Sensitivity of subchondral bone strain to softening in osteolytic and sclerotic regions: A finite element study of a Thoroughbred racehorse with extensive adaptive structural change and fatigue damage

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INTRODUCTION: Condylar stress fracture of the third metacarpal/tarsal bone (MC3/MT3) is a common catastrophic injury in Thoroughbred racehorses worldwide and a major cause of euthanasia. Large cyclic loads and microcrack formation in the distal end of the MC3/MT3 triggers new trabecular bone modeling as well as damage repair through bone remodeling. In some horses, possibly because of overtraining and lack of sufficient rest periods, microdamage accumulates faster than it is repaired through remodeling. Such imbalance results in subchondral bone fatigue injury (SBI) in the parasagittal grooves (PSG) seen as focal osteolysis lesions with standing computed tomography (sCT) imaging. sCT is a practical screening method with high sensitivity for detecting these concerning structural changes[1] that were shown to cause significantly higher PSG tensile strain *ex vivo*.[2] However, an objective nondestructive classification of horses at higher risk of stress fracture remains lacking. CT-based finite element (FE) modeling is widely used for patient-specific virtual mechanical testing and analysis, but the accuracy of such models is contingent on using a suitable density-modulus relationship. Although microcracks are not captured within the resolution of clinical CT, making the sclerotic volume the dominant factor in FE subchondral strain prediction[3], they have been shown to colocalize with sclerotic trabecular bone.[4] We hypothesized that defining compromised elastic modulus in the sclerotic and osteolytic regions improves accuracy of PSG strain prediction with CT-based patient-specific FE models and resolves the underestimation of strain in bones with PSG SBI.
METHODS: A thoracic limb specimen with extensive sclerosis and biaxial typical PSG SBI from a Thoroughbred racehorse that was euthanized because of catastrophic racetrack injury was selected for this study (Fig. 1A). Max principal PSG strain in the medial condyle was previously measured experimentally[2], and an sCT scan of the limb was acquired using the Equina scanner performing at exposure of 160 kVp and 8 mA, with 0.55 mm slice thickness. An electron density phantom with 4 plugs with 200, 800, 1250, and 1750 mg/cm^3 densities was scanned with the same parameters for calibration of CT number. The distal 2.5 inches of the MC3 was segmented in Mimics (v.26), and the sclerotic and focal osteolytic regions in both condyles were then separated (Fig. 1 B-C). Using 3-matic (v.18), 0.25 mm edge length triangular mesh was generated for all surfaces. The 3D FE model was created in FEBio (v.2.3) with 0.25 mm edge length linear tetrahedral elements in the osteolytic and sclerotic regions and 1 mm edge length linear tetrahedral elements elsewhere. Homogenous Poisson's ratio of 0.3 and sCT-based heterogenous elastic modulus was assigned using the density-modulus relationship established by Moshage *et al*.[5] To incorporate the compromised mechanical properties (due to fatigue damage) in the osteolytic and sclerotic regions, 3 different models were made with varying degrees of damage: 1) no damage, 2) 25% and 50% decrease in elastic modulus in the sclerotic and osteolytic regions, respectively, and 3) 50% and 75% decrease in the sclerotic and osteolytic regions, respectively (Fig. 1D). The proximal end of the model was constrained and a 7.5 kN load was applied to the palmar surface of the medial condyle at 60 degrees with respect to the frontal and transverse planes, replicating the experimental loading conditions.[2] 3D geometry of the osteolytic and sclerotic regions were visualized, and FE-predicted max principal medial PSG strain was evaluated.
RESULTS: Segmented volumes showed elliptical and conical shaped osteolytic regions in the subchondral bone in the medial and lateral PSG, respectively (Fig. 1B-C), extending from the linear fatigue cracks visible on the dissected joint surface (Fig. 1A). The sclerotic regions in both condyles had conical shapes propagating proximally from the joint surface in an oblique direction (Fig. 1C). All 3 FE models predicted the largest max principal strain in the PSG similar to previous findings[3] (Fig. 2A), however, the base model with no damage underestimated PSG strain levels compared to those measured experimentally.[2] The model with 25% and 50% damage in sclerotic and osteolytic regions, respectively, resulted in PSG surface strains comparable to the experimental measurements. The model with 50% and 75% damage overestimated PSG strain levels. Additionally, the models with incorporated damage predicted more extensive proximal propagation of high strain levels along the typical fracture path (Fig. 2B).
DISCUSSION: Incorporating a decrease in the elastic modulus of the sclerotic and osteolytic regions due to presence of microdamage/cracks[4] resolved the underestimation of PSG strain, as hypothesized. One limitation of the methodology presented here is homogenous damage incorporation in each region. Future studies should define heterogenous damage based on distance from the joint surface to resolve the artificially high strain created at the interface of these regions. Additionally, more specimens with varying degrees of PSG SBI and sclerotic volumes should be modeled for validation of the approach presented here.
SIGNIFICANCE/CLINICAL RELEVANCE: Finding the proper degree of fatigue damage-induced softening in the sclerotic and osteolytic regions is critically required for an accurate objective patient-specific assessment of MC3/MT3 subchondral strain by the virtual mechanical testing methodology presented here. Such a diagnostic tool is expected to improve identification of racing Thoroughbreds with high risk of stress fracture. This translational knowledge is also expected to advance stress fracture injury prevention in humans.
REFERENCES: [1] Irandoust *et al.* 2023, doi.org/10.1101/2023.11.09.566089; [2] Irandoust *et al.* 2023, doi.org/10.2139/ssrn.4555184; [3] Irandoust *et al.* ORS 2023; [4] Whitton *et al.* 2010, doi.org/10.1016/j.bone.2010.07.019; [5] Moshage *et al.* 2023, doi.org/10.1115/1.4062488.
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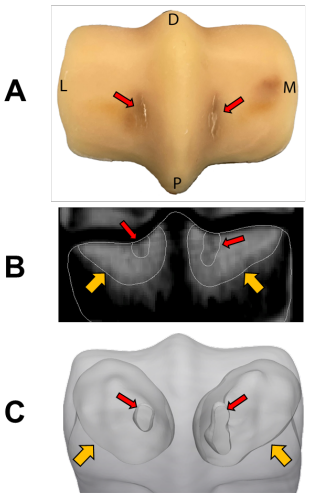

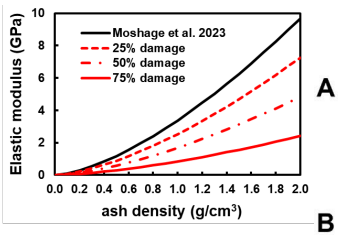

Fig. 1. Joint surface image with palmar/dorsal and medial/lateral sides indicated (A), sCT frontal slice (B) and segmented 3D model (C) of the distal MC3 used in this study. Osteolytic regions in the PSG (narrow red arrows) and sclerotic regions (orange arrows) are shown. A damage factor was defined in these regions (D).

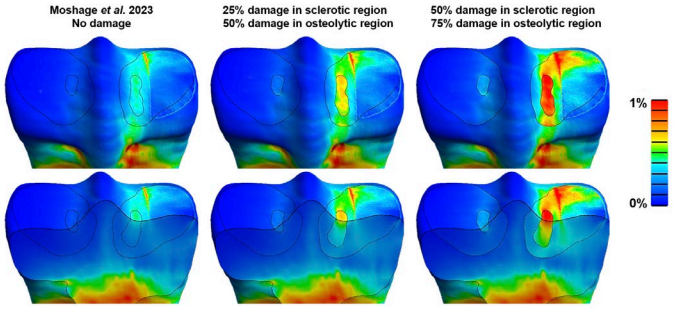

Fig. 2. Finite element prediction of the maximum (most tensile) principal strain in the distal MC3 is shown on the joint surface (A) as well as in a frontal slice (B). Three models were made with varying degrees of damage. The model with 25% and 50% damage in the sclerotic and osteolytic regions predicted surface strains comparable to those measured experimentally. Proximal propagation of elevated strain in the linear path of a typical fracture can be seen (B).