ACL Rupture by Tibial Compression: A Primer

Maerz Lab | University of Michigan | 2023

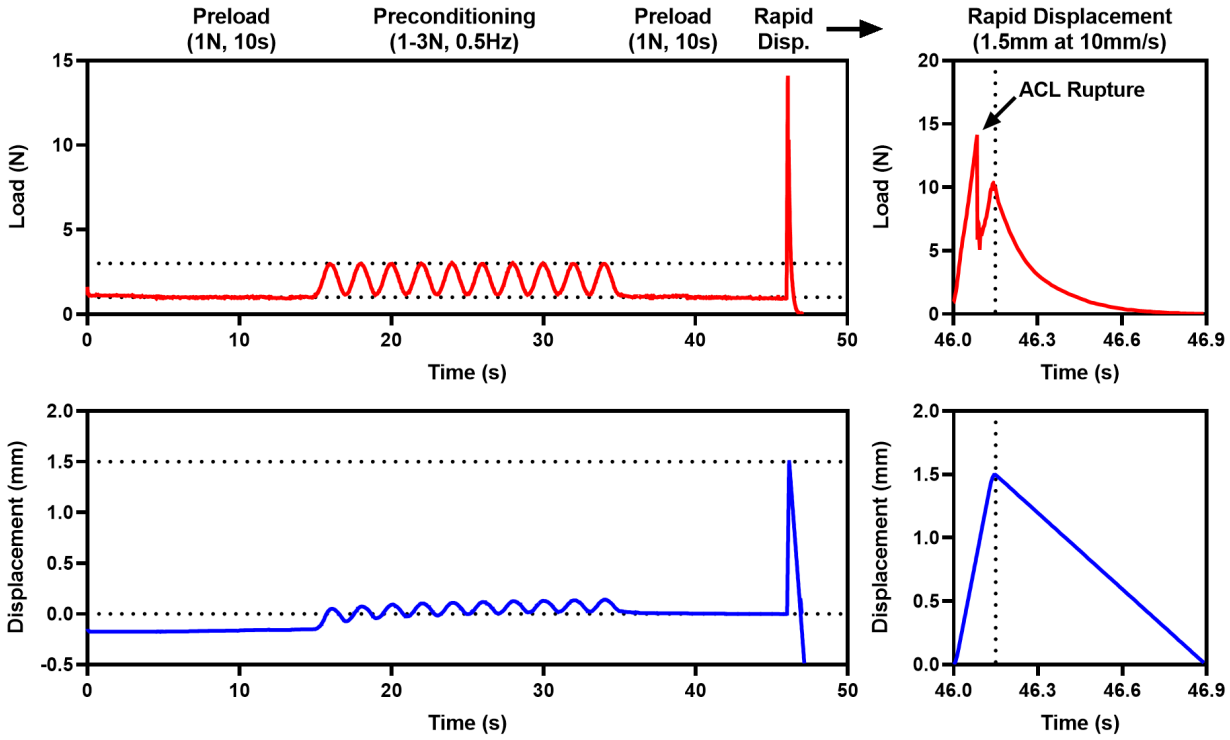

Figure 1. *Full mouse ACL rupture mechanical protocol (left), rapid displacement and ACL rupture (right).*

We utilize a model of non-invasive ACL rupture based on mechanical tibial compression, so named because the tibia is compressed while the ankle and knee are held in flexion, causing anterior tibial subluxation – see page 3 for a diagram. Tibial compression-induced ACL rupture was described in mice by Christiansen et al [1], and it was later adapted to a rat model by Maerz et al [2]. The protocol consists of a preload, preconditioning cycles, another preload, and finally a rapid 1.5mm displacement, followed by immediate unloading of the joint. The preload and preconditioning cycles are discussed in more depth on page 2. The rapid displacement causes the tibia to sublux anterior to the femur, stressing and eventually rupturing the ACL. Maerz et al [2] utilized motion capture in rats and demonstrated that the motion profile of this injury recapitulates the major proposed mechanism of clinical, sports-related ACL injury as described by Koga et al [3] (see page 4 of this document for a figure demonstrating the mechanism). *Rapid* displacement is crucial, as slower displacements can result in physeal displacement or avulsion fracture [2], where the ACL fails at the tendon-bone interface rather than midsubstance and pulls a chunk of bone out with it – both of these represent fundamentally different joint injuries and are not representative of clinical ACL rupture. In a perfect world, we might use a displacement strain, relative to the ankle-to-knee length of each individual animal, but this is not practical and the 1.5mm displacement works with a very high rate of success in rats of both sexes and a broad range of ages.

[1] Christiansen et al, Osetoarthrit Cartil 2012, doi: 10.1016/j.joca.2012.04.014
[2] Maerz et al, Ann Biomed Eng 2015, doi: 10.1007/s10439-015-1292-9
[3] Koga et al, Am J Sports Med 2010, doi: 10.1177/0363546510373570

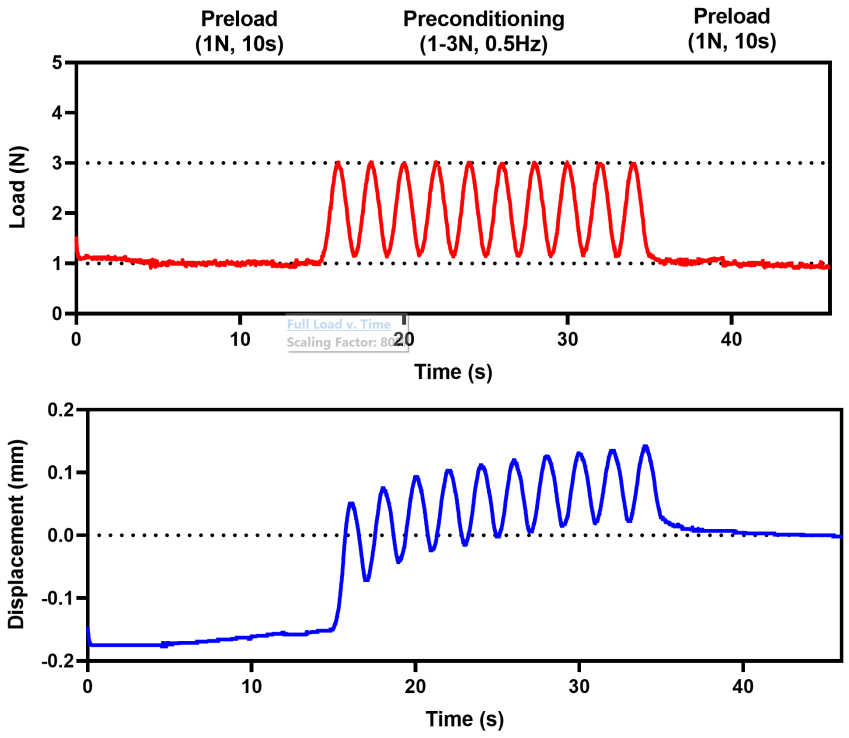

Figure 2. *Preload and preconditioning cycles only.*

Proper preconditioning of the tendon prior to the rapid displacement is important to ensure the tendon is fully elongated at the time of the displacement, increasing the chance of a successful, full thickness ACL rupture. Tendons are viscoelastic tissues, meaning that the rate of loading affects the mechanical response of the tendon. A tendon loaded quickly will behave more stiffly than one loaded more slowly, and a tendon held at a steady load will undergo creep, ie. the tendon will gradually elongate when held at a consistent load. The reasons for this mechanical behavior are very complex and not completely understood, but some combination of water egress and collagen fiber recruitment/alignment are understood to be involved. By applying preload and preconditioning cycles prior to rapid displacement, we can “warm up” or “condition” the tendon and attenuate the “viscous” portion of this response, to the tendon behaves more elastically during the displacement – this can be observed by comparing displacement during the initial preload, where the tendon undergoes considerable creep, to that during the final preload, where an approximate steady-state displacement is reached by the end. We can observe that the joint ends up being more compressed at the end of preconditioning compared to the beginning (with the tendon therefore in tension, due to anterior tibial subluxation). Similarly, during cyclic preconditioning, the tendon can be seen to elongate with each cycle (ie. the displacement increases), however by the final cycles, the rate of this elongation slows and approaches an equilibrium.

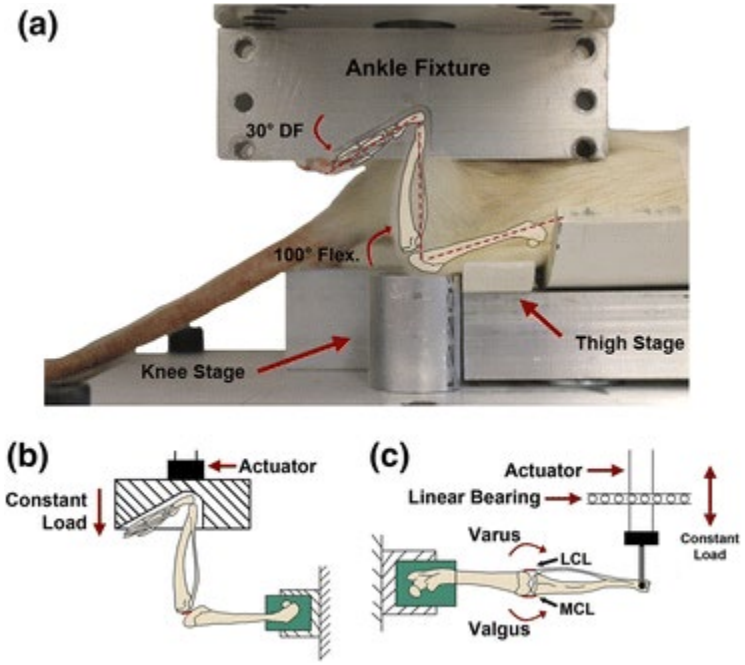

Figure 3. *Tibial Compression Model of ACL Rupture. From Maerz et al [2].*

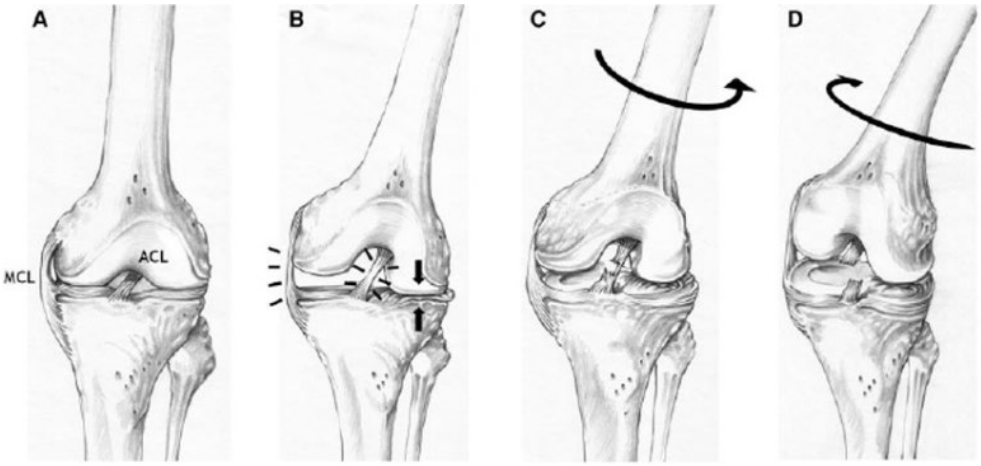

Figure 5. Our hypothesis for noncontact anterior cruciate ligament (ACL) injury mechanism. A, an unloaded knee. B, when valgus loading is applied, the medial collateral ligament becomes taut and lateral compression occurs. C, this compressive load, as well as the anterior force vector caused by quadriceps contraction, causes a displacement of the femur relative to the tibia where the lateral femoral condyle shifts posteriorly and the tibia translates anteriorly and rotates internally, resulting in ACL rupture. D, after the ACL is torn, the primary restraint to anterior translation of the tibia is gone. This causes the medial femoral condyle to also be displaced posteriorly, resulting in external rotation of the tibia.

Figure 4. Mechanism for clinical, non-contact ACL injury. From Koga et al [3].