

ACL Research Retreat V: An Update on ACL Injury Risk and Prevention, March 25–27, 2010, Greensboro, NC

Sandra J. Shultz, PhD, ATC, FNATA, FACSM*; **Randy J. Schmitz, PhD, ATC***;
Anh-Dung Nguyen, PhD, ATC†; **Ajit M. Chaudhari, PhD‡**;
Darin A. Padua, PhD, ATC§; **Scott G. McLean, PhD ||**;
Susan M. Sigward, PhD, ATC, PT¶

*Department of Kinesiology, University of North Carolina at Greensboro; †Department of Health and Human Performance, College of Charleston, SC; ‡Ohio State University Sports Medicine and Movement Analysis & Performance Program, Columbus; §Sports Medicine Research Laboratory, University of North Carolina at Chapel Hill; ||Injury Biomechanics Laboratory and Human Neuromechanics Laboratory, University of Michigan, Ann Arbor; ¶Musculoskeletal Biomechanics Research Laboratory, University of Southern California, Los Angeles

Multiple factors, whether individually or in combination, likely contribute to noncontact anterior cruciate ligament (ACL) injury. Although research has increased our understanding of contributing factors, much remains unknown, and continued research is needed. To that end, the fifth ACL Research Retreat was held at the University of North Carolina at Greensboro, March 25–27, 2010. The retreat's ongoing mission is to (1) present and discuss the most recent research on ACL injury risk and prevention and (2) identify new research directives aimed at understanding the epidemiology, risk factors, and prevention of noncontact ACL injury. This year, 75 clinicians and researchers representing 6 countries participated.

All the keynote presenters are expert scientists engaged in cutting-edge research on ACL injury risk and prevention. Edward Wojtyś, MD; Stephen W. Marshall, PhD; Darin A. Padua, PhD, ATC; and Christopher M. Powers, PT, PhD, focused on issues related to clinical and research considerations for ACL-injured pediatric and adolescent athletes, current trends in injury epidemiology, and new directions in risk-factor assessment and efficacy of injury-prevention programs. Forty podium and poster presentations were organized into thematic sessions: risk-factor assessment (specifically factors associated with spinal and trunk control, muscle strength and fatigue, anatomical and hormonal factors, and landing and cutting strategies), injury mechanisms, risk-factor screening, and prevention. A meeting hallmark is the substantial time provided for group discussion after each keynote address and thematic podium session. To close the meeting, participants revisited and updated the consensus statement from the 2008 ACL Research Retreat IV¹ and charted new directions for future research. Following are the updated consensus statement, keynote presentation summaries, and abstracts organized by topic and presentation order.

CONSENSUS STATEMENT

As at past retreats, the consensus statement was updated and further refined based on the input of all participants at

the meeting's end. Participants were divided into 3 interest groups: hormonal and anatomical risk factors, neuromechanical contributions to ACL injury, and risk factor screening and prevention. Within each group, the relevant sections of the previous consensus document were discussed and updated as to (1) what we know based on new evidence emerging in the literature and presented at the retreat, (2) what remains unknown about these factors, and (3) the important directions for future research needed to address these unknowns. Each group then presented its working draft to all participants for further discussion, and drafts were circulated after the meeting for final comment.

From these discussions, some global observations, themes, and recommendations emerged that deserve special note. First, in response to general themes raised previously,¹ we seem to be moving away from the purely descriptive sex-comparison studies that have tended to dominate the literature toward a better understanding of the underlying mechanisms associated with the observed sex differences and, more directly, ACL injury risk and prevention. However, a more integrated approach in our research is still desperately needed to better characterize the multifactorial nature of the ACL injury enigma: examine all relevant anatomical (eg, posture, structure, body composition) and structural (eg, tibial slope, condylar geometry) factors, as well as the associated neuromechanical outcomes (eg, integrated findings from kinetic, kinematic, and neuromuscular measures) used in assessing these risk factors. Studying multiple factors is particularly challenging, given the relatively low incidence of ACL injury. Thus, traditional risk-factor designs (eg, prospective cohort studies) are needed but may not advance our scientific understanding of the ACL injury mechanism rapidly enough to permit these injuries to be prevented. To offset these challenges, we must develop open-source databases (eg, video registries, software models) to pool our resources. Generating complementary new measurement paradigms is also important, both to more quickly advance our scientific understanding of the ACL injury mechanism

and related risk factors and to gain a more integrated understanding of this complex, multifactorial problem. Continued emphasis on computer and cadaveric modeling will allow multifactorial manipulation that is not possible in vivo, and state-of-the-art robotic simulations will help us to better understand complex relationships among muscle force production, joint and ligament morphology, and resultant ACL strain. More functional in vivo testing conditions are needed to describe cognitive, supraspinal, and spinal contributions to knee joint and ACL loading within the truly random movement environment of game play. The roles of behavior and cognition have to date received relatively little attention, yet the work of previous researchers^{2,3} and the keynote presentation by Chris Powers provide compelling evidence of the need to explore these areas. Addressing these and other unknowns is critical to the continued development and refinement of injury-prevention programs. Although most prevention programs to date have focused on eliminating certain movements (eg, valgus maneuvers) as a solution to the problem, the evidence supporting any one mechanism alone is quite weak. Thus, although these programs have shown some success and should continue, the ideal ACL injury-prevention program may not be realized until we have a better grasp on this complex, multifactorial problem.

Another general consideration raised at the retreat was increasing our focus on the youth athlete and taking more of a public health approach in our injury-risk screening and injury-prevention strategies in this population. The best time to identify and counter risk is posited to be during the adolescent years, but we still know very little about when relevant risk factors emerge, when intervention should be initiated, and how we can improve participation in, compliance with, and effectiveness of ACL injury-prevention programs in this critical target population. Children and adolescents perform athletic tasks differently than adults,⁴⁻⁷ and recent findings^{8,9} suggest that the response to injury-prevention programs may differ across age groups. Additionally, the continuing trends of inactivity and obesity (eg, loss of physical education classes in US schools) are of great concern with respect to their effect on acquisition of general motor programs, skill development, and prospective ACL injury risk. Another important public health concern is the consequences of ACL injury so early in life in both the short (eg, lost school time) and long term (eg, osteoarthritis, inactivity). Therefore, we call for a better understanding of the prospective risk factors for ACL injury in the maturing youth population; improved understanding of the cognitive, behavioral, and socioeconomic factors that influence the successful implementation of ACL injury-awareness and injury-prevention programs in youth sport participants; and a movement toward more translational approaches to implementing ACL injury-prevention programs into community settings to maximize public health.

Although only 2 years have passed since the last ACL retreat, a number of advancements in our knowledge of ACL injury risk and prevention have reshaped the important unknowns and future directions we need to take. These changes are reflected in the revised consensus statement. We hope that the insights and proceedings from this meeting will continue to strengthen the foundation upon which quality research and clinical interventions in ACL injury risk and prevention can be advanced.

NEUROMUSCULAR AND BIOMECHANICAL FACTORS

What We Know

1. The ACL is loaded by a variety of combined sagittal and nonsagittal mechanisms during dynamic sports postures considered to be high risk¹⁰⁻¹⁵ (abstracts #22, Yu et al, and #25, Myer et al).
2. In vivo strain of the ACL is related to maximal load and timing of ground reaction forces.^{16,17} Females typically display a more erect or upright posture when contacting the ground during the early stages of deceleration tasks.¹⁸⁻²¹ However, the magnitude of these differences may be task dependent (abstract #26, Benjaminse et al).
3. Maturation influences biomechanical and neuromuscular factors^{4-6,22-26} (abstract #33, Sigward et al).
4. Fatigue alters lower limb biomechanical and neuromuscular factors and is suggested to increase ACL injury risk.²⁷⁻³¹ The effect of fatigue is most pronounced when combined with unanticipated landings, causing substantial central processing and control compromise.^{3,32}
5. Trunk, core, and upper body mechanics influence lower extremity biomechanical and neuromuscular factors^{19,33-35} (abstract #1, Chaudhari et al).
6. Hip position and stiffness influence lower extremity biomechanical factors.³⁶⁻³⁸

What We Don't Know

1. Which biomechanical and neuromuscular profiles cause noncontact ACL rupture? An understanding of the causes is central to identifying how to screen at-risk individuals.
2. Although we understand that the trunk, core, and hip affect knee biomechanics in general, because of the limited number of research models estimating in vivo ACL strain, we still do not know how these trunk and hip biomechanical factors affect in vivo ACL strain during the highly dynamic activities known to cause ACL injury.
3. We do not yet understand the role of neuromuscular and biomechanical 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?
4. Slower reaction times, slower processing speed, and visual-spatial disorientation have been observed in athletes sustaining ACL injuries,² but we do not know if 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.³
5. Is gross failure of the ACL caused by a single episode or multiple episodes?
6. Is noncontact ACL injury governed by a single or potentially multiple high-risk neuromuscular and biomechanical profile(s)?

Where We Go From Here

1. To best understand movement patterns linked to noncontact ACL injury, researchers should include comprehensive kinetic, kinematic, and neuromuscular (strength, postural stability, activation, and timing) profiles (henceforth referred to as *neuromechanics*).
2. We need to improve our understanding of neuromechanical variability within (including between limbs) and among individuals as it relates to injury risk and injury mechanisms.
3. The interaction of anatomical structure, laxity, and neuromechanics needs to be better understood to fully appreciate joint-loading profiles.
4. Anatomical, hormonal, and neuromechanical factors and their interactions predictive of ACL injuries should be derived from prospective data. To that end, multifactorial prospective risk-factor studies are necessary, despite their expense and time-consuming nature.
5. To better understand how movement patterns and other structures in the kinetic chain affect ACL loads, we must continue to develop and improve quality models (eg, computational, cadaveric) that noninvasively estimate in vivo ACL forces and strain.
6. 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. Further, musculoskeletal models describing cause-and-effect relationships have to be studied explicitly within a realistic injury scenario.
7. Laboratory assessments of neuromechanical factors should use tasks that mimic the mechanical demands commonly associated with relevant sport-injury mechanisms, and care should be taken not to overgeneralize results from one specific task to other tasks with different mechanical demands.
8. We must identify any critical thresholds of structural or functional weakness at which compensatory strategies become evident.
9. 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).
10. The influence of the trunk and core on knee biomechanics and specifically ACL loads must be better characterized.
11. The influence of the maturational process on knee biomechanics and specifically ACL loads must be further understood.
12. Work that translates laboratory measures to the field and field measures to the laboratory will help with the interpretation of findings in both settings. Especially important is the validation of commonly performed field assessment (eg, squatting, landing) to known

neuromechanics profiles evident within the inherently random sport environment.

13. Technology must continue to advance and evolve to help us better understand in vivo mechanics, allow more precise transverse-plane measurements, and improve the accuracy and ease of use of measurement techniques generally.
14. We still do not have precise descriptions of the mechanisms of ACL rupture. The injury video is the only method available to extract biomechanical information from actual injury situations. Therefore, we must accumulate injury videos to allow us to better understand the injury mechanism.
15. 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.

ANATOMICAL AND STRUCTURAL FACTORS

What We Know

1. The female ACL is smaller in length, cross-sectional area, and volume than the male ACL, even after adjusting for body anthropometry.³⁹
2. The female's femoral notch height is larger and femoral notch angle is smaller than in males, which may influence femoral notch-impingement theory. Femoral notch width is a good predictor of ACL size (area and volume) in males but not in females. Femoral notch angle is a good predictor of ACL size in females but not in males.³⁹
3. The female ACL is less stiff (lower modulus of elasticity) and fails at a lower load level (lower failure strength), even after adjusting for age, body anthropometrics, and ACL size.⁴⁰
4. Ultrastructural analysis of the ACL shows that the percentage of area occupied by collagen fiber (area of collagen fibers/total area of the micrograph) is lower in females when adjusted for age and body anthropometrics.⁴¹
5. Compared with uninjured people, injured individuals have smaller ACLs (area and volume),⁴² greater posterior slope of the lateral tibia,^{43,44} similar slope of the medial tibia,^{43,44} reduced condylar depth on the medial tibial plateau,⁴³ and presence of an anterior medial ridge on the intercondylar notch.⁴⁵
6. Clear laxity differences have been observed between males and females, with females often displaying greater genu recurvatum,^{46,47} anterior knee laxity,^{48–52} and general joint laxity.^{53–55} Females are also reported to have 25% to 30% greater frontal-plane and transverse-plane laxity^{56–59} and less torsional stiffness^{56,60,61} than males, differences observed even when no sex differences in anterior knee laxity were present.^{56,58,62}

7. Greater magnitudes of joint laxity have been associated with altered knee-joint neuromechanics during weight bearing^{62–66} and increased risk of ACL injury.^{50,52,67–71}
8. Women have greater anterior pelvic tilt, hip anteversion, tibiofemoral angle, and quadriceps angle than men.^{46,72} No sex differences have been observed for tibial torsion,⁴⁶ navicular drop,^{46,47,72} and rearfoot angle.^{46,73}
9. Lower extremity alignments differ among maturation groups and develop at different rates in males and females among maturation groups.⁷⁴
6. We must continue to examine the influence of anatomical (eg, posture, structure, body composition) and structural factors (eg, tibial slope, condylar geometry) on knee-joint neuromechanics, both in adults and in maturing youth.
7. We need to understand the underlying factors that cause one to develop at-risk anatomical and structural profiles during maturation. Additional factors, such as the roles of physical activity, body composition changes, and muscle properties (eg, stiffness, slack [resting tissue] length, and fatty infiltration) should be considered.

What We Don't Know

1. Can physical activity influence these anatomical and structural factors, and if so, when, how, and for how long do the changes occur as a result of physical activity?
2. What effect does meniscal geometry have on ACL strain and failure during activity?
3. What variations in the anatomical and structural factors influence knee-joint neuromechanics to increase risk?
4. Some evidence indicates that elevated body mass index predicts future ACL injury in females⁵² and that artificially increasing body mass index encourages dangerous biomechanical strategies.^{35,75} Although body composition may be a relevant anatomical risk factor, we still know very little about its influence on lower extremity neuromechanical strategies and ACL injury risk.

Where We Go From Here

1. We should include all relevant lower extremity anatomical and structural factors along with neuromechanical factors in large-scale, prospective risk-factor studies. Developing more efficient, affordable, reliable, and readily available measurement methods of these anatomical and structural factors for use in these large-scale, prospective risk-factor studies is important.
2. Case-control study designs for examining structural factors should also be considered because structural factors are not acutely affected by ACL rupture.
3. We must continue to examine interactions among tibial slope (anterior-posterior, medial-lateral), ACL volume, ultrastructure, laxity, femoral notch geometry, condylar geometry, and lower extremity alignment for their effects on ACL strain and failure and predicting injury risk.
4. We should examine the influence of physical activity during maturation and across the life span on anatomical and structural factors.
5. The role of meniscus geometry on ACL strain and failure during activity must be described.

HORMONAL FACTORS

What We Know

1. The likelihood of suffering an ACL injury is not evenly distributed across the menstrual cycle; instead, the risk of suffering an ACL disruption is greater during the preovulatory phase of the cycle than during the postovulatory phase.^{76–80} During the preovulatory phase, hormone levels change dramatically, falling to their nadirs with the onset of menses and rising rapidly near ovulation.
2. Sex hormone (eg, estrogen, testosterone, relaxin) receptors are present on the human ACL.^{81–85}
3. Sex hormone (eg, estrogen, testosterone) receptors have been found in skeletal muscle.^{86–88}
4. Large individual variations in female hormone profiles should be appreciated in our study designs.⁸⁹ Although hormone profiles are substantially more consistent within an individual female from month to month, some variability still exists.⁹⁰ This within-subjects variability can be reduced by taking multiple samples over repeated days.⁹⁰
5. Consistent with individual variabilities in hormone profiles, substantial variations exist in the magnitude of change in laxity (ie, anterior knee laxity, genu recurvatum, general joint laxity) that females experience across the menstrual cycle.^{55,91} However, within an individual from month to month, the magnitude of cyclic variations in laxity is quite reproducible.⁵⁵
6. Because of the individual variabilities in hormone profiles across the menstrual cycle, a single measurement within a single phase (even with hormone confirmation) is not adequate to accurately characterize the same hormone profile or time point in a particular phase of the menstrual cycle for all females.
7. The mechanical and molecular properties of the ACL are likely influenced not only by estrogen but also by the interaction of several sex hormones, secondary messengers, remodeling proteins, and mechanical stresses.^{82,85,89,92–94}
8. A time-dependent effect for sex hormones and other remodeling agents influences a change in ACL tissue characteristics.^{85,89}

9. In animal models, interactions have been noted among mechanical stress, hormones, and altered ACL structure and metabolism.^{95–97}

What We Don't Know

1. What is the underlying mechanism for the increased likelihood of ACL injury in the preovulatory phase?
2. How do ACL injury rates vary in females who are eumenorrheic, oligomenorrheic, or using oral contraceptives?
3. What are the sex-specific hormonal, molecular, and genetic mechanisms of sex hormones on ACL structure, metabolism, and mechanical properties? Although the influence of hormones on ACL biology has been examined in a variety of animal models^{97–106} and relatively few human studies,^{94,107} consensus is lacking because of variations in study designs and the species examined.
4. What is the role of sex hormones on skeletal muscle structure and function in controlling dynamic motion? What, if any, changes occur in neuromuscular and biomechanical risk factors across the menstrual cycle? Although previous authors^{108–110} have suggested that cyclical changes in neuromuscular and biomechanical control may be negligible, these results may be incomplete because of the individual variations in hormone profiles (see “What We Know,” items 4–6).
5. Does the rate of increase or time duration of amplitude peaks in hormone fluctuation play a role in soft tissue changes?
6. Although cyclic variations in anterior knee laxity may be sufficient to alter knee joint neuromechanics,^{111,112} we do not yet fully understand the clinical implications of cyclic changes in knee laxity on weight-bearing knee-joint function.
7. What are the interactions among mechanical stress on the ACL, hormone profiles, and altered ACL structure and metabolism in physically active females?

Where We Go From Here

1. We must continue to consider the interactive effect of all relevant hormones on soft tissue structures and ACL injury risk, including hormonal, molecular, and genetic mechanisms.
2. The hormonal, molecular, and genetic mechanisms by which sex hormones may explain the observed sex-specific differences in ACL structure, metabolism, and mechanical properties should be defined (see also “Anatomical and Structural Factors”).
3. More studies using research designs relevant to the healthy, physically active female are needed to examine hormone effects on ACL structural, metabolism, and mechanical properties.
4. When examining hormone influences on knee-joint function and ACL injury risk, females using oral

contraceptives and those with irregular menstrual cycles (amenorrheic, oligomenorrheic) should also be examined. The type of contraceptive should be documented and both the endogenous and exogenous levels of sex hormones examined.

5. Future studies of hormonal risk factors should focus more on individual results, rather than mean values, because individual menstrual cycle characteristics vary markedly.
6. Improved methods of measuring individual hormone profiles to better match the complexity of the role of hormones in soft tissue changes must be developed. We need to verify phases of the cycle with actual hormone measures and consider all relevant hormones, including estrogen, progesterone, and possibly others. To confirm that the desired time in the cycle or a particular phase is truly captured in future study designs, hormone samples should be taken over multiple days rather than measured at a single time point.⁹⁰
7. When making female-to-male comparisons, variables should be collected during the early follicular phase, when hormone levels are at their nadirs (preferably 3–7 days postmenses) to decrease the potential for cyclic fluctuations in hormones that confound the anatomical, neuromuscular, and biomechanical outcomes of interest.
8. We must examine the interaction among hormones, mechanical loading, and ACL mechanical properties in the physically active female.
9. Examination of ACL injury in genome-wide association studies to establish any genetic components to ACL injury should be encouraged.

RISK-FACTOR SCREENING AND PREVENTION

What We Know

1. Training programs that incorporate elements of balance training, plyometric training, education, strengthening, and feedback alter biomechanical and neuromuscular variables thought to contribute to ACL injury.^{113–119}
2. Intervention programs have been shown to reduce the incidence of ACL injuries.^{78,120–125} Although these results are promising, ACL injury rates and the associated sex disparity have not yet diminished. The ideal ACL injury-prevention program has yet to be identified.
3. The protective effects of ACL injury-prevention training programs appear to be transient.^{126–128}
4. Field assessment and screening tools show promise for identifying individuals at increased risk for ACL injury (abstract #33, Padua et al).
5. Injuries to the ACL can have long-term effects, including contributing to the burden of osteoarthritis.^{129–132}

6. Cohort studies suggest that a prior history of ACL injury may be a risk factor for another ACL injury on the ipsilateral or contralateral side.^{133–135} Family history of ACL injury also appears to increase risk.^{136–138}

What We Don't Know

1. What are the mechanisms underlying the success of injury-prevention programs? Specifically, what elements of an injury-prevention program (strengthening, plyometrics, etc) produce the desired protective effect?
 2. How much training stimulus (eg, duration, timing) is required to produce the desired protective effect, and how long does the effect last?
 3. At what age should an injury-prevention program be implemented to reduce potential neuromuscular and biomechanical risk factors?
 4. Should intervention programs be tailored to specific sports, specific ages, or an individual athlete's needs? Evidence to date suggests that injury-prevention programs may be more effective for soccer than basketball.¹³⁹ However, most prevention programs have been designed for and tested in soccer and team handball athletes. Further, current programs do not appear to be as successful in pediatric age groups.^{8,9}
 5. Do intervention programs influence athletic performance?
 6. Although an individual's family history^{136–138} and personal history of ACL injury^{133–135} appear to increase the risk of ACL injury, the mechanisms and prospective risk factors associated with this elevated risk are not known.
 7. Are ACL injury mechanisms and prospective risk factors the same in pediatric and adult populations?
 8. What are the effects of physical activity (ie, inactivity) and the availability of physical education on prospective ACL-injury risk factors?
 9. What are the effects of individual, organizational, and socioeconomic factors on the successful implementation of ACL injury-prevention programs in various settings?
 10. What barriers and facilitators are associated with injury-prevention program compliance?
2. Expand screening and prevention research in the following ways:
 - a. Continue to develop and validate other field-assessment and screening protocols that identify individuals at risk for ACL injury. Should we develop and validate minimum standards for an ACL injury-risk screening protocol?
 - b. Increase knowledge regarding injury-prevention programs. What are key factors to modify with training? Should programs be population specific with regard to sport, maturation, and level of experience? What are the essential elements (exercises types, dose)? How can training programs address issues related to fatigue? What is the optimal timing with respect to season? What is the optimal duration and frequency (dose)? How long do the positive effects of training programs for preventing ACL injury last after an injury-prevention program is completed (ie, when is a booster needed)? What are the perceived barriers, attitudes, and motivations related to compliance with injury-prevention programs? Does increasing knowledge and awareness of programs increase compliance? Regarding emerging differential evidence of effectiveness, what are the characteristics of responders and nonresponders to an ACL injury-prevention program? What is the cost-effectiveness of ACL injury-prevention strategies?
 - c. Develop and evaluate new ACL injury-prevention interventions, including educational programs, sport-specific conditioning and training, modification of anatomical and hormonal factors, etc.

Where We Go From Here

1. Further define the epidemiology (burden and effect) of ACL injury, including individual risk factors, and assess interactions among risk factors (eg, predictive risk profiles). To that end:
 - a. Establish population-based registries of ACL injuries and strengthen and expand ongoing injury-surveillance systems to enable monitoring of long-term trends in ACL incidence, including
3. Evaluate if injury-prevention programs can positively or negatively affect athletic performance.
4. Begin translational research to implement ACL injury-prevention programs into community settings to maximize public health effects. Specifically

- a. Evaluate the benefits of a population-based approach (everyone receives the intervention) or a high-risk approach (only high-risk individuals receive the intervention) and determine best practices for dissemination of ACL injury-prevention strategies in the community.
- b. Apply and translate the Translating Research into Injury Prevention Practice (TRIIP) model of injury prevention for the ACL.¹⁴⁰
- c. Develop partnerships and systems approaches with organizations to better understanding barriers to implementation and to improve compliance and program effectiveness (sports governing bodies, coaches, and school-based organizations; professional organizations such as the National Athletic Trainers' Association, American Physical Therapy Association, and American Orthopaedic Society for Sports Medicine).
- d. Look to other countries for translation models that have successfully adopted programs for different communities to maximize program compliance and effectiveness in the United States.

ACKNOWLEDGMENTS

The ACL Research Retreat V was hosted by the Department of Kinesiology at the University of North Carolina at Greensboro. We gratefully acknowledge Innovative Sports Training, Inc; the Department of Kinesiology, School of Health and Human Performance, and the Office of the Provost and Executive Vice Chancellor of the University of North Carolina at Greensboro for their sponsorship and support of the meeting.

REFERENCES

1. Shultz SJ, Schmitz RJ, Nguyen AD. Research Retreat IV: ACL Injuries. The Gender Bias, April 3–5, 2008, Greensboro, NC. *J Athl Train.* 2008;43(5):530–537.
2. 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.
3. McLean SG, Samorezov JE. Fatigue-induced ACL injury risk stems from a degradation in central control. *Med Sci Sports Exerc.* 2009;41(8):1661–1672.
4. 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.
5. 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.
6. 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.
7. Yu B, McClure SB, Onate JA, Guskiewicz KM, Kirkendall DT, Garrett WE. Age and gender effects on lower extremity kinematics of youth soccer players in a stop-jump task. *Am J Sports Med.* 2005;33(9):1356–1364.
8. 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.
9. Grandstrand SL, Pfeiffer RP, Sabick MB, DeBeliso M, Shea KG. The effects of a commercially available warm-up program on landing mechanics in female youth soccer players. *J Strength Cond Res.* 2006;20(2):331–335.
10. Markolf KL, Burchfield DM, Shapiro MM, Shepard MF, Finerman GA, Slaughterbeck JL. Combined knee loading states that generate high anterior cruciate ligament forces. *J Orthop Res.* 1995;13(6):930–935.
11. McLean SG, Huang X, Su A, Van Den Bogert AJ. Sagittal plane biomechanics cannot injure the ACL during sidestep cutting. *Clin Biomech (Bristol, Avon).* 2004;19(8):828–838.
12. Shimokochi Y, Shultz SJ. Mechanisms of noncontact anterior cruciate ligament injuries. *J Athl Train.* 2008;43(4):396–408.
13. 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.
14. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA. The relationship between quadriceps muscle force, knee flexion, and anterior cruciate ligament strain in an in vitro simulated jump landing. *Am J Sports Med.* 2006;34(2):269–274.
15. Yu B, Garrett WE. Mechanisms of non-contact ACL injuries. *Br J Sports Med.* 2007;41(suppl):47i–51i.
16. 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.
17. 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.
18. Decker MJ, Torry MR, Wyland DJ, Sterett WI, Steadman RJ. Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. *Clin Biomech (Bristol, Avon).* 2003;18(7):662–669.
19. 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.
20. 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.
21. 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.
22. 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.
23. Barber-Westin SD, Noyes FR, Galloway M. Jump-land characteristics and muscle strength development in young athletes: a gender comparison of 114 athletes 9 to 17 years of age. *Am J Sports Med.* 2006;34(3):375–384.
24. 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.
25. Noyes FR, Barber-Westin SD, Fleckenstein C, Walsh C, West J. The drop-jump screening test: differences in lower limb control by gender and effect of neuromuscular training in female athletes. *Am J Sports Med.* 2005;33(2):197–207.
26. 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.
27. Benjaminse A, Habu A, Sell TC, et al. Fatigue alters lower extremity kinematics during a single-leg stop-jump task. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(4):400–407.
28. 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.
29. 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.
30. 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 Exerc.* 2007;39(3):502–514.

31. Orishimo KF, Kremenich IJ. Effect of fatigue on single-leg hop landing biomechanics. *J Appl Biomech.* 2006;22(4):245–254.
32. 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.
33. Blackburn JT, Padua DA. Influence of trunk flexion on hip and knee joint kinematics during a controlled drop landing. *Clin Biomech (Bristol, Avon).* 2008;23(3):313–319.
34. 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.
35. 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.
36. 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.
37. 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.
38. Sigward SM, Powers CM. Loading characteristics of females exhibiting excessive valgus moments during cutting. *Clin Biomech (Bristol, Avon).* 2007;22(7):827–833.
39. Chandrashekar N, Slaughterbeck J, Hashemi J. Sex-based differences in the anthropometric characteristics of the anterior cruciate ligament and its relation to intercondylar notch geometry: a cadaveric study. *Am J Sports Med.* 2005;33(10):1492–1498.
40. Chandrashekar N, Mansour JM, Slaughterbeck J, Hashemi J. Sex-based differences in the tensile properties of the human anterior cruciate ligament. *J Biomech.* 2006;39(16):2943–2950.
41. Hashemi J, Chandrashekar N, Mansouri H, Slaughterbeck JR, Hardy DM. The human anterior cruciate ligament: sex differences in ultrastructure and correlation with biomechanical properties. *J Orthop Res.* 2008;26(7):945–950.
42. 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.
43. 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.
44. Simon RA, Everhart JS, Nagaraja HN, Chaudhari AM. A case-control study of anterior cruciate ligament volume, tibial plateau slopes and intercondylar notch dimensions in ACL-injured knees. *J Biomech.* In press.
45. Everhart JS, Flanigan DC, 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.* In press.
46. Nguyen AD, Shultz SJ. Sex differences in clinical measures of lower extremity alignment. *J Orthop Sports Phys Ther.* 2007;37(7):389–398.
47. 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.
48. Beynon 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.
49. Rosene JM, Fogarty TD. Anterior tibial translation in collegiate athletes with normal anterior cruciate ligament integrity. *J Athl Train.* 1999;34(2):93–98.
50. 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.
51. Shultz SJ, Sander TC, Kirk SE, Perrin DH. Sex differences in knee laxity change across the female menstrual cycle. *J Sports Med Phys Fit.* 2005;45(4):594–603.
52. 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: a prospective four-year evaluation of 859 West Point cadets. *Am J Sports Med.* 2003;31(6):831–842.
53. Jansson A, Saartok T, Werner S, Renstrom P. General joint laxity in 1845 Swedish school children of different ages: age- and gender-specific distributions. *Acta Paediatr.* 2004;93(9):1202–1206.
54. Rikken-Bultman DG, Wellink L, van Dongen PW. Hypermobility in two Dutch school populations. *Eur J Obstet Gynecol Reprod Biol.* 1997;73(2):189–192.
55. Shultz SJ, Levine BJ, Nguyen A, et al. A comparison of cyclic variations in anterior knee laxity, genu recurvatum and general joint laxity across the menstrual cycle. *J Orthop Res.* In press.
56. Hsu W, Fisk JA, Yamamoto Y, Debski RE, Woo SL. Differences in torsional joint stiffness of the knee between genders: a human cadaveric study. *Am J Sports Med.* 2006;34(5):765–770.
57. 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.
58. Sharma L, Lou C, Felson DT, et al. Laxity in healthy and osteoarthritic knees. *Arthritis Rheum.* 1999;42(5):861–870.
59. Shultz SJ, Shimokochi Y, Nguyen A, Schmitz RJ, Beynon BD, Perrin DH. Measurement of varus-valgus and internal-external rotational knee laxities in vivo, part II: relationship with anterior-posterior and general joint laxity in males and females. *J Orthop Res.* 2007;25(8):989–996.
60. Park HS, Wilson NA, Zhang LQ. Gender differences in passive knee biomechanical properties in tibial rotation. *J Orthop Res.* 2008;26(7):937–944.
61. Schmitz RJ, Ficklin TK, Shimokochi Y, et al. Varus/valgus and internal/external torsional knee joint stiffness differs between sexes. *Am J Sports Med.* 2008;36(7):1380–1388.
62. 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.
63. 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.
64. 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.
65. Shultz SJ, Shimokochi Y, Nguyen AD, et al. Non-weight bearing anterior knee laxity is related to anterior tibial translation during transition from nonweight bearing to weight bearing. *J Orthop Res.* 2006;24(3):516–523.
66. Shultz SJ, Schmitz RJ, Nguyen AD, Levine BJ. Joint laxity is related to lower extremity energetics during a drop jump landing. *Med Sci Sports Exerc.* 2010;42(4):771–780.
67. 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.
68. 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.
69. 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.
70. Ramesh R, Von Arx 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.
71. 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.
72. Hertel JN, Dorfman JH, Braham RA. Lower extremity malalignments and anterior cruciate ligament injury history. *J Sports Sci Med.* 2004;3(4):220–225.

73. Astrom M, Arvidson T. Alignment and joint motion in the normal foot. *J Orthop Sports Phys Ther.* 1995;22(5):216–222.
74. Shultz SJ, Nguyen AD, 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.
75. 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.
76. Arendt EA, Bershadsky B, Agel J. Periodicity of noncontact anterior cruciate ligament injuries during the menstrual cycle. *J Gend Specif Med.* 2002;5(2):19–26.
77. 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.
78. Myklebust G, Engebretsen L, Braekken IH, Skjølberg 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.
79. 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.
80. Wojtyś EM, Huston L, Boynton MD, Spindler KP, Lindenfeld TN. The effect of the menstrual cycle on anterior cruciate ligament in women as determined by hormone levels. *Am J Sports Med.* 2002;30(2):182–188.
81. Dragoo JL, Lee RS, Benhaim P, Finerman GA, Hame L. Relaxin receptors in the human female anterior cruciate ligament. *Am J Sports Med.* 2003;31(4):577–584.
82. Faryniarz DA, Bhargava M, Lajam C, et al. Quantitation of estrogen receptors and relaxin binding in human anterior cruciate ligament fibroblasts. *In Vitro Cell Dev Biol Anim.* 2006;42(7):176–181.
83. 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.
84. 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.
85. 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.
86. Lemoine S, Granier P, Tiffocche C, Rannou-Bekono F, Thieulant ML, Delamarche P. Estrogen receptor alpha mRNA in human skeletal muscles. *Med Sci Sports Exerc.* 2003;35(3):439–443.
87. Sinha-Hikim I, Taylor WE, Gonzalez-Cadavid NF, Zheng W, Bhasin S. 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.
88. Wiik A, Glenmark B, Ekman M, et al. 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.
89. Shultz SJ, Kirk SE, Johnson ML, Sander TC, Perrin DH. Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc.* 2004;36(7):1165–1174.
90. Shultz SJ, Levine BJ, Wideman L, Montgomery MM. Some sex hormone profiles are consistent over time in normal menstruating females: implications for sports injury epidemiology. *Br J Sports Med.* In press.
91. 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.
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 Womens 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 Relat 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. *Osteoarthritis 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 CY, 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. Komatsuda T, Sugita T, Sano H, et al. Does estrogen alter the mechanical properties of the anterior cruciate ligament? An experimental study in rabbits. *Acta Orthop.* 2006;77(6):973–980.
99. Liu SH, Al-Shaikh RA, Panossian V, Finerman GA, Lane JM. Estrogen affects the cellular metabolism of the anterior cruciate ligament: a potential explanation for female athletic injury. *Am J Sports Med.* 1997;25(5):704–709.
100. Rau MD, Renouf D, Benfield D, et al. Examination of the failure properties of the anterior cruciate ligament during the estrous cycle. *Knee.* 2005;12(1):37–40.
101. Seneviratne A, Attia E, Williams RJ, et al. The effect of estrogen on ovine anterior cruciate ligament fibroblasts: cell proliferation and collagen synthesis. *Am J Sports Med.* 2004;32(7):1613–1618.
102. Slauterbeck J, Clevenger C, Lundberg W, Burchfield DM. Estrogen level alters the failure load of the rabbit anterior cruciate ligament. *J Orthop Res.* 1999;17(3):405–408.
103. Strickland SM, Belknap TW, Turner SA, Wright TM, Hannafin JA. Lack of hormonal influences on mechanical properties of sheep knee ligaments. *Am J Sports Med.* 2003;31(2):210–215.
104. Warden SJ, Saxon LK, Castillo AB, Turner CH. Knee ligament mechanical properties are not influenced by estrogen or its receptors. *Am J Physiol Endocrinol Metab.* 2006;290(5):1034–1040.
105. Wentorf FA, Sudoh K, Moses C, Arendt EA, Carlson CS. The effects of estrogen on material and mechanical properties of the intra- and extra-articular knee structures. *Am J Sports Med.* 2006;34(12):1948–1952.
106. Woodhouse E, Schmale GA, Simonian P, Tencer A, Huber P, Seidel K. Reproductive hormone effects on strength of the rat anterior cruciate ligament. *Knee Surg Sports Traumatol Arthrosc.* 2007;15(4):453–460.
107. Yu WD, Liu SH, Hatch JD, Panossian V, Finerman GA. Effect of estrogen on cellular metabolism of the human anterior cruciate ligament. *Clin Orthop Relat Res.* 1999;366:229–238.
108. Abt JP, Sell TC, Laudner KG, et al. Neuromuscular and biomechanical characteristics do not vary across the menstrual cycle. *Knee Surg Sports Traumatol Arthrosc.* 2007;15(7):901–907.
109. Chaudhari AM, Lindenfeld TN, Andriacchi TP, et al. Knee and hip loading patterns at different phases in the menstrual cycle: implications for the gender difference in anterior cruciate ligament injury rates. *Am J Sports Med.* 2007;35(5):793–800.
110. Dedrick GS, Sizer PS, Merkle JN, et al. Effect of sex hormones on neuromuscular control patterns during landing. *J Electromyogr Kinesiol.* 2008;18(1):68–78.
111. Park SK, Stefanyshyn DJ, Ramage H, et al. The relationship between knee joint laxity and knee joint mechanics during the menstrual cycle. *Br J Sports Med.* 2009;43(3):174–179.
112. Park SK, Stefanyshyn DJ, Ramage B, Hart DA, Ronsky JL. Alterations in knee joint laxity during the menstrual cycle in healthy women leads to increases in joint loads during selected athletic movements. *Am J Sports Med.* 2009;37(6):1169–1177.
113. Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes: decreased impact forces and increased hamstring torques. *Am J Sports Med.* 1996;24(6):765–773.
114. 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.

115. 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.
116. 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.
117. 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.
118. 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.
119. 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.
120. 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.
121. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. 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.
122. 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):490–498.
123. 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: 2-year follow-up. *Am J Sports Med.* 2005;33(7):1003–1010.
124. Olsen OE, Myklebust G, Engebretsen L, et al. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *BMJ.* 2005;330(7489):449.
125. 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.* 2009;18(6):824–830.
126. Graves JE, Martin AD, Miltenberger LA, Pollock ML. Physiological responses to walking with hand weights, wrist weights, and ankle weights. *Med Sci Sports Exerc.* 1988;20(3):265–271.
127. Häkkinen K, Alen M, Kraemer WJ, et al. Neuromuscular adaptations during concurrent strength and endurance training versus strength training. *Eur J Appl Physiol.* 2003;89(1):42–52.
128. Petitbois C, Deléris G. Effects of short- and long-term detraining on the metabolic response to endurance exercise. *Int J Sports Med.* 2003;24(5):320–325.
129. Brown TD, Johnston RC, Saltzman CL, Marsh JL, Buckwalter JA. Posttraumatic osteoarthritis: a first estimate of incidence, prevalence, and burden of disease. *J Orthop Trauma.* 2006;20(10):739–744.
130. Kessler MA, Behrend H, Henz S, Stutz G, Rukavina A, Kuster MS. Function, osteoarthritis and activity after ACL-rupture: 11 years follow-up results of conservative versus reconstructive treatment. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(5):442–448.
131. Lohmander LS, Osterberg A, Englund M, Roos H. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. *Arthritis Rheum.* 2004;50(10):3145–3152.
132. Lohmander LS, Englund PM, Dahl LL, et al. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. *Am J Sports Med.* 2007;35(10):1756–1769.
133. 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.
134. Sward P, Kostogiannis I, Roos H. Risk factors for a contralateral anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(3):277–291.
135. Wright RW, Dunn WR, Amendola A, et al. Risk of tearing the intact anterior cruciate ligament in the contralateral knee and rupturing the anterior cruciate ligament graft during the first 2 years after anterior cruciate ligament reconstruction: a prospective MOON cohort study. *Am J Sports Med.* 2007;35(7):1131–1134.
136. Flynn RK, Pederson CL, Birmingham TB, Kirkley A, Jackowski D, Fowler PJ. The familial predisposition toward tearing the anterior cruciate ligament. *Am J Sports Med.* 2005;33(1):23–28.
137. Harner CD, Paulos LE, Greenwald AE, Rosenberg TD, Cooley VC. Detailed analysis of patients with bilateral anterior cruciate ligament injuries. *Am J Sports Med.* 1994;22(1):37–43.
138. 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.
139. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. *Arthroscopy.* 2007;23(12):1320–1325, e1326.
140. Finch C. A new framework for research leading to sports injury prevention. *J Sci Med Sport.* 2006;9(1–2):3–9.

Address correspondence to Sandra J. Shultz, PhD, ATC, FNATA, FACSM, Department of Kinesiology, University of North Carolina at Greensboro, 1408 Walker Avenue, Greensboro, NC 27412. Address e-mail to sjshultz@uncg.edu.