INTRODUCTION:
Several mechanistic theories have been developed to describe bone modeling and remodeling to describe bone adaptation mechanisms. However, there is a lack of well-formed experimental models with minimal co-morbidity effects to validate these theories. The cortical defect healing in intact long bone is not yet well understood. In addition, the repair process seemed to follow a unique pathway [1] [2] and such process often remains incomplete with no effect on long bone structural strength [3]. We proposed that cortical defects repair follows a two-phase process. The initial phase is marked by an angiogenic response, characterized by micro-vascular invasion into the defect area followed by an intramembrance ossification process. The second phase is related to remodeling by transforming the woven bone into cortical bone. We hypothesized that the deformation gradient around the defect has a direct effect on vascular network and woven bone formation during the repair phase. In the remodeling phase, we assumed that bone material property and morphology are regulated by minimizing the strain energy gradient in bone around the defect while attempting to recover bone structural strength measured by the sectional modulus of the tubular structure. The objective of this study was to investigate the proposed defect repair and remodel regulating mechanisms using an animal model (canine with mid-tibia cortical defect) and a corresponding numerical simulation model (FEM).

METHODS: Fourteen skeletally matured adult male canines were used for this study. Unilateral longitudinal rectangular cortical bone defects were created in the antero-medial surface of the mid-diaphysis of the tibia. Defect length was equal to the tibial outer diameter (1 OD) and the defect width was 0.25 OD. One dog each was euthanized 1 and 2 weeks after surgery for microvascular analysis. The remaining 12 dogs were euthanized at 6 each at 4 and 16 weeks, respectively. The protocol was approved by Institutional Animal Care and Use Committee. A vascular corrosion casting method was used for the microvascular analysis. The specimens were coated gold for SEM study. Routine microradiographic biomechanical and histological analyses were performed for the 16-week group specimens.

A mid-diaphysis length of 5 OD was used for the analysis. Identical defect was created in the anterior-medial bone surface. Twenty-node reduced-integration brick elements were used in each FEM model. The transversely isotropic properties of the human cortical bone were used for the engineering constants. The defect had the same transversely isotropic material properties and only the elastic modules were changed to simulate the healing stages. The distal end was rigidly fixed and the proximal edge had coupled constraint under torsional load. ABAQUS CAE (Hibbitt, Karlsson & Sorensen, Inc., Pawtucket, RI) was used to create the FEM models for analysis and pose-process the results.

RESULTS:
There was dense micro-vascular formation at 1 week (Fig. 1) and it was transformed into woven bone in 2 weeks (Fig. 2). Capillaries formed from the medullary canal to the bone defect where the deformation gradient was maximal. At 16 weeks, the torsional strength was completely recovered as compared with the intact bone but with entirely different morphologic architecture (Fig. 3).

The stain energy density was distributed equally in the intact model, however, that of the defect model was increased dramatically in the surrounding of the defect part in comparison with that of the intact model (Fig. 4). The strain energy density surrounding the defect and at its corners were decreased significantly by the repair process. According to the increasing material properties of the repair tissues, the strain energy density decreased gradually around the defects. We applied 1000 N·mm as the torsional load and the torsional stiffness was calculated by dividing the torsional load by the rotational displacement, which also incorporated the "open section" effect. The torsional stiffness of the intact model at the defect center section was 0.248, while the defect model with no repair was 0.198. Under varying degrees of repair and remodeling at 10%, 50%, and 75% of defect recover (only on material modulus), the corresponding torsional stiffness values were increased to 0.238, 0.239, and 0.239, respectively.

DISCUSSION:
The high strain energy density around the defect corner was responsible for the initiation of the more active angiogenic activity in the repairing process. Therefore, the high deformation gradient may play a significant role to induce the vascular formation. In the modeling phase, the strain energy density was decreased while the material property of the repair tissue in the defect improved. However, complete bone cortex recovery may be altered by shape and morphology changes. Therefore, the cortical defect remodeling appears to be regulated by an adaptation mechanism to minimize the strain energy density while maintaining bone sectional modulus in response to functional demands. The torsional stiffness of the defect model was decreased by about 20% in comparison with the intact model, but with only partial increase of material property of the repair tissue, the structural stiffness could be recovered completely especially with new bone formation in the periosteal region. This may be one of the rare conditions where bone will heal with a scar.

REFERENCE:
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CORTICAL DEFECT – A MODEL TO STUDY BONE REPAIR & REMODELING

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Fig. 1: Vascular formation

Fig. 2: Woven bone formation

Fig. 3: Defect repair at 16 weeks (A) 5mm from defect, (B) defect edge, (C) ¼ defect, (D) defect center

Fig. 4: Strain energy density contour in torsion (1) defect, (2) 10% fill, (3) 50% fill, (4) 75% fill, (5) intact

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