the medial compartment of the tibiofemoral joint, a measure we argue to be only sensitive to individuals with varus alignment. To our knowledge, only one previous study has looked at the interaction of these two potent risk factors with respect to the loading at the knee joint (via the internal abduction moment.)
Therefore, the goal of this investigation was to examine the influences alignment and BMI have on the knee joint loads (measured by the bone-on-bone compressive forces and the internal knee abduction) in an obese older adult population while controlling for covariates, walking speed and gender. The hypothesis was that there would be an interaction between alignment and BMI on the knee joint loads after controlling for walking speed and gender.
METHODS

The Intensive Diet and Exercise for Arthritis (IDEA) study compared the effects of intensive dietary restriction-plus-exercise, intensive dietary restriction-only, and exercise-only interventions on inflammatory biomarkers and knee joint loads in overweight and obese adults with knee OA. This thesis utilizes the baseline data from a biomechanical subset of participants.

Study Sample

Participants in IDEA consisted of ambulatory, community-dwelling persons age \( \geq 55 \) years with: (1) K-L grade II-III radiographic OA in one or both knees; (2) BMI 27 to 42 kg/m\(^2\), either overweight or class I or II obese according to the latest NHLBI definitions\(^{73}\); and (3) a sedentary lifestyle, defined as not participating in a formal exercise program at least 3 days a week in the last 6 months.

Exclusion criteria included: (1) significant cognitive impairments (3MSE < 70); (2) significant co-morbid disease including active cancer, anemia, severe renal insufficiency, COPD, type 1 diabetes, severe CAD, severe hypertension, dementia, liver disease, blindness, or osteoporosis; (3) inability to walk; (4) previous acute knee injury or knee OA other than tibiofemoral; (5) excess alcohol use (>21 drinks/week); (6) conditions the prohibit obtaining a knee MRI; (7) unwillingness or inability to change eating and physical activity habits due to environment; (8) cannot read or speak English; (9) lives more than 50 miles from site or planning to leave area 3+ months during the next 18 months; and (10) failure to complete a graded exercise text (GXT) or obtaining a positive test. Further, persons with any type of injection (i.e. cortisone, hyaluronic acid,
etc.) within the past month will be excluded, as well as those with any type of knee surgery in the past six months. Person scoring 14-20 on the depression scale (CES-D) will also be excluded.

Eligible participants were randomized into one of three intervention groups: an intensive dietary restriction-plus-exercise (D+E) group, and exercise-only (E) group, or an intensive dietary restriction-only (D) group. Participants were recruited mainly through mass mailings and print media.

Screening Visits

After expressing interest in participating in IDEA, passing the initial eligibility criteria during the pre-screening visit, and signing the informed consent, each participant attended two screening visits to establish eligibility and a randomization visit. A randomized subset (50% of the total population) received additional testing including a MRI, CT scan, a full length lower extremity x-ray, grip strength and knee strength tests, and leg power tests. The full length lower extremity x-ray was used to assess knee alignment.

Prescreening Visit (PSV): Individuals who responded to advertising and contacted the recruiting office were asked questions focusing on major eligibility criteria. A screening visit appointment was made for participants meeting eligibility and they were sent a medical history form and medication form to complete.

Screening Visit One (SV1): Individuals were scheduled to visit the Clinical Research Center at Wake Forest University. SV1 included an explanation of the study and obtaining the informed consent. Medical history and medication forms were assessed as
well as height and weight (to calculate BMI), and a knee exam to determine the source of knee pain and exclude pes anserine and iliotibial band friction syndrome. Further, the 3MSE was administered and participants who scored < 70 were excluded from the study.

Those not excluded had an interview with an interventionist to assure their ability and willingness to comply. Participants then underwent a graded exercise test (GXT). A positive test was reason for exclusion and was defined as ≥ 2 mm ST depression at ≤ 4 METS, hypotension, or complex arrhythmias. These results and a written explanation were sent to the participant’s physician. Participants meeting all eligibility criteria had bilateral standing (semi-flexed view) and sunrise view knee x-rays done performed at the Outpatient Radiology at Wake Forest University Baptist Medical Center. A physician blinded to treatment groups was chosen to read all radiographs. After the knee x-rays were evaluated, those scoring a 2 (mild) or 3 (moderate), as defined by the radiographic criteria of Kellgren and Lawrence, were included in the study. At the end of SV1, the participants met with a nutritionist at the General Clinical Research Center at Wake Forest University Baptist Medical Center who trained them to track their food intake. They were given a 3-day food diary.

X-ray

Bilateral anterior-posterior (AP) weight-bearing knee x-rays were used to identify tibiofemoral arthritis and sunrise views to identify people with patellofemoral OA. Severity of OA was determined using the Kellgren and Lawrence (K-L) score. K-L scoring is as followed: grade 0: definite 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, and grade 4: severe x-ray changes. The radiological changes that were considered to be evidence of OA were: formation of osteophytes on the joint margins, narrowing of joint cartilage, sclerosis of subchondral bone, pseudocystic areas with sclerotic walls in the subchondral bone, periarticular ossicles, and altered shape of the bone ends 19,75. A single physician, masked to the treatment group, read the radiographs.

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

Figure 10: Plexiglas frame for foot fixation and rotation
**Figure 11:** Proper patient position for knee x-ray

The knee of interest was centered on film. Body weight was evenly distributed between both legs and the knees were flexed until the knees and anterior thigh contacted the Bucky (about 15 degrees). The source image distance was 100 cm, and was held constant throughout the study. The x-ray beam was centered on the joint line of the knee of interest posteriorly and was angulated caudally to 10 degrees.

Criteria for image evaluation included: 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 the joints including the osteophytes, were on the film; 6) left/right markers were on each film; 7) film was not under/over exposed; 8) the metallic beads on the center of the positioning guide were visualized on the radiograph.
Screening Visit Two (SV2): Participants returned to Reynolds Gymnasium at Wake Forest University to perform tests and complete questionnaires including: height and weight, 6-minute walk, demographic information, WOMAC, Physical Activity Scaled for the Elderly (PASE), FAST 23, Health Related Quality of Life (HRQL), Mini-Mental State Exam (MMSE), gait analysis, and hip and waist circumference. Half of the participants were randomly selected for additional testing including knee and grip strength, balance, the Digit Symbol Substitution Test (DSST), and the Short Physical Performance Battery (SPPB). A complete description of all of the tests can be found elsewhere (Messier et al., 2009). The tests that pertain to this study are described below.

**Height and Weight Measurements**

Height was measured with a vertically mounted stadiometer. Participants were asked to stand erect without shoes with feet together and height was recorded to the nearest 0.1 cm. Participants were weighted on an electric scale without shoes and wearing only light clothing and the weight was recorded to the nearest 0.1 kg. BMI was calculated as mass (measured in kilograms) divided by height squared (measured in meters).

**Gait Analysis**

All participants eligible for the study underwent a 3-D gait analysis test. Participants were prepped with a 37-relective maker set, arranged in the Cleveland Clinic full-body configuration and asked to wear a pair of shoes provided by the study to control for any variations that shoe type could have on the results. Three-dimensional high-speed videography (60 Hz) was accomplished using a 6-camera motion analysis system.
Kinematic data were collected, tracked, edited, and smoothed using EVaRT 4.6 software (Motion Analysis Corporation, Santa Rosa, CA) and raw coordinate data were smoothed using a low-pass Butterworth filter set at a cut-off frequency of 6 Hz. The processed data were compiled using OrthoTrak 6.0 beta4 clinical gait analysis software (Motion Analysis Corp., Santa Rosa, CA) to generate lower extremity kinematic and kinetic data.

During testing, participants walked at their preferred walking speeds on a 16.8 meter walkway located in the J.B. Snow Biomechanics Laboratory. Freely chosen walking speed was assessed using a Lafayette Model 63501-IR (Lafayette Instruments Co., Lafayette, IN) photoelectric control system with interfaced digital timers (Model 54035, Lafayette Instruments Co., Lafayette, IN) placed at waist level 7.3 meters apart. The average time for six trials was computed and a range of acceptable walking speeds was calculated from the average time ± 3.5%. During testing, the participant started no less than 2 meters before the first timer and walked past the beam of light to activate it. When they reached the second timer and crossed that beam of light, the timer was stopped and the time it took to walk from one to the other was recorded. Successful trials were defined placing the entire foot on the force platform during a normal stride while maintaining the freely chosen walking speed (± 3.5%). Three successful trials were collected and averaged for each side (left and right leg) to provide a representative trial for each participant.

A 6-channel force platform (OR6-5-1, Advanced Medical Technologies, Inc., Newton, MA) was integrated with the motion capture system and allowed simultaneous kinetic data collection at 480 Hz. OrthoTrak 6.0 software was used to calculate joint
moments and joint reaction forces. These data served as input to calculate the knee joint forces and moments. Kinematic and kinetic data were synchronized and an inverse dynamics model developed by DeVita and Hortobagyi was used to calculate knee joint forces and moments including internal knee extension and abduction moments, knee joint muscle forces, and knee joint compressive and shear forces.

Musculoskeletal Model

There are two basic components of the musculoskeletal torque-driven model used in this thesis. The first component calculates joint moments and joint reaction forces from kinematic, physiological, and force platform data collected during the gait visit. The second component uses these joint moments and joint reaction forces to generate individual muscular forces, and knee bone-on-bone compressive and shear forces. These computations include: 1) determining the forces of the quadriceps, hamstrings, and gastrocnemius muscles; 2) applying these forces along with joint reaction forces onto the tibia, and 3) determining the knee joint compressive and anteroposterior shear forces. Detailed information about this torque-driven model can be found in Appendix I.

MRI Visit Baseline (MR0): The half of the participants randomized for supplemental testing during the SV2 visit completed the additional measurements during the MRI visit. These tests included, but were not limited to, a full length lower extremity x-ray.

Full Length X-Ray

Full length x-rays were obtained to assess alignment at outpatient radiology at Wake Forest University Baptist Medical Center. A full length anteroposterior (AP) radiograph was obtained at baseline (and to be obtained later at follow up) using the Agfa
ADC System (Quantum Q-Rad CR-based imaging) approach. Participants were positioned using the methods of Sharma et al. 7 and both lower extremities were imaged simultaneously. The focus distance was set at 80 inches unless the hip joint was not visualized at 80 inches (was possible in long-legged individuals – then the focus distance was increased to include the hip). Alignment was defined as the measure of the angle formed by the intersection of the line connecting the centers of the femoral head and the intercondylar notch and connecting the centers of the ankle talus and tibial spines. A varus knee is an angle >2° in the varus direction (in the direction of a bowlegged appearance), and valgus in an angle >0° in the valgus direction (in the direction of a knock-kneed appearance). A neutral knee had an angle of 0-2° in the varus direction 79. All of the measurements were made by a single physician and done using the NIH ImageJ program.

**Figure 12**: Examples of neutral, varus, and valgus alignment (with permission from Lenna Sharma, M.D.)

Participants stand on a step stool without shoes and their back to the Bucky to ensure the ankle was included in the radiograph. Both tibial tubercles were made sure to
be facing directly forward. Two permanent marks were made on the step stool that were 6 inches apart to ensure the subject’s feet were 6 inches apart by lining up their great toes with these marks. Participants were asked to bear weight equally on both limbs. The central ray was placed perpendicular to the knee directed midway between the two knees at the level of the joint spaces and was ensured to be at 0 degrees. The position of the tibiofemoral joint space was found by locating the inferior border of the patella the superior margin of the tibial tuberosity.

Statistics

A variety of statistical analyses were used in order to examine the influence of both alignment and BMI had on the knee joint loading. First, distribution and simple statistics including means, standard deviations, and ranges were calculated on the forces, moments, and alignment measures to ensure normal distribution of the sample. Pearson correlations were calculated to evaluate the strength of the relationships between BMI and the force measures, as well as walking speed and the force measures.

Linear regressions were fitted to model the relationship between the outcome measures (joint loads) with the independent measures of BMI and knee alignment while controlling for gender and walking speed. The analysis permitted a direct relationship between the outcome and predictor variables and allowed us to control for several covariates. The regression coefficients for the independent measures were used to provide the magnitude and direction of the association.

Given a significant interaction did exist, after adjusting for gender and walking speed, the general form of the equation was: Knee Force or Moment measure = \( \beta_0 + \beta_1 * \)
gender + β₂ * walking speed + β₃ * Varus Alignment + β₄ * Valgus Alignment + β₅ * BMI + β₆ * (Alignment * BMI) where:

- Force or Moment measure = dependent or outcome variable of interest
- β₀ = intercept (constant)
- β₁ = either an increase or decrease in the outcome for females versus males
- β₂ = either an increase or decrease in the outcome, per unit difference in walking speed
- β₃ = either an increase or decrease in the outcome for varus versus neutral alignment
- β₄ = either an increase or decrease in the outcome for valgus versus neutral alignment
- β₅ = either an increase or decrease in the outcome, per unit difference in BMI
- β₆ = either an increase or decrease in the outcome, per interaction of alignment with BMI. The interaction indicates that the association of BMI with the outcome varies depending on the alignment.

Given a significant interaction did not exist, the main effects of each variable were examined. After adjusting for gender and walking speed, the general form of the equation was: Knee Force or Moment measure = β₀ + β₁ * gender + β₂ * walking speed + β₃ * Varus Alignment + β₄ * Valgus Alignment + β₅ * BMI, as defined above.
Through the analysis, the statistical significance level was less than 0.05. All statistical analyses were conducted using the SAS software package.
RESULTS

Descriptive Characteristics

The purpose of this cross-sectional investigation was to determine the influence of alignment and BMI on knee joint loads in overweight and obese adults with knee OA. Of the 454 participants enrolled in the IDEA clinical trial, 157 were assigned to full biomechanics testing and had full length lower extremity radiographs administered. Mean and standard deviations for selected descriptive characteristics for this subset are summarized in Table 1. Mean values and standard deviations for the alignment data (gathered from the full length radiograph) are summarized in Table 2. The alignment data were divided into three categories: varus, valgus, and neutral alignment. Having a knee alignment measure greater than or equal to 2 degrees was indicative of a varus knee, and those having a negative measure were classified as having a valgus knee. Measures between zero and less than 2 degrees varus were considered neutral.

Table 1: Descriptive characteristics of the population.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Alignment Cohort N = 157</th>
<th>Total IDEA N = 454</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>108 (68.8%)</td>
<td>325 (71.6%)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, n (%)</td>
<td>129 (82%)</td>
<td>377 (83%)</td>
</tr>
<tr>
<td>BMI (kg • m⁻²)</td>
<td>33.4 (3.7)</td>
<td>33.6 (3.7)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>92.6 (13.9)</td>
<td>92.9 (14.7)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>66 (6.2)</td>
<td>66 (6.2)</td>
</tr>
<tr>
<td>WOMAC pain</td>
<td>6 (2.9)</td>
<td>7 (3.1)</td>
</tr>
<tr>
<td>WOMAC function</td>
<td>22.9 (10.8)</td>
<td>24.2 (10.9)</td>
</tr>
<tr>
<td>Walking speed (m/s)</td>
<td>1.2 (0.2)</td>
<td>1.2 (0.2)</td>
</tr>
</tbody>
</table>
Table 2: Alignment data at baseline.

<table>
<thead>
<tr>
<th>Alignment</th>
<th>N</th>
<th>Percent (%)</th>
<th>Alignment Angle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>Varus</td>
<td>76</td>
<td>48</td>
<td>5.6</td>
</tr>
<tr>
<td>Neutral</td>
<td>42</td>
<td>27</td>
<td>1.23</td>
</tr>
<tr>
<td>Valgus</td>
<td>39</td>
<td>25</td>
<td>-2.69</td>
</tr>
</tbody>
</table>

The outcomes of interest from the baseline biomechanical testing are presented in Table 3. The kinetic variables included the peak values of the forces obtained from our inverse dynamics model (anteroposterior shear, compressive, and patellofemoral forces). Muscle forces were also calculated using the inverse dynamic model with hamstrings, quadriceps, and gastrocnemius noted below. The peak values of the internal abduction moment and internal knee extension moments are also included.
Table 3: Mean (SD) bone-on-bone and muscle forces, and knee internal moments during walking. Mean body weight = 912 N (93 kg).

<table>
<thead>
<tr>
<th></th>
<th>Mean N = 157 (%BW)</th>
<th>Standard Deviation</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compressive force (N)</td>
<td>2645 (2.9)</td>
<td>873</td>
<td>1241</td>
<td>6337</td>
</tr>
<tr>
<td>PF compressive force (N)</td>
<td>430 (0.47)</td>
<td>345</td>
<td>1.3</td>
<td>2300</td>
</tr>
<tr>
<td>Shear force (N)</td>
<td>408 (0.45)</td>
<td>156</td>
<td>88</td>
<td>894</td>
</tr>
<tr>
<td>Peak Knee Abductor Moment (Nm)</td>
<td>30</td>
<td>13</td>
<td>2.2</td>
<td>69</td>
</tr>
<tr>
<td>Peak Knee Extension Moment (Nm)</td>
<td>36</td>
<td>22</td>
<td>-12</td>
<td>122</td>
</tr>
<tr>
<td>Hamstring Force (N)</td>
<td>660 (0.72)</td>
<td>320</td>
<td>174</td>
<td>2085</td>
</tr>
<tr>
<td>Quadriceps Force (N)</td>
<td>1257 (1.38)</td>
<td>608</td>
<td>0</td>
<td>3926</td>
</tr>
<tr>
<td>Gastrocnemius Force (N)</td>
<td>712 (0.78)</td>
<td>154</td>
<td>366</td>
<td>1245</td>
</tr>
</tbody>
</table>

Our results showed that the mean peak compressive force was 2.9 times mean body weight, patellofemoral compressive force was 0.47 BW and shear force was 0.45 BM. In addition, quadriceps muscle force was 1.4 BW, hamstring force was 0.7BW and gastrocnemius force was 0.8BM.

Relationships of Alignment and BMI with Knee Joint Loads

Baseline measures of alignment and BMI were included in the linear regression models to predict the knee joint loads (via both bone-on-bone forces and joint moments) after adjusting for gender and walking speed. Beta coefficients, standard error, and p-values were calculated. Modeling results showed there was no significant interaction
between knee alignment and BMI in predicting peak knee joint forces, peak muscle forces, and knee internal extension and abduction moments (Table 4). For example, Figure 13 demonstrates that the relationship between BMI and the knee compressive forces is not significantly different among the three alignment tertiles. This suggests that regardless of the alignment, those with the largest BMI had the greatest knee compressive forces. Figure 14 signifies that the relationship between alignment and the internal abduction moment is not significantly different among the three increasing BMI categories, indicating that regardless of the individual’s BMI, those with varus alignment have larger abduction moments that those with neutral or valgus alignments. In contrast, the peak internal extension moment was not significant related to either BMI or alignment.

**Figure 13**: There was no significant interaction between knee alignment and BMI in predicting the compressive forces at the knee (p = 0.28). For each alignment category, higher BMIs resulted in greater knee compressive forces.
Figure 14: There was no significant interaction between knee alignment and BMI in predicting the knee abduction moment ($p = 0.65$). For each BMI category, varus alignment had the greatest knee abduction moment.

Relationship of Alignment with Knee Joint Forces and Moments

After adjusting for gender and walking speed, modeling results showed a significant association of alignment with the knee abduction moment, independent of BMI ($p < 0.0001$) (Table 4). The $\beta$ coefficients were largest for varus alignment ($\beta = 8.12$) such that individuals with varus alignment, on average had 8.12 Nm higher mean abduction moment than those with a neutral alignment. Those with valgus alignment, on average had abduction moments that were 10.07 Nm less than those with a neutral alignment ($p < 0.0001$, $\beta = 10.07$). Further, as depicted in Table 5 (the mean forces and moment for each alignment category), the mean abduction moment is greatest for varus, followed by neutral, and then valgus. All other kinetic gait characteristics were not significantly different between the alignment groups (Table 5). In the model, female
gender was associated with lower forces and moments (e.g. for compressive forces, $\beta = -730N$), and higher walking speed was associated with higher forces and moments (e.g. for compressive forces, $\beta = 2075$), except for the abduction moment ($p = 0.83$, $\beta = -1.03$).

**Table 4**: Modeling results between joint compressive forces and knee abduction moment with alignment and BMI. All $\beta$ coefficients for moments are in Nm and for joint forces are in N. Alignment is measured in degrees. BMI is reported in kg/m$^2$. P values are noted for covariates (gender and walking speed) and for varus and valgus alignment relative to a neutral alignment. *Walking speed obtained from gait analysis

<table>
<thead>
<tr>
<th>Joint Load</th>
<th>Gender [0 = male, 1 = female]</th>
<th>Speed (m/s)</th>
<th>Alignment</th>
<th>Valgus [0 = no, 1 = yes]</th>
<th>Varus [0 = no, 1 = yes]</th>
<th>p-value</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compressive Force (N)</td>
<td></td>
<td>2075</td>
<td>48</td>
<td>104.7</td>
<td>124.1</td>
<td>0.69</td>
<td>63</td>
</tr>
<tr>
<td>Shear Force (N)</td>
<td></td>
<td>287</td>
<td>-41</td>
<td>-8.8</td>
<td>26</td>
<td>0.33</td>
<td>10.1</td>
</tr>
<tr>
<td>PF Force (N)</td>
<td></td>
<td>617</td>
<td>30</td>
<td>71</td>
<td>59</td>
<td>0.48</td>
<td>9.7</td>
</tr>
<tr>
<td>Knee Abductor Moment (Nm)</td>
<td></td>
<td>-1.03</td>
<td>-10.07</td>
<td>8.12</td>
<td>&lt;0.0001</td>
<td>-0.03</td>
<td>-0.24</td>
</tr>
<tr>
<td>Knee Extension Moment (Nm)</td>
<td></td>
<td>43.63</td>
<td>4.31</td>
<td>3.78</td>
<td>0.69</td>
<td>0.75</td>
<td>0.43</td>
</tr>
<tr>
<td>Hamstring Force (N)</td>
<td></td>
<td>886</td>
<td>91</td>
<td>60</td>
<td>48</td>
<td>0.23</td>
<td>21.3</td>
</tr>
<tr>
<td>Gastrocnemius Force (N)</td>
<td></td>
<td>251</td>
<td>17</td>
<td>-26</td>
<td>18</td>
<td>0.07</td>
<td>16.6</td>
</tr>
<tr>
<td>Quadriceps Force (N)</td>
<td></td>
<td>1331</td>
<td>-22</td>
<td>72</td>
<td>96</td>
<td>0.58</td>
<td>28.3</td>
</tr>
</tbody>
</table>
Table 5: Mean (SD) bone-on-bone and muscle forces, and knee internal moments during walking in varus, valgus, and neutral alignment groups, controlling for BMI, gender, and walking speed. Mean body weight = 912 N (93 kg). Mean alignment for varus = 5.6°; valgus = -2.7°; neutral = 1.2°.

<table>
<thead>
<tr>
<th>Knee Force</th>
<th>Mean (SE)</th>
<th>Valgus</th>
<th>Neutral</th>
<th>Varus</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compressive Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2767 (106)</td>
<td>2719 (103)</td>
<td>2824 (74)</td>
<td></td>
<td></td>
<td>0.69</td>
</tr>
<tr>
<td>Shear Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>401 (22)</td>
<td>442 (22)</td>
<td>433 (15)</td>
<td></td>
<td></td>
<td>0.33</td>
</tr>
<tr>
<td>PF Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>455 (52)</td>
<td>425 (50)</td>
<td>496 (35)</td>
<td></td>
<td></td>
<td>0.48</td>
</tr>
<tr>
<td>Knee Abductor Moment (Nm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18.9 (1.8)</td>
<td>29.0 (1.8)</td>
<td>37.1 (1.3)</td>
<td></td>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Knee Extension Moment (Nm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35.8 (3.2)</td>
<td>38.1 (3.1)</td>
<td>39.1 (2.3)</td>
<td></td>
<td></td>
<td>0.69</td>
</tr>
<tr>
<td>Hamstring Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>732 (41)</td>
<td>641 (40)</td>
<td>700 (28)</td>
<td></td>
<td></td>
<td>0.23</td>
</tr>
<tr>
<td>Gastrocnemius Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>733 (16)</td>
<td>757 (15)</td>
<td>731 (11)</td>
<td></td>
<td></td>
<td>0.07</td>
</tr>
<tr>
<td>Quadricep Force (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1286 (82)</td>
<td>1308 (80)</td>
<td>1380 (57)</td>
<td></td>
<td></td>
<td>0.58</td>
</tr>
</tbody>
</table>

Relationship of BMI with Knee Joint Forces and Moments

When fitting the regression model for BMI and alignment while controlling for gender and walking speed, BMI was associated with compressive force, shear force, and hamstring, gastrocnemius, and quadriceps muscle forces, independent of knee alignment (Table 4). The β coefficient for the association between compressive force and BMI was 62.9 (p < 0.0001) such that with every 1 unit increase in BMI, there was a 63 N (14 lb) increase in compressive forces at the knee per step, independent of alignment. Overall, BMI affected the bone-on-bone joint forces, but alignment did not.

Table 6 shows the mean values for all gait characteristics between increasing BMI tertiles, after controlling for gender and walking speed. There was a significant positive
relationship between BMI and compressive forces (p = 0.001), shear forces (p = 0.005), hamstring muscle forces (p = 0.005), and gastrocnemius forces (p < 0.0001). The internal knee abduction moment did not show any significant increases in value with an increase in BMI.

Table 6: Mean (SD) bone-on-bone and muscle forces, and knee internal moments during walking in separate BMI tertiles, controlling for alignment, gender and walking speed.

Mean body weight = 912 N (93 kg).

<table>
<thead>
<tr>
<th>Knee Force</th>
<th>BMI (kg/m$^2$) Mean (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>27 – 29.9</td>
</tr>
<tr>
<td>Compressive Force (N)</td>
<td>2441 (113)</td>
</tr>
<tr>
<td>Shear Force (N)</td>
<td>373 (23)</td>
</tr>
<tr>
<td>PF Force (N)</td>
<td>410 (54)</td>
</tr>
<tr>
<td>Knee Abductor Moment (Nm)</td>
<td>27.0 (1.9)</td>
</tr>
<tr>
<td>Knee Extension Moment (Nm)</td>
<td>34.4 (3.4)</td>
</tr>
<tr>
<td>Hamstring Force (N)</td>
<td>573 (43)</td>
</tr>
<tr>
<td>Gastrocnemius Force (N)</td>
<td>660 (16)</td>
</tr>
<tr>
<td>Quadricep Force (N)</td>
<td>1178 (86)</td>
</tr>
</tbody>
</table>

The Pearson correlation coefficients were calculated between BMI and the knee force variables (Table 7). They show that there was a significant positive correlation between BMI and compressive forces (r = 0.19), shear forces (r = 0.19), and the gastrocnemius muscle forces (r = 0.33). There was a significant negative correlation between BMI and walking speed (r = -0.17). Table 7 also shows the Person correlations for walking speed with the force variables, showing that as walking speed increases, there was an increase in all force variables, except for the abduction moment (r = -0.007). BMI and walking speed were also significantly negatively correlated (r = -0.17).
Table 7: Pearson Correlation Coefficients for BMI and walking speed with each other and knee force measures.

<table>
<thead>
<tr>
<th>Knee Force</th>
<th>Pearson r p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td>Compressive Force (N)</td>
<td>-0.17</td>
</tr>
<tr>
<td></td>
<td>0.035</td>
</tr>
<tr>
<td>Shear Force (N)</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>0.017</td>
</tr>
<tr>
<td>PF Force (N)</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>0.57</td>
</tr>
<tr>
<td>Knee Abductor Moment (Nm)</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>0.54</td>
</tr>
<tr>
<td>Knee Extension Moment (Nm)</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>0.41</td>
</tr>
<tr>
<td>Hamstring Force (N)</td>
<td>0.15</td>
</tr>
<tr>
<td></td>
<td>0.06</td>
</tr>
<tr>
<td>Gastrocnemius Force (N)</td>
<td>0.33</td>
</tr>
<tr>
<td></td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Quadriceps Force (N)</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>0.1864</td>
</tr>
</tbody>
</table>
DISCUSSION

Malalignment of the knee increases the loading at the joint by concentrating the forces to a more focal area. Although, the effect that obesity has on OA incidence and progression has been less clear and may depend on the extent of malalignment. As expected, our results found BMI and alignment were associated with knee joint loading; however, these associations were detected in different measures. We also found that there was no interaction between BMI and alignment with knee joint loads after controlling for gender and walking speed, signifying that these two risk factors do not depend on or influence one another relative to knee joint loading, a major factor in the osteoarthritis disease pathway.

The interaction between obesity and malalignment was examined previously in a study by Moyer et al. (2010). The authors found a significant interaction with body mass and alignment on knee joint loading such that the there was a significant association between alignment and the external adductor moment in patients with the highest mass. We are unaware of any previous research that used a more direct measure of knee joint loading (i.e. the compressive forces at the knee) to examine these relationships.

Measures of Knee Joint Loading

As previously stated, the internal abduction moment of the knee is commonly used as a measure of internal medial compartment force because it predicts OA progression. Schipplein and Andriacchi proposed that the knee abduction moment is the primary determinant for medial compartment load during gait since it produces higher medial joint reaction forces at the knee. Previous studies, therefore, have suggested that
the internal abduction moment can be used as a surrogate measure for the loading at the knee joint\textsuperscript{47, 51, 54, 80}. Measures to examine the lateral compartment of the knee, to our knowledge, have not been validated.

We theorize that using the knee joint compressive force may be a more sensitive measure to examine the loading across the entire knee joint. The compressive forces at the knee, calculated by a biomechanical model developed by DeVita and Hortabagyi (2001)\textsuperscript{70}, depict the overall muscle forces acting across the entire knee joint. The abduction moment is primarily related to medial compartment loading and is only positively correlated with adults who have knee OA and varus malalignment\textsuperscript{12, 53, 81}.

Alignment and Knee Joint Forces and Moments

Sharma et al. (2001) conducted the first study to demonstrate that alignment can increase the risk of OA progression. Their study concluded that individuals with varus malalignment had an increased risk of medial OA progression and those with valgus malalignment had an increased risk of lateral OA progression\textsuperscript{5}. OA is a result of local mechanical factors acting with certain site-specific factors that determine how the load is distributed across the articular cartilage\textsuperscript{28}. The abduction moment had been linked to OA progression in several studies\textsuperscript{12, 53}, establishing its importance as a measure of joint loading. Our results showed that there was a significant association between varus alignment and the abduction moment after controlling for gender and walking speed (p <0.0001, $\beta = 8.12$). This suggests that individuals with varus alignment have on average, an abduction moment that is 8.12 Nm greater per step than those with a neutral alignment. In our population, this would predict a 27\% greater abduction moment (mean
abduction moment = 29.6 Nm) in those with varus malalignment relative to neutral. These data confirm the powerful influence varus malalignment has on the internal abduction moment \(^{38,55}\), and subsequently on OA disease progression.

Our results also showed a negative association with valgus alignment and the knee abduction moment (\(p < 0.0001, \beta = -10.07\)), suggesting that individuals with valgus alignment tend to have an abduction moment on average 10.07 Nm less per step than those with neutral alignment. This would predict a 33% reduction compared to a neutral alignment per step walked. This further demonstrates that the abduction moment is primarily sensitive to individuals with varus malalignment, since we would expect individuals with valgus alignment to still have excess loading in the lateral compartment of the knee. Also, these results were all independent of BMI (\(p = 0.90\)), suggesting that increases in the amount of varus alignment will increase the abduction moment, regardless of an individual’s BMI. Given these results, we suggest that the internal abduction moment may not be sensitive to changes in BMI, and that it may be measuring loading caused by frontal plane movements and not obesity.

There was a dose-response relationship (\(p < 0.0001\)) between the abduction moment and alignment, with the highest abduction moment occurring with varus malalignment (37 Nm), followed by neutral alignment (29 Nm), and valgus alignment (19 Nm). These data support the limited use of the abduction moment as a surrogate measure of knee joint loading to those with varus malalignment. The work of Moyer et al (2010) also showed a dose response relationship between alignment and the internal knee abduction moment \(^{13}\), support this.
Alignment had no significant association with knee compressive force after controlling for walking speed and gender (p = 0.69). This indicates that alignment does not have a significant effect on the bone-on-bone forces at the knee. Alignment also did not have an effect the shear or patellofemoral force or on any of the muscle forces (quadriceps, hamstrings, or gastrocnemius), that we would expect to be altered with an increase in knee joint loading.

**BMI and Knee Joint Forces and Moments**

Messier et al. (2005) conducted a prospective study examining the effects of weight loss on the knee joint loads in 142 sedentary overweight and obese individuals with knee OA. They concluded that for every 1 kg of weight loss there was a 40.6 N reduction in the knee compressive forces, further equated to for every one pound lost there is a 4-fold reduction in the load exerted on the knee per step. While our study did not examine the effects of weight loss on knee joint loading, we were able to detect similarities in the effect that body mass has on the knee joint. Our results found that for every 1 unit increase in BMI, there was a 63 N (14 lb) increase in compressive forces at the knee per step (p <0.0001, β = 63). This would account for a predicted 2% increase in knee compressive forces (mean compressive forces = 2644 N) per step. After observing that varus alignment alone predicts a 27% increase in the abduction moment, the predicted 2% increase appears as less drastic, but given the vast range of BMI values possible, the growing obesity epidemic, and the thousands of steps taken per day, a 2% increase per unit of BMI could be clinically important. These results were independent of knee alignment, indicating that the relationship between BMI and knee compressive forces was unchanged with varus, valgus, and neutral alignments. Further, there was also
a dose-response relationship in that as BMI increased, there was a significant increase in the knee compressive forces (p = 0.01).

![Figure 15: Mean Knee Compressive Forces by BMI groups, Independent of Alignment](image)

**Figure 15:** Figure signifying that with an increase in BMI, there was a subsequent increase in the knee compressive forces, independent of alignment (p = 0.001).

Mean BMI = 33.4 kg/m²; mean compressive forces: 2645 N.

Interestingly, we did not find a significant relationship between BMI and the knee abduction moment (the surrogate measure for knee joint loading) (p = 0.90) suggesting that BMI has no influence on the abduction moment. If obesity increases the overall loading at the knee, and the abduction moment is a surrogate measure of this loading, then it would follow that an increase in BMI would be reflected in the magnitude of the abduction moment. The lack of a significant relationship in our study implies that the abduction moment and the knee joint compressive forces are influenced by different OA risk factors.
BMI also had an influence on other kinetic gait variables including the shear forces as well as the modeled muscle forces. These relationships were also all independent of alignment, suggesting that they were similar in varus, valgus, and neutral knees. Interestingly, BMI (nor alignment) had no influence on the patellafemoral forces ($p = 0.15$). Given that individuals with patellafemoral OA (without also having tibiofemoral OA) were excluded from the IDEA trial due to some literature suggesting that other risk factors, such as quadriceps strength and Q-angle $^{82,83}$ may be more important for patellafemoral OA disease, these results are not surprising.

In our musculoskeletal model, the quadriceps, hamstrings, and gastrocnemius muscle forces account for a major portion of the knee compressive forces $^{77,84,85}$. Therefore, differences in the knee compressive forces will be due largely to differences in these muscle forces. Indeed, we saw a positive dose-response relationship between BMI and the compressive forces ($p = 0.001$), the shear forces ($p = 0.005$), the hamstring muscle forces ($p = 0.005$), and gastrocnemius muscle forces ($p > 0.0001$).

Messier et al. (2011) concluded that a 10% loss in weight in an osteoarthritic population elicits changes in the mechanical pathway to knee OA by not only lowering the knee compressive loads during walking, but that this reduction was due to reductions in hamstring co-contraction during the beginning of the stance phase. Our data further support these findings. We found BMI had a significant association with hamstring forces, such that as BMI increased, the hamstring muscle force increased significantly. However, there was no relationship between BMI and the quadriceps forces, suggesting that as BMI increased, there was no change in the quadriceps muscle force. This suggests
that individuals with higher BMIs use more co-contraction, resulting in increased knee joint forces.

The idea of increased co-contraction in obese individuals was also noted by DeVita et al. (2003). DeVita et al. observed that obese individuals had smaller knee extensor moments compared to normal weight individuals, and as obesity increased these internal knee extensor moments decreased, and at very high BMIs changed to an internal knee flexion moment, suggesting that knee joint stability was accomplished primarily with the hamstrings instead of the quadriceps—an idea termed “quadriceps avoidance” \(^6\). Our modeling results did not show significantly smaller knee extension moments with increases in BMI, after controlling for gender, walking speed, and alignment, however, the p-value suggests there was a trend (p = 0.08) with increasing BMI.

BMI also had a significant positive dose response relationship with the gastrocnemius muscle force (p < 0.0001). Since the gastrocnemius contracts eccentrically throughout most of the stance phase to control the forward movement of the body mass, an increase in these forces with increased BMI is not surprising, as more muscle force is required to control the larger mass.

**Interaction of Alignment and BMI with Knee Joint Forces and Moments**

We hypothesized that there would be a significant interaction between BMI and alignment on knee joint loading after controlling for gender and walking speed. As mentioned previously, our results found no significant interaction effect indicating that these two risk factors may influence joint loads differently. We did find that knee
alignment and BMI acted independently to affect different measures of knee joint loading.

In the study by Moyer et al. (2010), as stated before, they did find an interaction between alignment and body mass on the internal abduction moment. Moyer’s results suggested that in the group with the largest mass (over 100 kg) there was a 3.2 Nm increase in knee load (via the abduction moment) for every 1° increase in varus alignment. Further, for those with the most severe varus malalignment, there was a 0.4 Nm increase in knee load for every 1 kg increase in mass. They suggested that body mass clearly moderates the relationship between alignment and the abduction moment, but that alignment does not appear to moderate the relationship between body mass and the abduction moment as drastically. These results by Moyer et al. also found that body mass only accounted for 6-10% of the explained variance, where alignment accounted for up to 54%. Hence, it appears that body mass accounts for little of the variance in the abductor moment and that these results were dominated by alignment. These results could have also faired different from ours due to their larger sample size (487 participants) or that they used body mass while we used BMI.

In addition, while Moyer et al. found the relationship between alignment and the abduction moment to be larger in those with excess body mass (and we found this relationship across all body masses), out of the 487 participants in the study only 50 were classified of having valgus malalignment (10% of the total population), while the remainder were classified as having varus alignment. Previous research suggests that 25% of all osteoarthritic cases are in the lateral compartment, 50% in the medial compartment, and the other 25% being at the patellafemoral joint. Due to Moyer et al.
having a predominantly varus sample, using the abduction moment as their measure of knee joint loading may have picked up larger differences. Had they had more of a representative sample, one could suspect that perhaps the interaction between body mass and knee malalignment may not have been significant, similar to the results we have found. Our sample of 157 participants had 25% (39 individuals out of 147) of individuals classified as having valgus malalignment and thus making our population more representative of a true osteoarthritic sample, and therefore able to look at the true effect this interaction has on the abduction moment among all alignment categories.

Limitations

Limitations in the present cross sectional study should be acknowledged as well. Primarily, the inverse dynamics model developed by DeVita requires a number of assumptions and estimations. It must be understood that this model is a “lumped” muscle model and only created modeled, muscle forces of the quadriceps, hamstrings, and gastrocnemius. These muscles are the primary causes of the internal knee moments, but do not describe a complete representation of the muscle forces acting outside the knee joint. Not having these other muscle groups could lead to potential errors in the estimated forces of the knee.

Secondly, the magnitude, location, and moment of inertia in the segmental masses and mass centers of the lower extremity are estimated based on the relative segmental masses reported by Dempster et al. These estimations are subject to error for any subject who differs from the cadavers in Dempster’s study. Since our adults vary widely in age and BMI, this could likely be a source for error. Further, having obese subjects in
our study could also produce errors due to the fact that adipose tissue in obese individuals could prevent accurate placement of the reflective markers and cause oscillations of body segments that are not representative to the movement of the underlying bones.

Finally, our muscle model neglects to separate the loading in the medial and lateral compartments of the knee, and instead calculates the total tibiofemoral compressive and total anteroposterior shear forces. Our lack of an interaction in our data may be due to the lack of specific compartmental loading. If we were able to detect which compartment, medial or lateral, was loaded during gait, it may be more sensitive to detecting if an interaction with alignment and BMI existed.

Conclusion

Obesity is the most modifiable risk factor for the development and progression of knee OA. Several studies suggest the effect of obesity on knee OA is dependent on knee alignment. Malalignment is thought to progress OA by increasing the loading at the joint by concentrating the loading to a more focal area. The purpose of this investigation was to determine the influence of malalignment and obesity on the knee joint loads in overweight and obese individuals with knee OA. The results showed that larger BMIs were associated with increased peak compressive, shear, and knee muscle forces, and that these relationships were independent of alignment. Further, our data suggest that with every 1 unit decrease in BMI, there was a reduction in the compressive forces at the knee of 63 Nm, regardless of alignment. This stresses the importance of weight loss interventions in individuals with knee OA.
Alignment only had an association with the knee internal knee abduction moment, a surrogate measure of medial compartment knee joint loading in individuals who have knee OA and varus malalignment. However, BMI did not predict the abduction moment, which we would expect if it were a measure of knee joint loading. This result suggests that the knee compressive forces should be utilized in addition to the abductor moment when the effects of BMI on knee joint loading are being studied. When examining the effect of alignment on knee joint loading, the knee abductor moment remains an effective measure. Our results suggest that regardless of an individual’s alignment, obesity influences the tibiofemoral joint forces.

These results can assist researchers in selecting participants at a high risk of OA progression for future randomized clinical trials. When recruiting, researchers should aim for a cohort of individuals at a high risk for disease progression. For the IDEA study, individuals with high BMIs were targeted to examine what effect weight loss had on the knee joint loads. However, based on our data, alignment had the strongest association with the knee abduction moment, a surrogate measure for knee joint loading, and BMI was associated with knee compressive forces, an important component of the OA disease pathway. Therefore, researchers should aim to recruit a sample of osteoarthritic adults with both varus malalignment and high body mass since these risk factors appear to influence different measures of joint loading.
APPENDIX I

Inverse Dynamics Model/Biomechanical Knee Model

The biomechanical model of the knee calculates compressive, anterior-posterior shear, and resultant forces within the tibio-femoral compartment of the knee joint. The model includes two basic components. The first is a three-dimensional inverse dynamics analysis of the lower extremity to obtain the joint torques and reaction forces of the hip, knee, and ankle. The second uses these results along with the kinematic description of the lower extremity and related anatomical and physiological characteristics to calculate knee muscle, lateral ligament, and joint forces during the stance phase of walking.

The lower extremity was modeled as a rigid, linked segment system. Magnitude and location of the segmental masses and mass centers in the lower extremity along with their moments of inertia were estimated from the position data using a mathematical model and relative segmental masses reported by Dempster. Inverse dynamics using linear and angular Newtonian equations of motion were used to calculate the joint reaction forces and torques at each joint. This process included applying the ground reaction forces to the foot at the center of pressure and calculating the three dimensional net joint reaction forces and torques at the ankle applied to the foot. The net forces and torques were then reversed and applied to the leg and the process was repeated for the knee net reaction forces and torques. The knee kinetics were then used to calculate the hip forces and torques. This analysis is a standard biomechanical procedure.

The second component of the model calculates the forces in the three largest knee muscles and lateral soft tissue support structures (e.g. lateral collateral ligament) and
combines these with the knee joint reaction forces to determine bone-on-bone forces. The model is a “torque-driven” model in that it uses the joint torques from the inverse dynamic analysis to determine the muscle forces. The general technique has been successful in predicting forces in various joints and soft tissues of the lower extremity\textsuperscript{89, 90}. The three basic steps in this component of the model are: 1) determine the forces in the gastrocnemius, hamstrings, and quadriceps muscles and in the lateral support tissues of the knee, 2) apply these forces along with the joint reaction forces onto the tibia, and 3) determine the knee joint forces.

Figure 16: Schematic representation of the biomechanical musculoskeletal knee model used to calculate knee joint loads and muscle forces
The gastrocnemius force is determined from the plantar flexor moment at the ankle joint during the stance phase of gait. It is assumed that the plantar flexor torque in produced by the triceps surae (gastrocnemius and soleus muscles) and the tibialis anterior, that is co-active with the gastrocnemius during the initial 25% of the stance phase \(^48\). Tripe surae force was calculated as the quotient of the ankle joint torque and the moment arm for the triceps surae at the observed angular position of the ankle joint plus the additional 10% increase in force due to dorsiflexor coactivity. Muscle moment arm values for each ankle position were from moment arm-ankle joint position curves from the literature \(^91\). Gastrocnemius force was then calculated from triceps surae force based on its proportion of the total physiological cross sectional area (PCA) of the triceps surae which is 0.319 \(^92\).

The direction of the gastrocnemius force was determined from the heel and knee marker positions and is expressed as \(\alpha\), the angle between the gastrocnemius force and the tibia. The heel marker was used to represent the distal end of the gastrocnemius. The proximal end was positioned 0.020 m superior and 0.023 m posterior to the knee joint, along the line of the femur \(^93\). This position accounted for the gastrocnemius wrapping around the femoral condyles. The resultant direction of the gastrocnemius force was, on average, about \(\alpha = 3^\circ\) from parallel with the leg and so this force applied a relatively large compressive load by small shear load on the knee.

Hamstrings force was calculated from the extensor torque at the hip joint observed typically during the first half of stance. The predicted hamstrings force accounted for both the hamstrings PCA (physiologic cross-sectional area) relative to the total PCA of the hamstrings and gluteus maximus and the hamstrings moment arm at the
hip relative to the gluteus maximus moment arm. The total hamstrings proportion to the hip torque was calculated as:

\[ Hp = \left( \frac{\text{Ham PCA}}{\text{Ham PCA} + \text{GM PCA}} \right) \left( \frac{H_d}{\text{GMd}} \right) \]  \hspace{1cm} (Equation 2)

where \( Hp \) is the proportion of the hip extensor torque generated by the hamstrings, \( \text{Ham PCA} \) and \( \text{GM PCA} \) are the hamstrings and gluteus maximus PCAs, and \( H_d \) and \( \text{GMd} \) are the hamstrings and gluteus maximus moment arms. Values for each of these constants are obtained from the literature \(^{92, 94}\) and are: \( \text{Ham PCA} = 42.4 \text{ mm}^2 \), \( \text{GM PCA} = 17.36 \text{ mm}^2 \), \( \text{Ham d} = 0.042 \text{ m} \), and \( \text{GM d} = 0.047 \text{ m} \). The proportion of the hip extensor torque generated by the hamstrings (\( Hp \)) was equal to 0.63. The hamstrings force was then calculated as:

\[ H = Hp \frac{\text{Het}}{H_d} \]  \hspace{1cm} (Equation 3)

where \( H \) is the hamstrings force and \( \text{Het} \) is the hip extensor torque. Hamstrings force is assumed to be zero when the hip torque is in the flexor direction. The direction of the hamstrings force is a line parallel to the femur and at angle \( \beta \) to the tibia.

The quadriceps force was calculated from the observed knee joint torque produced by the inverse dynamics analysis and the hamstrings and gastrocnemius forces. This calculation took into account co-contracting knee flexors. The observed knee torque was the net torque at the knee and is a function of all muscles crossing the joint:

\[ K_t = Q(Q_d) - H(H_d) - G(G_d) \]  \hspace{1cm} (Equation 4)

where \( K_t \) is the net knee torque from inverse dynamics, \( Q, H, \) and \( G \) are the forces by the patellar tendon, hamstrings, and gastrocnemius muscles, and \( Q_d, H_d, \) and \( G_d \) are the
respective moment arms for the muscles at the knee. The force in the quadriceps, $Q$, was then calculated as:

$$Q = (K_t + H(H_d) + G(G_d))/Q_d \quad \text{(Equation 5)}$$

Moment arms at the knee were obtained from the literature by averaging the values from a number of studies and for each angular position of the knee joint\(^{95-99}\). The mean values throughout the knee ROM for the three moment arms were, $Q_d = 0.035$ m, $H_d = 0.032$ m, and $G_d = 0.018$ m. The direction of $Q$, $\Phi$, was determined from the literature\(^{96,99}\) and is also a function of knee angle.

The methods of Schipplein et al.\(^{54}\) were used to determine the distribution of frontal plane loads and in particular the force in the lateral support structures at the knee during the stance phase. The external loads place an adductor torque on the knee that is resisted by a combination of abductor torques from the quadriceps and the lateral structures. The quadriceps exerts a small to moderate abductor torque during walking because the center of pressure between the tibia and femur is located on the medial tibial plateau which is medial to the center of the patellar tendon. The moment arms for the quadriceps and lateral support structures to the center to pressure on the medial tibial plateau are estimated from the x-ray films for each subject. These distances average $\sim2.5$ cm and $\sim7.0$ cm, respectively. The quadriceps abductor torque (product of the quadriceps force and its frontal plane lever arm) is subtracted from the observed net internal abductor torque calculated by the inverse dynamics analysis. The remaining torque is distributed to the lateral knee tissues and the force in these tissues is calculated as the quotient of this torque and the lever arm. This force is considered to act parallel with the line of the tibia.
The final step was the calculation of knee joint forces. All muscle forces, the force in the lateral support structures, and the joint reaction forces identified with inverse dynamics were partitioned into their compressive (parallel with the tibia) and anteroposteior shear (perpendicular to the tibia in the frontal plane) components and summed. The equations were:

\[ K_s = G \sin \alpha - H \sin \beta + G \sin \Phi - K_z \sin \lambda + K_y \cos \lambda \]  
\[ \text{(Equation 6)} \]

\[ K_c = G \cos \alpha - H \cos \beta + G \cos \Phi - K_z \cos \lambda + K_y \sin \lambda + L_{ss} \]  
\[ \text{(Equation 7)} \]

where \( K_s \) and \( K_c \) are the shear and compressive forces at the knee, \( K_z \) and \( K_y \) are the vertical and horizontal knee joint reaction forces, and \( L_{ss} \) is the force in the lateral support structures. \( K_s \) is positive when the shear force applies an anterior load to the tibia and \( K_c \) is positive with the compressive force pushes into the tibia.

**Limitations of the biomechanical knee model**

The primary limitation was the absence of most knee ligaments. The absence of cruciate and medial collateral ligaments would have increased the knee muscle force predictions since these tissues resist all external loads. However, we argue that total knee loads will not be severely affected because they will be produced by the sum of all tissues crossing the joint regardless of whether these tissues are muscle or ligament. The model does not include the lateral collateral ligament, which is important and produces the principle, non-muscular restraint during the stance phase of walking.

A secondary limitation was the assumption of no co-activation by the hip flexors during stance. The assumption of no co-activity of the hip introduced some error due to
missing force production in the rectus femoris which also applied force at the knee. This issue was relevant during the initial part of the stance phase when the hamstrings are active and producing force. Thus, the predictions of the hamstrings force and subsequently, quadriceps and knee forces were probably underestimated. A sensitivity analysis was performed and found that an underestimate of hamstrings force by 25% (what we have established as our error in hamstring force) produced only a 5% error on knee force predictions. However, force in the rectus femoris during the first half of stance and relative EMG activation of this muscle among others, were relatively low. However, the model did include co-activation of knee flexors and extensors, and ankle dorsiflexors and plantar flexors during the stance phase.

While this model only estimates actual knee joint biomechanics, based on similarity between its predictions and those of other knee models, we conclude that it is a reasonable, valid, and appropriate for our purposes.
REFERENCES


CURRICULUM VITA

Mackenzie L. Hoops

Education

Graduate
Wake Forest University, M.S. (Health and Exercise Science)
2009-2011
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Advisor: Dr. Stephen P. Messier
GPA: 3.808
Thesis: The Influence of Obesity and Alignment on Knee Joint Loads during Osteoarthritic Gait

Undergraduate
University of Dayton, B.S. (Pre-Physical Therapy)
2005-2009
Dayton, OH
GPA: 3.49 (overall); 3.79 (major)
Undergraduate Thesis: Age, Sex, and Finish Time as Determinants of Pacing in the Marathon

Research Experience

2009-present
J.B. Snow Biomechanics Laboratory; Department of Health & Exercise Science; Wake Forest University
Research Assistant
Dr. Stephen P. Messier, Supervisor
Skills: Collected with a Six-camera Motion Analysis 3D motion capture system and editing with EVaRT video; post-processing using OrthoTrak; rearfoot motion processing using KinTrak; AMTI force platform; collection and knowledge with running and walking mechanics; skilled with isokinetic dynamometer (KinCom); conducted anthropometric measurements

TRAILS
The Runners and Injury Longitudinal Study (PI: Messier, S.P. Funding: Army Research Office).
Purpose: A prospective, longitudinal study to determine the causes of overuse injuries in runners. 180 non-injured runners will be tested (gait, strength, anthropometrics, runners’ history, injury history) and followed for up to 2 years. Injury status will be monitored biweekly. Comparison between those who become injured and runners remaining injury free will determine potential risk factors.
Role: Biomechanics Lab Technician
Responsibilities: data collection; data analysis; manual of operations development; testing protocol assistance; testing for reliability studies on Q-angle and isokinetic hip strength to be used in protocol; administration of questionnaires and informed consent; strong participant interaction
**IDEA**

*Intensive Diet and Exercise for Arthritis* (PI: Messier, S.P.
Funding: National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS).

**Purpose:** To compare the effects of intensive dietary restriction-plus exercise, exercise-only, and intensive dietary restriction-only interventions on inflammatory biomarkers and knee joint loads in 454 overweight and obese adults with knee OA.

**Responsibilities:** data entry; data analysis; data management; assistance in testing

**2008-2009 (Summers)**

United States Army Research Institute for Environmental Medicine (USARIEM), Natick, MA

**Biomechanics Lab Intern**

**Skills:** collected and processed using a 12-camera system with Qualysis Trak Manager (QTM); post-processed using Visual 3D; dual force-plates treadmill; assisted with balance testing using a force platform; experience with ParvoMedics metabolic cart; assistance in testing; 2D kinematic data (DartFish) for studies focusing on injury prevention based on biomechanical stresses; provided assistance in developing functional testing protocol for prospective study focusing on injury mechanisms in female collegiate athletes; conducted literature searches; assisted with laboratory tours; attended scientific and educational seminars

**2009**

Department of Health and Sports Sciences; University of Dayton

University of Dayton Independent Research Study

Dr. Paul Vanderbugh, Supervisor

*Age, Sex, and Finish Time as Determinants of Pacing in the Marathon*

**Purpose:** To determine the influences of age, sex, and run time on marathon pacing.

**Responsibilities:** data management; mined data from publicly available marathon websites; data analyses

Results presented at the 2009 National ACSM Conference in Seattle, WA

**Professional Experience**

2010-present

*Wake Forest University Department of Health and Exercise Sciences*

Teaching Assistant: Graduate and Undergraduate Biomechanics Lab

**Responsibilities:** Assist with Graduate Biomechanics Lab on isokinetic testing, 3D motion analysis with video and force platform systems, and anthropometric measures. Instructing and consulting Undergraduates through a Motion Capture workshop with instruction on 2-dimensional motion capture and digitizing.

2009-present

*Wake Forest Healthy Exercise and Lifestyles ProgramS (HELPS)*

Exercise Leader
Responsibilities: instruct patients with exercise and rehabilitation equipment; provide modalities such as resting and exercise EKG; greet patients; maintain clean facility; graded exercise testing

2007-2008

Miami Valley Hospital, Dayton, OH
Physiotherapy Volunteer
Observed physical and occupational therapists; interacted with patients

Teaching Experience

2010-present
Wake Forest University Department of Health and Exercise Sciences
*Biomechanics Lab*: Graduate and undergraduate required HES course
Graduate Lab Instructor

2009-present
Wake Forest University Department of Health and Exercise Sciences
*HES 101: Exercise for Health* required undergraduate course
Graduate Instructor

Fall 2006
University of Dayton
Adapted Physical Education
Assisted young girls (3rd - 4th grade) with special needs to improve physical fitness working to improve flexibility, strength, and endurance primarily; also worked to form good psychosocial relationships

Publications


Presentations


Certifications

2009 CPR and AED certified, American Heart Association