Fluid flow, not pressure causes bone resorption in a model for prosthetic loosening
+Fahlgren A., 2Bostom M.P.G, 2Yang X., 1Johansson L., 1Edlund, U., 1Aspenberg P.
1+Linköping University, Linköping, Sweden, +The hospital for Special Surgery, New York, NY.
Anna.Fahlgren@inr.liu.se

Introduction:
Instability after poor fixation of a joint prosthesis is a risk factor for late loosening. Several animal models have shown that micromotion of implants causes local bone resorption [1]. A possible cause of resorption is fluid dynamics. Changes in fluid pressure and flow play a key role in mechanotransduction and are involved in the adaptive response of bone [2]. Short daily episodes of compressing a fluid space adjacent to bone have led to dramatic resorption [3]. However, it has remained unclear which hydrostatic or dynamic conditions lie behind this response. We have previously used a model in which a piston is made to move perpendicular to an isolated bone surface on the proximal rat tibia in a cyclic fashion. The force has been applied intermittently, 20 times during 2 minutes twice daily. By this stimulus, the bone adjacent to the piston is largely resorbed after 5 days.

We have noted that areas of bone resorption are associated to bone cavities that existed at the start of the experiment. Resorption canals are formed from the pressurized space to the surrounding soft tissues outside the bone. These canals seldom form through cortical bone, but through more porous areas, where a high fluid flow can be expected, when a pressure gradient is applied.

Is resorption mainly caused by hydrostatic pressure fluctuations, or by the induced local fluid flow? To elucidate this, we have measured pressure, total flow rate and studied the 3-dimensional distribution of the bone lytic lesions.

Methods:
The rats received a titanium plate at the proximal tibia. A plug was inserted thought the plate. After 5 weeks of osseointegration, the plug was replaced with a pressure piston. The piston was subjected to a transcutaneous force of 8N, which created a velocity of approx. 9 mm/sec. The pressurized area was 6,6mm². Each episode of pressure comprised 20 pressure cycles at 0.17 Hz, applied twice a day. 30 rats were randomized to 3 groups with 10 rats. The rats were killed after 0, 5 or 14 days of pressuring. Immediately before the rats were killed, a pressure transducer was connected to the piston and the fluid pressure under the piston was measured. The mean pressure is standardized as the mean value of the 3rd to 5th episode of pressuring. The maximum pressure is the maximum pressure measured from the 1st episode.
The experimental area under the piston was scanned using the EVS micro-Ct system with a resolution of 39μm. The region of interest was 4 mm diam. Cylinder, 4 mm deep. The centre of the cylinder was placed where the centre of the piston had been (diameter of 3 mm; figure 1).

Bone volume and bone volume fraction (BVF) were measured for each region of interest. The results were analyzed by One-way ANOVA and with Tukey HSD post-hoc test. Institutional guidelines for care and treatment of experimental animals were followed.

Results:
14 days of pressure episodes decreased the bone volume and bone volume fraction with 45% and 31% compared to controls and 5 days of pressure respectively (Table1, Figure 2).

Discussion:
The present study strengthens earlier observation that fluid flow is an important factor in periprosthetic bone resorption [3]. The bone sideways from the pressurized area contains several cavities at the start of the experiment. These cavities became enlarged and confluent, to form a ring of more or less confluent resorption cavities around the pressurized area (Figure 2). For geometrical reasons, the flow must be higher in this region, but the pressure the same. Fluid flow velocity in our model was 50 times higher than calculated lacunocanalicular fluid flow involved in bone homeostasis, and 10 times higher than capillary blood flow [4, 5]. Fluid flow in our model lasted only a tenth of a second, and 20 such episodes twice a day were enough for a massive bone resorption. In patients, it is possible that short loading spikes during heel strike could cause such short high flow velocity episodes.

The magnitude of fluid pressure was in same order that was calculated in lacunocanalicular fluid pressure [4,5], as well as measured during different mechanical manoeuvres in patients with hip prosthesis such as walking and rising from a chair (155-776 mmHg) [6]. Clinical observations contradict the notion of a high fluid pressure as a cause of resorption around loose joint prostheses. Lytic lesions are often not contained, i.e. there is no possibility for static pressure to build, but small, fast motions of a loose prosthesis can create high local flow velocity in the soft interface tissue, independent of containment.

References:

Acknowledgements:
This investigation was supported by the Swedish Research Council, Materials in Medicine and Swedish Society for Medical Research.