

important conclusion of both studies is that the interaction of the magnetic molecules with the surface does not destroy the magnetic behaviour. In particular, in the study by Mannini *et al.* the single molecular magnets were connected to the surface by thiolate-terminated aliphatic chains that allow direct control of the magnetic interaction¹. This bonding was achieved through a wet-chemical synthesis making use of self-assembly effects.

In contrast, the supramolecular network studied by Gambardella and colleagues was constructed using self-assembly by evaporating a molecular precursor layer of terephthalic acid (TPA) followed by deposition of Fe atoms from an electron-beam evaporator under ultrahigh vacuum conditions. Interestingly, both studies

use X-ray absorption spectroscopy at modern third-generation synchrotron radiation facilities to reveal the secrets of their molecules. The X-ray magnetic circular dichroism technique provides the necessary sensitivity to probe the magnetic properties of the molecules in an element-specific manner.

With these techniques it is shown that Fe does indeed connect to the TPA molecules, and thereby the interaction with the Cu substrate is weakened². To support these findings on the electronic structure, the experimental results of Gambardella *et al.* are accompanied by solid density functional calculations and atomic multiplet calculations to model the experimental X-ray absorption spectra². In ref. 1 the experimental spectra reveal that the single molecular behaviour of the

Fe₄ complexes is not destroyed by their connection to the surface, owing to their structural stability and redox robustness. This is a clear advance over well-studied single molecular systems such as Mn₁₂.

In conclusion, both studies demonstrate that the hell that is surface science is not the worst place to be, and that fascinating science can emerge — as long as the place is cooled down sufficiently. □

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SHAPING FATE

The promise of stem-cell therapies for tissue regeneration hinges on the fact that, to a first approximation, stem cells can become any other cell type. But therein lies one of the biggest challenges — for how does a stem cell decide its fate? This decision is generally made in the body through complex biochemical pathways involving diffusing signalling molecules. One approach to stem-cell therapeutics is to manipulate these routes using either natural signalling factors or synthetic small molecules that serve the same role.

But there can be another, perhaps more surprising determinant of stem-cell fate. It may be influenced by purely mechanical means such as stretching or stressing cells, for example by altering the stiffness of the matrix in which they grow. Mesenchymal stem cells (MSCs), which are potential progenitors of many cell types including bone-growing osteoblasts, muscle-making myoblasts and tissue-making fibroblasts, will guide differentiation towards myoblasts in a soft matrix that resembles brain tissue, but towards osteoblasts in a hard, bone-mimicking matrix¹. This suggests a role for materials engineering in stem-cell therapy.

It's been long known that cells can sense and respond to deformation, for example via switchable ion

channels in their outer membranes. But more surprising is the fact that they seem responsive to texture and order. MSCs grown on nanopatterned polymer surfaces have been found to become more osteoblast-like when the surfaces are embossed with random arrays of nanopits, compared with regular, ordered arrays². That raises the prospect of using nanopatterned matrices to define the distributions of cell types in new tissue seeded from stem cells, for example in bone regeneration.

How does this work? Shu Chien and co-workers at the University of California at San Diego now think they have some clues³. They have found a new guiding factor for MSC differentiation that seems to be purely geometric. They grew human MSCs on substrates of aligned arrays of titanium dioxide nanotubes with varying diameter, from 30 to 100 nm, made electrochemically from thin films of titanium. The behaviour of the cells was strongly dependent on the nanotube size: for 30-nm tubes, they adhered well but didn't really differentiate at all, whereas for 100-nm tubes they became long, thin and osteoblast-like.

Elongation is the key. Chien and colleagues saw that the smaller nanotubes became quickly decorated around their open ends with blobs of



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protein: an extracellular matrix deposited by the cells, through which they can adhere to the surface. But these blobs were far less abundant on the wider tubes, simply because there is less space to put them. As a result, cells seeking to anchor themselves have to stretch further in the latter case, and the researchers think that this deformation triggers differentiation to a bone-forming lineage.

That not only suggests a way to guide bone growth by controlling nanostructure; because titanium nanotubes are themselves good candidates for a biocompatible bone-fostering implant material, they can do two jobs at once, providing both support and guidance. □

References

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