INTRODUCTION:
Altered mechanical loading due to injury of one structure of a joint may cause physiological and biomechanical changes to complementary structures in the joint. These changes may be even greater when the complementary structure is also injured. Anterior cruciate ligament (ACL) deficiency has been shown to cause biomechanical changes in the un-injured and injured medial collateral ligament (MCL) [1,2]. Even without direct injury to the MCL, ACL-deficiency resulted in increased MCL laxity and decreased ultimate tensile strength (UTS) after 14 weeks of this altered joint loading in a rabbit model [2]. Concurrently, a 13-fold increase in the blood flow to the MCL was observed comparing the ACL-deficient to ACL-intact knees [2]. When a gap-defect injury to the MCL accompanied the ACL-deficiency, increased MCL laxity and decreased UTS were observed after 14 weeks even though the failure loads of the healing MCLs from the ACL-intact and ACL-deficient knees were similar [1]. In addition to ACL-deficiency, posterior cruciate ligament (PCL) deficiency has also been shown to have a physiologic effect on un-injured MCLs [3]. While ACL-deficiency resulted in a 13-fold increase in blood flow, PCL-deficiency resulted in a 4-fold increase in blood flow [3]. Additionally, PCL-deficiency increased the water content of the un-injured MCL. In this study, we report on a small subset of rabbits (5 of 52 post-operative rabbits) where PCL rupture was observed at the time of the mechanical testing but 14 weeks earlier, at the time of the MCL gap-defect surgery, the knee had been stable. Our purpose in this study was to determine whether MCLs healing from a gap injury in a joint that became PCL-deficient at some time during the 14 weeks after creation of the MCL injury had inferior mechanical properties to MCLs healing from a gap injury in a PCL-intact joint. Our hypothesis was that MCLs healing in a PCL-deficient joint would have increased MCL laxity and UTS, similar to the response in ACL-deficient joints in the same model.

METHODS:
Twelve one-year-old female New Zealand White rabbits were used in this study approved by the institutional animal care committee. At the time of surgery, knee joints were manipulated to check for instability and none was detected. Then, animals underwent surgery to create a gap-defect in the midsubstance of the MCL of both hindlimbs [4]. Fourteen weeks after the MCL surgery, hindlimbs were harvested and frozen until tested. Upon dissection, knee joints determined to have a complete rupture of the PCL were assigned to the PCL-deficient group (n=5). The remaining limbs were assigned to the PCL-intact group (n=7). While the exact timing of this spontaneous PCL rupture is unknown, healing MCLs were exposed to joint instability for some portion of their 14 week post-operative period.

Knee joints were dissected without removing the menisci and ligaments. The ends of the ruptured PCL were not removed at this time. Knees were mounted at 70° of flexion in a mechanical testing system (MTS Systems Corp, Minneapolis, MN) with the long-axis of the MCL aligned with the direction of tensile loading. The joint underwent two cycles from -5N to +2N to determine joint laxity as the displacement between those two loads. Then, menisci, lateral collateral ligament, ACL and PCL were removed to isolate the MCL. Two additional cycles from -5N to +2N were performed to determine MCL laxity as the displacement between -0.1N and +0.1N. After stabilizing the environment chamber at 37°C and 99% relative humidity, the MCL underwent 30 cycles at 1Hz from +1N to a force corresponding to 5%UTS of 14-week MCL scar. Next, the MCL was elongated to failure at 20mm/min. The maximum force recorded during the failure test was normalized to the MCL cross-sectional area and failure force were not different between the stable and unstable joints. The MCLs that healed in a PCL-deficient joint had significantly lower UTS than the MCLs that healed in a PCL-intact joint (Figure 3; p=0.02).

RESULTS:
The joint laxity of PCL-deficient joints was greater than that of PCL-intact joints (Figure 1; p=0.007). The MCL laxity of healing MCLs from PCL-deficient joints was not statistically different than the MCL laxity of healing MCLs from PCL-intact joints (Figure 2). Healing MCL cross-sectional area and failure force were not different between the stable and unstable joints. The MCLs that healed in a PCL-deficient joint had significantly lower UTS than the MCLs that healed in a PCL-intact joint (Figure 3; p=0.02).

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