

# QCT-BASED COMPUTATIONAL BONE STRENGTH ASSESSMENT UPDATED WITH MRI-DERIVED 'HIDDEN' MICROPOROSITY

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## Introduction

Microdamage accumulated by cyclic loading or single overloading events contributes to bone fragility through a reduction in stiffness and strength [1]. Quantitative computed tomography (QCT) based computational modelling fails to incorporate *in vivo* microdamage due to limited resolution. Magnetic resonance imaging (MRI) on the other hand, is sensitive to pathophysiological changes to adjacent bone marrow that is 'hidden' to clinical CT imaging. In the case of repetitive trauma, signal hyperintensity in fluid sensitive sequences is indicative of a stress response where edema, haemorrhage and hyperaemia are present alongside microdamage [2]. Here, we aim to quantify this signal hyperintensity and use it to derive a pre-existing damage variable that represents the underlying tissue damage prior to *in silico* overloading. This variable is incorporated into an existing nonlinear constitutive law to investigate its influence on material and whole bone stiffness and strength.

## Methods

We use the equine athlete as a model for microdamage induced stress fracture where high-speed exercise induces subchondral microdamage. The distal metacarpals (MC3) from  $n=5$  Thoroughbred racehorses were scanned by clinical QCT (0.3 mm voxel size), calibrated to bone mineral density (BMD) and converted to bone volume fraction (BV/TV). MR images (T1w, STIR) were acquired at 3T (0.3 mm voxel size) and registered to the QCT data. Regions of 'dense' or 'sclerotic' subchondral bone, where microdamage coalesces [3], were segmented from T1w images (Fig 1a). A patch-based similarity method [4] was used to generate pseudoCT (pCT) images from the STIR images away from the dense subchondral bone in healthy tissue (Fig 1a). A relative increase in STIR intensity in the dense bone region returned a lower pCT-derived BMD than the QCT (Fig 1a). Such signal reflects the presence of underlying porosities such as microdamage and increased vasculature [2]. We derived a damage variable ( $D^{pex}$ ) from the difference of pCT and QCT BMD distributions. Voxel based FE meshes were generated and equipped with an isotropic BV/TV-dependent elasto-viscoplastic material model (UMAT, Abaqus v6.16) [5,6]. We update the material model to incorporate  $D^{pex}$  [7] and we performed *in silico* compression of the MC3 condyles before ( $D^{pex} = 0$ ) and after ( $D^{pex} > 0$ ) such update to investigate its influence on whole bone mechanical properties.

## Results

pCT BMD was lower in all MC3 bones. Incorporating  $D^{pex}$  resulted in a median reduction of material stiffness and strength of 20.3% and 20.9% respectively (Fig 1c).

Neither MC3 size nor volume of sclerotic bone correlated with QCT-only whole bone stiffness and strength.  $D^{pex}$  correlated with a reduction in whole bone stiffness ( $R^2 = 0.74$ ) and strength ( $R^2 = 0.89$ ) (Fig 1d).

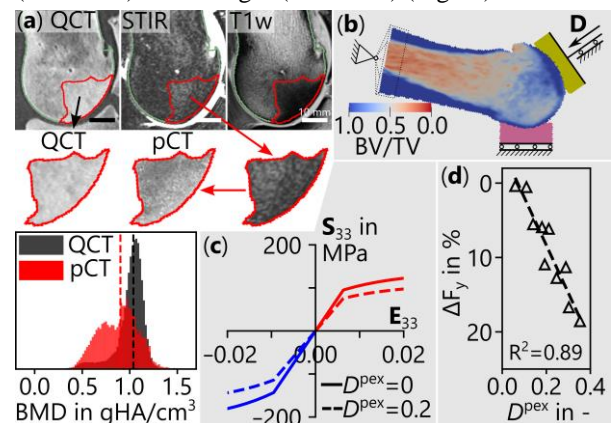


Figure 1. (a) Sagittal images of the distal MC3. Histograms show reduced BMD distribution of the pCT derived from the STIR image. (b) Boundary conditions used in whole bone modelling, superimposed onto a BV/TV map. (c) Uniaxial tensile (red) and compressive (blue) behaviour of QCT-only ( $D^{pex}=0$ ) and  $D^{pex}$  updated model. (d) Reduction in yield force,  $\Delta F_y$ , in %, correlates with  $D^{pex}$ .

## Discussion

We propose a methodology for incorporating MRI signal hyperintensity into a QCT-based FE models. The QCT-only modelling provided limited insights into mechanical properties of the MC3 whereas the detection and inclusion of  $D^{pex}$ , if corroborated by experimental findings, could provide a means to detect when microdamage accumulation overwhelms protective increases in bone mass induced by prolonged exercise. The results illustrate the complimentary value of multimodal imaging to potentially capture existing microdamage *in vivo*. As we use clinically available imaging techniques, our results may aid research beyond the equine model on fracture risk assessment in human diseases such as osteoarthritis and bone cancers.

## References

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