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. 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, 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°. 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.

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. In other sports, however, consideration of the 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. In other sports, however, consideration of the 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.

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. 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. 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.

Scott G. McLean
Jack T. Andrish
Antonie J. van den Bogert
Department of Biomedical Engineering, Department of Orthopaedic Surgery, Orthopaedic Research Center
The Cleveland Clinic Foundation
Cleveland, Ohio

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Authors’ Response: We very much appreciate the letter by McLean et al regarding our article, “Aggressive Quadriceps Loading Can Induce Noncontact Anterior Cruciate Ligament Injury” (March 2004). It is good to review some fundamental issues in the search for the mechanism of ACL injury. After
reviewing the issues, we feel comfortable with our initial conclusions.

First, McLean et al questioned whether a quadriceps force of 4500 N with the knee at 20° is possible. We used a conservative value of 4500 N for quadriceps force. We actually referred to Huberti et al., who estimated that forces of 6000 to 7000 N were possible. Quadriceps forces of more than 6300 N were deduced by Hoy et al., who incorporated known geometrical and physiological properties of muscle. Recent studies of human muscles show that the much smaller soleus muscle can generate 3330 N of force.5

Most estimates of muscle forces are obtained during isometric or isotonic conditions. In sport-specific ballistic movements, we have shown that activation as measured by integrated electromyography may be more than 200%, which might double muscle forces. In addition, high-speed eccentric action has the potential to create even more muscle forces. Zernicke et al9 actually captured a biomechanical analysis of a weight lifter who ruptured a patellar tendon while being analyzed by cinematography. Through kinematic modeling, the researchers determined that the force in the patellar tendon of an 82.2-kg lifter was actually 14 500 N at the moment of injury. McLean et al10 argue that maximum quadriceps strength occurs at 60° of flexion. Studies by Kulig et al11 do not show this result at all. Most classic studies of muscle strength measure joint torque, and knee extensor torques are certainly declining by 20° of flexion. However, even if muscle force were constant, the torques would be declining near extension because of the geometrical properties of the joint and its moment arm rather than because the actual muscle force is less. Lieber and Friden do point out that “both joint and muscle properties” must be considered. For example, human wrist extensor muscles generate maximal force in wrist extension where the muscles are shortest. Even though the muscle fibers are short, the muscle sarcomeres are in an optimal position for force production. Clearly, it is not possible to infer that joint torques and muscle forces can be assumed to be the same. Our article argues that the actual muscle force in the patellar tendon provides the anterior tibial shear force.

Third, McLean et al note that a large quadriceps force only exists when an external force resists knee extension. During landing of a high-speed stop-jump maneuver, ground reaction force applies a posteriorly directed component and an upwardly directed component on the tibia. It is exactly these ground reaction forces that result in such a large quadriceps activation near the time of heel strike when the knee is near extension. McLean et al refer to their computational model, which claims to show that sagittal plane loading cannot injure an ACL but that valgus load alone predicted ACL injury.7 This forward dynamic model will certainly be useful. We are not surprised that random variations of a relatively normal sidestep cut do not predict ACL injury. Anterior cruciate ligaments are usually injured in awkward, off-balance, or unanticipated conditions. We are, however, surprised that the authors concluded that valgus load perturbations alone are the predictor for noncontact ACL injury, which is apparently inconsistent with clinical observations of noncontact ACL injured knees. We feel strongly that the validity and reliability of this model are not powerful enough to corroborate conclusions about the mechanism. We also disagree with the way the model and other publications use the term valgus. Orthopaedic surgeons generally consider that valgus forces or alignment of the knee tend to force the tibia to rotate away from the midline of a sagittal axis through the center of the knee. In motion analysis studies, knee flexion and hip internal rotation give an apparent valgus angulation to the knee in the coronal plane that does not necessarily involve any rotation around the sagittal axis of the knee.

We thank McLean et al for focusing our attention on their questions. It is likely that a combination of forces cause the ACL to tear rather than any single force. Our article focused on the possibility of quadriceps forces alone, which are capable of injuring the ACL. Sports medicine practitioners may not be aware of the magnitude of muscle forces possible or the structure of the knee joint, which allows muscle forces to strain the ACL maximally near 20° of knee flexion.

REFERENCES