INTRODUCTION:
The natural history of isolated, traumatic, focal chondral defects in the knee is not completely understood. While progression of a chondral defect to osteoarthritis is multifactorial [1], many cartilage surgeons and researchers agree that unrepaired, full-thickness chondral defects progress to osteoarthritis by biomechanical overload of the defect rim with subsequent adjacent cartilage degeneration, subchondral bone (SCB) changes, and opposing articular surface overload [2].

Although several factors must be considered when deciding upon the most appropriate treatment for patients with articular cartilage lesions of the knee, defect size is a critical element. However, the evidence to support one cartilage repair or restoration technique over another based on defect size is inconclusive. Most current algorithms use 2 cm² as the threshold between marrow-stimulation techniques (MST) such as microfracture or drilling and cartilage restoration techniques such as autologous chondrocyte implantation (ACI), osteochondral autograft (OATS) or mosaicplasty, and osteochondral allograft [3,4]. Little biomechanical or clinical evidence exists to support the use of 2 cm² as the critical area beyond which stresses become damaging to adjacent cartilage and underlying bone.

The purpose of this study was to determine how defect size influences SCB contact within the defect. We hypothesized that the threshold at which significant subchondral bone contact occurs would not be 2 cm² and that SCB contact would be different for the medial and lateral femoral condyles due to the geometry of the articulating surfaces.

METHODS:
Full-thickness, femoral condyle chondral defects were created in 8 (4 left and 4 right) healthy, fresh-frozen bovine knees. Knees with cartilage softening, fissuring, fibrillations, or full-thickness defects were excluded. Once thawed, all skin and subcutaneous adipose tissue about the knee were removed. A medial and lateral parapatellar arthroscopy was made and the extensor mechanism was removed for joint access. Anterior tibialis and popliteus were removed. The menisci were removed at their respective anterior and posterior horn insertions. The cruciate (ACL, PCL) and collateral (MCL, LCL) ligaments were kept intact. All remaining soft tissue was dissected free from the distal femur and proximal tibia.

The knee was then positioned in full extension within a fabricated stainless steel frame and locked in a uniaxial material testing system (MTS-858 Bionix Test System [Eden Prairie, Minnesota, USA]). Coronal plane alignment was meticulously adjusted as necessary to prevent any eccentric valgus or varus alignment. K-Scan digital electronic pressure sensors (Tekscan, Boston, Massachusetts, USA) were placed between the articular surfaces of the femoral condyles and tibial plateau and were calibrated by applying a 1,000N load. The sensors were then removed, a 1,000N load was applied, and the femoral condyle contact areas were mapped using a surgical marker. These areas were bisected with use of an electronic digital caliper in the sagittal and coronal planes to locate the center of the intended defect.

Defects with areas between 0.2 cm² and 5.07 cm² were created with a set of mechanical cylindrical coring punches with diameters between 5 mm and 25.4 mm. After identifying the intended defect area, the femoral fixation apparatus was removed from the MTS to allow access to the articular surface, and a 0.2 cm² defect centered on the intersection of the lines bisecting the coronal and sagittal planes was created with the 5-mm punch. The SCB was not penetrated with the device and any leftover tissue was removed with a curette to ensure a vertical perpendicular defect rim. The specimen was then re-mounted on the MTS, and the Tekscan K-Scan sensors were reinserted into the joint. The knee was preloaded to 20N and then loaded at a rate of 20 N/s to 1,000 N, where it was held for 30 seconds and pressure readings were recorded. After the load returned to zero, the specimen was then unloaded from the MTS and the defect was concentrically enlarged until reaching a maximum size of 5.07 cm². A custom MATLAB program was used to compute the areas within the defect demonstrating SCB contact.

After detecting unequal variances in the SCB contact areas for the different defect sizes, we used the Kruskal Wallis test to evaluate the effect of defect size on the resultant SCB contact area, without distinguishing between medial and lateral femoral condylar defects. Post-hoc comparisons were made using Dunnett’s T3 test. Wilcoxon tests were performed to determine the difference in SCB contact area between medial and lateral defects. All data was analyzed with use of SPSS software, Version 16.0.1 (SPSS Inc., Chicago, Il).

RESULTS:
The mean SCB contact area for all defects larger than 1.99 cm² were significantly different from zero contact (p<0.006). No SCB contact was demonstrated in defects smaller than 1.27 cm². The mean SCB contact area of the 1.27 cm² and 1.61 cm² defects were not significantly different from zero contact (p=0.755 and p=0.297, respectively). The mean SCB contact area of the 1.99 cm² defect was significantly different from defects with no contact (p=0.008), but not from defects of 1.27 cm² (p=0.992).

Across all defect sizes, higher SCB contact occurred on the lateral condyle compared to the medial condyle (p=0.001) with an overall contact area increase of 65% in lateral defects (Figure 1). SCB contact was also significantly more variable on the lateral side (p<0.001).

DISCUSSION:
Clinically, the threshold for using different cartilage repair techniques has varied. Previously, lesions over 2 cm² were thought to be best treated with techniques other than microfracture, such as ACI, OATS or osteochondral allografts. Clinical studies have shown that microfracture may be still a valuable option up to 4 cm² [5]. The current study provides evidence for the use of a 2 cm² threshold size for treating chondral defects, as subchondral contact occurred in defects larger than 2 cm². Contact pressures greatly increased in defects of 2.87 cm². This finding may support why lesions over 2.87 cm² may have higher chances of failure with microfracture, as the subcondral bone contact pressures increase, which can cause further bone reaction and potentially subcondral thickening and sclerosis. This is the proposed mechanism for a three times greater incidence of failure of ACI after previously failed MST [6]. However, lateral defects experience greater and more variable SCB contact than medial defects and may progress more rapidly than medial equivalents, suggesting that 2 cm² may not be the appropriate size threshold on the lateral side.

This work was performed in a series of bovine knees under static loading, and the results may be different with differently sized knees and under dynamic loads. Future work should explore the relationship between the size of chondral defects and the size of the weight-bearing contact area to SCB pressure under static and dynamic loads and whether that relationship can be used to predict the progression of the defects in a series of prospective clinical studies.

REFERENCES: