MATERIALS

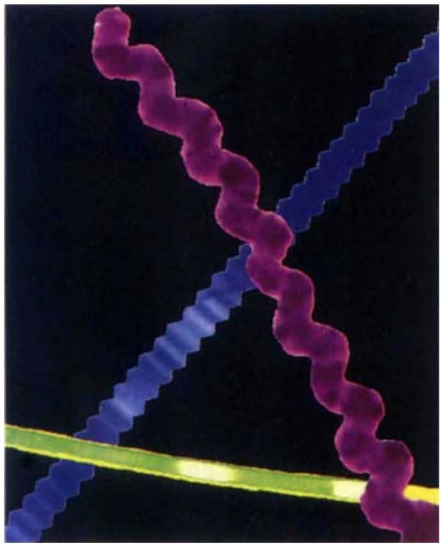
A fine set of whiskers

Philip Ball

THE ancient practice of putting chopped horsehair into plaster is a venerable example of how 'whisker reinforcement' toughens brittle materials. The engineering principle is older still: the paradoxical toughening of composite materials by the incorporation of weak interfaces presumably gave molluscs the same protection from predators millions of years ago as it does today.

Ceramic materials reinforced with fine whiskers of strong materials such as silicon carbide are typically twice as tough as the pure matrix material. Generally these whiskers have diameters of about half a micrometre, but on page 769 Charles Lieber and colleagues show how to make carbide whiskers ten to a hundred times thinner. As the toughness of a whisker-reinforced composite tends to increase with increasing whisker content, these ultrafine whiskers could provide the binder for still tougher composite materials.

The origin of whisker toughening lies in the strategy that it is better to yield a little than to risk catastrophic collapse. High strength in a material, as reflected in the bond energies that hold together the atomic lattice, is not enough by itself to prevent failure through cracking, because the stresses that accumulate at the tip of a propagating crack can be phenomenal. Rather than trying to prevent the progress of a crack tip, it is therefore better to absorb its energy by incorporating into the material weak interfaces that will fail when a crack reaches them. The crack then expends its energy in bringing about localized, small-scale failure rather than brittle fracture. In whisker composites, this dissipation of energy is primarily the consequence of whisker pull-out as the two faces of the crack are pulled apart to expose ever more of the whiskers bridging them.



Three varieties of nanorod as prepared by Lieber and colleagues, each just a few nanometres in width.

The fine whiskers made by Lieber and colleagues might be regarded as fossilized carbon nanotubes: they 'mineralize' the tubes through a reaction between the graphitic shells and volatile oxides or halides of the metallic component. The gases react to form a carbide structure that retains the general dimensions of the tubes; but the reaction proceeds to fill up the hollow tube interiors, turning them into solid rods. These structures are highly crystalline, with the result that they exhibit faceted surfaces. When the fastest-growing crystal facets do not lie more or less parallel to the tube axis, the result is a sawtooth structure, as shown in the photograph, the crimped surface of which would be expected to have a very high pull-out energy in composite materials. Mineralization of helical carbon nanotubes produces helical rods; it cannot be long before someone measures the mechanical properties of these strong 'nanosprings'.

The rods possess the same properties as the parent bulk phase: the iron carbide rods are ferromagnetic, the niobium carbide rods are superconducting, the titanium carbides are metallic. This raises the possibility of engineering intriguing properties into composite materials, for example by varying the nanorod content around the percolation threshold at which a network of metallic fibres forms a continuous conducting pathway through the sample. Such ideas have already been explored in carbon-fibre technology for making smart materials that show a large conductivity response to the appearance of small flaws. □

Philip Ball is an associate editor at Nature.

MULTIPLE SCLEROSIS

Presenting an odd autoantigen

Lawrence Steinman

MULTIPLE sclerosis (MS) is an autoimmune disease involving an integrated attack by T cells, B cells and macrophages on the myelin sheath that surrounds nerve fibres. Potentially inflammatory cytokines such as γ-interferon and tumour-necrosis factor-α are found at the sites of damage, and T cells and antibodies directed against myelin components can be isolated from inflamed regions in the central nervous system. Attempts to identify the components of the myelin sheath that provoke this misguided arsenal have yielded a number of candidates. On page 798 of this issue[1], van Noort *et al.* describe the isolation of a protein that is a prominent target at the disease site in the myelin sheath: this autoantigen turns out to be a small heat-shock protein, αB-crystallin, which is induced in the diseased white matter.

van Noort and colleagues reasoned that there could be an antigen present in the white matter of MS brain that is not found in normal white matter and which specifically activates T cells. They separated the proteins of the myelin sheath using reversed-phase high-performance liquid chromatography and discovered that a particular fraction in the myelin of MS brain, but not in the myelin taken from healthy brain, stimulated proliferation of T cells, causing them to release the pro-inflammatory cytokines γ-interferon and interleukin-2. αB-crystallin was identified as the myelin protein that elicited the strongest immune response. They then showed that αB-crystallin is expressed in glial cells from MS lesions but not in white matter from healthy individuals or in unaffected white matter from MS brain. αB-crystallin was found in oligodendroglial cells as well as in astrocytes in plaques from patients with acute and chronic MS.

These exciting results provide a strong case for αB-crystallin as a target of the immune response in MS, but they raise the question of the precise role of αB-crystallin in the pathogenesis of the disease. αB-crystallin is constitutively expressed in the lens of the eye, myocardial cells and kidney epithelium, and is inducible not only in MS tissue, but also in brains of patients with other neurological diseases, including Alzheimer's, Parkinson's and Huntington's diseases[1]. Degenerative diseases like Huntington's and Parkinson's do not appear to be mediated by the immune system, so it is not clear why αB-crystallin should be immunogenic in MS but not in these other degenerative conditions. One possibility is that the induced expression of αB-crystallin may be a reaction to some neuropathological stimulus that is not unique to MS. In MS additional local events may be required to allow the development of an immune response to αB-crystallin.

To assess the significance of the immune reaction against αB-crystallin in the pathology of MS, the immune response to other myelin components in MS needs to be considered. Myelin basic protein, proteolipid protein, transaldolase and 2′,3′-