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Reduced tissue hardness of trabecular bone is associated with severe osteoarthritis

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ABSTRACT

This study investigated whether changes in hardness of human trabecular bone are associated with osteoarthritis.

Twenty femoral heads extracted from subjects without musculoskeletal diseases (subject age: 49–83 years) and twenty femoral heads extracted from osteoarthritic subjects (subject age: 42–85 years) were tested. Sixty indentations were performed along the main trabecular direction of each sample at a fixed relative distance. Two microstructures were found on the indenting locations: packs of parallel-lamellae (PL) and secondary osteons (SO). A 25 gf load was applied for 15 s and the Vickers Hardness (HV) was assessed.

Trabecular tissue extracted from osteoarthritic subjects was found to be about 13% less hard compared to tissue extracted from non-pathologic subjects. However, tissue hardness was not significantly affected by gender or age. The SO was 10% less hard than the PL for both pathologic and non-pathologic tissues. A hardness of 34.1 HV for PL and 30.8 HV for SO was found for the non-pathologic tissue. For osteoarthritic tissue, the hardness was 30.2 HV for PL and 27.1 HV for SO. In the bone tissue extracted from osteoarthritic subjects the occurrence of indenting a SO (28%) was higher than that observed in the non-pathological tissue (15%).

Osteoarthritis is associated with reduced tissue hardness and alterations in microstructure of the trabecular bone tissue. Gender does not significantly affect trabecular bone hardness either in non-pathological or osteoarthritic subjects. A similar conclusion can be drawn for age, although a larger donor sample size would be necessary to definitively exclude the existence of a slight effect.

1. Introduction

Osteoarthritis (OA) is a common musculoskeletal disorder with considerable morbidity and mortality (Cooper et al., 1991).

OA is generally considered to be a disease of the articular cartilage. It has been hypothesised that the healing of micro-fractures, caused by the overloaded cartilage in the subchondral bone, might increase the bone stiffness in this area (Radin et al., 1972). However, it has been suggested that it might as well be the underlying subchondral bone that plays a significant role in the attenuation of the loads applied to the joint that is first affected by this disease (Fazzalari et al., 1987; Radin et al., 1970). Whichever is the pathogenesis of OA, it has been demonstrated that this disease affects the trabecular bone. In fact, it has been found that both the ratio between mineral and collagen (Brown et al., 2002) and the material density (Li and Aspden, 1997) are lower in OA subjects in comparison with subjects without pathologies. Furthermore, a biochemical analysis of the human femoral head suggests that the trabecular bone matrix in OA patients is subjected to an increased turnover (Mansell et al., 1997) and is metabolically very active (Mansell and Bailey, 1998), compared to non-pathological tissue. The changes in mineralisation and remodelling rate are associated with an alteration of trabecular bone microarchitecture. In particular, OA causes a decrease in trabecular number and an increase in trabecular thickness (Fazzalari and Parkinson, 1998; Perilli et al., 2007), which seem more marked in the weight-bearing region (Neilson et al., 2004). However, it is still not clear whether these effects lead to a similar (Perilli et al., 2007) or a higher trabecular bone apparent density (Li and Aspden, 1997) at a macroscopic scale.

At a nanometric scale bone is a composite material made of a stiff inorganic mineral phase of hydroxyapatite together with a softer organic phase (principally collagen type-I) and water (Currey, 2003; Fratzl et al., 2004).

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