**CHRONIC CHANGES IN THE RABBIT TIBIOFEMORAL JOINT FOLLOWING A SINGLE BLUNT IMPACT**

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**Introduction:** Currently, based on human cadaver experiments an automotive occupant safety criterion restricts compressive axial loads in the tibia below a fracture force level. These experiments document fractures in the knee at approximately 11 times body weight when the tibia and femur are artificially constrained to prevent anterior motion of the tibia during axial tibial compression. For the un-constrained knee, ACL failure occurs at approximately 60% of the bone fracture tolerance level. Clinically “bone bruises” are documented in knee MRIs in over 80% of patients with ACL tears, suggesting significant joint compression during the acute injury. Biopsies of articular cartilage overlaying these bone bruises show fissures and sequelae, also indicative of excessive, acute joint compressive loading. The objective of the current study was to develop an in vivo animal model to study chronic changes in the tibiofemoral (TF) joint following a single blunt force compressive loading, such as that generated during ACL rupture.

**Methods:** A single blunt impact was delivered to the TF joint of skeletally mature Flemish Giant rabbits using a gravity accelerated mass. The studies were approved by an All-University Committee on Animal Use and Care. The right leg was positioned so that the mass struck the distal femur with the knee flexed 90º with the impact force oriented axially in the tibia. Impacts were delivered with a 4 cm square deformable interface (1.2 MPa crush strength) to help insure uniform loading over the joint. Preliminary studies suggested that a 1.33 kg mass could be dropped from 70 cm without causing bone fracture in the knee or tibia of even non-impacted control rabbits. Impacted animals were used in this study. Impacted and control animals were exercised on a treadmill at 0.3 mph, five days a week for 10 minutes a day, using an established protocol. After 12 months and following gross examination of the joint tissues, the tibial plateau cartilage was stained with ink and photographed. The mechanical properties of the cartilage were then measured at four locations across the medial and lateral facets with related indentation tests. The gross photographs and histology sections were analyzed with software (SigmaScan) to measure the total length of surface fissuring on the cartilage. Finally, the joints were fixed, decalcified and prepared by normal histological procedures with a Saffranin O stain. Histology sections were quantified by three blinded readers. The thickness of cartilage and subchondral bone (SB), and the porosity of trabecular bone (TB) was also quantified (Fig 1).

**Results:** The impact load generated on the TF joint was 15.8 ± 1.5 x BW with a time to peak load of 6.6 ± 0.6 ms. No gross fractures of bone were observed. Gross photographs showed more fissuring medially and laterally on impacted versus control and non-impacted joint surfaces. The modiolus, impacted surface had 94.1 ± 29.0 mm of fissuring compared to 84.8 ± 22.0 mm of fissuring on the non-impacted limb, and 78.2 ± 28.0 mm in controls. Laterally, the impacted surface had 61.5 ± 16.5 mm of fissuring compared to 54.8 ± 17.4 mm and 52.9 ± 14.7 mm on the non-impacted and control facets, respectively. The histology sections indicated that, compared to controls, the impacted limbs had significantly (p<0.05) more tissue disruptions, as characterized by vertical and horizontal splits in the zone of calcified cartilage (ZCC), horizontal cracks located near the ZCC/SB interface, or compression damage beneath the articular cartilage surface (Fig 2). In addition, the contralateral, non-impacted joints also demonstrated a significant increase (p=0.04) in tissue disruptions compared to control joints. The modulus of impacted joint cartilage was also reduced by approximately 12% versus controls in uncovered areas on the tibial plateau, but these results were not statistically significant in this study. There was also no tendency for changes in the mechanical properties of covered areas after blunt loads. While only a slight thinning was evident in the impacted cartilage, the subchondral bone plate was 15% thicker than controls and 16% thicker than that of the non-impacted limb in the lateral compartment. The underlying trabecular bone, after impact loading, was also 5% more porous than controls, but had the same porosity as the non-impacted limbs. However, none of these bone morphology measures rose to a level of statistical significance in this study.

**Discussion:** The impact load generated in this study was approximately 50% of that required for bone fracture in constrained joints, and lower than that needed to produce an isolated ACL rupture (16.5 ± 5.2 x BW) in the rabbit model (data not shown). And yet one year after a single incident of joint compressive loading, the loaded joints showed significant fissuring and a slight mechanical softening of tibial plateau cartilage with trends towards changes in the underlying subchondral and trabecular bone. These post trauma changes in the TF joint tissues were consistent with clinical joint OA, which often develops following ACL rupture.

Previous studies by this laboratory also indicate that severe blunt impacts to the rabbit PF joint can result in acute fissures on the patellar cartilage with its subsequent mechanical softening and later thickening of underlying subchondral bone. The current study showed similar findings, but the sequence of these events was not investigated. In an earlier study, by another laboratory, the rabbit TF joint was subjected to physiological levels of cyclic loading and showed subchondral bone thickening that preceded changes in the overlying cartilage. In the current study impacted and non-impacted contralateral joints were also compared with un-impacted, control joints from separate animals. There was evidence of an increase in joint tissue disruptions in both the impacted and non-impacted joints versus controls, suggesting ipsilateral joint trauma influenced joint tissue homeostasis in the opposite, un-impacted limb in this animal model. The effect was not observed for severe, ipsilateral blunt trauma to the rabbit PF joint. These data will be important as we continue to study this model for the investigation of acute responses of the TF joint to compressive loads generated during ACL rupture.

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**References:**


**Figure 1.** Histology section showing subchondral bone thickness and trabecular bone porosity measurement.

**Figure 2.** Horizontal splitting at tidemark with compression ridges on left and splitting and cracking through the cartilage matrix.