ACL Research Retreat VI: An Update on ACL Injury Risk and Prevention
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Abstract:

It has been well recognized that multiple factors, whether individually or in combination, contribute to noncontact anterior cruciate ligament (ACL) injury. The ongoing mission of the ACL Research Retreat is to bring clinicians and researchers together to present and discuss the most recent advances in ACL injury epidemiology, risk factor identification, and injury-risk screening and prevention strategies and to identify future research directives. The sixth retreat held March 22–24, 2012, in Greensboro, North Carolina, was attended by more than 70 clinicians and researchers, including representatives from Canada, Iceland, Japan, The Netherlands, Norway, and South Africa. The meeting featured keynote presentations and discussion forums by expert scientists in ACL injury risk and prevention and 34 podium and poster presentations by attendees. Keynotes delivered by Ajit Chaudhari, PhD (The Ohio State University), Malcolm Collins, PhD (Medical Research Council and University of Cape Town, South Africa), and Tron Krosshaug, PhD (Oslo Sports Trauma Research Center, Norway) described their ongoing work related to proximal trunk control and lower extremity biomechanics, genetic risk factors associated with ACL injury, and methodologic approaches to understanding ACL loading mechanisms, respectively. Discussion forums led by Jennifer Hootman, PhD, ATC, FNATA, FACSM (Centers for Disease Control and Prevention) and Scott McLean, PhD (University of Michigan), focused on strategies for implementing injury-prevention programs in community settings and took a critical look at the strengths and limitations of motion-capture systems and how we might continue to refine our research approaches to increase the relevance and influence of our biomechanical research, respectively. Podium and poster presentations were organized into thematic sessions of anatomical, genetic, and hormone risk factors; the role of body position in ACL injury risk; pubertal and sex differences in lower extremity biomechanics; injury-risk screening and prevention; and methodologic considerations in risk factor research. Substantial time was provided for group discussion throughout the conference. From these discussions, the 2010 consensus statement[1] was updated to reflect recent advances in the field and to chart new directions for

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CONSENSUS STATEMENT

As in past retreats, participants were divided into 3 interest groups: anatomical, genetic, and hormonal risk factors; neuromechanical contributions to ACL injury; and risk factor screening and prevention. Within each group, relevant sections of the previous consensus document were discussed and updated as to important knowns and recent advances based on new evidence

emerging in the literature and presented at the retreat and important unknowns and future directions that are needed to advance our understanding. Working drafts from each group were then presented to all participants for further discussion and were recirculated after the meeting for further refinement.

From these discussions, the following global observations, themes, and recommendations emerged from the 2012 meeting. First, the biomechanical research community should consider the degree to which the movement patterns studied during the dynamic activities of risk factor screening tests should be expected to correspond with biomechanical loading profiles known to be associated with ACL strain. For example, cadaveric work overwhelmingly supports the notion that internal rotation of the tibia with respect to the femur loads the ACL; however, we do not know if those individuals at risk for future ACL injury commonly move with excessive amounts of knee internal rotation during screening tests or on the field. It is entirely possible that those individuals may adopt a movement strategy to avoid loading of the ACL or other structures during controlled screening tasks that is completely different from the movement strategies they use on the field in the course of athletic participation. That is, the movement strategies we prospectively identify as risk factors from screening tests may be different than the biomechanical mechanisms observed in vitro to increase strain on the ACL.

Another general biomechanical theme was the need to transfer relatively technical biomechanical modeling findings into a form that can be more easily accessed by the clinician or practitioner. Most importantly, risk factor screenings that incorporate biomechanical data should to some degree use clinician- and practitioner-friendly language in the interpretation and explanation of the findings. From an injury-prevention perspective, much remains unknown about which specific elements of successful injury-prevention programs (movement training, strengthening, plyometrics, etc) are necessary to produce the desired protective effects, why these programs are limited to short-term success, and to what extent program components need to be age, sex, and sport specific. If we are to streamline ACL injury-prevention programs to improve compliance and efficacy, thus making them more palatable to the public, high-quality randomized control trials are needed to address these critical questions. At the same time, we have yet to effectively translate our highly controlled ACL injury-prevention research to real-world community settings in which the public health benefit can be maximized.[1] To that end, it will be important to identify the barriers and facilitators (eg, feasibility, cost, and parent and coach buy-in) to maximize acceptance, compliance, and retention of these interventions within the context of these community settings.

Finally, as our understanding of this multifactorial problem continues to grow, the need for multidisciplinary, multicenter work is becoming more apparent. As a research community, we need to leverage our combined resources to bring together interdisciplinary teams and to achieve the population sizes needed for integrated examination of these different factors. Developing such integrated approaches is not without challenges, and concerted efforts are needed to identify and reduce the barriers that impede this important work.

Once again, we find that in the 2 years since the last ACL Research Retreat, many advances in our knowledge have reshaped the important unknowns and directions for future research. We hope that these proceedings will continue to foster quality research and clinical interventions.

Anatomical and Structural Risk Factors

The primary anatomical and structural factors examined relative to ACL injury include ACL morphology, tibial and femoral surface geometry, knee-joint laxity, and lower extremity structural alignments. Most of what is known is based on sex comparisons (driven by females' greater susceptibility to ACL trauma) and retrospective ACL-injured case-control studies.

Important Knowns and Recent Advances

1. *ACL Structure and Geometry*: Compared to noninjured controls, ACL-injured patients have smaller ACLs (area and volume).[2] When compared with males, females have smaller ACLs relative to length, cross-sectional area, and volume even after adjusting for body anthropometry.[3] After adjusting for age and body anthropometrics, the female ACL has less collagen fiber density (area of collagen fibers/total area of the micrograph)[4] and decreased mechanical properties, such as strain at failure, stress at failure, and modulus of elasticity.[5]

2. *Knee-Joint Geometry—Tibial Plateau*: Magnetic resonance imaging (MRI) studies (imaging both the lateral and medial tibial plateaus) demonstrate greater lateral posterior-inferior tibial plateau slopes (but not necessarily medial tibial slopes)[6–9] and reduced condylar depth of the medial tibial plateau[7] in ACL-injured patients versus controls. Compared with males, females have greater lateral and medial posterior-inferior tibial slopes[10,11] and reduced coronal tibial slopes.[10] Biomechanically, greater posterior-inferior lateral tibial slopes are associated with greater anterior joint reaction forces,[12] greater anterior translation of the tibia relative to the femur,[13,14] greater peak anterior tibial acceleration,[15] and when combined with a smaller ACL cross-sectional area, greater peak ACL strains.[16,182] Greater relative posterior-inferior slope of the lateral versus medial tibial plateau has been associated with greater peak knee-abduction and internal-rotation angles.[12]

3. *Knee-Joint Geometry—Femoral Notch*: Femoral notch dimensions have frequently been investigated as ACL injury-risk factors. Authors of the majority of prospective[17–20] and retrospective studies[8,21–23] have generally reported a smaller femoral notch width or notch width index in ACL-injured cases. The presence of an anterior medial ridge has also been noted on the intercondylar notch in ACL-injured patients versus controls.[8] When compared with males, the female's femoral notch height is taller, whereas their femoral notch angle is smaller, which may influence the femoral notch impingement theory.[3] Femoral notch width and angle are good predictors of ACL size (area and volume) in males but not in females.[3]

4. *Knee-Joint Laxity*: Greater magnitudes of anterior knee laxity,[20,24,25] genu recurvatum,[24,26–29] general joint laxity,[20,24,26,29,30] and internal-rotation knee laxity[31] have been reported in the contralateral knee of ACL-injured patients compared with control cases. Compared with males, females have greater sagittal-plane knee laxity (anterior knee laxity, genu recurvatum),[20,24,32–36] greater frontal (varus-valgus rotation)- and transverse (internal-external rotation)-plane knee laxity,[37–40] and greater general joint

laxity.[20,24] Sex differences in frontal- and transverse-plane knee laxity persist even when males and females have similar sagittal-plane knee laxity.[37,39,40] Greater magnitudes of knee laxity have been associated with the higher-risk landing strategies more often observed in females.[32,41–44]

5. *Lower Extremity Alignment*: Lower extremity alignments are different between maturation groups and also develop at different rates in males and females between maturation groups.[45] Fully mature females have greater anterior pelvic tilt, hip anteversion, tibiofemoral angle, and quadriceps angles.[35,46] No sex differences have been observed in tibial torsion,[35] navicular drop,[35,36,46] and rear-foot angle.[35,47] Currently, no compelling evidence links any one lower extremity alignment factor with ACL injury.

Unknowns and Directions for Future Research

1. Anatomical and structural factors have often been examined independently or in small subsets of variables. In order to determine the most important anatomical and structural risk factors for ACL injury, we need to conduct large-scale, prospective risk factor studies that account for all relevant lower extremity anatomical and structural factors to determine how they may combine or interact to pose the greatest risk to the ACL. Because most anatomical and structural factors are not acutely affected by the ACL rupture, large, multifactorial, case-control study designs are also ideal for examining structural factors.

2. To facilitate large-scale, multivariate risk factor studies, we need to develop more efficient, affordable, reliable, and readily available methods of measuring anatomical and structural factors.

3. The lack of uniform measurement techniques for determining intercondylar notch dimensions make it difficult to clearly identify which specific dimensions are most predictive of increased risk for ACL injury.[48] Specifically, we need to determine whether the size and geometry of the notch itself, the volume of the ACL, or some combination of these factors best characterizes risk for impingement and injury.

4. Recent researchers have begun to elucidate the influence of anatomical and structural factors on weight-bearing knee-joint neuromechanics;[12,15,16,43,44,49] which may be important in our injury-prevention efforts. Studies examining the combined effects of joint laxity, tibial geometry (lateral tibial slope, medial:lateral tibial slope ratio, coronal slope, medial condylar depth) and ACL cross-sectional area and volume, as well as interactions among these variables, on tibiofemoral joint biomechanics and ACL strain and failure are encouraged.

5. Investigations of knee-joint geometry are largely based on measures of subchondral bone. Recent research[50] suggests it may be important to also account for the overlying cartilage geometry.

6. Some evidence suggests that an elevated body mass index (BMI) is predictive of future ACL injury in females[20] and that artificially increasing BMI encourages dangerous biomechanical strategies.[51,52] Additionally, recent research suggests that body composition may influence knee-joint laxity,[53,183] potentially explaining why the combination of greater knee laxity and BMI substantially heightens the risk for ACL injury.[20] Continued research on the influence of body composition is warranted.

7. Although anatomical and structural factors are often considered nonmodifiable once a person is fully mature, we have limited knowledge of how these structural factors change during maturation or whether physical activity (or other chronic external loads) can influence this development over time, particularly during the critical growth periods. Prospective, longitudinal studies are needed to understand the underlying factors that cause one to develop at-risk anatomical and structural profiles during maturation while also taking into account relevant modifiable factors, such as body composition, neuromuscular properties, and physical activity.

Genetic Risk Factors

An ACL rupture is a multifactorial condition caused by a poorly understood interaction of both genetic and environmental (nongenetic) factors. The injury is most likely caused, at least in part, by environmental exposures and other stimuli interacting with a genetic (multiple-genes) background.[54] Mutations within the *COL1A1* and *COL5A1* genes cause rare Mendelian connective tissue disorders, suggesting that there is limited or no redundancy within the biology of the collagen fibril. Common polymorphisms within genes, such as the collagen genes that encode for structural components or regulators of the collagen fibril, which is the basic building block of the ACL, are ideal candidates for examining genetic predisposition to ACL ruptures.[54] Since the last ACL Research Retreat, considerable research has examined genetic associations with ACL injury.

Important Knowns and Recent Advances

1. A familial predisposition to ACL ruptures has been reported.[55]

2. A functional polymorphism within the first intron of the *COL1A1* gene is associated with risk for ACL ruptures in 2 independent white populations.[56–58] The *COL1A1* gene encodes for the α1(I) of type I collagen, which is the major building block of the collagen fibril of the ACL.
3. Although the sample sizes are small, polymorphisms within the *COL5A1* and *COL12A1* genes have been shown to associate with risk for ACL ruptures in white females. The *COL5A1* and *COL12A1* genes encode for the α1(V) chain of type V collagen and the α1(XII) chain of type XII collagen, respectively.[59,60] Both type V and XII collagens are important structural components of the collagen fibril.

4. The *COL5A1* polymorphism associated with ACL ruptures in females is located within a functional region of the 3′-untranslated region of the *COL5A1* gene. It has been proposed that the 3′-untranslated region regulates, at least in part, the amount of type V collagen

incorporated within the collagen fibril, which in turn alters the mechanical properties of the fibril.[61,62]

5. Inferred haplotypes constructed from functional variants within 4 matrix metalloproteinase (MMP) genes (*MMP10*, *MMP1*,*MMP3*, and *MMP12*), clustered together on human chromosome 11q22, have been shown to associate with the risk of ACL rupture. After adjusting for weight, age, and sex, the *MMP12* variant was independently associated with an increasing risk of noncontact ACL rupture.[63]

6. The traditional intrinsic risk factors associated with ACL ruptures are also to a lesser or greater extent determined by both genetic and nongenetic factors. For example, some early evidence indicates that the same genetic variants in *COL5A1*associated with ACL injury are also associated with joint laxity[64,184] and joint range of motion.[65,66]

Unknowns and Directions for Future Research

1. Most of the case-control genetic association studies published to date have used relatively small sample sizes, especially with respect to the sex-specific effects of *COL5A1* and *COL12A1*. These studies need to be replicated in other, larger populations, which may require the establishment of international consortia.

2. All the genetic studies to date have been done on European white populations, and the reported associations cannot be extrapolated to other populations. These studies therefore need to be repeated in other population groups.

3. Mutations within many of the collagen and noncollagen encoding genes cause rare Mendelian connective tissue disorders. Common variants within these genes, which cause less severe changes in the amount of proteins produced or the structures of the protein may be ideal candidates for determining the biological variation within the structure of the ACL and susceptibility to injury and should therefore be studied. Unlike other multifactorial disorders caused by the interaction of both environmental and genetic factors (eg, type 2 diabetes), the individual genetic effects that influence the predisposition to ACL rupture appear to be quite large.

4. Because most of the intrinsic risk factors are complex phenotypes, we need to better understand how genetic variants that partly determine these intrinsic risk factors alter susceptibility to ACL injury.

5. Molecular genetics should be viewed as one of many techniques that can elucidate the biological mechanisms of ACL ruptures. Genetic association studies may highlight biological processes and pathways for ACL injury, which require additional investigation using other methods. Multidisciplinary approaches should therefore be encouraged (eg, connecting genetics to cell biology to tissue function to whole-body function).

6. The effects of various stimuli, such as hormonal, mechanical loading, and other environmental stimuli, on the expression of genes associated with risk for ACL rupture

need to be investigated. These studies will assist us in understanding how the associated genetic variants interact with stimuli to influence ACL homeostasis and remodeling.

7. The interaction of hormones with genetic regulatory elements should be studied to explain female-specific anatomical differences (eg, small ACL) and increased risk for ACL ruptures.

Hormonal Risk Factors

Substantial differences in sex-steroid hormone concentrations likely underlie many of the sex-specific characteristics that emerge during puberty. In particular, the large magnitudes and monthly variations in estrogen and progesterone concentrations that females experience continue to be an active area of ACL injury risk factor research.

Important Knowns and Recent Advances

1. The risk of suffering an ACL injury appears to be greater during the preovulatory phase of the menstrual cycle than during the postovulatory phase.[67–71] However, there is no evidence that stabilizing hormone concentrations through the use of oral contraceptives protects against ACL injury.[72,73]

2. The risk of ACL injury may be higher in elite female athletes who have elevated serum relaxin concentrations.[74]

3. Sex hormone receptors on the human ACL (eg, estrogen, testosterone, and relaxin)[75–79] and skeletal muscle (estrogen, testosterone)[80–82] suggest that sex hormones have the potential to directly influence these structures.

4. Normal physiologic variations in sex hormone concentrations across the menstrual cycle have been associated with substantial changes in markers of collagen metabolism and production,[83] knee joint laxity,[40,84–88] and muscle stiffness.[85]However, large individual variations in hormone profiles across the menstrual cycle[88] are associated with substantial interparticipant variations in the magnitude of these phenotypic changes.[40,83,87,89]

5. Cyclic variations in knee laxity are of sufficient magnitude in some women to substantially alter their knee-joint biomechanics, particularly in the planes of motion in which the greatest magnitudes of knee-laxity change are observed.[49,90,91]

6. The mechanical and molecular properties of the ACL are likely influenced not only by estrogen but by the interaction of several sex hormones, secondary messengers, remodeling proteins, and mechanical stresses.[76,79,83,88,92–94] For example, interactions among mechanical stress, hormones, and altered ACL structure and metabolism have been observed in some animal models.[95–97]

7. A time-dependent effect for sex hormones and other remodeling agents influences a change in ACL tissue characteristics.[79,88]

Unknowns and Directions for Future Research

1. Although epidemiologic studies have consistently pointed to the preovulatory phase as the time when ACL injury is more likely to occur,[67–71] we know little of the underlying mechanism for this increased likelihood. Future researchers should examine the underlying sex-specific molecular and genetic mechanisms of sex hormones on ACL structure, metabolism, and mechanical properties and how mechanical stress on the ACL alters these relationships.

2. Although good evidence indicates that females who experience substantial cyclic changes in their laxity across the menstrual cycle also experience substantial changes in their knee-joint biomechanics,[49,90,91] it is not yet possible to clinically screen for these potentially high-risk individuals. We must understand the underlying processes that result in changes in ligament behavior (and other relevant soft tissue changes) so that we can better screen for these individuals and prospectively examine how these factors influence injury-risk potential. The effects of hormones and other stimuli on the synthesis of the less stable collagens and noncollagen proteins (eg, proteoglycans and other ground substance components) that regulate ligament biology should be investigated.

3. Oral contraceptives do not appear to be protective against ACL injury risk,[72,73] but they can vary substantially in the potency and androgenicity of the progestin compound delivered, which ultimately determines the extent to which they counteract the estrogenic effects.[98] Because many physically active females use oral contraceptives, we need to better understand how the different progestins influence soft tissue structures, knee function, and ACL injury risk. Relevant comparisons should then be made between oral-contraceptive users and eumenorrheic, amenorrheic, and oligomenorrheic females to determine if ACL injury risk or observed soft tissue changes vary between these groups.

4. Given the time-dependent effect of sex hormones on soft tissue structures, we ought to determine how the time of injury occurrence lines up with acute changes in ACL structure and metabolism or knee laxity changes and how the rate of increase or the time duration of amplitude peaks in hormone fluctuation across the menstrual cycle plays a role in the magnitude or timing of soft tissue changes. The actual hormonal targets in the ACL also need to be identified in order to understand the relatively quick and time-dependent hormonal effects on the ACL.

5. When examining hormone influences in physically active females, it is critical that we better match the complexity of interparticipant differences in timing, magnitude, and interactive changes in sex hormone concentrations across the cycle to our study designs. Future researchers should (1) verify phases of the cycle (or desired hormone environment) with actual hormone measurements (considering all relevant hormones, including estrogen, progesterone, and possibly others) rather than relying on calendar day of the cycle and (2) obtain multiple hormone samples over repeated days to better characterize hormone profiles within a given female.[99]

6. Because cyclic hormone concentrations affect soft tissues and knee-joint function, future studies comparing females with males should be conducted during the early follicular phase, when hormone levels are at their nadirs in females (preferably 3–7 days postmenses).

Neuromuscular and Biomechanical Factors Associated with the ACL Injury Mechanism

Neuromuscular and biomechanical (neuromechanical) factors, whether ascertained in vivo or in vitro, are generally derived from instrumented analyses of function that typically include kinematics, kinetics, and the timing and magnitude of the muscular activation and force production. Many of these measures are considered to be modifiable through training and have received considerable attention.

Important Knowns and Recent Advances

1. The ACL is loaded in vitro by a variety of isolated and combined compressive, sagittal and nonsagittal mechanisms during dynamic sport postures considered to be high risk.[100–106] This work collectively demonstrates high ACL strain under compression, tibial valgus, tibial internal rotation, and combined tibial valgus and internal rotation.[104,107–110]

2. Quantitative analyses of actual injury events demonstrate rapid tibial valgus and internal rotation.[111,112]

3. In vivo strain of the ACL is related to maximal load and timing of ground reaction forces.[113] A more erect (eg, upright) posture is commonly associated with increased vertical ground reaction forces.[114,115,185,186] Similarly, anterior tibial translation increases as demands on the quadriceps increase.[116] Thus, this upright posture when contacting the ground during the early stages of deceleration tasks has been suggested to be associated with the ACL injury mechanism.[117–120]

4. Given the inherent difficulties of measuring ACL strain in vivo, recent advances in our understanding of ACL loading have arisen from cadaveric and computer models of simulated landings. Such work has demonstrated that internal rotation results in greater ACL strain than external-rotation torque,[107] that mechanical coupling of internal tibial torque and knee valgus results in increased ACL loading,[108] and that combined tibial internal and valgus moments result in ACL strains near reported levels for tissue rupture.[110]

5. Maturation influences biomechanical and neuromuscular factors.[121–131,187,188]

6. Fatigue alters lower limb biomechanical and neuromuscular factors that are suggested to increase ACL injury risk.[132–135,189]The effect of fatigue on movement mechanics is most pronounced when combined with unanticipated landings, causing potentially adverse changes to central processing and control compromise.[136]

7. Hip, trunk, core, and upper body mechanics are associated with lower extremity biomechanical and neuromuscular factors.[51,118,137–141,190] Further, a recent modeling and

optimization study demonstrated that upper body kinematics influence valgus knee loading during sidestepping and that multiple kinematic changes occur simultaneously to reduce knee loading.[142]

Unknowns and Directions for Future Research

1. We still do not know the loads and neuromuscular profiles that cause noncontact ACL rupture, an understanding that is central to improving future injury-prevention strategies. Because we do not have precise descriptions of the mechanisms of in vivo ACL rupture, video from actual injury situations must be accumulated (along with control videos of these injured athletes before they were injured for comparison) to allow us to better understand the injury mechanism. Additionally, cadaveric, mathematical, in vivo kinematic, and imaging research approaches should be combined to best understand the loads and neuromuscular profiles that cause noncontact ACL rupture.[191]

2. Although translating laboratory biomechanical measures obtained during movement testing to the field is important, the optimal ways to assess movement in the laboratory environment are still being debated. We need to develop tasks designed to stress the joint systems that attempt to mimic injury mechanisms and are realistic to the mechanistic purpose of the study, as well as better techniques to measure the 3-dimensional movements and loading associated with these tasks. To better understand how movement patterns and other structures in the kinetic chain affect ACL loads, we must continue to develop, improve, and validate quality laboratory-based models (eg, computational, cadaveric) that noninvasively estimate in vivo ACL forces and strain. Care should be taken to not overgeneralize results from 1 specific task to other tasks with different mechanical demands.[192]

3. Although we understand how the lumbo-pelvic-hip (LPH) complex affects knee biomechanics in general, we do not know from the limited research models estimating in vivo ACL strain how these trunk and hip biomechanical factors affect in vivo ACL strain during highly dynamic activities known to cause ACL injury. The influence of the LPH complex on ACL loads must be better characterized. Additionally, we do not know if LPH mechanics are a cause of or a compensation for potentially dangerous knee biomechanics.

4. We do not yet understand the role of neuromechanical variability on the risk of indirect or noncontact ACL injury. Are there optimal levels of variability, and do deviations from these optimal levels increase the risk of injury? We may need to rethink our experimental design to take advantage of nontraditional analyses for assessing variability.

5. Even though decreased reaction times, processing speed, and visual-spatial disorientation have been observed in athletes sustaining an ACL injury,[143] whether noncontact ACL injury is an unpreventable accident stemming from some form of cognitive dissociation that drives central factors and the resulting neuromuscular and biomechanical patterns is unknown. We should continue to expand research models and analyses to include assessments of central processes (eg, automaticity, reaction time), cognitive processes

(eg, decision making, focus and attention, prior experience [eg, expert versus novice]), and metacognitive processes (eg, monitoring psychomotor processes).

6. We do not know if gross failure of the ACLis caused by a single episode or multiple episodes.

7. Although it is generally accepted that the ACL injury mechanism is multifactorial, resulting from the interplay of many different neuromuscular, biomechanical, anatomical, genetic, hormonal, and other factors, studies that consider only individual factors in isolation (eg, kinematic or kinetic or neuromuscular or anatomic) remain the norm in the literature. To best understand movement patterns linked to noncontact ACL injury, researchers should move toward a comprehensive collection of kinetic, kinematic, and neuromuscular data and as much data related to anatomy, genetics, hormones, and other factors as possible. These multifactorial studies will allow us to determine important interactions and interdependencies among factors.

8. In identifying potential factors that contribute to the injury mechanism, we should consider whether observed kinematics, kinetics, and muscle-activation strategies are root causes of increased ACL loading or compensations for deficiencies in other components of the kinetic chain. Studies specifically designed to evaluate cause and effect (ie, highly controlled human movement studies with 1 variable manipulated or simulation studies) could help advance this area.

9. Further insight into the dynamic-restraint systems are needed to more fully understand ACL loading mechanisms. Further work on muscle properties beyond absolute strength (eg, stiffness, muscle mass, rate of force production) is warranted.

10. We do not yet know whether females are at greater risk of noncontact ACL injury due to female-specific injury mechanisms or if the same injury mechanisms apply but the risk factors are merely more prevalent in females. We must continue to move away from purely descriptive sex-comparison studies and focus more on the underlying mechanisms associated with the observed sex differences and, more directly, ACL injury risk and prevention as appropriate.

11. Examining the influence of the maturational process on knee biomechanics and specifically ACL loads may allow unique insights into the observed difference in injury rates by sex occurring during the early stages of physical maturation and into mechanisms of injury across the continuum of physical attributes and capabilities.

12. The inability of most individual researchers to perform large-scale studies due to funding, personnel, and geographic restrictions has hindered our progress in understanding the ACL injury mechanism. Sharing datasets could potentially allow for investigations with the needed population sizes. Several actions that would facilitate such data sharing include but are not limited to the following:
    a. Common operational definitions of terms, such as *core stability*, *dominant limb*, *exposure*, *activity level*, *experience*, etc, need to be established.

b. Voluntary data-collection standards, including activities, methods, and demographics, are required to enable pooling of data.
   c. Creation of a central repository for neuromechanical datasets and a clearinghouse mechanism for using such datasets could greatly facilitate multicenter and transdisciplinary collaboration.

Risk Factor Screening and Prevention

Although intervention programs have been shown to reduce the incidence of ACL injuries,[69,144–149] overall ACL injury rates and the associated sex disparity have not yet diminished. There is still much we need to learn to maximize the effectiveness of these programs and to identify highly sensitive screening tools to target those at greatest risk for injury.

Important Knowns and Recent Advances

1. Clinically oriented screening tools (eg, Landing Error Scoring System (LESS) and tuck jump) show good agreement with laboratory-based biomechanics (concurrent validity).[150–152]

2. Clinically oriented screening tools are sensitive in detecting changes in movement quality over time.[153,154]

3. The ability of clinically oriented screening tools to identify individuals at risk for future ACL injury may be population specific (eg, sex, age, sport).[152,155,193]

4. Prospective biomechanical risk factors for ACL injury may include variables that are not directly associated with ACL loading or injury events.[156,194]

5. Neuromuscular control and strength of the hip musculature play an important role in knee biomechanics.[157–163,195]

6. Individuals with a personal history of ACL injury are at high risk for future ACL injury of the ipsilateral or contralateral leg.[164–166]

7. Multicomponent dynamic warm-up–style preventive training programs are safe and effective for reducing ACL injury rates.[144,147,167]

8. Preventive training programs with successful outcomes (eg, injury-rate reduction, improved neuromuscular control or performance) are conducted 2–3 times per week and last for 10–15 minutes at a minimum.[69,144,146–148,168–174]
9. Improvements in movement quality after 12 weeks of training do not appear to be retained once preventive training programs end. Thus, longer-duration or higher-intensity training programs may be required to better facilitate retention and transfer.[154]

10. Ensuring proper exercise technique and quality is an important factor for program effectiveness. Feedback should emphasize successful performance and ignore less successful attempts; this benefits learning because of its positive motivational effects.[175]

11. Real-time feedback can change landing biomechanics.[176–178,196]

12. The transition from conscious awareness during technique training sessions to unexpected and automatic movements during training or game involves complicated motor-control elements that might not fit in explicit learning strategies.[179]

13. Age-appropriate preventive training programs can be effective at modifying biomechanics in children.[153,180]

Unknowns and Directions for Future Research

1. We do not know which elements (eg, specific faulty movements, combination of faulty movements) of clinically oriented screening tools predict future ACL injury risk (predictive validity).

2. We do not know the reliability, validity, sensitivity, and specificity of current screening tools (LESS, tuck jump) and thresholds or cutoff points in order to determine whether a person is at high or low risk.[193,197]

3. We need to develop other clinically oriented screening tools that have good sensitivity and specificity for predicting future ACL injury risk.

4. We must understand how clinically oriented screening tools (eg, the LESS and tuck jump) predict other lower extremity injuries in addition to ACL injuries.

5. Various ACL injury-prevention programs that incorporate elements of balance training, plyometric training, education, strengthening, and technique training or feedback have been shown to reduce ACL injury[69,144–149] or alter biomechanical and neuromuscular variables thought to contribute to ACL injury.[168,170–174,181] However, we do not know which program elements are responsible for the reduced injury risk or biomechanical changes. Future research is necessary to determine which components are effective and necessary.

6. Technique training or feedback is frequently provided during preventive training programs to improve movement patterns. However, more study is needed to determine the most effective training variables (eg, frequency, timing, focus of attention) for improving movement patterns and optimizing the transfer of these learned movement patterns to sport-specific movements performed on the field.

7. We ought to continue to evaluate how a participant's sex, age, skill level, and type of sport should be considered in the type and variety of exercises prescribed and technique training or feedback provided.[153,180,198]

8. We need to identify the most most appropriate age to begin implementing preventive training programs.

9. We must determine the performance enhancement benefits associated with regularly performing preventive training programs.

10. We need to assess the effects of preventive training on reducing ACL injury rates in those with a history of ACL injury.

11. We should understand how preventive training programs influence lower extremity injuries in addition to ACL injuries.

12. We need to determine the cost effectiveness of current preventive training programs.

13. Because compliance has a strong influence on the success of ACL injury-prevention programs, research is essential to identify the barriers and motivational aspects that influence compliance (eg, type of feedback provided; coach or athlete knowledge, attitudes, and beliefs regarding prevention programs; design of prevention program; individual leading the prevention program). We need to learn if streamlining prevention programs, thus making them more palatable to the public, will improve compliance.

14. Although well-controlled ACL injury-intervention programs reduce the incidence of ACL injuries,[69,144,145,147,148] we have yet to effectively implement multifaceted programs in different settings that are sustainable over time (widespread implementation with high compliance rates and retention over the long term). Developing packaged preventive training programs that can be implemented broadly across different settings through appropriately educated and trained coaches or team leaders may improve compliance and efficacy. To that end, the following should be considered when developing large-scale injury-prevention programs in the future: (a) provide low-cost, brief time, packaged interventions; (b) adapt the program based on contextual factors for that setting (eg, sport, age, sex, environment); (c) incorporate lay people (eg, coaches instead of athletic trainers or strength and conditioning specialists) to implement the program for that setting and population; (d) educate and obtain organizational buy-in from all levels (eg, school, club, administrators, coaches, players, parents); (e) attempt to embed programs within an existing system when possible (part of the warm-up or conditioning program, team challenge, etc); and (f) develop written policies and procedures (specifics of program, when to perform, how often to perform, etc).

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REFERENCES

1. Shultz SJ, Schmitz RJ, Nguyen A, et al. ACL Research Retreat V: An Update on ACL Injury Risk and Prevention, March 25–27, 2010, Greensboro, NC. *J Athl Train*. 2010;45(5):499–508.

2. Chaudhari AM, Zelman EA, Flanigan DC, Kaeding CC, Nagaraja HN. Anterior cruciate ligament-injured subjects have smaller anterior cruciate ligaments than matched controls: a magnetic resonance imaging study. *Am J Sports Med*.2009;37(7):1282–1287.

3. Chandrashekar N, Slauterbeck J, Hashemi J. Sex-based differences in the anthropometric characteristics of the anterior cruciate ligament and its relation to intercondylar notch geometry. *Am J Sports Med*. 2005;33(10):1492–1498.

4. Hashemi J, Chandrashekar N, Mansouri H, Slauterbeck J, Hardy DM. The human anterior cruciate ligament: sex differences in ultrastructure and correlation with biomechanical properties. *J Orthop Res*. 2008;26(7):945–950.

5. Chandrashekar N, Mansour JM, Slauterbeck J, Hashemi J. Sex-based differences in the tensile properties of the human anterior cruciate ligament. *J Biomech*. 2006;39(16):2943–2950.

6. Khan MS, Seon JK, Song EK. Risk factors for anterior cruciate ligament injury: assessment of tibial plateau anatomic variables on conventional MRI using a new combined method. *Int Orthop*. 2011;35(8):1251–1256.

7. Hashemi J, Chandrashekar N, Mansouri H, et al. Shallow medial tibial plateau and steep medial and lateral tibial slopes: new risk factors for anterior cruciate ligament injuries. *Am J Sports Med*. 2010;38(1):54–62.

8. Everhart JS, Flanigan DC, Simon RA, Chaudhari AM. Association of non-contact ACL injury with presence and thickness of a bony ridge on the anteromedial aspect of the femoral intercondylar notch. *Am J Sports Med*. 2010;38(8):1667–1676.

9. Stijak L, Herzog RF, Schai P. Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. *Knee Surg Sports Traumatol Arthrosc*. 2008;16(2):112–117.

10. Hashemi J, Chandrashekar N, Gill B, et al. The geometry of the tibial plateau and its influence on the biomechanics of the tibiofemoral joint. *J Bone Joint Surg Am*. 2008;90(12):2724–2734.

11. Hudek R, Fuchs B, Regenfelder F, Koch PP. Is noncontact ACL injury associated with the posterior tibial and meniscal slope?*Clin Orthop Relat Res*. 2011;469(8):2377–2384.

12. McLean SG, Lucey SM, Rohrer S, Brandon C. Knee joint anatomy predicts high-risk in vivo dynamic landing knee biomechanics. *Clin Biomech (Bristol, Avon)*. 2010;25(8):781–788.

13. Dejour H, Bonnin M. Tibial translation after anterior cruciate ligament rupture. *J Bone Joint Surg Br*. 1994;76(5):745–749.

14. Giffin JR, Vogrin TM, Zantop T, Woo SL, Harner CD. Effects of increasing tibial slope on the biomechanics of the knee. *Am J Sports Med*. 2004;32(2):376–382.

15. McLean SG, Oh YK, Palmer ML, et al. The relationship between anterior tibial acceleration, tibial slope, and ACL strain during a simulated jump landing task. *J Bone Joint Surg Am*. 2011;93(14):1310–1317.

16. Lipps DB, Oh YK, Ashton-Miller JA, Wojtys EM. Morphologic characteristics help explain the gender difference in peak anterior cruciate ligament strain during a simulated pivot landing. *Am J Sports Med*. 2012;40(1):32–40.

17. LaPrade RF, Burnett QM. Femoral intercondylar notch stenosis and correlation to anterior cruciate ligament injuries. *Am J Sports Med*. 1994;22(2):198–302.

18. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears: a prospective study. *Am J Sports Med*. 1998;26(3):402–408.

19. Souryal TO, Freeman TR. Intercondylar notch size and anterior cruciate ligament injuries in athletes. *Am J Sports Med*.1993;21(4):535–539.

20. Uhorchak JM, Scoville CR, Williams GN, Arciero RA, St Pierre P, Taylor DC. Risk factors associated with non-contact injury of the anterior cruciate ligament. *Am J Sports Med*. 2003;31(6):831–842.

21. Ireland ML, Ballantyne BT, Little K, McClay IS. A radiographic analysis of the relationship between the size and shape of the intercondylar notch and anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthros*. 2001;9(4):200–205.

22. Souryal TO, Moore HA, Evans JP. Bilaterality in anterior cruciate ligament injuries. *Am J Sports Med*. 1988;16(5):449–454.

23. Domzalski M, Grzelak P, Gabos P. Risk factors for anterior cruciate ligament injury in skeletally immature patients: analysis of intercondylar notch width using magnetic resonance imaging. *Int Orthop*. 2010;34(5):703–707.

24. Scerpella TA, Stayer TJ, Makhuli BZ. Ligamentous laxity and non-contact anterior cruciate ligament tears: a gender based comparison. *Orthopedics*. 2005;28(7):656–660.

25. Woodford-Rogers B, Cyphert L, Denegar CR. Risk factors for anterior cruciate ligament injury in high school and college athletes. *J Athl Train*. 1994;29(4):343–346.

26. Kramer LC, Denegar CR, Buckley WE, Hertel J. Factors associated with anterior cruciate ligament injury: history in female athletes. *J Sports Med Phys Fit*. 2007;47(4):446–454.

27. Loudon JK, Jenkins W, Loudon KL. The relationship between static posture and ACL injury in female athletes. *J Orthop Sports Phys Ther*. 1996;24(2):91–97.

28. Myer GD, Ford KR, Paterno MV, Nick TG, Hewett TE. The effects of generalized joint laxity on risk of anterior cruciate ligament injury in young female athletes. *Am J Sports Med*. 2008;36(6):1073–1080.

29. Ramesh R, VonArx O, Azzopardi T, Schranz PJ. The risk of anterior cruciate ligament rupture with generalised joint laxity. *J Bone Joint Surg Br*. 2005;87(6):800–803.

30. Hewett TE, Lynch TR, Myer GD, K.R. F, Gwin RC, Heidt RS, Jr. Multiple risk factors related to familial predisposition to anterior cruciate ligament injury: fraternal twin sisters with anterior cruciate ligament ruptures. *Br J Sports Med*. 2010;44(12)7:848–855.

31. Branch TP, Browne JE, Campbell JD, et al. Rotational laxity greater in patients with contralateral anterior cruciate ligament injury than healthy volunteers. *Knee Surg Sports Traumatol Arthrosc*. 2010;18(10):1379–1384.

32. Rozzi SL, Lephart SM, Gear WS, Fu FH. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *Am J Sports Med*. 1999;27(3):312–319.

33. Shultz SJ, Kirk SE, Sander TC, Perrin DH. Sex differences in knee laxity change across the female menstrual cycle. *J Sports Med Phys Fit*. 2005;45(4):594–603.

34. Beynnon BD, Bernstein I, Belisle A, et al. The effect of estradiol and progesterone on knee and ankle joint laxity. *Am J Sports Med*. 2005;33(9):1298–1304.

35. Nguyen AD, Shultz SJ. Sex differences in lower extremity posture. *J Orthop Sports Phys Ther*. 2007;37(7):389–398.

36. Trimble MH, Bishop MD, Buckley BD, Fields LC, Rozea GD. The relationship between clinical measurements of lower extremity posture and tibial translation. *Clin Biomech (Bristol, Avon)*. 2002;17(4):286–290.

37. Hsu W, Fisk JA, Yamamoto Y, Debski RE, Woo SLY. Differences in torsional joint stiffness of the knee between genders: a human cadaveric study. *Am J Sports Med*. 2006;34(5):765–770.

38. Markolf KL, Graff-Radford A, Amstutz HC. In vivo knee stability: a quantitative assessment using an instrumented clinical testing apparatus. *J Bone Joint Surg Am*. 1978;60(5):664–674.

39. Sharma L, Lou C, Felson DT, et al. Laxity in healthy and osteoarthritic knees. *Arthrit Rheum*. 1999;42(5):861–870.

40. Shultz SJ, Schmitz RJ, Beynnon BD. Variations in varus/valgus and internal/external rotational knee laxity and stiffness across the menstrual cycle. *J Orthop Res*. 2011;29(3):318–325.

41. Shultz SJ, Carcia CR, Perrin DH. Knee joint laxity affects muscle activation patterns in the healthy knee. *J Electromyogr Kinesiol*. 2004;14(4):475–483.

42. Shultz SJ, Shimokochi Y, Nguyen A, et al. Non-weight bearing anterior knee laxity is related to anterior tibial translation during transition from non-weight bearing to weight bearing. *J Orthop Res*. 2006 24(3):516–523.

43. Shultz SJ, Schmitz RJ. Effects of transverse and frontal plane knee laxity on hip and knee neuromechanics during drop landings. *Am J Sports Med*. 2009;37(9):1821–1830.

44. Shultz SJ, Schmitz RJ, Nguyen A, Levine BJ. Joint laxity is related to lower extremity energetics during a drop jump landing.*Med Sci Sports Exer*. 2010;42(4):771–780.

45. Shultz SJ, Nguyen A, Schmitz RJ. Differences in lower extremity anatomical and postural characteristics in males and females between maturation groups. *J Orthop Sports Phys Ther*. 2008;38(3):137–149.

46. Hertel JN, Dorfman JH, Braham RA. Lower extremity malalignments and anterior cruciate ligament injury history. *J Sports Sci Med*. 2004;3(4):220–225.

47. Astrom M, Arvidson T. Alignment and joint motion in the normal foot. *J Orthop Sports Phys Ther*. 1995;22(5):216–222.

48. Smith HC, Vacek P, Johnson RJ, et al. Risk factors for anterior cruciate ligament injury: a review of the literature, part 1. Neuromuscular and anatomic risk. *J Sports Health*. 2012;4(1):69–78.

49. Shultz SJ, Schmitz RJ, Nguyen A, et al. Knee laxity and its cyclic variations influence tibiofemoral joint motion during weight acceptance. *Med Sci Sports Exer*. 2011;43(2):287–295.

50. Scheinman A, Beynnon BD, Smith HC, et al. The geometry of the articular cartilage of the tibial plateau and anterior cruciate ligament injury risk. Paper presented at:Orthopaedic Research Society 2012 Annual Meeting; February 4–7,2012; San Francisco, CA.

51. Kulas AS, Zalewski P, Hortobagyi T, DeVita P. Effects of added trunk load and corresponding trunk position adaptations on lower extremity biomechanics during drop-landings. *J Biomech*. 2008;41(1):180–185.

52. Kulas AS, Hortobagyi T, Devita P. The interaction of trunk-load and trunk-position adaptations on knee anterior shear and hamstrings muscle forces during landing. *J Athl Train*. 2010;45(1):5–15.

53. Mouton C, Seil R, Agostinis H, Maas S, Theisen D. Influence of individual characteristics on static rotational knee laxity using the rotameter. *Knee Surg Sports Traumatol Arthrosc*. 2012;20(4):645–651.

54. September AV, Schwellnus MP, Collins M. Tendon and ligament injuries: the genetic component. *Br J Sports Med*.2007;41(4):241–246.

55. Flynn RK, Pederson CL, Birmingham TB, Kirkley A, Jackonwski D, Fowler PJ. The familial predisposition toward tearing the anterior cruciate ligament. *Am J Sports Med*. 2005;33(1):23–28.

56. Collins M, Posthumus M, Schwellnus MP. The *COL1A1* gene and acute soft tissue ruptures. *Br J Sports Med*.2010;44(14):1063–1064.

57. Khoschnau S, Melhus H, Jacobson A, et al. Type I collagen alpha-1 SP1 plymorphism and the risk of cruciate ligament ruptures or shoulder dislocation. *Am J Sports Med*. 2008;36(12):2432–2436.

58. Posthumus M, September AV, Keegan M, et al. Genetic risk factors for anterior cruciate ligament ruptures: *COL1A1* gene variant. *Br J Sports Med*. 2009;43(5):353–356.

59. Posthumus M, September AV, O'Cuinneagain D, van der Merwe W, Schwellnus MP, Collins M. The *COL5A1* gene is associated with increased risk of anterior cruciate ligament ruptures in female participants. *Am J Sports Med*.2009;37(11):2234–2240.

60. Posthumus M, September AV, O'Cuinneagain D, van der Merwe W, Schwellnus MP, Collins M. The association between the*COL12A1* gene and anterior cruciate ligament ruptures. *Br J Sports Med*. 2010;44(16):1160–1165.

61. Collins M, Posthumus M. Type V collagen genotype and exercise-related phenotype relationships: a novel hypothesis. *Exerc Sport Sci Rev*. 2011;39(4):191–198.

62. Laguette MJ, Abrahams Y, Prince S, Collins M. Sequence variants within the 3′-UTR of the *COL5A1* gene alters mRNA stability: implications for musculoskeletal soft tissue injuries. *Matrix Biol*. 2011;30(5–6):338–345.

63. Posthumus M, Collins M, Van Der Merwe L, et al. Matrix metalloproteinase genes on chromosome 11q22 and the risk of anterior cruciate ligament (ACL) rupture. *Scand J Med Sci Sports*. EPub ahead of print.http://onlinelibrary.wiley.com/doi/10.1111/j.1600-0838.2010.01270.x/abstract.

64. Bell RD, Shultz SJ, Wideman L, Henrich VC. Collagen gene variants previously associated with ACL injury risk are also associated with joint laxity. *J Sports Health*. 2012;4(4):312–318.

65. Raleigh SM, Collins M, eds. Gene variants that predispose to Achilles tendon injuries: an update on recent advances: In: Čretnik A, ed. *Achilles Tendon*. Rejika, Croatia: InTech; http://www.intechopen.com/books/achilles-tendon.

66. Brown JC, Miller CJ, Schwellnus MP, Collins M. Range of motion measurements diverge with increasing age for *COL5A*1 genotypes. *Scan J Med Sci Sports*. 2011;21(6):266–272.

67. Arendt EA, Bershadsky B, Agel J. Periodicity of noncontact anterior cruciate ligament injuries during the menstrual cycle. *J Gender Spec Med*. 2002;5(2):19–26.

68. Beynnon BD, Johnson RJ, Braun S, et al. The relationship between menstrual cycle phase and anterior cruciate ligament injury: a case-control study of recreational alpine skiers. *Am J Sports Med*. 2006;34(5):757–764.

69. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R. Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. *Clin J Sport Med*. 2003;13(2):71–78.

70. Slauterbeck JR, Fuzie SF, Smith MP, et al. The menstrual cycle, sex hormones, and anterior cruciate ligament injury. *J Athl Train*. 2002;37(3):275–280.

71. Wojtys EM, Huston L, Boynton MD, Spindler KP, Lindenfeld TN. The effect of menstrual cycle on anterior cruciate ligament in women as determined by hormone levels. *Am J Sports Med*. 2002;30(2):182–188.

72. Agel J, Bershadsky B, Arendt EA. Hormonal therapy: ACL and ankle injury. *Med Sci Sports Exerc*. 2006 38(1):7–12.

73. Ruedl G, Ploner P, Linortner I, et al. Are oral contraceptive use and menstrual cycle phase related to anterior cruciate ligament injury risk in female recreational skiers? *Knee Surg Sports Traumatol Arthrosc*. 2009;17(9):1065–1069.

74. Dragoo JL, Castillo TN, Braun HJ, Ridley BA, Kennedy AC, Golish SR. Prospective correlation between serum relaxin concentration and anterior cruciate ligament tears among elite collegiate female athletes. *Am J Sports Med*.2011;39(10):2175–2180.

75. Dragoo JL, Lee RS, Benhaim P, Finerman GAM, Hame SL. Relaxin receptors in the human female anterior cruciate ligament. *Am J Sports Med*. 2003;31(4):577–584.

76. Faryniarz DA, Bhargave M, Lajam C, Attia ET, Hannafin JA. Quantitation of estrogen receptors and relaxin binding in human anterior cruciate ligament fibroblasts. *In Vitro Cell Dev Biol Animal*. 2006;42(7):176–181.

77. Hamlet WP, Liu SH, Panossian V, Finerman GA. Primary immunolocalization of androgen target cells in the human anterior cruciate ligament. *J Orthop Res*. 1997;15(5):657–663.

78. Liu SH, Al-Shaikh RA, Panossian V, et al. Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. *J Orthop Res*. 1996;14(4):526–533.

79. Lovering RM, Romani WA. Effect of testosterone on the female anterior cruciate ligament. *Am J Physiol Regul Integr Comp Physiol*. 2005;289(1):R15–R22.

80. Lemoine S, Granier P. Estrogen receptor alpha mRNA in human skeletal muscles. *Med Sci Sports Exer*. 2003;35(3):439–443.

81. Sinha-Hikim I, Taylor WE. Androgen receptor in human skeletal muscle and cultured muscle satellite cells: up-regulation by androgen treatment. *J Clin Endocrinol Metab*. 2004;89(10):5245–5255.

82. Wiik A, Glenmark B. Oestrogen receptor beta is expressed in adult human skeletal muscle both at the mRNA and protein level. *Acta Physiol Scand*. 2003;179(4):381–387.

83. Shultz SJ, Wideman L, Montgomery MM, Beasley KN, Nindl BC. Changes in serum collagen markers, IGF-I and knee joint laxity across the menstrual cycle. *J Orthop Res*. 2012;30(9):1405–1412.

84. Deie M, Sakamaki Y, Sumen Y, Urabe Y, Ikuta Y. Anterior knee laxity in young women varies with their menstrual cycle. *Int Orthop*. 2002;26(3):154–156.

85. Eiling W, Bryant AL, Petersen W, Murphy A, Hohmann E. Effects of menstrual cycle hormone fluctuations on musculoskeletal stiffness and knee joint laxity. *Knee Surg Sports Traumatol Arthrosc*. 2007;15(2):126–132.

86. Heitz NA. Hormonal changes throughout the menstrual cycle and increased anterior cruciate ligament laxity in females. *J Athl Train*. 1999;343(2):144–149.

87. Shultz SJ, Levine BJ, Nguyen AD, Kim HS, Montgomery MM, Perrin DH. A comparison of cyclic variations in anterior knee laxity, genu recurvatum and general joint laxity across the menstrual cycle. *J Orthop Res*. 2010;28(11):1411–1417.

88. Shultz SJ, Sander TC, Kirk SE, Johnson M, Perrin DH. Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc*. 2004;36(7):1165–1174.

89. Shultz SJ, Gansneder BG, Sander TC, Kirk SE, Perrin DH. Absolute hormone levels predict the magnitude of change in knee laxity across the menstrual cycle. *J Orthop Res*. 2006;24(2):124–131.

90. Park SK, Stefanyshyn DJ, Ramage H, Hart DA, Ronsky JL. The relationship between knee joint laxity and knee joint mechanics during the menstrual cycle. *Br J Sports Med*. 2009;43(3):174–179.

91. Shultz SJ, Schmitz RJ, Kong Y, et al. Cyclic variations in multiplanar knee laxity influence landing biomechanics. *Med Sci Sports Exerc*. 2012;44(5):900–909.

92. Romani W, Patrie J, Curl LA, Flaws JA. The correlations between estradiol, estrone, estriol, progesterone, and sex hormone-binding globulin and anterior cruciate ligament stiffness in healthy, active females. *J Women's Health (Larchmt)*.2003;12(3):287–297.

93. Slauterbeck JR, Hickox MS, Beynnon BD, Hardy DM. Anterior cruciate ligament biology and its relationship to injury forces.*Orthop Clin North Am*. 2006;37(4):585–591.

94. Yu WD, Panossian V, Hatch JD, Liu SH, Finerman GA. Combined effects of estrogen and progesterone on the anterior cruciate ligament. *Clin Orthop Rel Res*. 2001;383:268–281.

95. Comerford EJ, Tarlton JF, Avery NC, Bailey AJ, Innes JF. Distal femoral intercondylar notch dimensions and their relationship to composition and metabolism of the canine anterior cruciate ligament. *Osteoarthr Cartilage*. 2006;14(3):273–278.

96. Comerford EJ, Tarlton JF, Innes JF, Johnson KA, Amis AA, Bailey AJ. Metabolism and composition of the canine anterior cruciate ligament relate to differences in knee joint mechanics and predisposition to ligament rupture. *J Orthop Res*.2005;23(1):61–66.

97. Lee C, Liu X, Smith CL, et al. The combined regulation of estrogen and cyclic tension on fibroblast biosynthesis derived from anterior cruciate ligament. *Matrix Biol*. 2004;23(5):323–329.

98. Burrows M, Peters CE. The influence of oral contraceptives on athletic performance in female athletes. *Sports Med*.2007;37(7):557–574.

99. Shultz SJ, Wideman L, Montgomery MM, Levine BJ. Some sex hormone profiles are consistent over time in normal menstruating females: implications for sports injury epidemiology. *Br J Sports Med*. 2011;45(9):735–742.

100. Markolf KL, Burchfield DM, Shapiro MM, Shepard MF, Finerman GA. Combined knee loading states that generate high anterior cruciate ligament forces. *J Orthop Res*. 1995;13(6):930–935.

101. McLean SG, Huang X, Su A, VanDenBogert AJ. Sagittal plane biomechanics cannot injure the ACL during sidestep cutting.*Clin Biomech (Bristol, Avon)*. 2004;19(8):828–838.

102. Shimokochi Y, Shultz SJ. Mechanisms of noncontact anterior cruciate ligament injuries. *J Athl Train*. 2008;43(4):396–408.

103. Shin CS, Chaudhari AM, Andriacchi TP. The influence of deceleration forces on ACL strain during single-leg landing: a simulation study. *J Biomech*. 2007;40(5):1145–1152.

104. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA. The effect of an impulsive knee valgus moment on in vitro relative ACL strain during a simulated jump landing. *Clin Biomech (Bristol, Avon)*. 2006;21(9):977–983.

105. Yu B, Garrett WE. Mechanisms of non-contact ACL injuries. *Br J Sports Med*. 2007;41(Suppl 1):47–51.

106. Meyer EG, Haut RC. Anterior cruciate ligament injury induced by internal tibial torsion or tibiofemoral compression. *J Biomech*.2008;41(16):3377–3383.

107. Oh YK, Kreinbrink JL, Wojtys EM, Ashton-Miller JA. Effect of axial tibial torque direction on ACL relative strain and strain rate in an in vitro simulated pivot landing. *J Orthop Res*. 2012;30(4):528–534.

108. Oh YK, Lipps DB, Ashton-Miller JA, Wojtys EM. What strains the anterior cruciate ligament during a pivot landing? *Am J Sports Med*. 2012;40(3):574–583.

109. Shin CS, Chaudhari AM, Andriacchi TP. The effect of isolated valgus moments on ACL strain during single-leg landing: a simulation study. *J Biomech*. 2009;42(3):280–285.

110. Shin CS, Chaudhari AM, Andriacchi TP. Valgus plus internal rotation moments increase anterior cruciate ligament strain more than either alone. *Med Sci Sports Exerc*. 2011;43(8):1484–1491.

111. Koga H, Bahr R, Myklebust G, Engebretsen L, Grund T, Krosshaug T. Estimating anterior tibial translation from model-based image-matching of a noncontact anterior cruciate ligament injury in professional football: a case report. *Clin J Sport Med*.2011;21(3):271–274.

112. Koga H, Nakamae A, Shima Y, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. *Am J Sports Med*. 2010;38(11):2218–2225.

113. Cerulli G, Benoit DL, Lamontagne M, Caraffa A, Liti A. In vivo anterior cruciate ligament strain behaviour during a rapid deceleration movement: case report. *Knee Surg Sports Traumatol Arthrosc*. 2003;11(5):307–311.

114. Blackburn JT, Padua DA. Sagittal-plane trunk position, landing forces, and quadriceps electromyographic activity. *J Athl Train*.2009;44(2):174–179.

115. Shimokochi Y, Ambegaonkar JP, Meyer EG, Lee SY, Shultz SJ. Changing sagittal plane body position during single-leg landings influences the risk of non-contact anteior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc*. In Press.

116. Myers CA, Torry MR, Shelburne KB, et al. In vivo tibiofemoral kinematics during 4 functional tasks of increasing demand using biplane fluoroscopy. *Am J Sports Med*. 2012;40(1):170–178.

117. Decker MJ, Torry MR, Wyland DJ, Sterett WI, Steadman JR. Gender differences in lower extremity kinematics, kinetics, and energy absorption during landing. *Clin Biomech (Bristol, Avon)*. 2003;18(7):662–669.

118. Houck JR, Duncan A, De Haven KE. Comparison of frontal plane trunk kinematics and hip and knee moments during anticipated and unanticipated walking and side step cutting tasks. *Gait Posture*. 2006;24(3):314–322.

119. Pollard CD, Sigward SM, Powers CM. Gender differences in hip joint kinematics and kinetics during side-step cutting maneuver. *Clin J Sport Med*. 2007;17(1):38–42.

120. Schmitz RJ, Kulas AS, Perrin DH, Riemann BL, Shultz SJ. Sex differences in lower extremity biomechanics during single leg landings. *Clin Biomech (Bristol, Avon)*. 2007;22(6):681–688.

121. Barber-Westin SD, Galloway M, Noyes FR, Corbett G, Walsh C. Assessment of lower limb neuromuscular control in prepubescent athletes. *Am J Sports Med*. 2005;33(12):1853–1860.

122. Barber-Westin SD, Noyes FR, Galloway M. Jump-land characteristics and muscle strength development in young athletes. *Am J Sports Med*. 2006;34(3):375–384.

123. Hass CJ, Schick EA, Tillman MD, Chow JW, Brunt D, Cauraugh JH. Knee biomechanics during landings: comparison of pre- and postpubescent females. *Med Sci Sports Exerc*. 2005;37(1):100–107.

124. Hewett TE, Myer GD, Ford KR. Decrease in neuromuscular control about the knee with maturation in female athletes. *J Bone Joint Surg Am*. 2004;86(8):1601–1608.

125. Noyes FR, Barber-Westin SD, Fleckenstein C, Walsh C, West J. The drop-jump screening test: differences in lower limb contro by gender and effect of neuromuscular training in female athletes. *Am J Sports Med*. 2005;33(2):197–207.

126. Quatman CE, Ford KR, Myer GD, Hewett TE. Maturation leads to gender differences in landing force and vertical jump performance: a longitudinal study. *Am J Sports Med*. 2006;34(5):806–813.

127. Russell PJ, Croce RV, Swartz EE, Decoster LC. Knee-muscle activation during landings: developmental and gender comparisons. *Med Sci Sports Exerc*. 2007;39(1):159–170.

128. Swartz EE, Decoster LC, Russell PJ, Croce RV. Effects of developmental stage and sex on lower extremity kinematics and vertical ground reaction forces during landing. *J Athl Train*. 2005;40(1):9–14.

129. Ford KR, Myer GD, Hewett TE. Longitudinal effects of maturation on lower extremity joint stiffness in adolescent athletes. *Am J Sports Med*. 2010;38(9):1829–1837.

130. Ford KR, Shapiro R, Myer GD, VanDenBogert AJ, Hewett TE. Longitudinal sex differences during landing in knee abduction in young athletes. *Med Sci Sports Exer*. 2010;42(10):1923–1931.

131. Sigward SM, Pollard CD, Havens KL, Powers CM. The influence of sex and maturation on knee mechanics during side-step cutting. *Med Sci Sports Exerc*. 2012;44(8):1497–1503.

132. Chappell JD, Herman DC, Knight BS, Kirkendall DT, Garrett WE, Yu B. Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *Am J Sports Med*. 2005;33(7):1022–1029.

133. Kernozek TW, Torry MR, Iwasaki M. Gender differences in lower extremity landing mechanics caused by neuromuscular fatigue. *Am J Sports Med*. 2008;36(3):554–565.

134. McLean SG, Fellin R, Suedekum N, Calabrese G, Passerallo A, Joy S. Impact of fatigue on gender-based high-risk landing strategies. *Med Sci Sports Exer*. 2007;39(3):502–514.

135. Orishimo KF, Kremenic IJ. Effect of fatigue on single-leg hop landing biomechanics. *J Appl Biomech*. 2006;22(4):245–254.

136. Borotikar BS, Newcomer R, Koppes R, McLean SG. Combined effects of fatigue and decision making on female lower limb landing postures: central and peripheral contributions to ACL injury risk. *Clin Biomech (Bristol, Avon)*. 2008;23(1):81–92.

137. Blackburn JT, Padua DA. Influence of trunk flexion on hip and knee joint kinematics during. *Clin Biomech (Bristol, Avon)*.2008;23(3):313–319.

138. Chaudhari AM, Hearn BK, Andriacchi TP. Sport-dependent variations in arm position during single-limb landing influence knee loading: implications for anterior cruciate ligament injury. *Am J Sports Med*. 2005;33(6):824–830.

139. Chaudhari AM, Andriacchi TP. The mechanical consequences of dynamic frontal plane limb alignment for non-contact ACL injury. *J Biomech*. 2006;39(2):330–338.

140. McLean SG, Huang X, Van den Bogert AJ. Association between lower extremity posture at contact and peak knee valgus moment during sidestepping: implications for ACL injury. *Clin Biomech (Bristol, Avon)*. 2005;20(8):863–870.

141. Sigward SM, Powers CM. Loading characteristics of females exhibiting excessive valgus moments during cutting. *Clin Biomech (Bristol, Avon)*. 2007;22(7):827–833.

142. Donnelly CJ, Lloyd DG, Elliott BC, Reinbolt JA. Optimizing whole-body kinematics to minimize valgus knee loading during sidestepping: implications for ACL injury risk. *J Biomech*. 2012;45(8):1491–1497.

143. Swanik CB, Covassin T, Stearne DJ, Schatz P. The relationship between neurocognitive function and noncontact anterior cruciate ligament injuries. *Am J Sports Med*. 2007;35(6):943–948.

144. Gilchrist J, Mandelbaum BR, Melancon H, et al. A randomized controlled trial to prevent noncontact anterior cruciate ligament injury in female collegiate soccer players. *Am J Sports Med*. 2008;36(8):1476–1483.

145. Hewett TE. The effect of neuromuscular training on the incidence of knee injury in female athletes—a prospective study. *Am J Sports Med*. 1999;27(6):699–705.

146. Hewett TE, Ford KR, Myer GD. Anterior cruciate ligament injuries in female athletes: part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. *Am J Sports Med*. 2006;34(3):1–9.

147. Mandlebaum BR, SIlvers HJ, Watanabe DS, et al. Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes. *Am J Sports Med*. 2005;33(7):1003–1010.

148. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *BMJ*. 2005;330(7489):449.

149. Yoo JH, Lim BO, Ha M, et al. A meta-analysis of the effect of neuromuscular training on the prevention of the anterior cruciate ligament injury in female athletes. *Knee Surg Sports Traumatol Arthrosc*. 2010;18 (6):824–830.

150. Myer GD, Ford KR, Khoury J, Succop P, Hewett TE. Development and validation of a clinic-based prediction tool to identify female athletes at high risk for anterior cruciate ligament injury. *Am J Sports Med*. 2010;38(10):2025–2033.

151. Myer GD, Ford KR, Khoury J, Succop P, Hewett TE. Biomechanics laboratory-based prediction algorithm to identify female athletes with high knee loads that increase risk of ACL injury. *Br J Sports Med*. 2011;45(4):245–252.

152. Padua DA, Marshall SW, Boling MC, Thigpen CA, Garrett WE, Beutler AI. The Landing Error Scoring System (LESS) is a valid and reliable clinical assessment tool of jump-landing biomechanics: the JUMP-ACL study. *Am J Sports Med*.2009;37(10):1996–2002.

153. DiStefano LJ, Padua DA, DiStefano MJ, Marshall SW. Influence of age, sex, technique, and exercise program on movement patterns after an anterior cruciate ligament injury prevention program in youth soccer players. *Am J Sports Med*.2009;37(3):495–505.

154. Padua DA, DiStefano LJ, Marshall SW, Beutler AI, de la Motte SJ, DiStefano MJ. Retention of movement Pattern changes after a lower extremity injury prevention program is affected by program duration. *Am J Sports Med*. 2012;40(2):300–306.

155. Smith HC, Johnson RJ, Shultz SJ, et al. A prospective evaluation of the Landing Error Scoring System (LESS) as a screening tool for anterior cruciate ligament injury risk. *Am J Sports Med*. 2012;40(3):521–526.

156. Zazulak BT, Hewett TE, Reeves NP, Goldberg B, Cholewicki J. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. *Am J Sports Med*. 2007;35(7):1123–1130.

157. Palmieri-Smith RM, Wojtys EM, Ashton-Miller JA. Association between preparatory muscle activation and peak valgus knee angle. *J Electromyogr Kinesiol*. 2008;18(6):973–979.

158. Pollard CD, Sigward SM, Powers CM. Limited hip and knee flexion during landing is associated with increased frontal plane knee motion and moments. *Clin Biomech (Bristol, Avon)*. 2010;25(2):142–146.

159. Claiborne TL, Armstrong CW, Gandhi V, Pincivero DM. Relationship between hip and knee strength and knee valgus during a single leg squat. *J Appl Biomech*. 2006;22(1):41–50.

160. Russell KA, Palmieri RM, Zinder SM, Ingersoll CD. Sex differences in valgus knee angle during a single-leg drop jump. *J Athl Train*. 2006;41(2):166–171.

161. Zazulak BT, Ponce PL, Straub SJ, Medvecky MJ, Avedisian L, Hewett TE. Gender comparison of hip muscle activity during single-leg landing. *J Orthop Sports Phys Ther*. 2005;35(5):292–299.

162. Hollman JH, Ginos BE, Kozuchowski J, Vaughn AS, Krause DA, Youdas JW. Relationships between knee valgus, hip-muscle strength, and hip-muscle recruitment during a single-limb step-down. *J Sport Rehabil*. 2009;18(1):104–117.

163. Lawrence RK, Kernozek TW, Miller EJ, Torry MR, Reuteman P. Influences of hip external rotation strength on knee mechanics during single-leg drop landings in females. *Clin Biomech (Bristol, Avon)*. 2008;23(6):806–813.

164. Shelbourne KD, Gray T, Haro M. Incidence of subsequent injury to either knee within 5 years after anterior cruciate ligament reconstruction with patellar tendon autograft. *Am J Sports Med*. 2009;37(2):246–251.

165. Salmon L, Russell V, Musgrove T, Pinczewski L, Refshauge K. Incidence and risk factors for graft rupture and contralateral rupture after anterior cruciate ligament reconstruction. *Arthroscopy*. 2005;21(8):948–957.

166. Paterno MV, Rauh MJ, Schmitt LC, Ford KR, Myer GD, Hewett TE. Incidence of contralateral and ipsilateral anterior cruciate ligament (ACL) injury after primary ACL reconstruction and return to sport. *Clin J Sport Med*. 2012;22(2):116–121.

167. LaBella CR, Huxford MR, Grissom J, Kim KY, Peng J, Christoffel KK. Effect of neuromuscular warm-up on injuries in female soccer and basketball athletes in urban public high schools: cluster randomized controlled trial. *Arch Pediatr Adolesc Med*.2011;165(11):1033–1040.

168. Herman DC, Onate JA, Weinhold PS, et al. The effects of feedback with and without strength training on lower extremity biomechanics. *Am J Sports Med*. 2009;37(7):1301–1308.

169. Hewett TE, Stroupe AL, Noyes FR. Plyometric training in female athletes: decreased impact forces and increased hamstring torques. *Am J Sports Med*. 1996;24(6):765–773.

170. Hurd WJ, Chmielewski TL, Snyder-Mackler L. Perturbation-enhanced neuromuscular training alters muscle activity in female athletes. *Knee Surg Sports Traumatol Arthrosc*. 2006;14(1):60–69.

171. Myer GD, Ford KR, Brent JL, Hewett TE. The effects of plyometric vs. dynamic stabilization and balance training on power, balance, and landing force in female athletes. *J Strength Cond Res*. 2006;20(2):345–353.

172. Myer GD, Ford KR, McLean SG, Hewett TE. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. *Am J Sports Med*. 2006;34(3):445–455.

173. Oñate JA, Guskiewicz KM, Marshall SW, Giuliani C, Yu B, Garrett WE. Instruction of jump-landing technique using videotape feedback: altering lower extremity motion patterns. *Am J Sports Med*. 2005;33(6):831–842.

174. Pollard CD, Sigward SM, Ota S, Langford K, Powers CM. The influence of in-season injury prevention training on lower-extremity kinematics during landing in female soccer players. *Clin J Sport Med*. 2006;16(3):223–227.

175. Wulf G, Chiviacowsky S, Schiller E, Avila LT. Frequent external-focus feedback enhances motor learning. *Front Psychol*.2010;1:1–7.

176. Dowling AV, Favre J, Andriacchi TP. Inertial sensor-based feedback can reduce key risk metrics for anterior cruciate ligament injury during jump landings. *Am J Sports Med*. 2012;40(5):1075–1083.

177. Barrios JA, Crossley KM, Davis IS. Gait retraining to reduce the knee adduction moment through real-time visual feedback of dynamic knee alignment. *J Biomech*. 2010;43(11):2208–2213.

178. Crowell HP, Milner CE, Hamill J, Davis IS. Reducing impact loading during running with the use of real-time visual feedback. *J Orthop Sports Phys Ther*. 2010;40(4):206–213.

179. Beek PJ. Toward a theory of implicit learning in the perceptual-motor domain. *Int J Sport Psychol*. 2000;31:547–554.

180. DiStefano LJ, Blackburn JT, Marshall SW, Guskiewicz KM, Garrett WE, Padua DA. Effects of an age-specific anterior cruciate ligament injury prevention program on lower extremity biomechanics in children. *Am J Sports Med*. 2011;39(5):949–957.

181. Hertel JN, Guskiewicz KM, Kahler DM, Perrin DH. Effect of lateral ankle joint anesthesia on center of balance, postural sway, and joint position sense. *J Sport Rehabil*. 1996;5(2):111–119.

182. Lipps DB, Oh YK. Ashton-Miller JA, Woytys EM. Influence of knee morphological characteristics on peak ACL strain during simulated pivot landings. *J Athl Train*. 2012;47(5):e1. doi:10.4085/1062-6050-47.5.13.

183. Pye ML, Schmitz RJ, Shultz SJ. Less lower extremity muscle mass is associated with greater knee laxity and lower stiffness in the frontal and transverse planes. *J Athl Train*. 2012;47(5):e1. doi:10.4085/1062-6050-47.5.13.

184. Bell R, Wideman L, Henrich VC, Shultz SJ. Exploratory analysis of joint laxity and genotypes associated with ACL injury. *J Athl Train*. 2012;47(5):e2. doi:10.4085/1062-6050-47.5.13.

185. Lyle MA, Stearns KM, Powers CM. Sex differences in lower extremity stiffness during a single limb drop jump maneuver: evidence for heightened feedforward control in females. *J Athl Train*. 2012;47(5):e3. doi:10.4085/1062-6050-47.5.13.

186. Norcross MF, Lewek MD, Padua DA, Shultz SJ, Weinhold PS, Blackburn JT. The influences of knee flexion angle at initial contact and sex on knee kinetics. *J Athl Train*. 2012;47(5):e7. doi:10.4085/1062-6050-47.5.13.

187. DiStefano LJ, Trojian TH, Nguyen A, Boling MC. Pubertal and sex differences in lower extremity kinematics during a cutting task. *J Athl Train*. 2012;47(5):e3. doi:10.4085/1062-6050-47.5.13.

188. Pfile KR, Hertel J, Hart JM, Kerrigan DC, Ingersoll CD. Adult female athletes display different hip and knee biomechanics during sidestep cutting compared to adolescent and prepubescent female athletes. *J Athl Train*. 2012;47(5):e3. doi:10.4085/1062-6050-47.5.13.

189. Greska E, Cortes N, Samaan M, Onate JA, Ringleb S, Van Lunen B. The effects of fatigue and leg dominance on hip and knee neuromechanics. *J Athl Train*. 2012;47(5):e10. doi:10.4085/1062-6050-47.5.13.

190. Frank BS, Bell DR, Norcross MF, Goerger BM, Blackburn JT, Padua DA. Lumbopelvic hip complex neuromuscular control is associated with triplanar knee loading. *J Athl Train*. 2012;47(5):e6. doi:10.4085/1062-6050-47.5.13.

191. Chandrashekar N, Cassidy K, Sabharwal P. A combined in-vivo/in-vitro method to study the biomechanics of ACL injury. *J Athl Train*. 2012;47(5):e11. doi:10.4085/1062-6050-47.5.13.

192. Jamison ST, McNally MP, Schmitt LC, Chaudhari AMW. Correlation of frontal plane knee moments and angles between drop vertical jump and run-to-cut maneuvers. *J Athl Train*. 2012;47(5):e10. doi:10.4085/1062-6050-47.5.13.

193. Whale C, Oliver G, Tripp PM. Predictive value of the jump task for injury among Division-I female soccer athletes. *J Athl Train*.2012;47(5):e9. doi:10.4085/1062-6050-47.5.13.

194. Padua DA, Boling MC, Goerger BM, et al. A prospective evaluation of knee flexion and valgus mechanics as predictors for anterior cruciate ligament injury risk. *J Athl Train*. 2012;47(5):e8. doi:10.4085/1062-6050-47.5.13.

195. Stearns KM, Powers CM. The influence of hip abductor muscle performance on the knee valgus moment during landing: a gender comparison. *J Athl Train*. 2012;47(5):e4. doi:10.4085/1062-6050-47.5.13.

196. Ericksen HM, Lepley AS, Doebel SC, et al. Immediate decreases in peak vertical ground reaction force following real-time feedback during jump landing. *J Athl Train*. 2012;47(5):e6. doi:10.4085/1062-6050-47.5.13.

197. Nilstad A, Kristianslund E, Krosshaug T, et al. A simple clinical screening test can identify elite female football players with increased knee valgus angles. *J Athl Train*. 2012;47(5):e5. doi:10.4085/1062-6050-47.5.13.

198. Benjaminse A, Gokeler A, Cortes N, Otten E. Effects of stiff and soft landing techniques on knee loading during a single-leg cross-over hop. J Athl Train. 2012;47(5):e4. doi:10.4085/1062-6050-47.5.13.