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The ACL Injury Enigma: We Can't Prevent What We Don't Understand

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Multiple risk factors, both modifiable and nonmodifiable, are known to manifest within the noncontact anterior cruciate ligament (ACL) injury mechanism.[1] I will primarily address neuromechanical contributions to injury risk, which are often a key focus at meetings of this nature; such factors are amenable to training and, hence, largely modifiable. I hope, however, that as the reader progresses through the document, the critical importance of underlying nonmodifiable factors within the resultant neuromechanical strategy will not be lost.

Sexual dimorphism in modifiable neuromuscular factors linked to ACL injury is well documented, with the "female" movement pattern interpreted as riskier. Females, for example, land in a more extended posture,[2] are more quadriceps dominant,[1–3] and demonstrate altered muscle activation and coactivation[4] and greater out-of-plane knee motions[5] and loads[6–9] than males. Neuromuscular training strategies continue to evolve in line with these findings and represent an ever-increasing and equally important research focus.[10–12] Recent epidemiologic data, however, suggest that in spite of these ongoing initiatives and reported early successes,[13,14] ACL injury rates and the associated sex disparity have not diminished.[15] If current prevention methods delivered reasonable efficacy, one would assume that a noticeable reduction in these rates would already be evident. It appears, therefore, that current strategies fail to counter key factors implicated within the injury mechanism. In particular, understanding of the precise contributions of neuromuscular control and resultant biomechanics to the injury mechanism and their integration with nonmodifiable structural and hormonal factors remains limited.

The current lack of insight into the neuromechanical contributions to noncontact ACL injury risk and, thus, how they can be effectively countered appears to arise through several key factors. The remainder of this paper will focus on some of these factors, in the hope that researchers of ACL injury mechanisms and prevention will begin to address them.

Currently, potential neuromechanical predictors of injury risk are generated primarily from the laboratory-based assessment of "safe" movement tasks. Although much can be gained from evaluating high-risk sport postures within a controlled laboratory setting, inferring injury risk from such assessments is questionable. Hence, research that more effectively brings together the laboratory and field environments appears warranted. Sports in which ACL injuries are common are largely governed by a random and often complex series of dynamic events, requiring an equally complex, centrally coordinated response.[16,17] Integrating more sport-relevant factors within the in vivo experimental testing environment may, therefore, provide further crucial insights into the causal factors of noncontact ACL injury, facilitating the development of more effective and adaptable prevention methods. Authors of recent studies have begun to acknowledge this fact by regularly incorporating into the experimental design fatiguing[8,18,19] and decision-making[20–22] tasks, factors inherent in realistic sport participation. Because each of these factors promotes substantial adaptation in the neuromechanical profile and, in particular, exaggerates variables considered high risk, including them when assessing injury predictors is critical. Further, recent data suggest that the combined effect of these tasks may represent a worst-case scenario in terms of injury risk, in which substantial compromise of spinal and, specifically, supraspinal control promotes ineffective decision, response, and resultant movement strategies.[16]

Along with increasing efforts to develop more realistic laboratory testing environments, a similar research groundswell is bringing the laboratory to the field. Such developments not only permit biomechanical assessments during actual ACL injury scenarios but also contribute substantially to the screening and, ultimately, diminution or elimination of high-risk neuromechanical factors. Model-based image-matching techniques, for example, using commercially available software applications, can estimate joint kinematics with reasonable accuracy from videos of actual injury events.[23] Combining these data with neuromuscular measures obtained for similar movements within the laboratory setting may provide helpful insights into the neuromechanical profile of the ACL injury. Other recent developments, such as markerless motion capture techniques[24] and wearable motion sensors,[25] may similarly allow the assessment of lower limb joint mechanics during actual sport participation and, possibly, true injury scenarios. These devices may also provide an excellent method of screening for high-risk, sport-relevant neuromechanical profiles and, further, countering them through dynamic, real-time feedback techniques. These possibilities alone suggest that additional exploration of such methods is warranted.

Current modeling methods, while obviously not representing an actual on-field assessment, also afford an important extension beyond in vivo, laboratory-based experimental methods. The recent development and

validation of participant-specific forward dynamic simulations of high-risk sporting postures, for example, have provided a fast and relatively inexpensive means to study knee joint injury risk while controlling all aspects of the neuromuscular profile.[26] These models permit us to study cause and effect, something that is virtually impossible within the current in vivo laboratory-based paradigm. These models also enable us to explicitly study the effect of specific neuromuscular interventions on resultant knee mechanics.[27] Hence, as authors of experimental studies continue to provide links between neuromuscular control and ACL injury, musculoskeletal modeling techniques will become increasingly applicable and effective.[28]

Another factor that may skew our ability to target and counter realistic neuromechanical contributions to ACL injury risk is the continued and possibly isolated focus on sex. The sex-based disparity in ACL injury rates has understandably precipitated extensive research comparing both modifiable and nonmodifiable factors in males and females. As neuromuscular intervention strategies continue to evolve in line with this work, however, assuming a safe, homogeneous, and typically male-based movement strategy may be extremely problematic.[6] For example, considering that knee joint mechanics are governed by a combination of underlying geometric, laxity, and tissue factors (which themselves demonstrate a degree of sex dependence), the existence of a safe, generic overriding neuromuscular strategy is unlikely. Females have a less round and narrower intercondylar notch than males,[29,30] increasing the risk of ACL impingement.[31] Also, knee joint articular surfaces are 20% to 35% smaller in females,[32] promoting a dangerously small lever arm between the tensile load on the ACL and the compressive load on the lateral condyle during valgus loading. Increases in female femoral anteversion and valgus malalignment may similarly elevate risk.[33] Increased knee joint laxity, which is common in women[34,35] and influenced by hormonal factors,[36,37] also prospectively predicts ACL injury risk.[38] Thus, although it is indeed possible that altered neuromuscular control patterns in females contribute directly to their increased risk of ACL injury compared with males, it is equally plausible that they reflect a compensatory mechanism to accommodate for hazardous joint mechanical variations. If the latter holds true, then simply teaching women to ''move like men'' may be largely ineffective and potentially catastrophic. Hence, the evaluation of joint mechanical contributions to injury risk and the subsequent formulation of an accommodative neuromuscular profile should extend beyond an isolated sex focus, to include an integrative assessment of individual injury predisposition based on readily screenable anatomical and laxity measures. This approach, in turn, will promote more effective neuromuscular training strategies that encourage safe joint loading postures within the context of individual joint vulnerabilities.

In order to consider the integrated effect of structural and neuromechanical contributions to noncontact ACL injury, we need to develop methods that can adequately assess and counter these mechanisms. The potential for using musculoskeletal models to assess neuromuscular contributions to injury risk within a directional (cause-and-effect) pathway has already been highlighted above. Expanding such methods to incorporate an anatomically relevant knee joint,[39,40] capable of accommodating individual variations in knee anatomy and laxity, would provide immediate and much-needed insights into participant-based injury risk predictions. Current technologies indeed make the development of such models a realistic possibility, as demonstrated by Borotikar and van den Bogert (abstract 22). The recent initiation of surrogate modeling methods, coupling forward dynamic musculoskeletal and structurally relevant tissue deformation (finite element) models,[41] will also provide unique and otherwise impossible insights into the interactions among neuromuscular control, joint mechanics, and resultant ligament loading. Exploring such methods will lead to improved predictions of injury risk and the potential for participant-specific screening and prevention modalities and should, thus, be strongly encouraged.

CONCLUSIONS

Considering the continued increases in quantity and likely quality of research directed at noncontact ACL injuries, it seems intuitive that our ability to identify and subsequently counter the mechanisms of this injury should also progress. Current data, however, suggest that this progression may be less than optimal. Obstacles we must overcome to attain a better understanding of ACL injury include inferring risk from standardized, laboratory-based assessments of ''safe'' movement postures; an isolated focus on sex within the study design; and failure to consider the integrated effect of individual joint vulnerabilities on the resultant neuromechanical profile. Existing and evolving technologies should directly assist in our understanding of realistic neuromechanical contributions to ACL injury. Methods that afford increased congruency between the laboratory and the field appear promising and should be strongly encouraged. Further, the continued development of musculoskeletal models that integrate structurally relevant joint and ligament behaviors will enable us to explicitly examine injury cause-and-effect relationships. If these important steps are taken, then elucidating and subsequent preventing neuromechanical contributions to noncontact ACL injury risk will no longer simply be possible but, instead, probable.

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