The Influence of Static Stretching of Knee Flexors on Knee Biomechanics

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THE INFLUENCE OF STATIC STRETCHING OF KNEE FLEXORS ON KNEE BIOMECHANICS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science

by

JOSHUA DAVID PERRIN

B.S., Wright State University, 2015

2018

Wright State University
I HEREBY RECOMMEND THAT THE THESIS PREPARED UNDER MY SUPERVISION BY Joshua David Perrin ENTITLED: The Influence of Static Stretching of Knee Flexors on Knee Biomechanics BE ACCEPTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF Master of Science.

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ABSTRACT

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There is a greater incidence of non-contact anterior cruciate ligament (ACL) injury in female athletes compared to males. The higher rate of ACL injury in female athletes is most likely multifactorial and is influenced by intrinsic and extrinsic risk factors. This study examines the effects of an extrinsic factor — static stretching of the hamstrings on knee biomechanics.

Twelve female athletes performed drop vertical jump (DVJ) tasks before and after a hamstrings static stretching protocol. Knee kinematics and kinetics were recorded during the contact phase of the DVJ. The results of this study revealed stretching had no significant effect on knee kinetics or kinematics. Our initial hypothesis that static stretching of the knee flexors would increase the incident of biomechanical ACL injury risk factors was not supported.
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I. INTRODUCTION

The knee is the largest joint in the body [1, 2]. It is a modified hinge type of synovial joint located between the two longest lever arms: the femur and the tibia [3]. Though the knee is designed to sustain large loads, it is extremely vulnerable to injuries [1, 2]. Injury to the knee can damage one of the primary knee stabilizers — the anterior cruciate ligament or ACL [3].

The ACL is one of the most commonly injured knee structures [4]. ACL injury can occur from contact or noncontact [4]. Contact injuries can be defined as forces from another person being applied to the athlete’s knee or person [4]. Non-contact injuries can be defined as an injury due to indirect forces like higher ground reaction forces and internal muscle forces — these types of injuries are more common and are the focus of this study [4]. The majority of non-contact injuries occur during cutting, landing, or running and jumping maneuvers because these activities occur in multiples of body mass that can be greater than two to three times normal bodyweight [5, 6]. Both contact and non-contact injuries can be debilitating [7].

Generally, there are two methods of treatment after injuring an ACL: rehabilitation without surgical intervention or reconstructive surgery [8, 7]. A study published in The Journal of Bone and Joint Surgery determined ACL reconstruction surgery to be more cost-effective than rehabilitation alone when taking into account
societal and economic factors, however, the procedure is still costly and comes with a significant recovery period [8].

Several studies have taken a look at the impacts of ACL reconstruction surgery [8, 7, 4]. One study evaluated 229,446 outpatient arthroscopic ACL reconstructions over a 9-year period [7]. The median immediate procedure cost (defined as a 3-day window centered around the surgery) was $9,399.49. Total health care utilization (defined as 3 months prior to surgery and 6 months after) was 13,403.38. Over the course of the study (2005-2013) the immediate procedure costs increased by 41% and the total cost of health care utilization increased by 34%. Along with the cost of reconstructive surgery, patients faced a course of rehabilitation that typically lasted 6 months [7].

Often overlooked are the life-long effects of ACL injury and reconstruction. When taking into account societal and economic factors, the mean lifetime cost to society for a typical patient undergoing ACL reconstruction can be as high as $38,121 [8]. This value illustrates the immediate and long-term economic impact of an ACL injury. Though any athlete can incur this economic burden, a quick review of the literature reveals a greater incidence of non-contact ACL injuries in female athletes compared to males [9, 10, 11].

Title IX is a federal law that was passed in 1972 [12, 11]. The law prohibits discrimination on the basis of sex in any federally funded educational program or activity [12, 11]. As a result, there was an increase in female athletic participation [12, 11]. Along with the increasing participation was an increased rate of female sport-related injuries when compared to males [9, 11, 10]. Many researchers noticed this trend and wanted to
know why. Two researchers, in particular, Elizabeth Arendt and Randall Dick, wanted to determine if females were more susceptible to ACL injury than males.

In 1995, Arendt and Dick wanted to test their hypothesis. To do so, they evaluated the incidence of ACL injuries in men and women’s sports across a broad sample over the course of several years. What they found was that females were much more likely to sustain an ACL injury in soccer (0.31 vs 0.13) and basketball (0.29 vs 0.07). These injury rate values represent the number of injuries per 1000 athlete-exposures [11, 10]. Many others continued to evaluate the ACL injury gender disparity and found similar results [13, 14, 15, 16, 17].

The higher rate of ACL injury in females is most likely multifactorial and includes both extrinsic and intrinsic factors. Extrinsic factors are external to the athlete and intrinsic factors are inherent [18, 19]. In order to have an understanding of the factors that affect ACL injury rates, one must have a basic understanding of knee anatomy.

**Knee Joint Anatomy**

The largest joint in the human body is the knee [2, 1]. It is a complex modified hinge type of synovial joint that allows for flexion and extension in the sagittal plane as well as a small amount of varus and valgus rotation about the frontal plane [2, 1, 3]. The joint also permits a small amount of rotation around a vertical axis during flexion and extension [2, 1, 3].

At its core, the knee is a three joint structure composed of the medal and lateral femorotibial joints and the patellofemoral joint [2, 1, 3]. The femorotibial joint bears
most of the body’s weight and is formed by the articulation of the medial and lateral condyles of the femur and tibia [2, 3]. The second joint, patellofemoral, is formed by an articulation between the patella and the patellar surface of the femur [2, 3]. This articulation increases the mechanical advantage of the quadriceps muscles [2, 3].

Unlike many other joints, the bony articulations that form the knee are relatively incongruent [2]. As a result, the knee joint is stabilized by associated structures. Primarily stabilizing the knee joint are ligaments [3].

Ligaments

Ligaments are fibrous bands of tissue that connect bone to bone and are thought to play a role in joint proprioception through cutaneous receptors [3]. There are several ligaments that surround the knee, each of which provides stability in a specific direction. The ligaments of the knee can be divided into extracapsular and intracapsular [2].

There are five extracapsular ligaments: patellar, medial collateral (MCL), lateral collateral (LCL), oblique popliteal, and arcuate popliteal [2]. The latter two ligaments strengthen the joint capsule posterolaterally [2]. The remaining three ligaments play a larger role in knee stabilization [2].

The patellar ligament is a continuation of the quadriceps femoris tendon and the anterior ligament of the knee joint [3]. It originates from the apex of the patella and inserts into the tibial tuberosity of the tibia [3]. Inserting into the patellar ligament are the medial and lateral patellar retinacula [2]. These aponeurotic expansions of the vastus
medialis and vastus lateralis play an important role in maintaining alignment of the patella relative to the patellar surface of the femur [2].

Reinforcing the medial and lateral aspects of the knee are the collateral ligaments [2, 3]. The MCL extends from the medial epicondyle of the femur to the medial condyle and the superior part of the medial surface of the tibia [2]. It provides stability to the medial aspect of the knee and prevents excessive valgus movement during external rotation [2, 3]. It’s counterpart, the LCL, originates from the lateral epicondyle of the femur and extends inferiorly to the lateral surface of the fibular head [2]. This ligament provides stability to the lateral aspect of the knee and prevents excessive varus movement and external rotation at all positions of knee flexion [2, 3].

Within the knee joint capsule are two strong intracapsular ligaments: PCL and ACL [2]. Also known as the cruciate ligaments, they obliquely cross each other within the joint capsule, like the letter “X” [2]. Because of this crossing pattern, they limit medial rotation of the tibia in relation to the femur to roughly 10° and permit 60° of lateral rotation [2]. The oblique orientation of the cruciate ligaments also ensures one of them is tense at any angle of knee flexion [2]. These ligaments are responsible for maintaining contact between the tibia and femur during flexion of the knee [2]. This makes these ligaments very important knee stabilizers.

The PCL specifically protects against posterior displacement of the tibia in relation to the femur [3]. It arises from the posterior intercondylar area of the tibia and attaches to the anterior part of the lateral surface of the medial condyle of the femur [2]. Preventing excessive anterior and rotational displacement of the tibia in relation to the femur is the ACL [3]. This ligament arises from the anterior intercondylar area of the
tibia [2]. It then extends superiorly, posteriorly, and laterally to attach to the posterior part of the medial side of the lateral condyle of the femur [2].

The purpose of ligaments surrounding the knee is to prevent excessive movement. This is achieved passively, as each ligament prevents movement in a specific direction [2, 3]. During movement of the knee, ligaments can become at risk for injury. To combat the increased risk of injury, ligaments work in conjunction with muscles. Muscles, with their attaching tendons, dynamically reinforce the knee through movement [2, 3].

Muscles

Most movement of the knee exists in the sagittal plane as flexion and extension [3]. To a smaller degree, the knee experiences rotation about the transverse plane as well as varus and valgus stress about the frontal plane [3]. There are many muscles that act on the knee to perform these movements, however, there are arguably two groups of muscles that are more important than the others: quadriceps and hamstrings.

On the anterior aspect of the knee joint is the insertion site of four large muscles found on the anterior thigh: the quadriceps [2]. The rectus femoris, vastus medialis, vastus lateralis, and vastus intermedius work in the sagittal plane [2]. Contraction of these muscles produces extension [2]. Differential contraction of these muscles produces varus/valgus movements. Contraction of the vastus lateralis tends to have a valgus moment arm and contraction of the vastus medialis tends to have a varus moment arm.

On the posterior aspect of the knee are a group of muscles that act in opposition to the quadriceps: the hamstrings [2]. Flexion is produced when the biceps femoris,
semitendinosus, and semimembranosus contract [2]. Just like the quadriceps, differential contraction of these muscles produces varus and valgus movements. Contraction of the lateral hamstring muscles, the biceps femoris, tends to produce a valgus moment arm and contraction of the medial hamstring muscles, the semitendinosus and semimembranosus, tends to produce a varus moment arm.

**Kinematic and Kinetic factors behind ACL injury**

In a prospective study by Hewett et al., adolescent female athletes who actively participated in high-risk sports such as soccer, basketball, and volleyball were measured for neuromuscular control using joint angles and joint moments during a jump-landing task. Out of 205 participants, 9 went on to rupture their ACL during follow-up. The 9 injured participants had significantly different knee posture and loading compared to the participants who did not suffer injury. At landing, the injured athletes had knee abduction angles that were 8° greater when compared to their uninjured counterparts. Injured athletes also had a 2.5 times greater knee abduction moment and 20% higher ground reaction force (GRF). The stance time was 16% shorter. In short, increased motion, force, and moments occurred more quickly. Findings from this study and others are important because they identify kinematic and kinetic mechanisms for ACL injury [17].

Kinematics can be described as motion without referencing the forces causing the motion [1, 6]. Several studies, just like the one above, found dynamic valgus to be a kinematic mechanism for ACL injury. In *Understanding and Preventing Noncontact*
*Anterior Cruciate Ligament Injuries*, dynamic knee valgus was once again identified as a risk factor for ACL injury.

Dynamic valgus is a combination of movements involving the hip, knee, and ankle joints [17]. An individual experiencing dynamic valgus will have femoral adduction, knee abduction, and eversion of the ankle [17]. Dynamic valgus can also be described as the position or motion of the distal femur (at the knee joint) toward the midline of the body and the distal tibia away from the midline of the body [17]. Dynamic valgus has also been correlated with increased anterior tibial translation when combined with low knee flexion angles, which puts the ACL under tension [6].

Kinetics is a study based on kinematics [1, 6]. It incorporates the forces that cause motion [1, 6]. One common force associated with ACL injury is ground reaction force (GRF). GRFs are the opposing linear forces applied from the ground while weight bearing [6]. As we saw in the study above, higher GRFs are correlated with increased ACL injury [17]. The mechanism by which GRFs are applied also plays a role in ACL injury risk. Non-contact ACL injuries typically occur when landing from a jump, cutting, pivoting, or with an unplanned change of direction [6]. A joint moment is another force to consider when discussing ACL injuries. Moments are the net effect of the eccentric forces of all structures acting on a joint to produce rotational motion [6]. Since the ACL prevents anterior tibial translation, many studies have focused on flexion and extension moments produced by the hamstrings and quadriceps. Reviewing the literature reveals more anterior tibial translation occurs with smaller knee flexor moments [6].

As we will discuss in the following sections, there are extrinsic and intrinsic factors that affect the rate at which an individual experiences these risky kinematic and
kinetic mechanisms [18, 19]. Extrinsic factors can include the weather, type of playing surface, or the type of shoe the athlete is wearing [19, 20]. Recent research suggests fatiguing of the muscles surrounding the knee joint through exercise and stretching may also be a factor that increases the likelihood of ACL injury [21]. Intrinsic factors can be divided into anatomical, hormonal, age at menarche, neuromuscular, genetic, cognitive, and previous injury [18, 19]. Each factor, extrinsic or intrinsic, contributes to ACL injury risk in its own way.

Intrinsic ACL Injury Risk Factors

Anatomical

It is thought that there exists a difference in ACL injury rates between men and women because of natural anatomic variations between sexes [18]. Due to the location of the ACL, many researchers speculated that one of the anatomic variations that exist would be appreciated in the intercondylar notch of the femur [18]. It had been suggested that a smaller intercondylar notch would impinge the ACL, limiting its range of motion, and putting it at a higher risk for injury at specific knee positions [18]. Several studies aimed at measuring intercondylar notch fell to the same conclusion: individuals with a smaller intercondylar notch were at a higher risk for ACL injury and females, on average, had a smaller intercondylar notch than their male counterparts [18, 22]. Another anatomical factor that has been shown to affect ACL injury rates is general joint laxity [18, 9]. An increase in knee laxity has been associated with increased ACL injury risk.
As with the previous risk factor, this too comes with a gender disparity. Females have greater knee and generalized joint laxity than males [9, 3]. Having increased knee joint laxity puts them at a greater risk for ACL injury. A third anatomic variant that was found between sexes is the effect of BMI on ACL injury risk [22].

In a study by Uhorchak et al., 895 United States Military Academy cadets (739 men, 120 women) were evaluated over a 4-year period for non-contact ACL injury risk factors. Over the course of the study, 29 complete ACL tears were found (21 in men, 8 in women). Smaller notch width and increased generalized joint laxity were recognized as factors for increased ACL injury and a significant main effect for sex was found. A higher than normal body mass index (BMI) also indicated an increased risk for ACL injury. Interestingly, a higher than normal BMI only affected female cadets. Throughout the study a general trend of increased ACL injury risk in females when compared to males can be found, however, the gender disparity was at a climax when risk factors were combined and when evaluating relative risk. In this study, 100% of females who displayed notch width, BMI, and generalized laxity risk factors experienced complete ACL tears. None of the males with the combination of these risk factors were injured [22].

Though this was one study, taking these data at face value reveals an even greater risk for ACL injuries in females when compared to males than previously thought. Continuing to evaluate individual risk factors is a must and more research into how stacked individual risk factors affect ACL injury risk need to continue.
One basic difference between the sexes is the presence of a cyclic hormonal cycle in females [23]. The menstrual cycle consists of several phases, each of which has characteristic rises and falls in different hormone levels [23]. At the beginning of the menstrual cycle (day 1), estrogen and progesterone are at their lowest [23, 24]. During the follicular phase of the menstrual cycle estrogen is at its highest and during the luteal phase is when the highest levels of progesterone can be appreciated [23, 24]. High levels of these hormones have been correlated with increased ACL injury, which is thought to be because of increased ACL laxity [24]. At peak estrogen and progesterone levels, an increase in ACL laxity can be appreciated [24]. High levels of estrogen have also been shown to affect muscle strength, tendon and ligament length, aerobic and anaerobic capacity, endurance, and isokinetic strength [14]. These data suggest that estrogen affects systems outside of the ACL that may increase injury risk rates.

Despite the amount of data, the point at which females are at their highest risk for ACL injury is still up for debate. A consensus statement from the Hunt Valley II meeting stated that there was evidence to support the hypothesis that the highest incidence of ACL injury occurred during the early and late follicular phases. Contradicting this statement was a case-control study published after the conference. Befuddling, as it may seem, the findings just echo the need for more research.
Menarche & Age of

It would be impossible to discuss the effects hormones play on ACL injury risk without mentioning the effects of puberty. In Hewett et al.’s article *Anterior Cruciate Ligament Injuries in Female Athletes*, the effect hormones have on biomechanics and neuromuscular characteristics were explored. During puberty, the tibia and femur grow at rapid rates. Increasing the length of these bones, as well as others, will increase the height of the individual. A taller individual will have a higher center of mass and, typically, a greater overall mass. Increasing the two longest lever arms in the body, raising the center of mass, and increasing overall mass will increase the amount of force on the knee. In order to compensate for the increased force, increased dynamic lower extremity control is needed. The increased demand for lower extremity control is the reason ACL injury rates increase for both genders, however, girls have a higher rate of ACL injury immediately after the growth spurt. The appreciable difference in ACL injury rates between males and females can be attributed to a divergence of neuromuscular patterns that manifests in the form of girls having decreased adaptation after puberty. After puberty, males demonstrate increased power, strength, and coordination with age. Females, on the other hand, show little change throughout maturation [14]. There is an abundance of research that agrees with the conclusion that females undergo a change at puberty that increases their risk for ACL injury [9, 25, 15, 16, 26].

Wanting to understand this relationship more, Hewett et al tested middle school and high school-aged girls and boys for dynamic knee movements and lower limb length. The results revealed increased shank and thigh length for males and females as well as
increased height and mass. Despite the increase in anthropometric measures, postpubertal female peak muscular torque about the knee remained steady with increasing age. Males had significantly greater quadriceps torque with increasing age. The dynamic movements at the knee also favored the males and prepubertal females. Postpubertal females had significantly more medial rotation and increased valgus moments at the knee. These data support the hypothesis that postpubertal females have poor neuromuscular control of the knee joint when compared to males and prepubertal females [26].

Recent research from our lab suggests earlier age at menarche negatively affects biomechanical and neuromuscular development [27, 28]. The narrowing of the gait base of support is a normal part of pubertal development [29], and affects dynamic knee alignment [27, 28]. Menarche is associated with an increase in estrogen levels, which induces the cessation of skeletal muscle growth [30, 31, 32]. If estrogen levels increase too soon, as in the case with early menarche, then the narrowing of the gait base of support is interrupted. Interrupting normal gait narrowing will result in retention of a more immature and wider base of support [27]. The wider base of support translates into more valgus knee angles and moments in the frontal plane [27]. Increased valgus has been identified to increase the risk for ACL injury [26]. These findings are suggestive of earlier age at menarche increasing the risk for ACL injury.

**Neuromuscular**

Many studies document a post-pubertal increase in neuromuscular strength and coordination for males; however, this is not always the case for females [14]. Because of
this gender disparity, much of the research into ACL injuries concerns neuromuscular risk factors. One of the most commonly researched neuromuscular risk factors concerns the hamstrings to quadriceps (H:Q) ratio.

The hamstring muscles, producing flexion of the knee, share an agonistic relationship with the ACL [2, 3, 14, 6]. Research supports the hypothesis that increasing the strength of the hamstrings will decrease the stress placed on the ACL [33, 34]. Inversely, the quadriceps muscles, producing extension of the knee, have an antagonistic relationship with the ACL [2, 3, 14]. Increased quadriceps activation has been associated with increased anterior tibial translation and increased ACL elongation [12]. Despite the danger to the ACL, the forces produced by the quadriceps are immensely important for locomotion. Without them, extension at the knee joint would be impossible. To allow for extension at the knee and protection of the ACL, a proper H:Q ratio must be maintained. This supports the theory that when the ratio of hamstrings strength to quadriceps strength is unbalanced, injuries can occur.

Some research has suggested age at menarche in females has an effect on their H:Q ratio by affecting the rate at which their musculature develops, which in turn influences neuromuscular control of the knee [25, 26]. After menarche, females have a lower H:Q ratio than males because the strength of their quadriceps increases more than the strength of their hamstrings [25, 26]. This can make them more susceptible to ACL injury due to an increase in valgus alignment and loading of the knee [26]. Moreover, recent work in our lab suggests that earlier menarche is associated with greater valgus alignment and loading [27, 28], pointing to the possibility that the timing of menarche is related to the H:Q ratio.
Asymmetry

In 1970, S. R. Chibber and Inderbir Singh studied the weight of right and left lower limbs of 10 cadavers. The results showed one limb being heavier than the other, suggesting limb dominance. Interestingly, in this study, the heavier lower limb was often on the contralateral side of the dominant upper limb. From this, it was concluded that there was lower limb asymmetry, however, there was no correlation between dominance in the upper and lower limbs. Findings such as these made it clear that there was a difference between right and left limbs, however, dominant and non-dominant may not be the correct way to distinguish them [35].

In a study by Fort-Vanmeerhaeghe, Gabriel Gual, Daniel Romero-Rodriguez, and Viswanath Unnitha, subjective lower limb dominance and objective lower limb strength were compared. Males and females were used for this study. The results of the study showed that there was little correlation between subjective dominance (leg dominance as reported by the participant) and objective leg strength. Out of all participants, only 40% described the dominant leg as being the one to score higher in strength tests. These findings once again supported the conclusion that there was lower limb asymmetry, but they also showed subjective lower limb dominance might not always be the strongest leg. Another important finding was that females had higher rates of asymmetry than males [36]. Other studies comparing males to females have come to similar conclusions. For example, in a study by Ford et al, females experienced more valgus at the dominant leg than the non-dominant leg. They postulated the dominant leg might be able to experience
higher forces and torques while the non-dominant leg may be able to manage only average forces [37]. Pappas and Carpes found similar results in their study.

The reasons for increased lower limb asymmetry in females are most likely multifactorial. As previously discussed, after puberty, females have significantly lower neuromuscular development compared to their male counterparts [14]. Cowley et al. studied these neuromuscular differences and found females to have increased GRF and valgus of the dominant leg during a cutting maneuver. Interestingly, during a drop vertical jump, the non-dominant leg was the one experiencing higher GRF. The change in kinetics may indicate a change in lower limb dominance depending on the type of activity [38].

Understanding asymmetry is important because recent research into lower limb asymmetry has shown a side-to-side strength disparity may increase injury risk (Hewett, Stroupe, Nance, & Noyes, 1996; Hewett, et al., Biomechanical Measures of Neuromuscular Control and Valgus Loading of the Knee Predict Anterior Cruciate Ligament Injury Risk in Female Athletes, 2005; Hewett, Myer, & Ford, Decrease in Neuromuscular Control About the Knee with Maturation in Female Athletes, 2004). More research into this topic needs to be done. In order to stratify participant populations, a standardized method of determining limb dominance needs to be established. This may include subjective dominance through questioning or measuring the strength of the limb. However, each of these methods must be taken with trepidation because the possibility of lower limb dominance reversal exists. Perhaps the best method of dealing with limb asymmetry is through preventative training programs — in lieu of retrospective research.
A study has even shown that a proper plyometric training program can be effective at correcting lower limb asymmetry [39].

**Extrinsic ACL Injury Risk Factors**

*Fatigue*

Fatigue is an extrinsic factor that changes the biomechanics of the knee by affecting mechanisms centrally and peripherally [40]. These changes manifest in the form of decreased force production, impaired neuromuscular coordination and precision, slower muscle reaction times, increased joint laxity, and weakened proprioceptive capabilities [21, 41, 42, 43, 44, 45, 46, 47]. Given what is known, the effects of fatigue on ACL injury rates have been voraciously studied.

In a study by Chappell et al., recreational athletes were fatigued and then assessed for knee joint angles, resultant forces, and moments. The results showed males and females had significantly increased peak proximal tibial anterior shear forces, valgus moments, and decreased knee flexion angles during 3 stop-jump tasks when fatigued. These fatigued athletes demonstrated altered motor strategies, possibly resulting in the increased anterior tibial shear forces. The increased anterior tibial shear forces correlated with increased strain on the ACL. The results suggest subjects had experienced fatigue-induced increased ACL injury risk because of decreased knee flexion angle. Interestingly, the data also suggest females experienced knee valgus moments in conjunction with decreased knee flexion angles. The increased knee valgus moments in females puts them
at increased ACL injury risk when compared to males [21].

Stretching

Stretching of skeletal muscle prior to engaging in physical activity is a common practice. Two common types are dynamic and static stretching [48, 49]. Evidence suggests both types have a positive impact on overall joint mobility/range of motion (ROM) [48, 49]. Data suggest both types of stretches also affect the peak torque (PT) of a muscle, however, they do so in different ways [49].

Affecting the PT of a muscle may or may not affect overall athletic performance by changing neuromuscular control [48]. In regards to the knee, changing PT may indirectly affect the ACL through changes in the H:Q ratio [50]. As previously discussed, the H:Q ratio is the ratio of hamstrings strength to quadriceps strength. The hamstring muscles are agonists to the ACL and the quadriceps are antagonists. When the ratio of the strength in these muscle groups becomes unbalanced, particularly when the quadriceps is exceedingly stronger than the hamstrings, more stress is placed on the ACL [12, 34, 3, 2, 6]. In the following paragraphs, I will discuss the different types of stretches and, using current literature, speculate answers to the questions about whether or not a specific pre-exercise stretch will affect PT, affect the H:Q ratio, affect overall athletic performance, or increase the risk of ACL injury.

Two dynamic stretching methods are commonly recognized: active and ballistic [49]. Active stretching is the process of gently moving a limb through its entire range of motion several times [49]. This method of stretching, when performed properly, has been
shown to increase ROM [49, 48, 51, 52]. The effect it has on PT of a muscle and athletic performance is somewhat controversial. Some studies report positive effects [49, 53, 54, 55, 56, 57]. One study reports no effect at all [58]. Another study even reports possible negative effects as a result of dynamic stretching [59]. Despite the existence of one negative study, most researchers can agree that active dynamic stretching does not appear to have a detrimental effect on PT or athletic performance.

The effects of dynamic stretching have also been applied to the H:Q ratio. One article directly tackled the topic and found dynamic stretching had no effect on the H:Q ratio [50]. There are data that support the theory of dynamic stretching having a positive influence. Because of these findings, it can be speculated that dynamic stretching does not negatively affect ACL injury rates. Contrary to active dynamic stretching, ballistic stretching has been correlated with increased risk for injury [49]. It involves rapid, not gentle, movements and is typically not recommended anymore [49].

Static stretching is one of the most common types of stretching. It involves holding a specific position that applies tension to a muscle or muscle group for a short period of time [49]. The process is repeated several times to achieve the desired result [49]. The stretch can be performed actively (without a partner) or passively (with a partner) [49]. Static stretching has been correlated with increased range of motion, just like dynamic stretching, however, that is where the similarities end [49, 48].

The effects of static stretching become confusing because the majority of research suggests the activity has negative effects [60, 61, 62, 56, 54, 63, 64, 53, 57, 65], while some studies report no effect at all [66, 67]. The lack of agreeable data can be attributed to the many variables that exist between research studies. Some of the prominent
variables include participant population, type of stretch, duration of stretch, and other
activities performed in conjunction with the static stretch exercise. Despite some
confusion about static stretching’s effects, it has been shown to affect the H:Q ratio [60].

Several trends can be identified when looking at studies that report no negative
effects from static stretching. The first, and perhaps easiest to identify, is the population
of participants. Two studies, in particular, used elite athletes [66, 68]. The data from these
athletes revealed no changes at all from static stretching. This was evidence suggesting a
trained athlete was unaffected by the activity. The second and perhaps more important
category is the duration of the stretch. Research has shown static stretching for less than
45 seconds does not produce negative effects [69, 70]. On the other hand, stretching for
60 seconds or more, with increased frequency, can produce negative effects [70]. These
negative effects can manifest in the form of increased ACL injury.

It is well supported that static stretching can decrease the PT of a muscle [71].
Further research has determined that static stretching of the hamstrings can adversely
affect the H:Q ratio by decreasing the maximum torque of the hamstrings [50]. The
decreased maximum torque of the hamstrings increases the chances for an ACL injury
because they normally act to stabilize the knee, decrease valgus moment, and dampen
anterior tibial translation [3, 2].

Interestingly, the effects of static stretching are not permanent or irreversible.
Some studies report negative effects on performance lasting as long as 24 hours post-
stretch [56]. One theory that is supported with data is that static stretching with sport
specific activity or warm-up can reverse or mitigate negative effects [72, 73, 74]. Athletes
that perform static stretching with a sport specific activity almost always mitigate or completely reverse the effects of static stretching [72, 73].

**Hypothesis**

The higher rate of ACL injury in females is most likely multifactorial and includes both extrinsic and intrinsic factors. Extrinsic factors are external to the athlete and can include weather, type of playing surface, or the type of shoe the athlete is wearing. Other external factors include fatiguing and stretching of muscles and surrounding structures. Intrinsic factors can be divided into anatomical, hormonal, age at menarche, neuromuscular, genetic, cognitive, and previous injury. This study was interested in the effects of static stretching due to its effects of altering neuromuscular control.

It is well supported that static stretching can decrease the PT of a muscle. Further research has determined that static stretching of the hamstrings can adversely affect the H:Q ratio by decreasing the maximum torque of the hamstrings. The decreased maximum torque of the hamstrings increases the chances for an ACL injury because they normally act to stabilize the knee, decrease valgus moment, and dampen anterior tibial translation. In our study, a static stretching protocol was completed to assess for changes in kinematic and kinetic data. Due to the known effects of static stretching, we have postulated that static stretching of the knee flexors will increase biomechanical ACL injury risk factors.
II. MATERIALS AND METHODS

Participants

Twelve active females (aged = 25.08 ± 3.8 years, mass = 60.44 ± 10.02 kg, height = 162.28 ± 6.96 cm, body mass index = 22.98 ± 3.83) volunteered for this study. All participants were Wright State University students, aged 18 years or older, who were healthy with no history of anterior cruciate ligament injury or any other injuries that might affect lower limb biomechanics. Inclusion criteria for the study included recreational activity, participating in organized sports and/or moderate exercise at least two times per week. Informed consent was obtained, and Wright State University’s Institutional Review Board approved the study.

Data Collection and Instrumentation

After acquiring informed consent, anthropometric measurements were obtained using standard methods [75]. The measurements included height (cm), sitting height (cm), weight (kg), bi-iliac breadth (cm), right foot length (cm), and left foot length (cm).

Biomechanical data were obtained using the LHRC’s three-dimensional motional capture laboratory. The laboratory is equipped with six Osprey cameras (Motion Analysis Corp., Santa Rosa, CA) that are synchronized with three force plates embedded in a 15m
walkway (two AMTI OR6-7-1000, Advanced Medical Technology Inc., Watertown, MA; and one Kistler Type 9281B11, Kistler Instruments, Winterthur, Switzerland). Cameras captured subject motion at a rate of 120 frames per second, recording the movement of reflective markers placed on precise anatomical landmarks. Cameras captured the motion of 25 reflective markers placed on each subject. The Helen Hayes marker set was used, including bilateral markers at the head of the second metatarsal, heel, medial and lateral malleoli, shank, medial and lateral femoral epicondyles, thigh, anterior superior iliac spine, acromion process, lateral humeral epicondyle, mid-wrist, and a unilateral marker on the sacrum. After static trials, the medial femoral epicondyle and medial malleoli markers were removed.

Participants were required to perform six trials pre-stretching routine (three per leg) and six trials post-stretching routine (three per leg). Any trials with poor marker recognition were removed from the dataset. For each variable, participant averages across viable trials for each leg were used in the analysis. Biomechanical data were collected using Cortex 6.0 software and was processed using MacGait 1.0 software (Motion Analysis Corp., Santa Rosa, CA). Reported moments are external moments, which were calculated using inverse dynamics and normalized for body mass. Ground reaction forces are expressed as multiples of body weight. This study uses the following conventions for angle and moment signs: positive values refer to adduction/varus and flexion; negative values refer to abduction/valgus and extension.
Experimental Procedure

Data were collected during a single visit to Wright State University’s Lifespan Health Research Center. Prior to marker placement, participants completed a questionnaire regarding menstrual history, sports participation history, and ACL injury history. After completing the questionnaire, participants were taken to an exam room and anthropometric measurements were obtained. Once anthropometric measurements were obtained, makers were placed on the subject and they performed a drop vertical jump (DVJ) task. Participants immediately transitioned into the stretching protocol and then completed a second DVJ task, completing the protocol. A visual representation of the procedure is represented in Figure 3.

Drop Vertical Jump

To perform the DVJ task, participants were instructed to jump down from a height of 31 cm onto a force plate and then jump as high as they could with arms stretched vertically after landing with both feet at the same time. DVJ is demonstrated in Figure 1. To prevent bias, participants were told to jump onto the “gray square on the floor” and were unaware of the presence of a force plate. The participant landed on the ground with both feet hitting the ground at the same time with each trial, however, only one foot landed on the force plate. The participant landed on the force plate with her dominant foot three times and then landed on the force plate with her non-dominant foot three times for a total of six trials. Prior to data collection, each subject performed practice
jumps to ensure they understood the procedure and were able to land properly on the force plate.

**Stretching Protocol**

Stretching of the hamstrings was induced by having subjects perform a routine consisting of five unassisted stretches. The five unassisted stretches included (1) a standing unilateral toe-touch to the dominant leg; (2) a standing unilateral toe-touch to the non-dominant leg; (3) a bilateral toe touch; (4) a seated hurdler stretch to the dominant leg; (5) a seated hurdler stretch to the non-dominant leg. Each stretch is demonstrated in Figure 2. Each stretch was held for 30 seconds, four times, with 15 seconds of rest between. The stretches were held with some discomfort but not pain, as described by the participant.

The hamstring stretches and duration that they would be held was chosen based on previous static stretching research. Several studies evaluating the effects of static stretching on the hamstrings revealed a decrease in maximum muscle torque after holding each stretch for 30 seconds, four times, with a short rest period in between [54, 50]. A comprehensive review of static stretching times reveals no effect of static stretching less than 45 seconds with some effects being noticeable after 60 seconds of total stretching [70]. Our participants performed each stretch for a total of 2 minutes. This is much greater than 60 seconds.

After completing the first DVJ task, participants immediately start the static stretching protocol. The instructor demonstrated the stretching exercises for the
participant. Each participant verbalized understanding of the stretching exercise. After completing the stretching protocol, participants once again completed a DVJ task.

Statistical Analysis

Statistical analysis of the data began with the identification of the contact phase of the DVJ task. Initial contact (IC) was determined as the first frame in which ground reaction forces (GRF) were measured. Toe-off (TO) was determined as the final frame in which GRF was measured. DVJ trials were grouped according to leg dominance. Leg dominance was determined by which leg the participant would use to kick a soccer ball.

Data for the knee and the hip were analyzed. At the knee, frontal and sagittal angles, as well as frontal knee moments, were analyzed at IC, TO, maximum, and minimum for the duration of the contact phase, and at peak vGRF using paired t-tests. The peak vGRF was also analyzed via a paired t-test, with an additional sensitivity analysis to test for the effects of removing an outlier. Statistical analysis was conducted using the JMP 12.0 and SAS 9.3 integrated suite of statistical software.
Figure 1.

(A)

(B)
Figure 1.

Drop vertical jump (DVJ) task performed three times per leg by participants. (A) displays a series of images from Cortex 6.0 software showing movement of reflective markers placed on precise anatomical landmarks and showing force vectors during the contact phase of the DVJ task. (B) shows the corresponding video images of the DVJ task. Images are in chronological order from left to right. [76]
Figure 2.
Figure 2.

The five unassisted stretches included (A) a standing unilateral toe-touch to the dominant leg; (B) a standing unilateral toe-touch to the non-dominant leg; (C) a bilateral toe touch; (D) a seated hurdler stretch to the dominant leg; (E) a seated hurdler stretch to the non-dominant leg.
Figure 3.
Figure 3.

Timeline of events that occurred during a participant’s visit to the LHRC. Numbers on the horizontal axis represent time in minutes.
III. RESULTS

During the stretching protocol, participants completed 5 unassisted stretches. Each of the stretches was held for 30 seconds, four times, with 15-second rest periods in-between. The stretches were held to a point of discomfort but not pain, as described by the participant. Knee kinematics and kinetics were measured for all 12 subjects. Knee kinematics and kinetic measurements are summarized in Table 1.

The results of our data revealed stretching had no significant effect. Knee abduction angle showed no significant change at IC (P= 0.50), TO (P= 0.81), Max (P= 0.39), and Min (P= 0.38). Knee flexion angles in the sagittal plane also showed no change at IC (P= 0.80), TO (P= 0.43), Max (P= 0.98), and Min (P= 0.95). Knee abduction moment was not significantly affected by stretching at IC (P= 0.98), TO (P= 0.43), Max (P=0.84), or Min (P= 0.43).

Effect size for repeated measures were calculated. Hedges gAV values for knee abduction angle at IC (g= 0.07), TO (g= 0.03), Max (g= 0.09), and Min (g= 0.17) showed a very small effect. Hedges gAV values for knee flexion angle at IC (g= 0.06), TO (g= 0.09), Max (g= <0.01), and Min (g= 0.01) also showed a very small effect. Finally, hedges gAV values for knee abduction moment at IC (g= 0.01), TO (g= 0.20), Max (g= 0.03), and Min (g= 0.16) showed a very small effect. Hedges gAV values are summarized in Table 2.
The probabilities that any person would have a higher value post-stretching than they would pre-stretch routine were also calculated. The probability that someone would have a higher value post-stretch than pre-stretch for knee abduction angle at IC (0.56), TO (0.52), Max (0.57), and Min (0.58) were essentially equal to 50%. The probability that someone would have a higher value post-stretch than pre-stretch for knee flexion angle at IC (0.52), TO (0.57), Max (0.50), and Min (0.51) were approximately equal to 50%. Finally, the probability that someone would have a higher value post-stretch than pre-stretch for knee abduction moment at IC (0.50), TO (0.57), Max (0.52), and Min (0.57) were also approximately equal to 50%. The probability of a higher value post-stretch than pre-stretch is summarized in Table 2.

Pearson correlation values for knee abduction angle pre- and post-stretching routine at IC (r= 0.87944), TO (r= 0.84885), Max (r= 0.88931), and Min (r= 0.6415) are summarized in Figure 3. Pearson correlation values for knee flexion angle pre- and post-stretching routine at IC (r= 0.34599), TO (r= 0.84374), Max (r= 0.93623), and Min (r= 0.76675) are summarized in Figure 4. Pearson correlation values for knee abduction moment pre- and post-stretching routine at IC (r= 0.43931), TO (r= 0.43931), Max (r= 0.67209), and Min (r= 0.52648) are summarized in Figure 5.
<table>
<thead>
<tr>
<th>Contact Phase Event</th>
<th>Pre-</th>
<th>Post-</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Knee Abduction Angle (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>6.0 ± 2.5</td>
<td>6.2 ± 3.0</td>
</tr>
<tr>
<td>TO</td>
<td>5.5 ± 2.6</td>
<td>5.6 ± 4.1</td>
</tr>
<tr>
<td>Maximum</td>
<td>8.1 ± 2.8</td>
<td>8.4 ± 3.0</td>
</tr>
<tr>
<td>Minimum</td>
<td>−1.2 ± 2.8</td>
<td>−1.8 ± 4.1</td>
</tr>
<tr>
<td><strong>Knee Flexion Angle (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>31.2 ± 4.7</td>
<td>30.9 ± 4.9</td>
</tr>
<tr>
<td>TO</td>
<td>26.3 ± 10.8</td>
<td>27.4 ± 10.6</td>
</tr>
<tr>
<td>Maximum</td>
<td>96.4 ± 13.6</td>
<td>96.4 ± 14.5</td>
</tr>
<tr>
<td>Minimum</td>
<td>24.3 ± 9.3</td>
<td>24.2 ± 7.1</td>
</tr>
<tr>
<td><strong>Knee Abduction Moment (Nm/Kg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>0.1 ± 0.2</td>
<td>0.1 ± 0.1</td>
</tr>
<tr>
<td>TO</td>
<td>0.1 ± 0.3</td>
<td>0.2 ± 0.2</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.3 ± 0.7</td>
<td>1.3 ± 0.7</td>
</tr>
<tr>
<td>Minimum</td>
<td>−0.6 ± 0.3</td>
<td>−0.5 ± 0.3</td>
</tr>
</tbody>
</table>

*Note.* A negative value for knee abduction angle or moment indicates a knee abduction/valgus angle or moment, respectively.
Table 1.

The effects of static stretching on mean (± SD) knee kinematic and kinetic variables at various time points of the DVJ contact phase.
<table>
<thead>
<tr>
<th>Contact Phase Event</th>
<th>Hedges gAV</th>
<th>Probability Higher</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Knee Abduction Angle (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>0.07</td>
<td>0.56</td>
</tr>
<tr>
<td>TO</td>
<td>0.03</td>
<td>0.52</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.09</td>
<td>0.57</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.17</td>
<td>0.58</td>
</tr>
<tr>
<td><strong>Knee Flexion Angle (°)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>0.06</td>
<td>0.52</td>
</tr>
<tr>
<td>TO</td>
<td>0.09</td>
<td>0.57</td>
</tr>
<tr>
<td>Maximum</td>
<td>&lt;0.01</td>
<td>0.5</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.01</td>
<td>0.51</td>
</tr>
<tr>
<td><strong>Knee Abduction Moment (Nm/Kg)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>0.01</td>
<td>0.5</td>
</tr>
<tr>
<td>TO</td>
<td>0.2</td>
<td>0.57</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.03</td>
<td>0.52</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.16</td>
<td>0.57</td>
</tr>
</tbody>
</table>
Table 2.

Effect sizes and the probability of a higher value post-stretch than pre-stretch at various time points of the DVJ contact phase.
Figure 4.

Note. KAA: Knee Abduction Angle. IC: Initial Contact. TO: Toe Off
**Figure 4.**

Pearson correlation values of knee abduction angle pre- and post- stretching routine at various time points of the DVJ contact phase.
Figure 5.

Note. KFA: Knee Flexion Angle. IC: Initial Contact. TO: Toe Off.
**Figure 5.**

Pearson correlation values of knee flexion angle pre- and post- stretching routine at various time points of the DVJ contact phase.
Figure 6.

Note. KAM: Knee Abduction Moment. IC: Initial Contact. TO: Toe Off
Figure 6.

Pearson correlation values of knee abduction moment pre- and post- stretching routine at various time points of the DVJ contact phase.
IV. DISCUSSION

The purpose of this study was to examine the effects of static stretching of the hamstring on knee biomechanics. Previous studies have explored the effects of static stretching and concluded there to be a possibility of decreased maximum muscle torque for a period of time. Many of these studies have assessed overall athletic performance by measuring power output and timed events, such as sprinting. Many did not determine if the effects of static stretching were evident in the biomechanics of a risky athletic maneuver. The results of this study do not support our initial hypothesis that static stretching of the knee flexors would increase the incident of biomechanical ACL injury risk factors. The null hypothesis was supported in that no effect of static stretching on knee biomechanics was found.

Effects of Static Stretching on Kinetics and Kinematics of the Knee

The results of this study indicate that there was no change in knee abduction angle, knee abduction moment, and no change in knee flexion angle after our stretching routine. Furthermore, the static stretching protocol did not affect or change the rate of lower limb asymmetry. The results of this study both agree with and contrast with much of the research regarding static stretching.
Much of the research discussing the effects of static stretching can be organized into two categories: showing effects and showing no effects. The results of our study fall into the latter. Our results are similar, but not identical, to another study conducted by Knudson et al. In this study, biomechanical data of a vertical jump were analyzed before and after a static stretching routine. The results of the study revealed stretching prior to activities like a vertical jump resulted in small decreases in performance in some subjects; however, there were no significant biomechanical changes [67].

It has been theorized that static stretching reduces the stiffness of a muscle by affecting the musculotendinous unit (MTU) [71]. Through our protocol, if stiffness had been reduced, we might have found decreased knee flexion angles with subsequent changes in knee abduction angles and moments [71, 67]. Decreased knee flexion angles were not found in the study by Knudson et al. or our own. This result suggests minimal to no change in stiffness occurred.

The remaining articles that report no change from static stretching do so by assessing athletic performance, rather than biomechanics. This makes comparing our data with that of other studies difficult. Collectively, these articles tested participants for variables such as peak muscle torque (PT) and sprint times. Each of them presents a different explanation as to why no change in performance was found.

Some research suggests a sport-specific or dynamic warm-up prior to static stretching will mitigate or completely erase the effects of static stretching [72, 73, 74]. In an article by Blazevich, participants completed a comprehensive warm-up routine prior to engaging in either a short-term static stretch, longer-term repeated static stretch, dynamic stretch, or no stretch at all. Immediately following the stretching routine, participants
completed a higher intensity warm-up. This higher intensity warm-up consisted of 2 minutes of moderate jogging, 5 seconds of high knees and 5 seconds of heel-to-butt kick drills. Immediately after finishing the higher intensity warm-up, participants were tested for flexibility, vertical jump, sprint running and change of direction. The results of the study suggested static stretching was unlikely to affect an athlete when performed in conjunction with a comprehensive physical preparation routine [72]. Samson et al. also came to a similar conclusion. In their study, participants completed similar activities mixed with stretching exercises. The inclusion of a specific warm-up — high knee skipping, high knee running, and butt kick running — resulted in an increase in performance. No static stretch-induced deficit was found. In fact, the participants in this study actually saw both an increase in performance and increased range of motion when the specific warm-up and static stretch were combined. This was not found for dynamic stretching conditions [73]. Taylor et al. once again came to a similar conclusion, but only after a sport-specific warm-up were simultaneously used.

Our protocol did not consist of specific warm-ups such as high knee maneuvers or butt kicks; however, our participants did complete a drop vertical jump trial run prior to engaging in the static stretching routine. We asked the participants to complete the DVJ to the best of their ability, which may have included 100% effort. Given this, and the data presented in previous research, it is possible that the inclusion of practice DVJs could have mitigated the effects of static stretching. Work done by Wallman et al. supports this theory. In their research, participants completed a 5-minute walk on a treadmill at 3.5 mph. They then ran a baseline 40-yard sprint with a subsequent self-paced 10-minute walk. They then completed a stretching routine and then repeated the 40-yard sprint.
There were no statistically significant differences for static stretching, ballistic stretching, or dynamic stretching [58]. I would argue this study inadvertently elicited a specific warm-up that mitigated the results of stretching, just as we could have.

Perrier et al. who tested for counter-movement jumps (CMJ) found no difference in CMJ when comparing static stretching trials to no stretching trials. Their protocol did call for a 5-minute jog on a treadmill prior to completing a routine [51]. This also agrees with our findings. Finally, the research project by Knudson et al., most similar to our own, utilized a 3-minute cycle warm-up followed by 3 practice vertical jumps. They also found no effects from static stretching [67]. Future protocols should explore the idea of having participants come in on different days to eliminate a possible mitigating warm-up effect.

The second reason no change in performance was found was due to the population of participants. The participants of nearly every study were recreationally active, however, a few studies utilized high-level athletes. The results of these studies showed no changes as a result of a static stretching routine [66, 68]. The lack of a static stretch effect was primarily thought to be due to the participants being highly trained athletes. Directly contrasting this research is Sekir et al. who employed high-level female athletes. They were put through a static stretching routine and a dynamic stretching routine. The static stretching routine produced detrimental effects while the dynamic stretching routine improved performance [55]. This is just one example of the variability that exists within the research.

In our pool of participants were division I female athletes and recreationally active females. The level of activity varied greatly among our participants, as some of
them participated in organized sports 5-7 days a week and others engaged in self-directed exercise 2-3 times a week. Given this, and taking into the account previous research using highly trained athletes, if there was an effect from the stretching protocol we may have seen it affect the less trained participants first. However, the data showed, uniformly, no correlation between static stretching and increased risk for ACL injury. It is possible that the stretching routine we employed was ineffective, or, the athletes utilized in the aforementioned studies were more trained than the few that we employed.

Last and perhaps the most important reason researchers saw no change from a stretching protocol was due to short stretching times. Anthony Kay and Anthony Blazevich explored various static stretching routines and found a possible dose-response effect. Articles were separated according to the amount of static stretching performed by participants. The first group stretched for less than 30 seconds, the second group stretched for 30-45 seconds, and the last group stretched for a total of 60 seconds or more. They came to the conclusion that stretching for 45 seconds or less was not significantly correlated with detrimental effects. On the other hand, there was a sharp increase in the incidence of detrimental effect at 60 seconds, with a plateau occurring at 120 seconds [70]. Another article by Costa, Grave, Whitehurst, and Jacobs agrees with these findings. In this article, they concluded a stretching protocol consisting of 45-second hold durations did not produce adverse effects. [69]. Future projects should not hesitate to consult this empirical evidence when designing a stretching protocol. Likewise, these resources should be consulted when reviewing other research articles, as some of them employed inadequate stretching protocols.
Wallman, Christensen, Perry, and Hoover utilized 2 repetitions of 15 seconds for each leg for a total of 30 seconds and did not see an improvement in their study [58]. Blazevich et al. had participants perform either 5 seconds of static stretching or 30 seconds total of static stretching. No effect was seen and neither of the stretching times which came close to a 60-second threshold [72].

Up until this point, our project has been similar to one conducted by Knudson et al. Their stretching protocol consisted of 3, 15-second repetitions, totaling 45 seconds. Our study had participants hold each stretch for 30 seconds, 4 times. This totaled 2 minutes per stretch. This puts our procedure well beyond a 60-second threshold. Future research projects could examine if the total time the muscle was placed under stress from stretching or if the amount of time spent stretching during a repetition is more important when attempting to elicit a static stretching induced deficit.

In contrast to the articles that have been discussed so far, much of the research concerning static stretching reports measuring changes as a result. The overwhelming majority of these articles report an acute decrease in athletic performance as a result of static stretching. These articles, just like many of the ones that report no change, typically did not assess biomechanics; rather, they assessed for variables such as peak muscle torque and timed events. Also, the same variables exist for these articles in that they employed non-high level athletes, did not use sport-specific warm-ups and had stretching routines that were longer in duration.

It is very possible that our stretching routine decreased the athletic performance of our participants; we just did not know it. Future research could employ the use of a dynamometer to definitively determine whether or not maximum muscle torque was
affected. Using this method in conjunction with biomechanical data can help determine whether or not static stretching, and changing the MTU, actually affects the biomechanics of the knee. If the results of such an endeavor revealed decreased peak torque and still no change in biomechanics, then that would be suggestive of no increased ACL injury risk with a static stretching routine. No changes in EMG activity would indicate that our stretching routine was either ineffective or there was another variable diminishing the effects of stretching, like a warm-up routine.

The final point of thought in regards to our results comes in the form of what actually affects biomechanics. It is possible that our static stretching routine was effective and changed the stiffness of the MTU; however, biomechanics of the knee joint may have remained intact. If our protocol was indeed effective, yet we saw no results, this indicates another variable that we have not taken into account is the real reason biomechanics change. For instance, some form of neuromuscular inhibition may change biomechanics more readily than muscle stiffness.

**Influence of Participant Bias**

One possible source of error that needs to be addressed is the possibility of participant bias. Most of the participants were unaware of the possible detrimental effects of static stretching, just like much of the general public. In fact, many of the participants were under the impression that a static stretching routine would improve performance. On the other hand, a few of the higher-level athletes had been made aware of static stretching’s effects at one point or another prior to data collection. This gap in
information could have affected the data, however, our data showed no correlation between stretching and increased incidence of biomechanical ACL injury risk factors. This supports the idea that our routine was not affected by participant bias. In another article, a small increase in performance after static stretching was found in some of the subjects [67]. This was attributed to the possibility of a reverse placebo effect caused by many participants expecting to improve performance after stretching [67]. We did not appreciate such a phenomenon in our study.

**Variation in Static Stretching Protocols and Analysis**

It is important to mention the variability throughout the aforementioned research. Two of the biggest variables are differences in stretching protocols and methods of analyses. As reviewed by Kay et al., the duration of the static stretch is extremely important when wanting to elicit a response. Anything less than 60 seconds in total has been shown to have no detrimental effects, however, anything 60 seconds or more greatly increases the chance of having detrimental effects [70]. Another factor that might have implications is the application of assisted stretches.

Several articles utilized assisted stretches — also known as passive stretching performed by a trained professional — to elicit a response. Many of them were successful at producing some sort of detrimental effect. Given this, we can speculate about possible a relationship between passive stretching and producing a static stretching effect. Passive stretching may allow the individual to stretch even further, possibly generating the static
stretch response. We did not use passive stretching in our work. Future iterations of our research topic may compare the effects of passive stretching to active stretching.

Analytical methods varied greatly throughout the literature. Many researchers measured peak muscle torque using a dynamometer [50, 60, 61, 62, 64, 66]. Some measured peak torque along with other variables such as EMG, MMG, and ROM [54, 63, 55]. Some measured participants for sprint times, jump tests, ROM, or countermovement jumps [56, 53, 57, 65, 74, 73, 58]. One research article by Costa et al. even measured stretching’s effect on balancing ability [69]. While our stretching protocol is similar to others in duration and type of stretch, our analytical method was different. We did not test out participants for peak muscle torque nor did we collect any sort of sprint or jump performance data. We did collect data for ground reaction forces and joint biomechanics. Given the variability of all the research, future work should strive to generate a more standardized method of measurement. A standardized method of measurement would make comparing data easier and more effective.

Practicality of the Research

Much of the research in regards to static stretching employs protocols utilizing assisted stretches. It could be argued that a protocol that consisted of active stretching, rather than passive stretching, is more practical and useful to the general public. If the research is focused on elite athletes that always have access to trainers for passive stretching, then the argument can be made that the results of static stretching will be mitigated anyway, based on previous research. Our research protocol is practical in the
sense that the majority of recreationally active individuals, aged youth to adult, will perform active stretching.

One must also take into consideration the analytical methods that are being used. Recreationally active individuals do not routinely sprint 100 meters or push themselves to 100% of capacity. Our drop vertical jump test has been shown to be a good measure of ACL loading, however, it could have been supplemented with a common cutting maneuver or short duration sprint to better evaluate each participant.

Overall, our research proves practical due to its simplicity and ease of replication. The stretches performed are ones that can be done at home or in an athletic facility without proper training or assistance. And, the drop vertical jump produces forces that each individual may encounter at some point during a recreational activity or strenuous workout.

**Study Limitations**

There are several limitations to this study that should be considered when evaluating the results. The static stretches themselves were taken from other protocols, however, the combination of stretches and them being performed actively is unique to this study. It is possible that an effect would have been found with passive stretching or a different stretching protocol.

In regards to the participants, they were all from different backgrounds of varying degrees of athletic participation. Some of the participants knew how to perform the stretches prior to instructor explanation while others needed more guidance. This
variable, which was unaccounted for, could have produced varying degrees of stretching. The individuals who already knew how to perform the stretch could have done better, producing more of a static stretch effect. As we saw with the data, however, there was no correlation between the stretching routine and increased ACL injury risk. Nonetheless, this variable should be taken into account during future research.

Some research has shown a sport specific warm-up or a dynamic warm-up of some kind can mitigate or extinguish the effect of static stretching. We may have inadvertently elicited this phenomenon by having our participants perform practice drop vertical jumps before completing the stretching protocol. Future research should explore the idea of having participants come to the lab for two separate visits. One day to practice the drop vertical jump and become familiar with the stretches, and then a second to actually perform the procedure. This introduces variables that we did not account for because we completed everything in one visit, but is an idea that should be explored and may be beneficial if done properly.

To the best of our ability, we reduced the chance of variables outside of the lab; however, some of the participants had obligations that could not be ignored. We attempted to schedule participants on a day and at a time that suited them and would also allow them to come into the lab without being active earlier in the day. Unfortunately, we could not control whether or not a patient was active outside of the lab, as many of them were obliged to participate in organized sporting activities. It is possible some of the participants already presented to the lab with a minimal amount of undisclosed fatigue, whether they knew it or not.
Conclusions

The findings of this study do not support our hypothesis that static stretching of the knee flexors would increase the level of biomechanical ACL injury risk factors. Based on this study, the following conclusions can be made:

• Active static stretching protocols lasting greater than 60 seconds in total time may not induce changes in frontal plane knee kinetics and kinematics.

• Landing strategies may be maintained even after bouts of 60 seconds or longer of static stretching.

• Limb dominance has no significant correlation with the effects of active static stretching on knee biomechanics.

• Differences in stretching protocols and analytical practices make it difficult to compare results among studies evaluating lower limb biomechanics and the effects of static stretching.
V. RESULTS


