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In situ permeability measurement of the mammalian lacunar-canalicular system.

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Abstract
Bone is capable of adapting its mass and structure under mechanical cues. Bone cells respond to various mechanical stimuli including substrate strain, fluid pressure, and fluid flow (shear stress) in vitro. Although tissue-level strains are well documented experimentally, microfluidic parameters around bone cells are quantified mainly through theoretical modeling. A key model parameter, the Darcy permeability of the bone lacunar-canalicular system (LCS), is difficult to measure using traditional methods due to the co-existence of the larger vascular and smaller LCS porosities. In this paper, we developed a novel method to measure the LCS permeability by rapid compaction of intact mammalian bones and recording the intramedullary pressure (IMP). Six canine metacarpals were subjected to three step compression tests with peak loads of 50, 100, or 200lbs, while the IMP was simultaneously recorded using a catheter pressure transducer. The loading ramp time was chosen to be ~2ms, which was long enough to allow pressure equilibrium to be established between the marrow cavity and the vascular pores, but short enough to observe the LCS fluid flowing into and out of the vascular pores. This loading scheme permitted us to differentiate the contribution of the two intermingled porosities to the IMP responses. The time constant of the IMP pressurization and relaxation due to the LCS was found to be 8.1+/-3.6s (n=18). The mid-shaft cortex of the metacarpals mainly consisted of osteons with an average radial thickness of 65+/-27microm, which served as the characteristic distance for the LCS fluid to relax. The LCS permeability was obtained via poroelastic analysis to be 2.8+/-1.8x10(-)(23)m(2), which was smaller than previous theoretical predictions (order of 10(-)(19) to 10(-)(22)m(2)), but within the range of previous experimentally based estimations (order of 10(-)(22) to 10(-)(25)m(2)). Our results also show that osteoblasts and osteocytes experience hydraulic pressures that differ by three orders of magnitude under physiological compressive strains. These estimates of the in vivo mechanical environments may be used to design in vitro models for elucidating the cellular and molecular mechanisms of bone adaptation and pathological bone loss.

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