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Letter to the Editor

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Letter to the Editor

Dear Editor:

We read with great interest the article by DeMorat et al, "Aggressive Quadriceps Loading Can Induce Noncontact Anterior Cruciate Ligament Injury" (March 2004, pages 477-483). In an elegant experiment, 4500 N of quadriceps force was applied while the knee joint was flexed 20°, and ACL injury was detected visually as well as by laxity measurements. We agree with the authors that aggressive quadriceps activation can cause ACL injury during the "falling-back" position in alpine skiing, but we disagree with the authors' conclusion that "the same fundamental principles hold true when addressing non-contact ACL injuries in other sports." There are 3 important differences between the cadaveric protocol used by DeMorat et al and in vivo muscle function during sports movements that must be considered before such a conclusion can be made.

First, DeMorat et al assumed that an athlete can generate 4500 N of quadriceps force while the knee is at 20° of flexion. It is known that maximal quadriceps strength occurs at about 60° of flexion.^{7,8} At 20°, however, the muscle fibers are shortened below their optimal length, and quadriceps strength is at most 20% to 40% of its maximal value,⁸ suggesting that 4500 N of force at such an angle is impossible.

Second, even without strength limitation, a large quadriceps force can only exist when an external force resists knee extension. During the falling-back position in skiing, the external ground reaction force (GRF) is applied to the tail of the ski, with a large lever arm to effectively oppose knee extension. Without skis, however, the lever arm of the GRF vector with respect to the knee joint is much shorter, especially when the knee is near full extension. This finding is consistent with results of in vivo movement analyses of deceleration, cutting, and landing movements, in which the peak extensor moment occurs at much larger flexion angles of 50° to 70°. ^{1,2,10} At these large flexion angles, the patellar tendon is no longer anterior to the tibial axis, and quadriceps force is no longer harmful to the ACL, regardless of magnitude.^{3,5}

Third, the experiment of DeMorat et al was conducted with the flexion angle fixed and the other degrees of freedom left free. This condition implies that knee extension was resisted by a pure moment rather than a GRF, as would occur in vivo. The contribution of this GRF to ACL loading, and thus injury risk, is potentially important. In skiing, it is possible to have a GRF that resists knee extension and pushes the tibia into anterior drawer at the same time.⁴ In other sports, however, consideration of the moment balance at the knee will show that a GRF that resists knee extension must necessarily act posteriorly, rather than anteriorly, on the tibia. Therefore, in nonskiing sports, there are 2 opposing forces acting along the anterior-posterior axis of the tibia: the patellar tendon force, which loads the ACL, and a GRF, which is proportional to the quadriceps force and which unloads the ACL. The presence of the latter will significantly reduce the likelihood that the ACL is injured.

We feel, therefore, that the experiment conducted by DeMorat et al has overestimated the risk of quadriceps-induced ACL injury substantially because (1) the magnitude of applied quadriceps force in this experiment was unrealistically high for the flexion angle chosen, and (2) the protective effect of the GRF, which exists

in sports other than skiing, was neglected. In a computational model that includes these effects, we have shown that ACL injury is not possible because of sagittal plane loading during a sidestep cutting movement.⁹ Valgus load, on the other hand, was predicted to exceed known injury thresholds in certain conditions. This result is consistent with a recent prospective study, which found that dynamic valgus load is a prospective predictor of injury risk but flexion angle at initial contact is not.⁶ We do not exclude the possibility that the quadriceps is an important intrinsic contributor to overall ACL injury risk, but we feel that the results presented by DeMorat et al are not representative of in vivo injury mechanisms in sports movements, and caution should be advised before translating these findings into specific strategies for injury prevention.

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