THE RELATIONSHIP OF LOWER EXTREMITY MALALIGNMENTS IN COLLEGE STUDENTS WITH A HISTORY OF ACL INJURY

A Thesis in Kinesiology

by

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ABSTRACT

Anterior cruciate ligament (ACL) injuries are a common and debilitating knee injury. Non-contact ACL injuries are 2-8 times more common in females for reasons that are currently unknown. It has been hypothesized that structural differences between males and females may lead to the increased injury rate in females. The purpose of this study was to examine lower extremity malalignments in college students who had previously suffered an ACL injury. In addition, previous ankle injury was hypothesized to be a contributing factor to ACL injury so ankle injury history information was also collected.

Fifty-two college age students (33 females, 19 males) with a history of an ACL injury and 33 females controls with no history of knee injury volunteered for this study. Participants filled out a survey of knee and ankle history and then had 16 different measures of lower extremity malalignment and flexibility taken.

My most clinically relevant finding when comparing injured males and females was a significant association between ACL injury history and ankle sprain injury history. Those with a history of ACL sprain were more likely to have had an ankle sprain. To explore the relationship between malalignments and ACL injury, a discriminant analysis was performed. Thirty-five and two-tenths percent of the variation in ACL injury history was explained by twelve variables. Of those 12 variables, 21.1% of that association was explained by greater generalized laxity, greater genu recurvatum, and lack of iliotibial band (ITB) flexibility.

If there is a kinetic chain relationship between previous ankle injury and risk for ACL injury, ACL prevention programs can be added to the ankle rehabilitative programs currently used. Future research on the relationship of knee and ankle injury risk factors is needed.
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CHAPTER 1

INTRODUCTION

Anterior cruciate ligament (ACL) injuries are common occurrences in athletics. Although both males and females are susceptible to ACL injuries, there is a much higher rate of injury in females. In addition, close to 85% of all ACL injuries in females are non-contact in nature. During a non-contact ACL injury, the mechanism is usually described as the athlete suddenly stopping and pivoting to change directions. In this position, with the foot fixed on the ground, the knee is placed in “a position of no return” and an ACL injury can occur. With the foot planted on the ground, the knee gets put into an extreme valgus position with tibial external rotation, thus stressing the ACL to the point of rupture.

To date, several studies have examined ACL injuries at the high school, collegiate, and recreational levels, but no one has been able to determine conclusively why these injuries are more common in females. Researchers have examined intrinsic and extrinsic factors that may lead to injury, but the common conclusion is that these injuries are probably multifactoral in nature. Gender differences in anatomy, biomechanics, hormonal influences, and neuromuscular performance have all been reported in the literature. Although gender differences have been identified, which combination of factors are related to ACL injury is still unknown.

Statement of the Problem

Non-contact ACL injuries occur 2-8 times more frequently in females than they do in males (Zilmer). It is believed that ACL injuries are multifactoral in nature, but we still do not know which factors are associated with these injuries and why these injuries are more common in females. Previous studies have examined differences between males and females to determine
why there is a higher rate of ACL injuries in females. Despite several years and millions of dollars put into research, the question remains largely unanswered.

**Statement of the Purpose**

It has been hypothesized that anatomical differences in lower extremity alignment between males and females may be a factor related to the high incidence of non-contact ACL injuries in females. The purpose of this study was to examine lower extremity alignment in college students with and without a history of ACL injury to determine if structural differences existed between groups. Additionally, the relationship between ankle sprain history and ACL history was examined to determine if a kinetic chain injury relationship was present.

**Significance**

Lower extremity malalignments that predispose females to ACL injury should be examined to determine if they can be altered to reduce the risk for injury. If there is a relationship between ankle sprain history and predisposition for ACL injury, people with a history of ankle sprain should be put on an ACL injury prevention program to reduce their risk of injury.

**Research Hypotheses**

1) Females with a history of ACL injury will have different lower extremity alignments compared to uninjured females.

2) There will be a relationship between history of ankle sprain and ACL injury. Both males and females with a history of ACL injury will be more likely to report history of a previous ankle sprain.
**Limitations**

1) This was a retrospective study. We were not able to take malalignment measures prior to injury or prior to ACL surgery. Some malalignments could be adaptive as a result of injury or surgery.

2) Subjects were drawn from a college-aged population and therefore conclusions from this study may only be representative of this group.

3) Visual observation was used for many malalignment measures and radiographs may be more accurate way to report lower extremity alignment.

4) Length of time after surgery was not standardized. Subjects had to be cleared for athletic participation (> 6 months post operative), however some subjects had surgery more than 3 years prior to this study. Some malalignments could be adaptive over time.

**Assumptions**

1) All subjects filled out the injury questionnaires honestly and accurately.

2) All subjects gave maximal effort on the two performance measures.

3) All subjects were surgically corrected in a similar manner and surgical technique did not alter lower extremity alignment.

4) Malalignments are structurally inherent and not a result of surgery or injury.
CHAPTER 2
LITERATURE REVIEW

Epidemiology

It is estimated that there are more than 100,000 ACL injuries in the United States each year and more than half of individuals sustaining an ACL injury require surgery, with an estimated annually cost close to one billion dollars. (Urochak). Despite the research efforts over the past 15 years resulting in over 4000 publications, there is still no clear understanding of the etiology of ACL injuries.

In recent years there has been an increase in the number of women participating in athletics. The combination of higher levels of competition and more physical styles of play may lead to higher rates of knee injuries in females. The NCAA reported during the 1996-1997 academic year more than 130,000 women participated in college athletics and there was an average knee injury rate of 1 per 1000 exposures (1 for every 10 female athletes) (Arendt, 1995). In this model there is an expected anterior cruciate ligament (ACL) injury rate of .22 per 1000 exposures or 2200 injuries per year. The cost of treatment for ACL reconstruction and rehabilitation is approximately $17,000 per patient, which would total $37 million per year in collegiate athletics alone (Arendt, 1995).

In 1999, results from the NCAA Injury Surveillance System (ISS) showed that female basketball and soccer players are 3 times more likely to tear their ACL than males athletes in the same sports (Arendt, 1999). The NCAA ISS study was completed in two, 5 year time periods; the first from 1989-1993 and the second from 1994-1998. During the first five-year period, women’s basketball players suffered 189 ACL injuries at a rate of .29 per 1000 exposures and men’s basketball players suffered 49 ACL injuries at a rate of .07 per 1000 exposures. During the
same reporting period, women’s soccer players suffered 97 ACL injuries at a rate of .31 per 1000 exposures and men’s soccer players suffered 81 ACL injuries at a rate of .13 per 1000 exposures (Arendt, 1999).

During the second five year period, women’s basketball players suffered 194 ACL injuries at a rate of .29 per 1000 exposures and men’s basketball players suffered 75 ACL injuries at a rate of .10 per 1000 exposures. During the same reporting period, women’s soccer players suffered 158 ACL injuries at a rate of .33 per 1000 exposures and men’s soccer players suffered 77 ACL injuries at a rate of .12 per 1000 exposures (Arendt, 1999).

In 1996, the National Federation of State High School Associations (NFSHSA) reported 2 million high school female athletes participating in athletics programs. (NFSHSA, 1996) Serious knee injury in high school females was reported to be 1 in 100 which is 5 times the incidence in high school males (Chandy). Chandy et al. reported that in the mid 1980’s, knee surgery comprised 70% of all surgeries for female high school athletes.

ACL injury rates in the military have also been examined. A study by Gwinn et al. evaluated the relative risk of ACL injuries in female and male midshipmen at the United States Naval Academy from 1991-1997. Results from this study revealed 159 ACL injuries during this 6-year time period where overall, women had a significantly greater relative risk (2.44) of injury. For intercollegiate soccer, women had an incidence rate of .768 and a significantly greater relative risk of 5.37. Men had an incidence rate of .081. For intercollegiate basketball, women had an incidence rate of .478 and a significantly greater relative risk of 9.48. Men had an incidence rate of .089 (Gwinn).

Uhorchak et al performed a prospective four-year evaluation of 859 cadets at the United States Military Academy from 1995-1999 and recorded 24 non-contact ACL injuries among
cadets. The incidence rate of non-contact ACL injury was .66 in women and .21 in men yielding a 3 to 1 ratio of female to male injuries (Urochak).

Soderman et al. examined ACL injury rates in young female soccer players in Sweden and reported that between 1994-1998, 398 females under the age of 19 had suffered an ACL tear. Thirty-nine percent of these injuries were suffered by females under the age of 16 participating on senior level teams. The authors concluded that young female soccer players may become injured when playing on senior level teams due to older, heavier, and stronger opponents involved (Söderman, 2002).

Sport injury rates between men and women are typically thought of as sport specific rather than gender specific, but there is a 2-8 higher predisposition to ACL injuries in women than there are in men participating in the same sport (Zilmer).

**Anatomy**

**Macrostructure**

The ACL is an intra-articular and extrasynovial ligament. The average length of the ACL is between 31 to 38 mm (Arnoczky). The ligament is composed of two bundles that attach the tibia to the femur. These bundles appear in a three dimensional shape and are made up of longitudinal fascicles that have a broad tibial attachment compared to the proximal femoral attachment. The bundles are labeled the anteromedial (AM) bundle and posterolateral (PL) bundle. There is a reported functional difference between these bundles as the AM bundle is taut in flexion while the PM bundle is taut in extension (Danylchuk).

Both the tibial and femoral insertions can be described as having a banded or zoned attachment that provides mechanical stability. Cooper and Misol described four zones of the MCL, that the ACL seems to replicate (Cooper, 1970). The first zone is purely ligament
substance which moves into the second zone which contains ligament and fibrocartilage. The third zone moves from fibrocartilage to mineralized fibrocartilage and the fourth zone is pure bone.

**Microstructure**

The microstructure of the ACL represents a highly organized collagen framework similar to other soft connective tissues. These collagen fibrils are arranged in a mostly parallel orientation running longitudinally over the length of the ligament. Individual fibrils are organized into the main structural unit called a fiber. Fibers run in both the parallel and oblique directions in relation to the longitudinal axis and form a larger unit known as the subfasicle. The endotenon surrounds the subfasicle and is made of loose connective tissue. Several subfascicles make up the fasciculus which is surrounded by the epitenon. The outermost layer of the ACL is called the paratenon and is made of thicker connective tissue which is ultimately encased by the synovial membrane (Danylchuk). An important structural finding in the ACL fibers is a wave pattern called crimping. This orientation of the fibers leads to biomechanical features of the ACL such as increased cross sectional area and increased tensile strength (Yahia).

Lyons et al evaluated the microstructural differences between the cells of the ACL and cells of the medial collateral ligament (MCL). They reported some structural differences which may help to explain why the MCL heals well after injury. It was found that the cells of the MCL are morphologically similar to fibroblasts while the cells of the ACL are more similar to fibrocartilage cells (Lyons). Fibrocartilage cells have a decreased blood supply and a decreased regeneration ability.

The ACL is surrounded by a fold of synovial membrane that resembles a mesentery and incompletely divides the joint in the sagittal plane. Even though the ligament in intrarticular, it is
extrasynovial. The synovial membrane is richly interlaced with blood vessels primarily from the medial genicular artery and secondarily from the lateral inferior genicular artery (Arnoczky). These vessels extend into the ACL and branch into a network of longitudinal vessels that run parallel to the collagen bundles within the ligament and surround the ligament in a web-like fashion (Arnoczky). The infrapatellar fat pad and synovial membrane also give blood supply to the ACL. In a canine model as well as the human samples, the blood supply appears to originate from the soft tissue structures rather than the ligament bone junctions in the femur and tibia (Arnoczky).

The ACL is innervated by branches of the tibial nerve in the posterior aspect of the knee. Mechanoreceptors such as the Golgi tendon organ (GTO), Ruffini and Pacinian corpuscles, and free nerve endings are found within the ligament (Arnoczky).

Through histological studies, authors (Shutte, Shultz) described mechanoreceptors present within the tissue. Schultz examined ten anterior cruciate ligament specimens taken from autopsy of amputation patients. The ACL’s contained fusiform corpuscles about 200 micrometers long and seventy-five micrometers in diameter and having morphological similarities to Golgi tendon organs. The corpuscles consisted of a single axon, of which some terminated within the capsule, while others left from a single space and attached with larger nerve bundles outside the capsule. They found the mechanoreceptors to lie only on the surface of the ligament amidst the fatty and vascular tissue. The mechanoreceptors were found mostly at the insertions of the ligament, mostly at the femoral site. They found at most three receptors and usually only one present on each ligament. No receptors were found within the joint capsule.

Schutte examined 6 cadavera specimens obtained during autopsy and found three distinct types of mechanoreceptors: two types of Ruffini end organs, Pacinian corpuscles, and free nerve
endings. These mechanoreceptors were present not only at the surface of the ACL, but throughout the ligament itself. The fibers enter the ligament by way of an axon from a nerve of the surrounding connective tissue. Each receptor had distinct structural characteristics.

The ACL is known to have a distinct “twist” throughout the ligament. If the femoral attachment is the zero point, the twist begins at 5-7 mm. From 0-5 mm, free nerve endings were present, while few organelles existed in the twist region. From 12-26 mm, mechanoreceptors were the most predominant neural structure lying mostly near the tibial insertion. All receptors and nerve endings within the ligament constituted 1% of the area of the ACL.

The first type of receptor is a slowly adapting Ruffini end organ. These receptors have a low sensitivity to changes in tension and play a role in signaling the proximity of the joint through its end ranges of motion. At the end ranges of motion, these receptors have the greatest response (Schutte).

A second type of Ruffini receptor that is morphologically different than the first described above is the pilo-Ruffini complex, characterized by the hair-like appearance. This receptor highly resembles a Golgi Tendon Organ and is also slow adapting (Schutte).

The third type of receptor which was found most often is the Pacinian corpuscle. This receptor is rapidly adapting activated by any movement of the joint regardless of position. These receptors have a very low threshold at the beginning and end of movement (Schutte).

Schultz et al. also observed that the ACL contained a small number of free nerve-endings within the network of the ACL (Schultz). Because of the small number of pain-receptors within the ligament, the authors suggest that the ligament is relatively insensitive to pain. Smith et al. added that this may be the reason why patients who tear their ACL may not experience pain initially following injury, but only after joint effusion occurs (Smith). In addition,
mechanoceptors are responsible for kinesthesia and not pain, which is why an isolated ACL rupture initially causes a pop and instability without the presence of pain. As previously noted, as joint effusion increases and the joint becomes distended, pain increases (Schutte).

**Estrogen Receptors**

Liu et al first identified estrogen receptors on the human ACL in 1995 by a process known as primary immunolocalization. Seventeen ACL specimens of both men and women were obtained during routine knee procedures, dehydrated, and embedded in paraffin. Commercial monoclonal anti-estrogen receptor antibodies were used. These antibodies were shown to be reactive against human estrogen receptors. All 17 specimens contained cells that demonstrated positive staining for estrogen although three of the specimens had less than 15% of staining (Liu, 1996).

Estrogen positive target cells included fibroblasts, synoviocytes, and cells in the blood vessel wall of the ACL. Fibroblasts in the perisynovial tissue demonstrated the highest percentage of staining (Liu, 1996, Slauterbeck, 1999).

**Androgen Receptors**

Hamlet et al reported androgen target cells in the human ACL by the same immunolocalization process as Liu used in 1995. Thirty-one specimens were studied (17 female and 14 male) to determine if the androgen receptor protein was present. There was a positive staining for the androgen receptor in 8 of the specimens. Interestingly however, androgen receptors were only located in young men between the ages of 18-24 years old. No positive identification was seen in older men or women of any age. However, in this sample, there were only two women less than 25 years older so a larger, more diverse sample may reveal androgen receptors. The receptors were located in the same places as the estrogen receptors; the
synoviocytes of the synovium, fibroblasts in the ligament stroma, and in the cells lining the blood vessel walls of the ACL (Hamlet). Even though no studies have identified the role of testosterone in human ligaments, this finding suggests that male hormones, specifically testosterone, may play a role in ACL injuries to men between the ages of 18-24.

There may be a catabolic-anabolic relationship in the ACL of males as compared to females. Since we now know that only males have both estrogen and testosterone receptors, we can look at this relationship further. As previously mentioned, it has been demonstrated that estrogen has catabolic properties as it has been shown to alter collagen synthesis. Although the role of testosterone has not been studied on human ligaments, testosterone in general has anabolic properties. This has been demonstrated by the role androgens have in the growth and development of fibroblasts within the skin, prostate, and other tissues throughout the body (Kasperk, Levine, Liang, Story). It can be hypothesized that males may have a balance between estrogen and testosterone in the build up and break down of collagen whereas females may only have breakdown. Again, more research is needed in this area to determine the functions of estrogen and progesterone receptors in the human ACL.

**Estrogen Receptors in Other Tissues**

Estrogen is known to play a role in certain musculoskeletal disorders leading to joint instabilities in the knee and shoulder (Arendt, 1995; Dyer, Ernst, Fischer, 1973; Liu, 1997). Anterior cruciate ligament injuries, shoulder instability, osteoporosis, and generalized osteoarthritis are all gender-specific diseases of the musculoskeletal system that are affected by estrogen levels. Degenerative spondylolisthesis is also 4 to 5 times more common in females, so Nadaud et al. studied 14 patients undergoing lumbar spinal fusion to determine if facet joint capsular ligaments contained estrogen receptors. None of the specimens, however, contained
estrogen receptors so the authors concluded that although degenerative spondylolisthesis may have hormonal influence, estrogen does not seem to play a direct role in the pathogenesis of the disease (Nadaud).

**Functions of the ACL**

The primary function of the ACL is to resist anterior tibial translation. The ACL acts synergistically with the hamstrings to prevent anterior tibial translation and posterior femoral translation. If a force is applied to try to push the tibia anteriorly, the hamstrings contract to pull it posteriorly (Gray, Huston, 2000, Ireland). The ACL is also a secondary restraint against tibial external rotation and protects against varus and valgus forces.

**Mechanism of Injury**

The majority of ACL injuries (approximately 85%) in females are non-contact usually involving a rotational mechanism of the tibia on the femur with the foot fixed on the ground. The majority of these injuries occur when landing from a jump. The other 15% of injuries may occur by a contact injury and usually involve an isolated mechanism with extreme force. The mechanisms of a contact ACL injury include: a force that drives the tibia anteriorly, a hyperextension force, or a valgus force. Again, these mechanisms are usually isolated but can occur in combination (Gray, Huston, 2000, Ireland).

**Extrinsic Factors Leading to ACL Injury**

Several extrinsic factors have been hypothesized to lead to the higher incidence of female ACL injuries including: poor neuromuscular control, increased knee stiffness, improper landing techniques, and muscular activation patterns differing from those observed in males (Caraffa, Hewett, 1996; Huston, 2000, Ireland). Extrinsic factors, in contrast to intrinsic factors, can to some extent be modified. Extrinsic also include the type of sport, playing surface,
conditioning level, experience, skill, and equipment used (Arendt, 1999). For example, jump landing training and the use of shorter cleats in football have been hypothesized to decrease the risk of knee injury.

**Neuromuscular Control**

Strength and muscle activation patterns are two extrinsic factors which have been investigated more recently. Female athletes rely less on their hamstrings and more on their quadriceps and gastrocnemius than male athletes do (Hewett, 1996, Hewett, 1999). Huston and Wojtys reported that female athletes relied more on quadriceps muscle contraction to resist anterior tibial translation as compared to male athletes as well as non-athletic males and females who relied on their hamstrings for initial knee stabilization (Huston, 1996). If ACL injuries occur when the tibia is forced anteriorly on a fixed femur, the inability of female athletes to contract their hamstrings quickly may lead to higher injury rates. Since female athletes tend to become quadriceps dominant, activities focusing on both eccentric and concentric hamstring strength are important to minimize risk of ACL injury. If the quadriceps fire without the hamstrings, the tibia may be pulled anteriorly which increases the force on the ACL (Liu).

Anderson et al found that male high school basketball players were able to generate higher peak torque and average power than females even when adjustments for body weight were made. In addition the hamstring to quadriceps muscle ratio at 60°/second was significantly higher for males indicating females had weaker hamstrings (Anderson). However, conclusions from this study must be made with caution as the muscle ratios were assessed in the seated position in the open chain which is not a functional position for lower extremity injuries.

Moul et al examined quadriceps and hamstring strength in male and female division 1 basketball players and found significant differences in the eccentric hamstring to eccentric...
quadriceps ratio (Moul). Males were significantly stronger when compared to females. Many ACL injuries occur when landing from a jump or when changing directions. In females, the hamstrings may be inefficient at decelerating the tibia during these movements.

**Knee Stiffness**

Knee stiffness is another important part of knee stability that has been reported to be different in males and females. Normally, as muscles that cross the knee contract, they dissipate forces to prevent excess strain on the ACL. Stability of the knee requires active muscle stiffness which can be controlled through voluntary muscle recruitment. (Duan, Wagner) The hamstring muscle group can contract concentrically to prevent anterior translation of the tibia to help prevent an ACL tear. Although joint stability requires both passive (ligamentous and capsular restraint and skeletal alignment properties) and active stiffness, active stiffness can be controlled through training and warrants further investigation. In addition, passive restraints are able to provide adequate joint stability at low loads, but running, jumping, and cutting require activation of the dynamic stability or muscular stability of the joint (Butler, Smith, 1993)

Mechanoreceptors in the knee ligaments and joint capsule control muscle stiffness through muscle spindle activation. (Johansson, Sojka). Differences in muscle stiffness may also be due to variations in lower extremity muscle mass, (Such) the elastic properties of the muscles and tendons, (Winter) or unknown factors related to gender (Wojtys). Muscle stiffness in the knee is also dependent on both the number of actin and myosin cross bridges and the excitation of the alpha and gamma motor neurons (Huston, 2000). A greater number of cross bridges and the greater control over muscle contractions may lead to increased stiffness.

Females however, are thought to have inadequate joint stiffness, which may predispose them to injury. Granata et al examined active stiffness of the quadriceps and hamstrings during
isometric knee flexion and extension exercises in healthy male (N=12) and female (N=11),
volunteers between the ages of 21-33. Stiffness was measured using EMG data recorded from
the quadriceps and hamstrings as well as motion recorded from an accelerometer attached to the
heel. Females exhibited 56-73% of the effective stiffness of male subjects at each of the load
conditions (0kg, 6kg, and 20% of maximal voluntary excursions). (Granata, 2002-1)

Granata et al completed a follow-up study in 2002, of leg stiffness during two-legged
hopping tasks in healthy (N=11) males and females (N=10) between the ages of 21-31. Two
hopping tasks were performed at the subjects preferred rate and at the rate of 3 Hz measured on a
metronome. Leg stiffness was measured by methods previously reported by McMahon and
Cheng (1990) and Farly (1991). The results indicated that stiffness values were significantly
greater in males than in females at each of the hopping frequencies. Leg stiffness in females was
approximately 77% of the leg stiffness in males (Granata, 2002-2).

Cutting, Jumping and Landing Tasks

Different cutting, jumping and landing characteristics are also thought to predispose
females to higher rates of ACL injury. (Feretti, Gray). Females have been shown to run and cut
with a more valgus knee position, less hip flexion, and greater quadriceps activation (Feretti,
Gray). During landing, when ground reaction forces can reach 3 to14 times body weight, these
dynamic faults seen during running and cutting may increase the risk for injury. Female athletes
also have greater abduction and adduction moments at the knee when landing from a jump than
male athletes (Huston, 1996). James found that females exhibited greater knee joint forces than
the males (James). Higher knee joint forces combined with weaker quadriceps musculature and
delayed hamstring activation in females athletes may lead to a higher incidence of knee injury.
Using 2D motion analysis to record knee flexion angles in the sagittal plane, Huston et al examined gender differences in knee angle when landing from a drop jump (Huston, 2001). Females landed with significantly less knee flexion than men. When landing from a 60 cm height, knee flexion angles were 7° and 16° for women and men respectively. Knee flexion angle is correlated with ground impact forces. As knee flexion angle decreases, ground impact forces increase by 1%. Since females landed 9° straighter than males, there could be a 9% increase in impact load per unit of body weight for females landing from a 60 cm height. Since ACL strain is greater in knee extension (Beynnon, Renström), females could be at increased risk for ACL injury due to the increased strain placed on the ligament during landing compared to males who land with a more flexed knee.

Conversely, Fagenbaum et al. found that female collegiate basketball players landed with approximately 10-14° greater knee flexion than male collegiate basketball players when landing from three different jumps (maximum vertical jump forward 25cm, drop jump from a 25 cm platform, drop jump from a 51 cm platform) (Fagenbaum). This study was limited however by a small subject number (8 females, 6 males) who were from the same institution and probably shared similar training techniques and unlike the Huston et al study, only competitive athletes were tested.

Ford et al examined valgus knee motion during landing in high school female and male basketball players and found that female high school basketball players displayed more valgus knee motion compared to males (Ford). Eighty-one participants (47 females, 34 males) dropped off a box 31 cm from the ground onto both feet and then immediately performed a maximal vertical jump as if they were rebounding a basketball. Three dimensional motional analysis recorded frontal plane motion. In addition to a greater valgus motion in females, females also
showed significant side-to-side differences in valgus knee angles. The dominant legs (leg used to kick a ball) had significantly greater knee valgus angles compared to the non-dominant leg. This is a unique finding as the dominant leg (used to kick a ball) is not the leg a basketball player would use for balance during layups or single leg activities, therefore the dominant leg may be weaker resulting in greater valgus angle at landings. The non-dominant limb (used to balance to kick a ball) in basketball players could be expected to have better control as that is the limb used for take-off during lay-ups.

Chaudhari et al. examined the effects of dynamic limb alignment of knee moments during single limb landing in healthy subjects between the ages of 18-29 years old. Nine males and twelve females performed 90° lateral run-to cut maneuvers while frontal plane movement was recorded with a video camera and a 3D opto-electronic system measured joint kinetics. The results showed that women were more likely to be valgus landers and the valgus landers had a significantly higher knee abduction moment compared to neutral or varus landers (Chaudhari).

Malinzak et al examined knee joint motion during running, side-cutting, and cross-cutting in 11 male and 9 female, recreational athletes (mean age of 24.5 years) and found that females had less knee flexion angle compared to males during all three athletic tasks. In addition, females had a significantly greater knee valgus motion and a higher activation of their quadriceps (Malinzak). The authors concluded that females have different motor control strategies that may alter knee motion patterns and may predispose females to a higher rate of ACL injuries.

A follow up study by Chappell et al. compared knee kinetics between male and female recreational athletes in three stop-jump tasks and found that females land with a more extended knee and greater knee valgus (Chappell). Ten males and ten females completed a two-legged forward jump, backward jump and vertical jump while 3D videography recorded motion. Female
athletes exhibited a knee extension moment during landing while males displayed a knee flexion moment. Similarly, females had a knee valgus moment while males had a knee varus moment upon landing. These results also demonstrated that females have a greater proximal tibial shear force during the landing phase of stop jump tasks which may place excessive strain on the ACL. It can be hypothesized that the greater knee extension moment and greater proximal tibial shear found in females is due to increased quadriceps activation in females. This has been consistently demonstrated in the literature.

Gender differences in strength and lower extremity kinematics were also examined by Lephart et al. Fifteen, female division one athletes and 15 male controls performed two landing tasks (single leg landing off a 20 cm platform onto a “X” and a forward hop 45% of their height away from an “X”). A 3D motion analysis system measured angular displacement and motion. Females landed with significantly less knee flexion. In addition females had more hip internal rotation with tibial external rotation, which is consistent with the mechanism for a non-contact ACL injury (Lephart).

In summary, the gender differences in neuromuscular control previously discussed are thought to contribute to differences in ACL injury risk although the exact contributions are unknown. Females have different strength and recruitment patterns than males. Female athletes are more quadriceps dominant and often rely less on their hamstrings to control the deceleration of the tibia. Females also generate less peak torque per body weight and have lower hamstring to quadriceps ratios. Furthermore, females have less knee stiffness and greater joint laxity when. Joint stiffness is a measure of joint stability and females that have more laxity and less stiffness are thought to have a more unstable joint which may increase the risk of injury. The combination of delayed hamstring muscle recruitment and decreased joint stiffness may be two factors related
to an increase in female ACL injury. Finally, the combination of altered strength and recruitment with increased laxity and decreased knee stiffness may lead to the observed gender differences in cutting and landing techniques. Females land and cut with a more valgus knee position and less knee flexion when compared to males. This position can increase joint reaction force and place greater stress on the ACL. Greater joint reaction forces combined with inefficient hamstring activation and less joint stiffness can lead to the increased ACL injury rate observed in females.

Other Extrinsic Factors

Conditioning, skill, and experience are the final three extrinsic factors to be discussed. Investigations into the conditioning level ACL injury have been primarily concerned with fatigue. It is hypothesized that as an athlete becomes more fatigued they will have less dynamic joint stabilization, which could lead to injury (Rozzi, 1999). It is unknown whether males and females are affected differently by fatigue mostly because the protocols that have induced fatigue have varied so much. However, it has been demonstrated that both male and female athletes exhibit decreases in proprioceptive activity and also have altered neuromuscular ability when fatigued (Rozzi, 1999). These factors may both decrease dynamic joint stabilization, which could lead to injury. It may be that for female basketball and soccer players, the effects of fatigue may lead to an increase rate of ACL injury because these female athletes have greater knee joint laxity and diminished joint kinesthesia as compared to males.

Skill and experience are two extrinsic factors that have not been proven to be related to ACL injury in females but were once hypothesized as factors. In the past, fewer females participated in athletics so it was thought that lack of experience would translate to less skill and coordination which could lead to injury (Garrick). Skill is not well defined and is difficult to measure but it was shown that there were no differences in ACL injury rates among NCAA
division I, II, or III levels when comparing the same gender and sport (Harmon). Even though in
the last few decades females have seem to caught up to their male counterparts in athletic
experience, someone that may not be as skilled or has had less experience may be at an increased
risk for knee injury. More recently, Söderman et al. examined ACL injury rates in young female
soccer players in Sweden and reported that between 1994-1998, 398 females under the age of 19
had suffered an ACL tear. Thirty-nine percent of these injuries were suffered by females under
the age of 16 participating on senior level teams. The authors concluded that young female
soccer players may become injured when playing on senior level teams due to older, heavier, and
stronger opponents involved (Söderman, 2002).

**Intrinsic Factors leading to ACL injury**

Intrinsic factors are those factors that may not be able to changed and include hormonal
influences, joint laxity, limb alignment, and femoral notch size. Although one single factor has
not been determined to be the cause of the higher incidence of ACL injuries in females,
examining ACL injuries as a multifactoral cause is necessary.

**Hormonal Effects and the Menstrual Cycle**

Throughout the three phases of the menstrual cycle there are fluctuating levels of
hormones present. A normal menstrual cycle lasts approximately 28 days and begins with
menses and ends with the onset of menses again. During the beginning of first phase of the
menstrual cycle called the follicular phase, estradiol and progesterone are at their lowest levels.
Then, just before the start of the second phase of the cycle around days 10-13, estradiol and
lutenizing hormone (LH) surge to initiate ovulation. It is at this time, approximately 3 days
before ovulation that estradiol is at the highest level and progesterone is at its lowest level
(Karageanes).
Ovulation is the second phase of the menstrual cycle and describes the period of time when the egg is released from the ovary. It typically occurs about 14 days after menses and it is during this time that pregnancy could occur. Ovulation occurs somewhere between 12-48 hours after the LH surge (Karageanes).

The last phase of the menstrual cycle, the luteal phase, is where progesterone levels are at their highest. As progesterone increases, LH is slowly decreasing. Estradiol begins to rise at a rate similar to progesterone and then tapers 4-5 days before menses. This phase is similar in length to the follicular phase of approximately 14 days (Karageanes).

**Effects of Estrogen**

Estrogen has a variety of effects on the growth and development of several tissues such as bone, muscle, and connective tissue (Lebrun, Shitaka). For example, in two studies on rat tissue, estrogen has been shown to influence collagen and elastin metabolism (Shitaka, Yamamuro). It has also been shown to enhance the sensitivity to relaxin by increasing the concentration of relaxin receptors.

There are several possible mechanisms by which local concentrations of estrogen may weaken the human ACL. First, estrogen has been shown to acutely decrease total collagen and collagen synthesis in rat tissue (Dyer, Fischer, 1973; Fischer, 1977; Hama, Shitaka, Yamamuro). Second, there is evidence that estrogen promotes collagen cross linking (Hama, Yamamuro). This may lead to changes in the strength and type of collagen in the ACL. Type III collagen is structurally weaker than Type I collagen and it is believed that estrogen enhances the production of Type III collagen. Third, estrogen increases the amount of elastin in the rat aorta and rat hip joint capsule which could lead to increase injury rates (Shitaka, Yamamuro). Changes in the elasticity of the human ACL may lead injury. Finally, estrogen has been shown to increase the
concentration of relaxin receptors in the rat myometrium which could make it more sensitive to relaxin. Relaxin reduces the organization of collagen leading to increased elasticity (Shitaka, Yamamuro).

**Estrogen effects on Metabolism**

Estrogen receptors in the blood vessel walls supplying the ACL may effect the metabolism of these cells. Estrogen is a vasodilator and causes increased permeability in the cells of the blood vessel walls of the uterus (Fischer, 1973). Increases in the permeability in the blood vessel walls of the ACL may lead to increased water content which could ultimately disrupt homeostasis and lead to injury (Liu, 1996).

Estrogen has also been shown to affect collagen synthesis. Liu et al in 1997, found there was a decrease in cellular proliferation and total collagen synthesis in a rabbit ACL when treated with estradiol for 2 weeks (Liu, 1997). In a similar in vitro study on human ACL’s, Yu et al reported a significant decrease in fibroblast proliferation and Type I procollagen levels with 17β-estradiol administration (Yu). In this study, Type 3 procollagen was unaffected by estradiol which suggests that differences in fibroblast metabolism cause changes in Type 1 procollagen. The combined effect of decreased fibroblast proliferation and decreased Type I collagen levels could lead to reduced strength of the ACL which may lead to increased risk for injury. In a similar study Yu et al. found that estrogen has a dominant inhibitory effect on ACL fibroblast proliferation and Type I procollagen synthesis. However, in this study, when progesterone was increased, there was an increase in ACL fibroblast proliferation and Type I procollagen synthesis.

The proper function of the ACL depends on the type, crosslinking, and remodeling of collagen. Type I collagen relates to a greater mechanical strength than Type 3 collagen and
Type 3 collagen leads to greater elastic properties. A larger ratio of Type 1 to Type 3 collagen is thought to lead to greater strength where a lower ratio may lead to increased laxity (Laros, Liu, 1995).

**Hormones and Injury Rates**

The relationship these hormones have on each other and their effect on the ACL is still undetermined. In addition, although some studies report that ACL injuries may occur at higher rates during certain phases of the menstrual cycle, there is no consistency in methodology among investigators. Wojtys et al. obtained urine samples from athletes within 24 hours of ACL injury and used hormone analysis to determine which phase of the menstrual cycle the athletes were currently in (Wojtys, 1998). Chi-square analyses were used to calculate the expected and observed frequencies of ACL injury based on menstrual cycle phase. Wojtys et al reported that ACL injury rates were higher among females in the ovulatory phase compared with the luteal or follicular phases. These results clinically seem relevant, because during ovulation there is an estrogen surge that may lead to an increase in injury. Estrogen is known to have catabolic properties of certain tissues in the body. Estrogen decreases collagen synthesis, decreases metabolism, and promotes Type III collagen cross linking. The effects of estrogen on the ACL will be discussed in the following section. This study is severely limited however due to urine collection procedures. Without collecting daily urine samples, it is difficult to determine the exact phase of the menstrual cycle.

Slauterbeck et al., collected saliva samples from athletes within 72 hours of ACL injury and found that 26 of the 37 athletes tested injured their ACL in the first half of their menstrual cycle but the occurrence in the follicular phase was closer to menses than ovulation. This does
not seem to follow the clinical logic because during the first half of the follicular phase, estrogen levels remain fairly low (Slauterbeck, 2002).

**Joint Laxity**

Greater ACL injury rate in females has also been attributed to the amount of joint laxity they possess. Although there is no known link between increased laxity and ACL injury, it has been suggested that females have greater joint laxity than males. Uhorchak et al. measured knee laxity and generalized joint laxity in 859 West Point cadets. Knee laxity was measured using a KT2000 knee arthrometer and generalized joint laxity was determined by a score of 5 or more (bilaterally in three joints) on the following measures: 5th finger MCP hyperextension, elbow hyperextension, knee hyperextension, and thumb hyperextension and abduction to the volar aspect of the arm. Females had greater joint laxity and generalized laxity. Moreover, non-contact ACL injured subjects had greater joint laxity and generalized laxity than non-injured ACL subjects (Uhorchak).

Woodford-Rogers et al. examined risk factors for ACL injury in high school and college athletes and found that the best predictors of ACL group membership for football players were increased navicular drop and increased joint laxity at 20 lbs. of force on a KT1000 knee arthrometer as well as increased laxity with a manually anterior drawer. This classification usually these three variables corrected predicted 71.4% of athletes into the ACL-injured and ACL-uninjured groups. For the female athletes, that the best predictors of ACL group membership were increased navicular drop and increased joint laxity at 20 lbs. of force on a KT1000 knee arthrometer as well as increased laxity with a manually anterior drawer. This classification usually these three variables corrected predicted 87.5% of athletes into the ACL-injured and ACL-uninjured groups (Woodford-Rogers).
Rozzi et al. used a KT1000 knee arthrometer and examined knee laxity in male and female collegiate basketball and soccer players and found females to have more anterior-posterior translation of the tibia (Rozzi, 1999). Rosene and Fogarty conducted a similar study on collegiate male and female athletes participating in women’s basketball, women’s soccer, women’s softball, women’s volleyball, men’s soccer and men’s volleyball (Rosene). They found that females had significantly greater ligament laxity than males with measurement of anterior tibial translation using the KT-1000 knee arthrometer. Laxity was greater in the females when using forces of 67 N, 89 N and 134 N.

Some authors have reported that greater laxity may increase the risk of injury. In 1970, Nicholas found that professional football players categorized as having “loose joints” incurred more knee injuries than teammates with “tight joints” (Nicholas). This study was completed in 1970 however and the methods for determining “looseness” are questionably subjective. Five tests were used to determine joint laxity: flexing the spine so the palms touched the floor, genu recurvatum past 20º with the athlete lying prone (with the knees off the table), sitting in the “W” or “lotus” positions, turning the heels away from each other at a 180º angle (knees and hips externally rotated), and elbow hyperextension with the shoulder flexed and forearm supinated (so the hypothenar eminence was to incline cephalad in a vertical plane with the elbows extended and the forearm supinated).

Grana et al tested a group of male and female high school basketball players and non-athletes for ligament laxity using the same series of five laxity tests previously developed by Nicholas (Grana). They followed the subjects for two years and calculated the total number of injuries within each group of subjects. They found that both groups of girls in the study (basketball players and non-athletes) had significantly more laxity than the boys, however, the
female basketball players had less laxity than the female non-athletes. Of the female basketball players, the ones who sustained the most sprains had “looser” joints. The ligament laxity tests contain measurements of both the upper and lower extremities and are mostly subjective flexibility tests and not actual quantitative measurements of joint laxity. Also, none of the injuries were described in detail by the author.

Although it has been hypothesized that females may exhibit more joint laxity than males, as stated previously, we do not know if increased joint laxity leads to injury. Some authors have reported no correlation between joint laxity and injury in athletes (Decoster, Harner, Hopper). Measurement technique must be questioned when looking at the results of the previous studies as some studies used methods or machines that have not been previously validated (Harmon).

**Hormones and Joint Laxity**

Heitz et al. did not report higher injury rates but did report that females had the greatest anterior laxity in the luteal phase, however laxity also increased with increasing levels of circulating estrogen in the follicular phase. Blood samples were taken on days 1, 10, 11, 12, 13, 20, 21, 22, and 23 of the menstrual cycle from 7 active females between the ages of 21 to 32 years of age. Laxity measurements were taken on the same days using the KT2000 arthrometer (Heitz). This study however did not take any blood samples or laxity measurements between the days of the follicular and luteal phases (days 15-20) which may mean that the laxity seen during the luteal phase was just a delayed effect of the estrogen surge seen at ovulation.

In contrast, Karageanes et al. found that there was no significant change in laxity from the follicular to luteal phases of the menstrual cycle (Karageanes). Subjects consisted of 26 athletes on various sports teams in junior high and high school. Knee laxity was tested by the KT1000 arthrometer each day before competition and athletes recorded their menstrual
characteristics on a calendar. The three phases of the menstrual cycle were determined from this information. No phase in the menstrual cycle clinically effected the ACL more than another. This study contradicts the previous two, however subjects were younger and hormones levels were not measured.

Despite conflicting reports, there are several things that need to be accounted for when determining knee laxity, injury rates, and hormonal levels throughout the menstrual cycle. The methodology, subject homogeneity, and cycle characteristics need to be monitored closely. The following questions also need to be answered: Does knee laxity lead to an increase incidence of ACL injury? How do fluctuating hormone levels affect the ACL?

Several authors have found no relationship between joint laxity and injury rates in both male and female athletes, however methodology and subject characteristics were highly variable (Decoster, harner, Hopper). Rozzi et al did find that female basketball and soccer players had more anterior laxity as measured on the KT1000 when compared to men (Rozzi, 1999, Rozzi, 1999). We also know that female basketball and soccer players suffer ACL injuries at higher rates than other sports so therefore we can possibly conclude that increased laxity may lead to increase in ACL injury. Rosene et al found the same results with a larger group of athletes (Rosene). Despite these previous findings, the methods used to test laxity were all done statically and different instruments were used. Without consistent measurement techniques it is difficult to make solid conclusions. Finally, is increased laxity necessarily bad? Could more laxity lead to more tolerated ligament stretch during an external force? Further research is needed in this area as well.
**Static Postural Alignment**

Biomechanical abnormalities of the lower extremity have been hypothesized to be related to ACL injuries. A retrospective study by Loudon et al measured seven variables: standing pelvic position, hip position, standing sagittal and frontal knee position, hamstring length, prone subtalar joint position, and navicular drop. Twenty ACL injured females were compared to 20 age matched controls. A conditional step-wise logistical regression analysis indicated that the ACL injured group was best predicted by excessive sagittal plane knee position, and excessive navicular drop along with excessive subtalar joint pronation (Loudon). Female athletes demonstrating this posture maintain the knee in a more hyperextended position with subtalar joint pronation. This combination can place an increased stress on the ACL and potentially cause injury.

Woodford-Rogers et al. also found that increased navicular drop may predispose athletes to ACL injury. Authors found that the best predictors of ACL group membership for football players were increased navicular drop and increased joint laxity at 20 lbs. of force on a KT1000 knee arthrometer as well as increased laxity with a manually anterior drawer. This classification usually these three variables corrected predicted 71.4% of athletes into the ACL-injured and ACL-uninjured groups. For the female athletes, that the best predictors of ACL group membership were increased navicular drop and increased joint laxity at 20 lbs. of force on a KT1000 knee arthrometer as well as increased laxity with a manually anterior drawer. This classification usually these three variables corrected predicted 87.5% of athletes into the ACL-injured and ACL-uninjured groups (Woodford-Rogers).

A large quadriceps angle (Q angle) may contribute to increased incidence in female ACL ruptures, although two studies examined this relationship and found no correlation (Gray,
Huegel). The Q angle is an angle formed by the line from the anterior superior iliac spine to the midpoint of the patella and the line from the tibial tuberosity to the midpoint of the patella. In females an angle of more than 20° is thought to be problematic. Theoretically, larger Q angles increase the lateral pull of the quadriceps on the patella and may increase stress to the lateral knee.

In a study of 45 recreational basketball players it was determined that athletes sustaining knee injuries had larger Q angles than those athletes that were injury free (Shambaugh). In addition to a large Q angle, females have wider pelvises, more femoral anteversion, increased external tibial torsion, and more overpronation when compared to males (Ireland). However, even though there have been no studies that show that these factors alone lead to a higher rate of ACL injury, a combination of these anatomical factors may increase the risk for injury.

Some investigators measure Q angle with the knee in 30° of knee flexion to place the knee in a more function position that ACL injuries may occur. When measuring Q angle in knee flexion, the same landmarks are used. Moul et al examined Q angle with the knee extended and with the knee flexed 30° in male and female division 1 basketball players and found significant differences in Q angle measurements between males and females with the knee flexed to 30° (Moul). Males had significantly smaller Q angles when compared to females. There were no significant differences in Q angle with the traditional measurement technique.

In summary, combinations of static alignments may increase the risk of ACL injury. It has been hypothesized that a static posture consisting of an anterior pelvic tilt, inflexible hamstrings, genu recurvatum, and subtalar pronation may place the individual at risk for an ACL tear (Loudon). If the knee is in a hyperextended position caused by genu recurvatum and the tibia has increased internal rotation caused by pes planus or hyperpronation, the ACL can be put under
greater stress during dynamic movements. Anterior pelvic tilt can cause the hamstrings to be put on stretch making them less efficient at controlling tibial deceleration. Loudon et al concluded that the combination of hyperpronation and genu recurvatum were significantly associated with ACL injury risk in female athletes (Loudon, 1996).

In addition to being associated with genu recurvatum, increased anterior pelvic tilt places the hamstrings in an elongated position. Lengthening of the hamstrings may slow their neuromuscular response time (Trontelj), and thus, their capacity to serve as dynamic agonists to the ACL. Conversely, anterior tilt is associated with shortening of the hip flexors, including the rectus femoris (Lee). This may allow for faster neuromuscular facilitation of this muscle (Trontelj) and contribute to the phenomenon of quadriceps dominance hypothesized by Huston and Wojtys (1996).

**Femoral Notch Size**

The size of the femoral notch and the size of the ACL are two other factors that may predispose female athletes to ACL rupture, however the association between a small notch size and incidence of injury is not necessarily related (Huston, 2000). When the knee is in flexion, the ACL comes in contact with the medial margin of the lateral femoral condyle and in extension the ACL comes in contact with the anterior intercondylar notch. It is thought that a small notch increases the chances for impingement during knee motion if the ACL is normal size and shape (Norwood).

It was once thought that a female with a small notch probably has a proportionally small ACL, however this has been recently refuted. Muneta et al. reported that ACL size does not change regardless of the size of the notch (Muneta). Cadavers with a small or normal intercondylar notch width had similar sized ACL’s. Anderson et al confirmed these findings as
he concluded that notch characteristics measured in male and female high school basketball players do not contribute to the gender differences in ACL injury. (Anderson). In both males and females, ACL dimensions did not change with the size of the notch. It was concluded that if notch size contributes to ACL injury, the normal sized ACL in a stenotic notch is probably the cause.

Houseworth et al radiographically measured the areas of the anterior notch opening, posterior arch, and distal femur and reported a significant difference in the ratios of the posterior arch to the total area of the distal femur in people suffering an ACL injury (Houseworth). A smaller posterior notch was identified as a predisposing factor to ACL injury.

In a similar study by Lund-Hanssen et al it was determined that females handball players with 17mm of less anterior notch width at the level of the popliteal groove were six times more susceptible to ACL injury than female handball players with wider notch widths (Lund-Hanssen).

Other authors have also concluded that a smaller anterior outlet of the intercondylar notch may put athletes, both male and female, at an increased risk for ACL injury (Shelbourne, LaPrade, Souryal, Anderson, Good,). This however does not explain the gender difference in ACL injury rates.

**Ankle Instability and ACL Injury Risk**

Since ankle sprains and knee sprains are two of the most common occurrences in athletics, there may be an underlying relationship between the ankle sprains and ACL injuries. Previous literature has demonstrated proximal changes in the hip after lateral ankle sprain and these neuromuscular changes may also affect the knee. It has been described by some authors that the “knee is a slave to the hip” and it is plausible to think that ankle injuries may not only
affect the hip, but also affect the knee. As the gluteal and hamstring muscles control motion of the femur, improper control causing the femur to become internally rotated may lead to adaptations in tibial internal rotation and forefoot pronation thus placing the ACL at risk for injury.

Bulluck-Saxton examined hip muscle function following severe ankle sprain in a group of 20 males with a history of unilateral sprain compared to a healthy group (Bullock-Saxton). EMG was recorded for the gluteus maximus, biceps femoris, and erector spinae muscles bilaterally. Results showed that the combined right and left gluteus maximus activity was delayed in the injured group compared to the uninjured group. The author’s conclusions were that this decreased gluteus maximus activity could be due to an altered gait pattern adapted during the time of initial ankle injury or due to a centralized reflex chain of events in both proximal limbs after distal unilateral injury. If ankle injury can lead to changes in hip muscle recruitment, knee mechanics may also be altered.

A study by Richards et al found that there was a relationship between ankle joint dynamics and patellar tendinopathy in elite volleyball players further supporting the idea that the ankle can affect the proximal knee joint (Richards). In this study, three-dimensional kinematics of the ankle, knee, and hip showed that a high inversion moment at the ankle at the time of landing from a spike jump was a significant predictor of patellar tendinopathy. This study provides evidence that kinematics at the ankle can affect the knee proximally.

Distal changes at the ankle after LAS may also affect lower extremity kinematics. Previous literature has demonstrated that changes at the ankle/subtalar joint complex may affect pronation at the foot and tibial internal rotation which may place stress on the ACL (Loudon, Woodford-Rogers). Subtalar joint instability after LAS has also been reported (Hertel, Ishi,
Meyer). Again, altered mechanics at the subtalar joint can affect the kinetic chain proximally. In addition, postural control changes after LAS and chronic ankle instability have been previously reported in the literature (Hertel). Altered postural control may be a risk factor for ACL injury although further research in this area is needed.

**Conclusions**

After a thorough literature search there are still several questions left unanswered and coming closer to answering the question as to why females suffer more ACL injuries has not been accomplished. What is known is that female athletes suffer 2 to 8 times more ACL injuries than their male counterparts and that ACL injuries in females usually involve a non-contact mechanism and the etiology is believed to be multifactorial in nature.

The combined role of intrinsic and extrinsic factors may lead to a higher injury rate in females. What the combination of these factors is, however, is not known. Future research in this area is needed to identify possible combinations of hormonal and biomechanical factors that may be inherent to females. Future studies should focus on improving both the methodology of measuring hormones, laxity, and biomechanics, and on recruiting a homogenous group of subjects controlling for age, menstrual cycle characteristics, and activity level. The relationship between previous ankle sprain and history of ACL injury warrants further research. In addition research should begin to examine the role of oral contraceptives and their affect on the menstrual cycle and injury, as well as taking a more longitudinal approach to the problem of female ACL injuries.
CHAPTER 3

METHODS

Subjects

Fifty-two, physically active participants (33 females, 19 males, age = 22.1 ± 3.1 years, height = 171.9 ± 8.8 centimeters, weight = 72.9 ± 13.7 kilograms) with a history of ACL injury volunteered for this study. All ACL injuries occurred within 5 years of the onset of the study. For the females, there were 27 participants with a unilateral ACL injury and 6 participants with bilateral ACL injuries for a total of 39 injured limbs and 27 uninjured limbs. For the males, there were 16 participants with a unilateral ACL injury and 3 participants with bilateral ACL injuries for a total of 22 injured limbs and 16 uninjured limbs. Thirty-three, physically active participants volunteered as female controls. All subjects signed an informed consent form approved by the Pennsylvania State University Institutional Review Board. Subjects had to be free from lower extremity injury or surgery in the previous five months.

Protocol

All participants reported to the Athletic Training Research Laboratory for a 30-minute session. Participants signed an informed consent form, completed a survey about their knee and ankle injury history and then had 16 different measures of lower extremity malalignment and flexibility taken. All measurements were taken by the same examiner who was blinded to the subjects injury history.

Five categories of intrinsic or anatomical risk factors were measured or observed: generalized laxity, lower extremity malalignment, lower extremity range of motion and flexibility, functional reach, and static balance. Within the five categories of intrinsic risk factors,
sixteen dependent measures were assessed. All measures are commonly used in sports medicine practice and have been previously reported as reliable measures of lower extremity alignment.

**Generalized laxity**

For generalized laxity using the Beighton laxity scale (Beighton) an overall laxity score was used as the dependent variable. Visual observation was used to record laxity in different joints throughout the body. All observations were made bilaterally. A “yes” or “no” was circled if laxity was observed in the following joints: opposition of thumb to palmar forearm, hyperextension of 5th MCP joint beyond 90°, hyperextension of elbow beyond 10°, hyperextension of knee beyond 10°, palms flat on floor with trunk flexion and knee extension. A total score was recoded as a generalized laxity measurement.

**Lower extremity malalignments**

For lower extremity malalignments, foot type, Morton’s toe, navicular drop, pelvic tilt, leg length, tibial varum, standing Q angle, genu recurvatum, and femoral anteversion were used as the nine dependent variables.

Foot type was categorized by visual observation using the visual observation methods reported by Dahle (Dahle). The examiner recorded foot type as pes planus, pes cavus, or pes rectus. Each foot was classified separately. Morton’s toe was also categorized as “yes” or “no” by visual observation. If the second toe was longer than the first, a “yes” was recorded.

Navicular drop, which is a measure of pronation, was measured with a Vernier caliper (Mitutoyo America Corporation, Mitotoyo, Japan) using the methods previously described by Brody (Brody). This test involves marking the midpoint of the navicular tuberosity of the foot with the athlete in a seated position with their knees and hips at a 90° angle. Subtalar joint neutral is maintained and the Vernier caliper is used to record a starting position in millimeters. The
subject is then asked to stand up so the foot relaxes and the Vernier caliper is used to take a final position in millimeters. Navicular drop is the difference between the starting and final positions. Each foot was measured separately.

Pelvic tilt was measured with the subject using a Palm PALpation Meter (Performance Attainment Associates, St. Paul, MN) using methods previously reported by Krawiec (Krawiec). The Palm PALpation meter measures sagittal plane rotation of the innominate bone. This tool is made of an inclinomter and two caliper arms. The bubble inclinomter is a semi-circular arc with one-degree gradations that range from 0º- 30º on either side of the midline. The caliper tips allow for direct palpation of bony landmarks, the ASIS and PSIS. Each hip was measured separately and the amount of pelvic tilt was recorded in degrees.

Leg length was measured with the subject in a supine position using a tape measure. Each leg was measured separately and the distance from the ASIS to the medial malleolus was recorded in centimeters. For leg length discrepancy, the difference in centimeters (to the nearest tenth) between the right and left legs was used.

Standing Q angle was measured with the subject in a standing, relaxed position with a goniometer and a tape measure. A line was drawn from the ASIS so that it bisected the midpoint of the patella. A second line was drawn from the midpoint of the tibial tuberosity so that it bisected the midpoint of the patella. A standard goniometer (Baseline Assessment Products, Montreal, Canada) was used to measure the angle formed by the two lines.

Tibial varum was measured with methods reported by McPoil (Tomaro). The subject stood on a firm surface in a comfortable bilateral stance. The posterior, distal one-third of the leg was bisected at the widest point of the gastrocnemius and again between the medial and lateral malleolus. A line was drawn to connect the midpoints. Each leg was measured separately and a
goniometer was used to measure the angle between the bisection of the distal one-third of the leg and the vertical.

Genu recurvatum was measured with the athlete standing. The examiner aligned the axis of the goniometer over the lateral joint line and the stationary arm of the goniometer in line with the greater trochanter of the femur and the movable arm of the goniometer with the lateral malleolus of the fibula. Each leg was measured separately and the angle formed between the two lines was recorded with the goniometer.

Femoral anteversion was measured by methods illustrated by Ruwe (Ruwe). The subject was placed in a prone position with the knee flexed to 90°. While maintaining the knee in flexion, the examiner used her other hand to palpate the greater trochanter at the point where it was halfway between its most lateral and medial position, indicating the hip was in a neutral position. Each leg was measured separately and a standard goniometer was used to measure the angle between the vertical and the line drawn from the tibial tuberosity to the bisection of the medial and lateral malleolus.

**Lower extremity range of motion and flexibility**

For lower extremity range of motion and flexibility, ankle dorsiflexion range of motion, the 90/90 test, the Thomas test, and the Ober’s Test were the four dependent variables.

Ankle dorsiflexion range of motion was used to quantify gastrocnemius flexibility and was measured using a fluid-filled inclinometer (Fabrications Enterprises Inc., Irvington, NY) with methods previously reported by Denegar (Denegar). The subject was positioned supine so the knee was extended and the ankle was placed at 90°. The inclinometer was fastened to a Velcro strap placed around the subject’s foot. The subject was then asked to actively dorsiflex the ankle and the angle of dorsiflexion was then recorded. Each ankle was measured separately. The
starting position was 0° (when the ankle was placed at 90° by the examiner) and the angle increased as the foot was actively moved into dorsiflexion.

The 90/90 test was used to measure hamstring flexibility. The subject was positioned supine with the knee and hip each at a 90° angle. The subject was then asked to actively extend the knee. The examiner placed an inclinometer fastened to a Velcro strap over the midpoint of the calf to measure active knee extension which helps determine hamstring flexibility. The inclinometer was aligned at 90° of knee flexion and full knee extension was recorded as 180°. Each leg was measured separately.

The Thomas test was used to measure the flexibility of the hip flexors. The subject was positioned standing with their buttocks against the end of a table and asked to lie back onto the table pulling both knees to the chest and then holding on to one leg while the other leg relaxed into hip extension. The test leg was the leg that was lowered. The examiner made sure the lumbar spine was in contact with the table and then placed an inclinometer fastened to a Velcro strap over the lateral midpoint of the thigh parallel to the femur to measure passive hip extension. At the starting position, the hip would was at 0° when the femur and trunk were parallel to each other. As the hip passively extended or flexed from the 0° starting position, the angle was recorded on the inclinometer. Each leg was tested separately.

The Ober’s test was used to measure iliotibial band (ITB) and tensor fascia latae (TFL) flexibility. The subject was positioned side lying (on the opposite side being tested) at the long edge of a table with the examiner standing behind the athlete. The examiner stabilizes the pelvis with one hand and grasps the femur above the knee with the other hand. The examiner abducts and extends the hip and then allows it to passively adduct, making sure no internal or external rotation of the leg occurred. The examiner placed an inclinometer fastened to a Velcro strap over
the lateral midpoint of the thigh parallel to the femur to measure passive hip adduction which helps determine ITB and TFL flexibility. At the starting position, the hip would be at 0° (parallel to the table) and would increase in degrees as the hip became adducted towards the table. Each leg was tested separately.

*Functional Reach*

For functional reach, an anterior reach test was used as the dependent variable. The subject was positioned standing with both feet together to begin the anterior reach. A tape measure was placed on the floor in front of the subject and was used to record reach distance in centimeters. The subject was asked to maintain single leg stance and reach as far as possible with the opposite leg along the tape measure on the floor. The subject was asked to keep the stance foot flat on the ground but was given permission to slightly flex the knee to complete the reach with the opposite leg. The reach foot touched the line as lightly as possible without any weight transfer to the reach leg. The subject then returned to a bilateral stance. The average of the three anterior reaches was calculated for the function reach score. Each leg was measured separately.

*Static Balance*

For static balance, the balance error scoring system (BESS) was used as the dependent variable. The BESS assessed single leg balance with the eyes closed while standing on a firm surface. Subjects were asked to position their hands on their hips while maintaining a single leg stance. The subjects were asked to stand quietly and as motionless as possible for a 20 second trial. They were instructed to make corrections if they lost their balance. A stopwatch recorded the 20-second trial and time began when the subjects closed their eyes. The total score for the BESS was assessed by giving a point for each error made during the 20 second trial. An error was recorded if any of the following occurred during the trial: eyes opened, hands cam off hips,
touchdown of non-stance leg, hip abduction > 30°, or losing position for more than 5 seconds. Each leg was tested separately.

**Statistical Analysis**

The 14 continuous dependent variables (generalized laxity, navicular drop, pelvic tilt, leg length discrepancy, tibial varum, standing Q angle, genu recurvatum, femoral anteversion, dorsiflexion ROM, hamstring flexibility, ITB flexibility, hip flexor flexibility, anterior reach and BESS) were analyzed using one-way ANOVA’s to compare the injured and uninjured groups. Additionally, the data were divided into thirds (Table 3.1) and chi-square analyses were performed to identify associations between the dependent measures and group status. The three categorical variables (foot type, Morton’s toe, and ankle injury history) were analyzed using a chi-square analysis. For both analyses, the alpha level was set a priori at .05.

The 12 variables that were identified as significantly different between injured and uninjured limbs in the preliminary analyses were then entered into a series of discriminant analyses. The goal of these analyses was to identify factors that most highly related to ACL injury. The predictor variables were entered together into the initial discriminant analysis. The variable offering the least relationship to injury status was removed and another discriminant analysis was run until only the single most predictive variable was identified. Eleven, separate analyses were calculated and the alpha level was set a priori at .05.
Table 3.1 Malalignment Measures Divided into Thirds

<table>
<thead>
<tr>
<th>LE Malalignment Measures</th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beighton Laxity Scale</td>
<td>≤ 3</td>
<td>4 to 5</td>
<td>&gt; 6</td>
</tr>
<tr>
<td>Navicular Drop</td>
<td>≤ 5</td>
<td>6 to 7</td>
<td>&gt; 8</td>
</tr>
<tr>
<td>Tibial Varum</td>
<td>≤ 4</td>
<td>5 to 7</td>
<td>&gt; 8</td>
</tr>
<tr>
<td>Q Angle</td>
<td>≤ 12</td>
<td>13 to 14</td>
<td>&gt; 15</td>
</tr>
<tr>
<td>Genu Recurvatum</td>
<td>≤ 4</td>
<td>5 to 7</td>
<td>&gt; 8</td>
</tr>
<tr>
<td>Femoral Anteversion</td>
<td>≤ 9</td>
<td>10 to 11</td>
<td>&gt; 12</td>
</tr>
<tr>
<td>Pelvic Tilt</td>
<td>≤ 9</td>
<td>10 to 11</td>
<td>&gt; 12</td>
</tr>
<tr>
<td>Leg Length</td>
<td>≤ -.5</td>
<td>-.4 to .4</td>
<td>&gt; .5</td>
</tr>
<tr>
<td>Dorsiflexion ROM</td>
<td>≤ 10</td>
<td>11 to 14</td>
<td>&gt; 15</td>
</tr>
<tr>
<td>90/90 Test</td>
<td>≤ 150</td>
<td>151 to 169</td>
<td>&gt; 170</td>
</tr>
<tr>
<td>Thomas Test</td>
<td>≤ 9</td>
<td>10 to 15</td>
<td>&gt; 16</td>
</tr>
<tr>
<td>Ober's Test</td>
<td>≤ 18</td>
<td>19 to 22</td>
<td>&gt; 23</td>
</tr>
<tr>
<td>Anterior Reach</td>
<td>≤ 69</td>
<td>70 to 73</td>
<td>&gt; 74</td>
</tr>
<tr>
<td>BESS</td>
<td>≤ 1</td>
<td>2 to 3</td>
<td>&gt; 4</td>
</tr>
</tbody>
</table>
CHAPTER 4

RESULTS

Injured vs. Uninjured Subjects

When comparing the injured and uninjured females, a one-way ANOVA revealed significant group differences ($p < .05$) for the following variables: laxity, tibial varum, hamstring flexibility, ITB flexibility, Q angle, and pelvic tilt. (Table 4.1) The chi square analyses revealed significant associations for the following variables: laxity, tibial varum, Q angle, genu recurvatum, femoral anteversion, dorsiflexion ROM, hamstring flexibility, ITB flexibility, hip flexor flexibility, foot type, and ankle injury history. (Table 4.2-4.13)

Ankle Sprain History and ACL Injury History

When comparing ACL injured males and females with uninjured controls, results of the chi-square analysis revealed a significant association between ACL injury history and ankle sprain history ($\chi^2 = 5.97$, df = 1, $p = .015$, Cramer’s V=.244). (Table 4.14) Those with a history of ACL sprain were more likely to also have a history of ankle sprain. Of the 61 ACL injured limbs, 34 had a previous history of ankle injury (55.7%). Of the 61 ACL injured limbs, 27 did not have previous history of ankle injury (44.3%). Of the 39 uninjured matched limbs, 12 had a previous history of ankle injury (30.8%). Of the 39 uninjured matched limbs, 27 did not have a previous history of ankle injury (69.2%). The Cramer’s V of .244 explains the strength association between ACL sprain and ankle sprain history. A Cramer’s V of 0 to .1 explains a weak association, a .1-.3 Cramer’s V explains a moderate association, while a Cramer’s V of .3 and greater explains a strong association.
Predictive Factors of ACL Injuries

When comparing the injured and uninjured females, the 12 variables (generalized laxity, pelvic tilt, tibial varum, standing Q angle, genu recurvatum, femoral anteversion, dorsiflexion ROM, hamstring flexibility, ITB flexibility, hip flexor flexibility, foot type, and ankle injury history) that were identified in the preliminary analyses were then entered into a discriminant analysis to identify factors that most highly related to ACL injury.

The 12 dependent variables that were entered into the discriminant analysis as a group, explained 35.2% of ACL group membership. The discriminant regression model that best describes the factors most related to ACL injury history (in descending order from strongest relationship) are generalized laxity ($r^2$ change = .073), genu recurvatum ($r^2$ change = .069), and ITB flexibility ($r^2$ change = .069). Of the 35.2% association between ACL group membership explained by the twelve variables, 21.1% was explained by an increase in generalized laxity (7.3%), increased genu recurvatum (6.9%), and decreased ITB flexibility (6.9%). (Table 4.15) This discriminant regression model correctly predicted 67% of cases with history of ACL injury and 70% of cases without ACL injury.

To further validate this model we performed a backward stepwise linear regression analysis of the relationship of the twelve dependent variables to ACL group membership. Using a cutoff value of $p<0.05$, we eliminated individual predictor variables that did not significantly relate to the association of group membership in the presence of other significant predictor variables. The three statistically significant contributors were: generalized laxity ($p=.004$), ITB inflexibility ($p=.01$), and genu recurvatum ($p=.029$).
Ankle sprain history as a predictive factor of ACL injury history

When comparing the injured and uninjured females, comparing ankle injury history to other potential factors such as malalignments and laxity, ankle injury history did not have as strong of a relationship ($r^2$ change = .018) to ACL injury history as generalized laxity, genu recurvatum, ITB flexibility, tibial varum, q-angle, and femoral anteversion. A series of discriminant analyses were subsequently calculated using ankle sprain as the grouping variable to see if these other measures were also strongly related to ankle sprain history.

The six dependent variables that were entered into the analysis as a group, explained 12.9% of ankle injury group membership. The discriminant regression model that best describes the factors most related to ankle injury group membership were generalized laxity ($r^2$ change = .069) and ITB flexibility ($r^2$ change = .053). Of the 12.9% association between ankle injury group membership explained by the six variables, 12.2% can be explained by an increase in generalized laxity (6.9%) and decreased ITB flexibility (5.3%). (Table 4.16) The discriminant regression model with these 2 variables correctly identified 59% of subjects with a history of ankle sprain and 65% of subjects without a history of ankle sprain.
Table 4.1 Injured vs. Uninjured Females (One-way ANOVA)

<table>
<thead>
<tr>
<th>Malalignment Measures</th>
<th>Injured Mean (SD)</th>
<th>Control Mean (SD)</th>
<th>F Value</th>
<th>P Value</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beighton Laxity Scale</td>
<td>5.23 (2.55)</td>
<td>3.81 (2.18)</td>
<td>11.61</td>
<td>.01*</td>
<td>.65</td>
</tr>
<tr>
<td>Navicular Drop</td>
<td>6.29 (2.86)</td>
<td>6.50 (2.72)</td>
<td>.19</td>
<td>.66</td>
<td>.08</td>
</tr>
<tr>
<td>Tibial Varum</td>
<td>5.33 (3.11)</td>
<td>7.00 (2.51)</td>
<td>11.47</td>
<td>.01*</td>
<td>.67</td>
</tr>
<tr>
<td>Q Angle</td>
<td>12.95 (2.41)</td>
<td>14.19 (2.21)</td>
<td>9.50</td>
<td>.01*</td>
<td>.56</td>
</tr>
<tr>
<td>Genu Recurvatum</td>
<td>6.12 (4.90)</td>
<td>6.22 (2.50)</td>
<td>.03</td>
<td>.88</td>
<td>.04</td>
</tr>
<tr>
<td>Femoral Anteversion</td>
<td>10.18 (2.95)</td>
<td>11.11 (2.72)</td>
<td>3.50</td>
<td>.06</td>
<td>.34</td>
</tr>
<tr>
<td>Pelvic Tilt</td>
<td>10.35 (3.30)</td>
<td>11.69 (3.87)</td>
<td>4.64</td>
<td>.03*</td>
<td>.35</td>
</tr>
<tr>
<td>Leg Length</td>
<td>87.97 (5.40)</td>
<td>86.61 (3.56)</td>
<td>2.94</td>
<td>.09</td>
<td>.38</td>
</tr>
<tr>
<td>Dorsiflexion ROM</td>
<td>13.46 (4.93)</td>
<td>12.14 (3.79)</td>
<td>2.97</td>
<td>.09</td>
<td>.35</td>
</tr>
<tr>
<td>90/90 Test</td>
<td>162.56 (14.90)</td>
<td>156.62 (12.86)</td>
<td>6.01</td>
<td>.02*</td>
<td>.46</td>
</tr>
<tr>
<td>Thomas Test</td>
<td>11.99 (8.82)</td>
<td>12.85 (9.06)</td>
<td>.31</td>
<td>.58</td>
<td>.09</td>
</tr>
<tr>
<td>Ober's Test</td>
<td>19.33 (5.12)</td>
<td>22.47 (5.74)</td>
<td>11.01</td>
<td>.01*</td>
<td>.55</td>
</tr>
<tr>
<td>Anterior Reach</td>
<td>72.00 (6.47)</td>
<td>70.44 (9.32)</td>
<td>1.24</td>
<td>.27</td>
<td>.17</td>
</tr>
<tr>
<td>BESS</td>
<td>3.18 (2.63)</td>
<td>3.08 (2.90)</td>
<td>.05</td>
<td>.83</td>
<td>.03</td>
</tr>
</tbody>
</table>

* p< .05

Table 4.2 Overall Chi Square Results for Injured vs. Uninjured Females

<table>
<thead>
<tr>
<th>LE Malignment Measures</th>
<th>χ2 Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beighton Laxity Scale</td>
<td>13.39</td>
<td>0.01*</td>
</tr>
<tr>
<td>Navicular Drop</td>
<td>0.6</td>
<td>0.74</td>
</tr>
<tr>
<td>Tibial Varum</td>
<td>15.08</td>
<td>0.01*</td>
</tr>
<tr>
<td>Q Angle</td>
<td>9.75</td>
<td>0.01*</td>
</tr>
<tr>
<td>Genu Recurvatum</td>
<td>13.21</td>
<td>0.01*</td>
</tr>
<tr>
<td>Femoral Anteversion</td>
<td>8.9</td>
<td>0.01*</td>
</tr>
<tr>
<td>Pelvic Tilt</td>
<td>0.62</td>
<td>0.73</td>
</tr>
<tr>
<td>Leg Length</td>
<td>2.1</td>
<td>0.35</td>
</tr>
<tr>
<td>Dorsiflexion ROM</td>
<td>7.86</td>
<td>0.02*</td>
</tr>
<tr>
<td>90/90 Test</td>
<td>6.5</td>
<td>0.04*</td>
</tr>
<tr>
<td>Thomas Test</td>
<td>6.5</td>
<td>0.04*</td>
</tr>
<tr>
<td>Ober's Test</td>
<td>12.4</td>
<td>0.01*</td>
</tr>
<tr>
<td>Anterior Reach</td>
<td>2.83</td>
<td>0.24</td>
</tr>
<tr>
<td>BESS</td>
<td>2.53</td>
<td>0.28</td>
</tr>
<tr>
<td>Ankle History</td>
<td>5.27</td>
<td>0.02*</td>
</tr>
<tr>
<td>Morton's Toe</td>
<td>2.8</td>
<td>0.1</td>
</tr>
<tr>
<td>Foot Type</td>
<td>6.36</td>
<td>0.04*</td>
</tr>
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</table>

*p< .05
### Table 4.3 Beighton Laxity Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>14</td>
<td>18</td>
<td>34</td>
<td>13.39</td>
<td>.01*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>14/66=35%</td>
<td>18/66=27.3%</td>
<td>34/66=51.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>26</td>
<td>26</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>26/66=39.4%</td>
<td>26/66=39.4%</td>
<td>14/66=21.2%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
* p< .05

### Table 4.4 Tibial Varum Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>30</td>
<td>22</td>
<td>14</td>
<td>15.08</td>
<td>.01*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>30/66=45.5%</td>
<td>22/66=33.3%</td>
<td>14/66=21.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>10</td>
<td>29</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>10/66=15.2%</td>
<td>29/66=43.9%</td>
<td>27/66=40.9%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
* p< .05

### Table 4.5 Q Angle Chi Square

<table>
<thead>
<tr>
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<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
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<th>P Value</th>
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</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>30</td>
<td>22</td>
<td>14</td>
<td>9.75</td>
<td>.01*</td>
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<tr>
<td>% of Injury</td>
<td>30/66=45.5%</td>
<td>22/66=33.3%</td>
<td>14/66=21.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>20</td>
<td>15</td>
<td>31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>20/66=30.3%</td>
<td>15/66=22.7%</td>
<td>31/66=47%</td>
<td></td>
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</tr>
</tbody>
</table>
* p< .05

### Table 4.6 Recurvatum Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>24</td>
<td>14</td>
<td>28</td>
<td>13.21</td>
<td>.01*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>24/66=36.4%</td>
<td>14/66=21.2%</td>
<td>28/66=42.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>16</td>
<td>34</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>16/66=24.2%</td>
<td>34/66=51.5%</td>
<td>16/66=24.2%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
* p< .05
### Table 4.7 Anteversion Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
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<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
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<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>30</td>
<td>15</td>
<td>21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of Injury</td>
<td>30/66=45.5%</td>
<td>15/66=22.7%</td>
<td>21/66=31.8%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>14</td>
<td>24</td>
<td>28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>14/66=21.2%</td>
<td>24/66=36.4%</td>
<td>28/66=42.4%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p< .05

### Table 4.8 Dorsiflexion Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>22</td>
<td>9</td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of Injury</td>
<td>22/66=33.3%</td>
<td>9/66=13.6%</td>
<td>35/66=53%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>25</td>
<td>20</td>
<td>21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>25/66=37.9%</td>
<td>20/66=30.3%</td>
<td>21/66=31.8%</td>
<td></td>
<td></td>
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</tbody>
</table>

* p< .05

### Table 4.9 90/90 Test Chi Square

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<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>18</td>
<td>22</td>
<td>26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of Injury</td>
<td>18/66=27.3%</td>
<td>22/66=33.3%</td>
<td>26/66=39.4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>31</td>
<td>20</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>31/66=47%</td>
<td>20/66=30.3%</td>
<td>15/66=22.7%</td>
<td></td>
<td></td>
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</tbody>
</table>

* p< .05

### Table 4.10 Thomas Test Chi Square

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<thead>
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<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
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<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>25</td>
<td>21</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of Injury</td>
<td>25/66=37.9%</td>
<td>21/66=31.8%</td>
<td>20/66=30.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>13</td>
<td>33</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>13/66=19.7%</td>
<td>33/66=50%</td>
<td>20/66=30.3%</td>
<td></td>
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</tr>
</tbody>
</table>

* p< .05
Table 4.11 Ober’s Test Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Lower Third</th>
<th>Middle Third</th>
<th>Upper Third</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>29</td>
<td>19</td>
<td>18</td>
<td>12.4</td>
<td>.01*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>29/66=43.9%</td>
<td>19/66=28.8%</td>
<td>18/66=27.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>11</td>
<td>23</td>
<td>32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>11/66=16.7%</td>
<td>23/66=34.8%</td>
<td>32/66=48.5%</td>
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* p< .05

Table 4.12 Ankle History Chi Square

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<tr>
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<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>34</td>
<td>32</td>
<td>5.27</td>
<td>.02*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>34/66=51.5%</td>
<td>32/66=48.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>21</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>21/66=31.8%</td>
<td>45/66=68.2%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p< .05

Table 4.13 Foot Type Chi Square

<table>
<thead>
<tr>
<th></th>
<th>Planus</th>
<th>Rectus</th>
<th>Cavus</th>
<th>$\chi^2$</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL Injury (YES)</td>
<td>27</td>
<td>33</td>
<td>6</td>
<td>6.36</td>
<td>.04*</td>
</tr>
<tr>
<td>% of Injury</td>
<td>27/66=40.9%</td>
<td>33/66=50%</td>
<td>6/66=9.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>18</td>
<td>32</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>18/66=27.3%</td>
<td>32/66=48.5%</td>
<td>16/66=24.2%</td>
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</tr>
</tbody>
</table>

* p< .05

Table 4.14 Chi-Square for ACL History vs. Ankle Sprain History (Males and Females)

<table>
<thead>
<tr>
<th></th>
<th>Ankle Injury (YES)</th>
<th>Ankle Injury (NO)</th>
<th>$\chi^2$</th>
<th>P Value</th>
<th>Cramer V</th>
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<td>34</td>
<td>27</td>
<td>5.971</td>
<td>.02*</td>
<td>.24</td>
</tr>
<tr>
<td>% of Injury</td>
<td>34/61=56%</td>
<td>27/61=44%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACL Injury (NO)</td>
<td>12</td>
<td>27</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of No Injury</td>
<td>12/39=31%</td>
<td>27/39=69%</td>
<td></td>
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* p< .05
Table 4.15 Discriminant Analysis (Factors Most Related to ACL Injury History)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Canonical Correlation</th>
<th>$r^2$</th>
<th>$r^2$ change</th>
<th>Variable Removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>0.593</td>
<td>35.2</td>
<td>0.4</td>
<td>Thomas</td>
</tr>
<tr>
<td>11</td>
<td>0.59</td>
<td>34.8</td>
<td>0.5</td>
<td>Dorsiflexion</td>
</tr>
<tr>
<td>10</td>
<td>0.586</td>
<td>34.3</td>
<td>0.5</td>
<td>90/90</td>
</tr>
<tr>
<td>9</td>
<td>0.581</td>
<td>33.8</td>
<td>0.5</td>
<td>Tilt</td>
</tr>
<tr>
<td>8</td>
<td>0.577</td>
<td>33.3</td>
<td>0.7</td>
<td>Foot Type</td>
</tr>
<tr>
<td>7</td>
<td>0.571</td>
<td>32.6</td>
<td>1.8</td>
<td>Ankle</td>
</tr>
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<td>6</td>
<td>0.555</td>
<td>30.8</td>
<td>2</td>
<td>Anteversion</td>
</tr>
<tr>
<td>5</td>
<td>0.537</td>
<td>28.8</td>
<td>3</td>
<td>Q angle</td>
</tr>
<tr>
<td>4</td>
<td>0.508</td>
<td>25.8</td>
<td>4.7</td>
<td>Tibial Varum</td>
</tr>
<tr>
<td>3</td>
<td>0.459</td>
<td>21.1</td>
<td>6.9</td>
<td>Obers *</td>
</tr>
<tr>
<td>2</td>
<td>0.377</td>
<td>14.2</td>
<td>6.9</td>
<td>Recurvatum *</td>
</tr>
<tr>
<td>1</td>
<td>0.27</td>
<td>7.3</td>
<td>7.3</td>
<td>Laxity *</td>
</tr>
</tbody>
</table>

* Substantial predictors of ACL injury history group membership

Table 4.16 Discriminant Analysis (Factors Most Related to Ankle Injury History)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Canonical Correlation</th>
<th>$r^2$</th>
<th>$r^2$ change</th>
<th>Variable Removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>0.359</td>
<td>12.8</td>
<td>0</td>
<td>Tibial Varum</td>
</tr>
<tr>
<td>5</td>
<td>0.359</td>
<td>12.8</td>
<td>0</td>
<td>Q angle</td>
</tr>
<tr>
<td>4</td>
<td>0.358</td>
<td>12.8</td>
<td>0.2</td>
<td>Anteversion</td>
</tr>
<tr>
<td>3</td>
<td>0.355</td>
<td>12.6</td>
<td>0.4</td>
<td>Recurvatum</td>
</tr>
<tr>
<td>2</td>
<td>0.349</td>
<td>12.2</td>
<td>5.3</td>
<td>Obers *</td>
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<tr>
<td>1</td>
<td>0.262</td>
<td>6.9</td>
<td>6.9</td>
<td>Laxity *</td>
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</table>

* Substantial predictors of ankle injury history group membership
CHAPTER 5
DISCUSSION

Predictive Factors of ACL Injuries

The discriminant analysis used to identify factors most highly related to ACL injury revealed that of the 35.2% association between ACL group membership explained by the twelve variables initially entered into the analysis, 21.1% was explained by an increase in generalized laxity (7.3%), increased genu recurvatum (6.9%), and decreased ITB flexibility (6.9%). This discriminant regression model correctly predicted 67% of cases with history of ACL injury and 70% of cases without ACL injury.

Increase in Generalized Laxity

Although it has been hypothesized that females may exhibit more joint laxity than males, we do not know if increased joint laxity leads to injury. Some authors have reported no correlation between joint laxity and injury in athletes (Decoster, Harner, Hopper) while others have reported that increased laxity may lead to an increase in injury risk (Nicholas, Grana, Urochak, Woodford-Rogers). However, measurement technique must be questioned when looking at the results of the previous studies as some studies used methods or instruments that have not been previously validated (Harmon).

Nicholas found that football players categorized as having “loose joints” incurred more knee injuries than teammates with “tight joints” (Nicholas). Using the Beighton scale, Grana et al (Grana) tested a group of male and female high school basketball players and non-athletes for ligament laxity. They followed the subjects for two years and calculated the total number of injuries within each group of subjects. Of the female basketball players, the ones who sustained the most sprains had “looser” joints. The ligament laxity tests contain measurements of both the
upper and lower extremities and are mostly subjective flexibility tests and not actual quantitative measurements of joint laxity. Also, none of the injuries were described in detail by the author.

Uhorchak et al. assessed knee laxity and generalized joint laxity in 859 West Point cadets. Knee laxity was measured using a KT2000 knee arthrometer and similar to the Beighton scale, generalized joint laxity was determined by subjects having a score of 5 or more (bilaterally in three joints) on the following measures: 5th finger MCP hyperextension, elbow hyperextension, knee hyperextension, and thumb hyperextension and abduction to the volar aspect of the arm. There was a significant main effect when looking at non-contact ACL injured subjects versus healthy controls for both knee laxity and generalized laxity where injured subjects showed increases on both measures. Injured subjects had both greater knee laxity and greater generalized laxity (Uhorchak).

*Increase in Genu Recurvatum*

Since knee hyperextension is one of the generalized laxity measures, it is not surprising to find genu recurvatum to be a predictor of ACL group membership. Soderman et al (Soderman, 2001) examined risk factors for leg injuries in female soccer players and found that both generalized joint laxity and knee hyperextension (>10°) significantly increased the risk for traumatic leg injuries in soccer. Generalized joint laxity was observed using a modified Beighton scale where knee hyperextension was an included measure.

Loudon et al also found a strong association between noncontact injuries to the ACL in females who display a standing posture of genu recurvatum with subtalar joint overpronation. (Loudon, 1996). The resting position of knee hyperextension may produce a preloading effect on the ACL since it increases tension in the ligament (Beckett).
Also, knee hyperextension which may be related to an greater anterior pelvic tilt, could make the hamstrings less efficient by placing them in an elongated position. Lengthening of the hamstrings may slow their neuromuscular response time (Trontelj), and thus, their capacity to serve as dynamic agonists to the ACL. Conversely, anterior tilt is associated with shortening of the hip flexors, including the rectus femoris. (Lee) This may allow for faster neuromuscular facilitation of this muscle (Trontelj) and contribute to the phenomenon of quadriceps dominance hypothesized by Huston and Wojtys (1996).

**Decrease in ITB Flexibility**

The final predictor of ACL injury group membership in this study is a decrease in ITB flexibility. It has been hypothesized that reinforcing motor patterns that cause altered lower extremity position can result in overuse of the TFL, tightness in the ITB, and weakness of the gluteus medius (Host). It is possible that the neuromuscular sequelae of ACL injury include the onset of hypertonicity within the TFL and ITB.

**Unexplained Factors Related to ACL Injury**

Although the twelve variables entered into the discriminant analysis explained 35.2% association of ACL group membership, 64.8% of ACL group membership is unexplained. Since ACL injury is related to multiple factors, other areas must be further explored to better predict people with an ACL injury. In addition to assessing lower extremity malalignments as we did in this present study, other factors that have been previously examined are gender differences in neuromuscular control, strength, hormonal fluctuations throughout the menstrual cycle, jumping and landing techniques, and other structural differences. The factors or combination of factors that will correctly predict whether a person has or does not have an ACL injury is still unknown.
Ankle Sprain History and ACL Injury History

Our most interesting finding when comparing ACL injured males and females, was a significant association between ACL injury history and ankle sprain history. Those with a history of ACL sprain were more likely to also have a history of ankle sprain. Söderman et al (Söderman 2001) also found that subjects with a history of ACL injury were also more likely to have suffered a previous ankle sprain. Although only five subjects were examined, three of them had suffered an ankle sprain during the three months preceding the study period.

Since ankle sprains and knee sprains are two of the most common occurrences in athletics, there may be an underlying relationship between the ankle sprains and ACL injuries. Previous literature has demonstrated proximal changes in the hip after lateral ankle sprain and these neuromuscular changes may also affect the knee. It has been described by some authors that the “knee is a slave to the hip” and it is plausible to think that ankle injuries may not only affect the hip, but also affect the knee. As the gluteal muscles control rotation of the femur and the hamstrings control rotation of the tibia, altered control causing these bones to internally rotate may lead to adaptations in the lower extremity including pronation thus placing the ACL at risk for injury.

Bulluck-Saxton examined hip muscle function following severe ankle sprain in a group of 20 males with a history of unilateral sprain compared to a healthy group (Bullock-Saxton). EMG was recorded for the gluteus maximus, biceps femoris, and erector spinae muscles bilaterally. Results showed that the combined right and left gluteus maximus activity was delayed in the injured group compared to the uninjured group. The author’s conclusions were that this decreased gluteus maximus activity could be due to an altered gait pattern adapted during the time of initial ankle injury or due to a centralized reflex chain of events in both
proximal limbs after distal unilateral injury. If ankle injury can lead to changes in hip muscle recruitment, knee mechanics may also be altered.

A study by Richards et al. found that there was a relationship between ankle joint dynamics and patellar tendinopathy in elite volleyball players further supporting the idea that the ankle can affect the proximal knee joint (Richards). In this study, three-dimensional kinematics of the ankle, knee, and hip showed that a high inversion moment at the ankle at the time of landing from a spike jump was a significant predictor of patellar tendinopathy. This provides evidence of distal alterations increasing injury risk at the knee.

Distal changes at the ankle after LAS may also affect lower extremity kinematics. Previous literature has demonstrated that changes at the ankle/subtalar joint complex may affect pronation at the foot and tibial internal rotation which may place stress on the ACL (Loudon, Woodford-Rogers). Subtalar joint instability after LAS has also been reported (Hertel, Ishi, Meyer). Again, altered mechanics at the subtalar joint can affect the kinetic chain proximally. In addition, postural control changes after LAS and chronic ankle instability have been previously reported in the literature (Hertel). Altered postural control may be a risk factor for ACL injury although further research in this area is needed.

When we entered ankle sprain history into our ACL injury discriminant analysis, there was not as strong of an association to ACL group membership as we hypothesized. Since several of our subjects reported both a history of ACL injury and a previous ankle sprain, we hypothesized that some of the factors that predicted ACL group membership would also predict ankle injury group membership. Therefore, we calculated a second discriminant analysis to identify factors commonly related to both ACL injury and ankle injury.
The discriminant analysis used to identify factors most highly related to ankle injury revealed that of the 12.9% association between ankle injury group membership explained by the six variables initially entered into the analysis, 12.2% can be explained by generalized laxity (6.9%) and ITB flexibility (5.3%). Both increased generalized laxity and decreased ITB flexibility were present as predictors of ACL group membership as well. The kinetic chain relationship previously described helps explain why factors that cause knee injury could be related to ankle injury and vice versa.

**Clinical Implications**

When grouped with all other risk factors measured in this study, ankle sprain as a risk factor only contributed approximately 2% to ACL group membership and can be viewed as a weak predictor of ACL injury. However, when ankle sprain history was viewed individually, a strong association was found between ACL injury history and previous ankle sprain. This is a clinically significant finding. For the clinician, taking a good medical history and screening for generalized laxity, genu recurvatum, and a tight ITB in those with a history of LAS, may help identify those at risk for an ACL injury.

If there is a kinetic chain relationship between previous ankle injury and ACL injury, ACL prevention programs can be added to the ankle rehabilitative programs currently used. If an athlete suffers an ankle sprain, they are at increased risk for an ACL tear. Since the recovery time after an ankle sprain is usually 1-2 weeks, rehabilitation programs may only last as long as the time lost from activity. Additionally, most ankle rehabilitation focus primarily on restoring ankle strength and function and do not address the entire kinetic chain. ACL prevention programs focus on addressing kinetic chain factors that can be modified such as hamstring and gluteal
strength and power, decreasing landing forces, and teaching proper neuromuscular control during landing and cutting (Ireland, 1999, Hewett, 1996).

**Suggestions for Future Research**

Since this was a retrospective study the major limitation was that we were not able to identify lower extremity position prior to ACL injuries or surgery or previous ankle injuries. Some alignments may be adaptive rather than structurally inherent. Without a rehabilitation history, it is difficult to have a homogenous group of subjects. In addition, our subject number was small (N=52) and we defined our population very generally as “physically active”. Future research should focus on designing a large scale, prospective study examining ACL risk factors across different age groups and activity levels. In addition to measuring structural malalignments, analyzing menstrual cycle characteristics, lower extremity injury history and rehabilitation, and neuromuscular control factors is important.

**Conclusion**

This paper provides evidence that a kinetic chain relationship between previous ankle sprain and risk for an ACL injury does exist. Athletes with a history of ankle sprain should be screened for dynamic malalignments such as increased generalized laxity, genu recurvatum, and decreased ITB flexibility, which may also place them at risk for an ACL injury. Future research should focus on the efficacy of ACL prevention programs in people with a history of lower extremity injury such as ankle sprains.
Bibliography


Heitz MS, Eisenman PA, Beck CL. Hormonal changes throughout the menstrual cycle and increased anterior cruciate ligament laxity in females. *J Athl Train.* 1999; 34:144-149.


Moul JL. Differences in selected predictors of anterior cruciate ligament tears between male and female NCAA division 1 collegiate basketball players. 1998: J Athl Train. 33: 118-121.


Appendix A: Informed Consent Form

INFORMED CONSENT FORM FOR SOCIAL SCIENCE RESEARCH
The Pennsylvania State University

Title of Project: Correlation between ACL injury and lateral ankle sprain in collegiate athletes

Principal Investigator: Lauren C. Olmsted, MEd, ATC
Department of Kinesiology
269 Recreation Hall
University Park, PA 16802
lco100@psu.edu
814-863-1758

Other Investigator(s): Jay Hertel, PhD, ATC
Department of Kinesiology
269A Recreation Hall
University Park, PA 16802
jnh3@psu.edu
814-865-8816

1. Purpose of the Study: The purpose of this research is to determine if there is a relationship between anterior cruciate ligament injury (ACL) and lateral ankle sprain in athletes.

2. Procedures to be followed: If I take part in this study, I will report to 16 Recreation Building, one time for approximately 30 minutes. I will fill out a questionnaire related to my knee injury history and ankle injury history. Demographic information of age, height, and weight will be collected. I will then have measurements taken on my hip, knee, and ankle. These measurements include observation of joint position, flexibility testing, and range of motion testing.

3. Discomforts and Risks: There are no associated risks with this research.

4. Benefits:
   a. There is no direct benefit to me for participating in this study.
   b. My participation in this study will benefit society by helping clinicians understand the relationship between knee injuries and ankle injuries in athletes.

5. Duration/Time: My participation will take approximately 30 minutes on one day.

6. Statement of Confidentiality: My participation in this research is confidential. Only the investigators will have access to my identity and to the information that can be associated with my identity. In the event of publication of the research, no personally identifying information will be disclosed.
7. **Right to Ask Questions**: I may ask questions about the research procedures and the questions will be answered. I may also decline to answer any of the questions the researchers ask me. Further questions should be directed to Lauren Olmsted at lco100@psu.edu. I may also contact the Office of Research Protection, 212 Kern Graduate Building, University Park, PA 16802, (814) 865-1775 for additional information concerning my rights as a research participant.

8. **Compensation**: I realize that there is no compensation, financial or otherwise, for participating in this study.

9. **Voluntary Participation**: I understand that participation is voluntary. I can therefore withdraw from the study at any time by notifying the principal investigator. I have been given an opportunity to ask any questions I may have and all such questions or inquiries have been answered to my satisfaction.

I must be 18 years of age or older to consent to participate in this research study. This is to certify that I consent to and give permission for my participation as a volunteer in this program of investigation. I understand that I will receive a signed copy of this consent form. I have read this form, and understand the content of this consent form.

You will be given a copy of this consent form to keep for your records.

____________________________________  _____________________  
Participant Signature     Date

I, the undersigned, verify that the above informed consent procedure has been followed.

____________________________________  _____________________  
Investigator Signature     Date
Appendix B: Knee History Questionnaire

Project Title: Correlation between ACL injury and previous lateral ankle sprain in college students

Subject #: _____

Knee Injury History Questionnaire

Have you ever sprained your ACL?  Yes / No

If yes, which one?  Right / Left

Initial injury

What was the mechanism of injury?  Contact / Non Contact

How long ago did it occur?

Was your injury diagnosed by a physician?  Yes / No

What was the diagnosis?

What was your course of treatment?

How long were you out of activity?

Have you had any recurrent problems?  Yes / No

Feeling of instability during activity?  Yes / No

Feeling of instability on uneven surfaces?  Yes / No

Pain?  Yes / No

Swelling?  Yes / No

Weakness?  Yes / No

Decreased function?  Yes / No

Do you participate in activity with a brace?  Yes / No

Do you wear orthotics?  Yes / No
Appendix C: Ankle History Questionnaire

**Project Title:** Correlation between ACL injury and previous lateral ankle sprain in college students

**Subject #: _____**

### Ankle Injury History Questionnaire

- **Have you ever sprained your ankle?** 
  - Yes / No
- **If yes, which one?** 
  - Right / Left

#### Initial injury

- **How long ago did it occur?**
- **Was your injury diagnosed by a physician?** 
  - Yes / No
  - **What was the diagnosis?**
- **What was your course of treatment?**
- **How long were you out of activity?**
- **How many times have you sprained your ankle since your initial injury?**

#### Recurrent Signs and Symptoms

- **Do you currently have “giving way”, “rolling” or instability?** 
  - Yes / No
- **Do you have any of the following problems?**
  - **Feeling of instability during activity?** 
    - Yes / No
  - **Feeling of instability on uneven surfaces?** 
    - Yes / No
  - **Pain?** 
    - Yes / No
  - **Swelling?** 
    - Yes / No
  - **Weakness?** 
    - Yes / No
  - **Decreased function?** 
    - Yes / No
Appendix D: Data Collection Sheet

PENN STATE ATHLETIC TRAINING RESEARCH LAB
MALALIGNMENT AND FLEXIBILITY FORM

Subject #: __________________    Date:___________________
Sport:______________________    Examiner: ______________
Gender:  M  F

Generalized Laxity Scale:

<table>
<thead>
<tr>
<th>Test</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opposition of thumb to palmar forearm</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Hyperextension of 5th MCP beyond 90º</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Hyperextension of elbow beyond 10º</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Hyperextension of knee beyond 10º</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Palms flat on floor with trunk flexion and knees straight</td>
<td>Y</td>
<td>N</td>
</tr>
</tbody>
</table>

Total Score:__________

Foot Structure

<table>
<thead>
<tr>
<th>Test</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morton’s Toe</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Foot Type</td>
<td>Planus</td>
<td>Rectus</td>
</tr>
</tbody>
</table>

Malalignments

<table>
<thead>
<tr>
<th>Test</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Navicular Drop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tibial Varum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q-Angle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genu Recurvatum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Femoral Anteversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pelvic Tilt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leg Length</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Range of Motion

<table>
<thead>
<tr>
<th>Test</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle Dorsiflexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Flexion (90/90)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Extension (Thomas)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Adduction (Ober’s)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Functional Tests

<table>
<thead>
<tr>
<th>Test</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior Reach</td>
<td></td>
</tr>
<tr>
<td>Balance Error Scoring System in Single Leg Stance</td>
<td></td>
</tr>
<tr>
<td>(20 second trial with eyes closed on firm surface; Tally each error)</td>
<td></td>
</tr>
<tr>
<td>Eyes Opened</td>
<td></td>
</tr>
<tr>
<td>Hands Off Hips</td>
<td></td>
</tr>
<tr>
<td>Touchdown of non-stance leg</td>
<td></td>
</tr>
<tr>
<td>Hip Abduction &gt;30º</td>
<td></td>
</tr>
<tr>
<td>Out of Position &gt;5 seconds</td>
<td></td>
</tr>
<tr>
<td>Total Errors</td>
<td></td>
</tr>
</tbody>
</table>
Vita

Lauren Caryl Kramer

Education

2004  The Pennsylvania State University: Doctor of Philosophy in Kinesiology
2000  The University of Virginia: Master of Education in Athletic Training
1998  The Pennsylvania State University: Bachelor of Science in Kinesiology

Professional Experience

Clinical Coordinator, Athletic Training Education Program, The Pennsylvania State University
2002-2004

Instructor in Kinesiology, The Pennsylvania State University
2001-2004

Per Diem Certified Athletic Trainer, Department of Athletics, The Pennsylvania State University
2001-2003

Assistant Athletic Trainer, Marist College, Poughkeepsie, New York
2000-2001

Graduate Assistant Athletic Trainer, Washington and Lee University, Lexington, Virginia
1999-2000

Recent Publications


