

## Combined finite element and musculoskeletal predictive structural modelling of the femur: potential mechanobiology applications

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### Introduction

In the 1870's, Wolff [1] formulated a 'trajectory theory' for trabecular bone architecture which can be succinctly written as follows: bone adapts its structure under loading to align with the principal stress directions. Later, Frost [2] introduced the concept of a target strain towards which bone would adapt. In this study, it is assumed that the human femur is optimally adapted to the loading conditions experienced due to a range of activities. An initially randomized structural mesoscale finite element model of the femur was iteratively adapted to reach a target strain when submitted to muscle and joint contact forces derived for a range of activities, using a validated musculoskeletal model [3]. Although the approach is phenomenological, it is expected that it can be extended to study several aspects of mechanobiology, such as osteoarthritis and osteoporosis conditions, through altering the rates of bone apposition and resorption in the converged model.

Physiologically, bone adaptation is made possible by the activity of bone resorption cells (osteoclasts) and bone formation cells (osteoblasts) [4]. In addition to the adaptation to load, osteoclasts and osteoblasts continuously perform an active renewal of the bone tissue, called 'bone remodelling'. The osteocytes embedded within the bone matrix might be responsible for triggering this dynamic process [4,5], in response to the formation of microcracks or bone disuse [6].

The aim of this study was to build a predictive mesoscale structural model of the human femur that would overcome the trade-off between resolution and computational efficiency that arises in continuum modelling approaches. A subsequent aim is to study the changes in the resulting bone architecture when submitted to various bone remodelling rates, representative of osteoporotic or osteoarthritic conditions [5,7].

### Methods

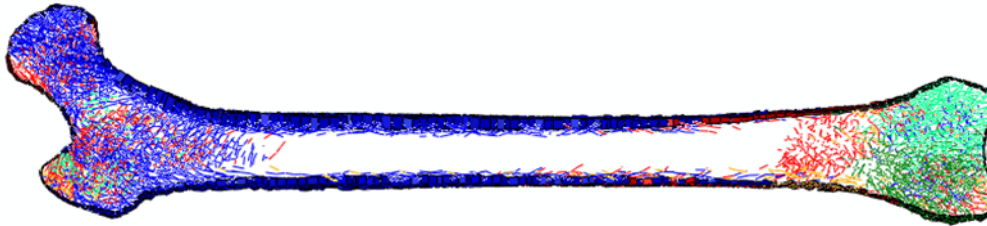
Gait cycles describing walking, stair ascent and descent, sit to stand and stand to sit, were recorded on a volunteer. Inverse dynamics and static optimisation analyses were carried out using a validated musculoskeletal model of the lower limb [3] in OpenSim. Muscle and joint contact forces were computed and loading conditions experienced by the bones of the limb segments were derived for each activity.

A structural FE model of the femur was generated using a web of truss elements within a layer of shell elements, and the cross-sectional properties of these elements were iteratively adapted to reach a target strain under the loading scenarios derived through the musculoskeletal simulations, according to the methodology presented by Phillips et al. [8,9].

## Results

The converged model is presented in Figure 1. A good comparison with clinical imaging is observed in the distribution of the cortical layer thickness. The main trabeculae groups including the greater trochanter, primary compressive and primary tensile groups identified in the proximal femur by numerous studies [10] can also be observed.

Bone elements were mapped according to the activity primarily responsible for their growth. Results are illustrated in Figure 1. They indicate that walking is primarily responsible for the structure of the proximal femur while the architecture of the distal femur is strongly influenced by the activities of sit to stand and stand to sit.



**Figure 1: Mapping of the bone elements according to the activity primarily responsible for their growth (blue: walking; red: stair ascent; yellow: stair descent; light green: sit to stand; dark green: stand to sit)**

## Mechanobiology applications

Bone remodelling will be introduced as a probabilistic process in the converged model. The probability of bone remodelling occurring will include a physiologically informed term related to microcrack formation and a strain-dependant term relating to bone disuse [6].

An imbalance between the rates of bone apposition and resorption will be introduced to model osteoporotic condition [5]. Changes in the architecture of the femur will be observed as well as the evolution of the fracture risk. The influence of the nature of the physical activities performed will be studied. This study will serve as a proof of concept concerning the potential applications of the present structural modelling approach in mechanobiology.

It has been observed that osteoarthritis is related to an increase in bone remodelling activity in a first stage, followed by an imbalance in remodelling rates in favour of bone apposition [7]. These phenomena will be implemented in future work.

As osteocytes shear resulting from fluid flow is of greater amplitude than shear caused by matrix strain [11], the previous studies would benefit from the derivation of the fluid flow generated by bone stress and strain. Given the order of magnitude of the diameter of the canaliculi (small canals within bone elements) compared to an individual trabecular size, continuum poroelasticity approaches may be of interest in such attempts [11].

## REFERENCES

- [1] Wolff, J. et al. (1869), *Clinical Orthopaedics and Related Research*, 468, 1056-1065.
- [2] Frost, H. (1987), *The Anatomical Record* 219:1, 1-9.
- [3] Modenese, L. et al. (2011). *Journal of Biomechanics* 44:12, 2185-2193
- [4] Duncan, R.L. and Turner, C.H. (1995), *Calcif Tissue*, 57:344-358.
- [5] Rachner. T.D. et al. (2011), *Lancet* 373, 1276-1287.
- [6] Huiskes, R. et al. (2000), *Nature* 405, 704-706.
- [7] Burr, D. B. and Gallant M.A., (2012), *M. A. Nat. Rev. Rheumatol.* 8, 665-673.
- [8] Phillips, A.T. M. et al, (2012), *Engineering and Computational Mechanics* 165: 147-154
- [9] Phillips, A.T.M. et al. (2014, *submitted* )
- [10] Singh, M. et al., (1970). *The Journal of Bone & Joint Surgery* 52:3, 457-467.
- [11] Adachi, T. et al. (2010), *Phil. Trans. R. Soc. A* 368, 2669-2682.