**Small minerals make strong bones**

Small can also mean strong. Huajian Gao (Max Planck Institute for Metals Research, Stuttgart, Germany), Peter Fratzl (University of Leoben, Leoben, Austria), and colleagues find that the small size of the mineral particles in bone, teeth, and nacre (mother-of-pearl) is the key to their strength, and that the same principle can be applied to the construction of artificial materials.

Bones, teeth, and nacre are all biocomposites. A protein matrix absorbs and distributes shocks, but its softness is buttressed by hard, embedded minerals. Somehow the resulting combination is a material that can be thousands of times less susceptible to fracture than the pure mineral.

Fracture of the pure mineral initiates at flaws. “It’s inevitable that a mineral will be flawed,” says Gao. “No material is pure. Just from entropic effects you will have impurities.” This is especially true in biocomposites, where proteins will sneak into and disrupt a mineral matrix.

Gao calculated how the fracture strength of such a flawed mineral would vary with changing crystal thickness. After an insult, the amount of energy absorbed by a given crystal depends on the crystal’s volume. Much of that energy will be concentrated at the most serious flaw, perhaps leading to its rupture and thus a catastrophic failure.

As mineral size decreases (left to right) to reach that found in tooth enamel (inset), the fracture resistance increases.

**Selectins with a catch**

Not content simply with a good fit, evolution has apparently designed adhesion molecules that stick optimally only when tugged upon. This property, say Bryan Marshall and Cheng Zhu (Georgia Institute of Technology, Atlanta, GA), and Tadayuki Yago and Rodger McEver (University of Oklahoma, Oklahoma City, OK), and colleagues, may stop leukocytes and platelets from lingering in stagnant backwaters in the blood where there is little or no flow.

Blood is a medium dominated by flow and shear forces. “If you think flow is disruptive, then greater flow should make cells roll faster,” says Zhu. “That’s the case at the high end, but at the low end it reverses.”

The Georgia Tech group found that this effect could be attributed to the behavior of individual adhesion molecules such as P-selectin and its partner P-selectin glycoprotein ligand-1 (PSGL-1). Leukocytes roll along vessel walls when their PSGL-1 binds to the P-selectin found on activated endothelial cells.

At high shear forces, increasing shear leads to decreasing binding lifetime. But the Georgia Tech group found that this “slip” mode was reversed at lower shear forces. In this “catch” mode, found only below a certain limit of shear force, binding lifetime increased with increasing shear force.

For the individual binding events measured, the catch mode shear forces were small—as small as those produced by a few molecular motors. But Zhu feels that the catch mode is relevant in the real world, where the larger shear forces are distributed across many binding events per cell.

The crystal structure of the binding complex does not point to an obvious catch mechanism, although molecular dynamics simulations may yield clues.

Zhu is also interested in exploring the binding energy landscapes. Any bound state is a kinetic trap, so perhaps the force both raises the bottom of this energy well and, even more, elevates the energy barrier blocking escape. Or force might make a short lifetime exit path inaccessible. Whatever the mechanism, the product is a cell that inspects closely only when it has a moving target.

Reference: Marshall, B.T., et al. 2003. *Nature* 423:190–193.