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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 Wojtys, 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 IV[1] 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 risk[10–15] (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]

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 females[52] 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.
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.

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?

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 sex differences. Information regarding ACL injuries should include type, mechanism, risk factors, reconstruction procedures, and outcomes.
    b. Develop standard operational definitions for ACL injury-incidence and injury-prevalence studies and mechanisms in order to facilitate cross-study comparisons (eg, direct contact, indirect contact, noncontact injury).
    c. Define ACL injury risk factors across different populations: age, maturation, sex, sport, and experience level.
    d. Understand prospective risk factors for ACL reinjury. Are they the same as for the initial ACL injury?
    e. Understand the consequences of ACL injury on future health and other outcomes (lost school time, etc).
    f. Define the direct and indirect costs of sport-related ACL injuries.
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.
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 (TRIPP) 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.

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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.