FATIGUE-CAUSED MICRODAMAGE IN TRABECULAR BONE

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Introduction

Bone quality and quantity are the main predictors of the bone mechanical properties, such as strength and fracture resistance. These factors deal with the morphology and chemical composition of bone and can be assessed by DXA, providing the clinical parameters BMD and TBS. These parameters, and in particular the BMD, are currently used as clinical indicators of the fracture risk, but do not provide information on the fatigue life. Yet, bone is continuously subjected to cyclic loading, which can influence the fracture risk. Accumulation of microcracks, and diffuse damage are respectively the consequences of single static overloading event and repetitive physiological (fatigue) loading, that lead to the degradation of mechanical properties. Understanding the different kinds of damages is complex, but fundamental. Formation of microcracks and diffuse damage activate different remodelling activities combined with toughening mechanisms that allow bones to dissipate energy. Bone fractures associated with bone fragility can be either prevented or detected, monitored, and treated. Fatigue-induced micro-damage might be crucial in fragility fractures and falls in the elderly might be the consequences of such fractures [1-2].

Methods

To probe the effect of fatigue-induced damage on bone microarchitecture and elucidate the effect of such damage on the bone clinical parameters, we combined fatigue testing on ex-vivo porcine trabecular bone samples, DXA measurements, and μCT imaging. Besides, we also performed interrupted fatigue testing at different load levels, to highlight the trend of the microstructural parameters during the fatigue life.

Results

Our results suggest that the current invasive and non-invasive diagnosis protocols, i.e. μCT and DXA, respectively, are not able to assess the amount of fatigue-induced damage. This can be due to the fact that such techniques provide global parameters, whereas fatigue-induced damage is a local phenomenon, strictly connected to the microarchitecture. We found a similar fatigue life behavior compared to human trabecular bones. By considering the effective area, which is defined as in Equation 1:

$$A_{eff,i} = A_{n,i} \times BVTV_i \qquad (1)$$

, we could define the effective stress amplitude, $\sigma_{a,eff}$, as in Equation 2:

$$\sigma_{a,eff} = \frac{F_{max} - F_{min}}{2A_{eff}} \qquad (2)$$

Fatigue life curve for the effective stress amplitude normalized with respect to the initial elastic modulus is shown in Figure 1. Our results also showed that the regions with the lowest BV/TV values are the weakest parts of the specimens, where the micro-cracks start to accumulate, leading to mechanical failure, confirming a previous literature study [3]. After the final catastrophic failure, BV/TV increased suddenly in those regions, while its value is almost constant in other parts of the specimen. The local increase in BV/TV, caused by failure, also increased the mean value of BV/TV for each specimen, as observed in our previous study [4]. Our results confirmed that no single diagnosis method can be used for the detection of fatigue-induced damages. The local variation bone volume fraction calculated from micro-CT images suggest a need for the introduction of local sensitive parameters for BMD and TBS rather than an average value.

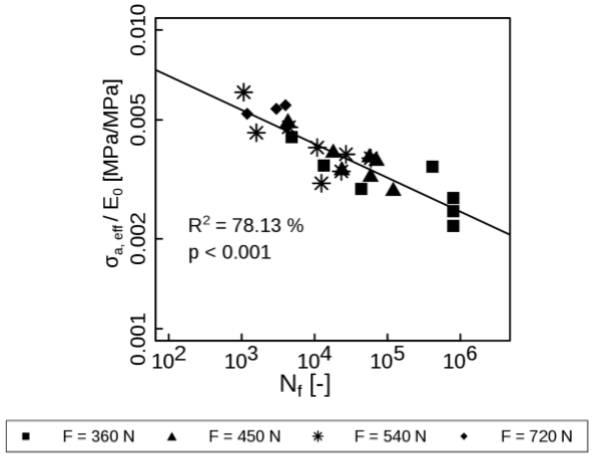

Figure 1: Fatigue life curve for the effective stress amplitude normalized with respect to the initial elastic modulus. The plot shows the following law $\sigma/E_0 = aN^b$ in log-log scale; $a = -0.01$ and $b = -0.1$ are the regression coefficients ($R^2 = 0.66$; p-value < 0.001).

References

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