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

March 22–24, 2012; Greensboro, NC

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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 future research. Following is the updated consensus statement. The presentation abstracts organized by topic and presentation order appear online at http://nata.publisher.ingentaconnect.com/content/nata/jat.

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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