

Nanoparticles pin cracks in a proxy for living tissues

A crack tip is a stress concentrator. Once initiated, it keeps on growing until it has released the whole available energy. A crack in a windshield is an example of a fracture that hardly stops and is stimulated by a wet environment. Biopolymer hydrogels have physico-chemical properties akin to that of soft living tissues, hence are widely used in biomedical applications (patches for drug release, scaffolds for tissue regeneration...). The physiological environment contains many nanoparticles in suspension (proteins, cells, inorganic colloid artefacts...). A team of INSP has shown that silica nanoparticles can interact with a crack very efficiently so as to prevent its growth.

A gelatin hydrogel consists of an elastic network of macromolecules which self-assembles via the formation of triple helices, reminiscent of the native collagen protein. The network, with a mesh size of order 10 nm, is swollen by an aqueous solvent which is the natural route for environmental interactions. Fracture proceeds via helix unzipping and polymer chain extraction. When the crack opens in air, the energy cost for the lack of chain hydration results in a slowing down of the crack. In a liquid environment, chains are rehydrated and the crack speeds up. We have observed that silica nanoparticles of diameters a few 10 nm can slow down a crack and even pin it on the spot. How come?

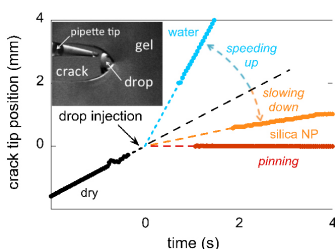


Figure 1

The rate of crack growth in a hydrogel depends drastically upon the environment. Whereas pure water speeds up the crack, the latter is strongly slowed down by nanoparticles (NP) and even pinned on the spot if the concentration is large enough.

Colleagues at ESPCI-ParisTech have proven that silica nanoparticles were able to stick (suture) two pieces of gel or tissue (liver) owing to their huge specific area which promotes adhesion. Curiosity, which has led our INSP group to introduce the same nanoparticles into a crack, enabled us to discover unexpected phenomena. By adjusting the sign of the electric charges borne by the protein network and changing the size of the negatively charged nanoparticles, we have identified two original mechanisms:

1. Whatever their relative state of charge, particles are filtered by the network. They clog it, thereby preventing water from rehydrating the chains. Like an engine with a clogged air filter, the crack slows down.
2. When cracks are of opposed signs, particles adhere strongly to the unzipped chains, thereby restoring cohesion. Here again crack growth is hindered.

Why are these mechanisms, acting at the very end ($< \mu\text{m}$) of the tip, so efficient? The tip end is the seat of a strong depression which pumps water into the gel and advects particles. If their diameter is slightly larger than the network meshsize (as is the case in our study), they are filtered out but kept in place by the depression, forming a clog. Thus, any surface flaw will activate the pump and generate a self-healing clog out of the environment.

This study is a nice case of multiscale process: nanometer sized particles, focussed at the micrometer scale onto the crack tip, are able to control the velocity of a centimeter scale crack. Biomechanical implications on the tearing of soft, collagen-rich tissues (skin, amniotic membrane, vocal folds, arterial walls, tendons...) remain to be assessed.

Reference

« Environmental Nanoparticle-Induced Toughening and Pinning of a Growing Crack in a Biopolymer Hydrogel »
O. Ronsin, I. Naassaoui, A. Marcellan, and **T. Baumberger**
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<https://journals.aps.org/prl/abstract/10.1103/PhysRevLett.123.158002>

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