by a prostaglandin-independent mechanism[6].

Gordon *et al.* demonstrate astrocyte-mediated dilation of arterioles in solutions containing 20% oxygen, which produces approximately physiological levels of oxygen in brain slices[7]. They also find that astrocyte-mediated constriction occurs in solutions containing oxygen levels well above the physiological (95%). Why has evolution produced the latter pathway, in which neural activity decreases blood flow? And will physiological tissue concentrations of oxygen ever be high enough to activate this pathway? It turns out that 20-HETE-mediated arteriole constriction is inhibited[8] by nitric oxide (NO), a molecule that is released by neurons in response to glutamate secreted by neighbouring neurons (and which can also directly dilate arterioles). The 20-HETE-mediated pathway may therefore be better viewed as a mechanism producing a basal constriction of arterioles that can then be modulated by NO to provide another pathway for activity-dependent dilation.

Future work is likely to focus on how changes in the levels of lactate, adenosine, oxygen and NO interact to coordinate blood flow and hence the brain's energy supply. Some clues can be found in previous data. For example, NO released by neurons inhibits the conversion of arachidonic acid to epoxygenase derivatives that evoke dilation[9]. As NO production in neurons requires oxygen[10], at low oxygen levels this mechanism will be inhibited, promoting dilation. Moreover, oxygen is needed for the synthesis of both constricting (20-HETE) and dilating (prostaglandin $E_2$ and epoxygenase) derivatives of arachidonic acid. At low oxygen levels, however, the production of 20-HETE is inhibited more strongly than that of prostaglandin $E_2$ and epoxygenase derivatives[8], increasing dilation. Finally, in blood capillaries, where contractile cells called pericytes may regulate blood flow[11], lactate causes constriction at high oxygen levels, but dilation at low levels[12]. There is, therefore, an array of switching mechanisms that promote brain energy supply when oxygen levels fall.

In a wider context, Gordon and colleagues' observations raise questions for both cognitive neuroscientists and neurologists. Could the initial dip in local oxygen concentration that accompanies neural activity[13] affect astrocyte signalling rapidly enough to contribute to the increase in blood flow that generates the signals seen in functional imaging of the brain? And could our new understanding of astrocyte signalling lead to better therapies for correcting disorders of blood flow in the brain, such as those that occur after stroke and in vascular dementia?

Gordon *et al.*[1] have opened a fresh chapter in our investigation of how blood flow is regulated in the brain. But their work has a broader implication: physiological studies using solutions bubbled with 95% oxygen may be altering the operation of signalling pathways in the brain, producing misleading results. ■

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MATERIALS SCIENCE

# Deformation of the ultra-strong

Subra Suresh and Ju Li

***In situ* electron microscopy observations of the extrusion of single nanocrystals from graphitic cages show that these crystals deform near their theoretical strength limits. The question is how this happens.**

Many nanostructured materials can sustain specimen-wide stress up to more than a tenth of their ideal strength for a considerable time. For example, a test performed on a monolayer of graphene yielded a strength value very close to its ideal strength calculated by quantum mechanics[1]. But the way that these ultra-strong materials respond to deformation at high temperatures remains mysterious because performing temperature-controlled mechanical tests of nanostructures *in situ* is not easy.

Writing in *Physical Review Letters*[2], Sun *et al.* report observations of plasticity — the permanent, irreversible deformation of a material, as opposed to elastic deformation in which atomic bonds are stretched but not broken — of nanometre-sized metallic crystals inside a transmission electron microscope (TEM). They ascribe the observed deformation to the activity of short-lived, string-like defects in the crystals, known as dislocations.

In 1926, Jacov Frenkel estimated[3] the ideal (maximum attainable) shear strength of a perfect crystal to be about a tenth of its shear modulus (initial rigidity). But at that time, tests performed on real materials yielded strengths two to three orders of magnitude lower. This discrepancy was attributed to dislocations, which are boundaries of planar fault regions in the crystal structure where atoms slip out of position when the material is strained. But dislocations were directly observed in the TEM only 30 years later[4]. Once created, dislocations move and multiply easily on their own as the material is subjected to loading. Common metal objects — for example, a kitchen fork — contain many dislocations to start with, and thus deform at stresses much lower than their ideal shear strengths.

Relatively large characteristic structural dimensions, such as micrometre-sized grains in bulk materials, facilitate the continuous generation, entanglement and storage of dislocations during plastic deformation. However, as the characteristic scale (such as the crystal grain size or the smallest dimension of a thin film) shrinks below 100 nm, dislocations are 'fatally attracted' to internal interfaces (such as crystal grain boundaries) and surfaces of the specimen. Consequently, it becomes much more difficult to sustain a permanent population of mobile dislocations — which are the vehicles of plastic deformation during straining — inside the material[5,6]. In these cases, deformation can be achieved only if new dislocations are nucleated afresh, usually from the same internal interfaces and surfaces that also absorb and annihilate them[7,8]. The continual need to nucleate new dislocations in these tiny crystals results in a significant increase in the material's strength.

In their experiment, Sun and colleagues[2] initially confined individual, three-dimensional crystals (as small as 10 nm in diameter) of materials, including gold and platinum, in spherical graphitic shells. Subsequent puncturing and irradiation of the shells by a focused electron beam at different temperatures led to the extrusion of the crystals from the capsules (Fig. 1a, b). From direct comparison of the lattice spacings in the gold nanocrystals inside and outside the capsules, the authors inferred a prevailing pressure of about 20 gigapascals (about 200,000 times the standard atmospheric pressure) in the capsule when this was irradiated at about 300 °C. This is an extremely high stress for gold, considering that its ideal shear strength is only about 1 gigapascal. There is thus no question that these systems are ultra-strong.

More controversial, however, is the mechanism of deformation during extrusion. With

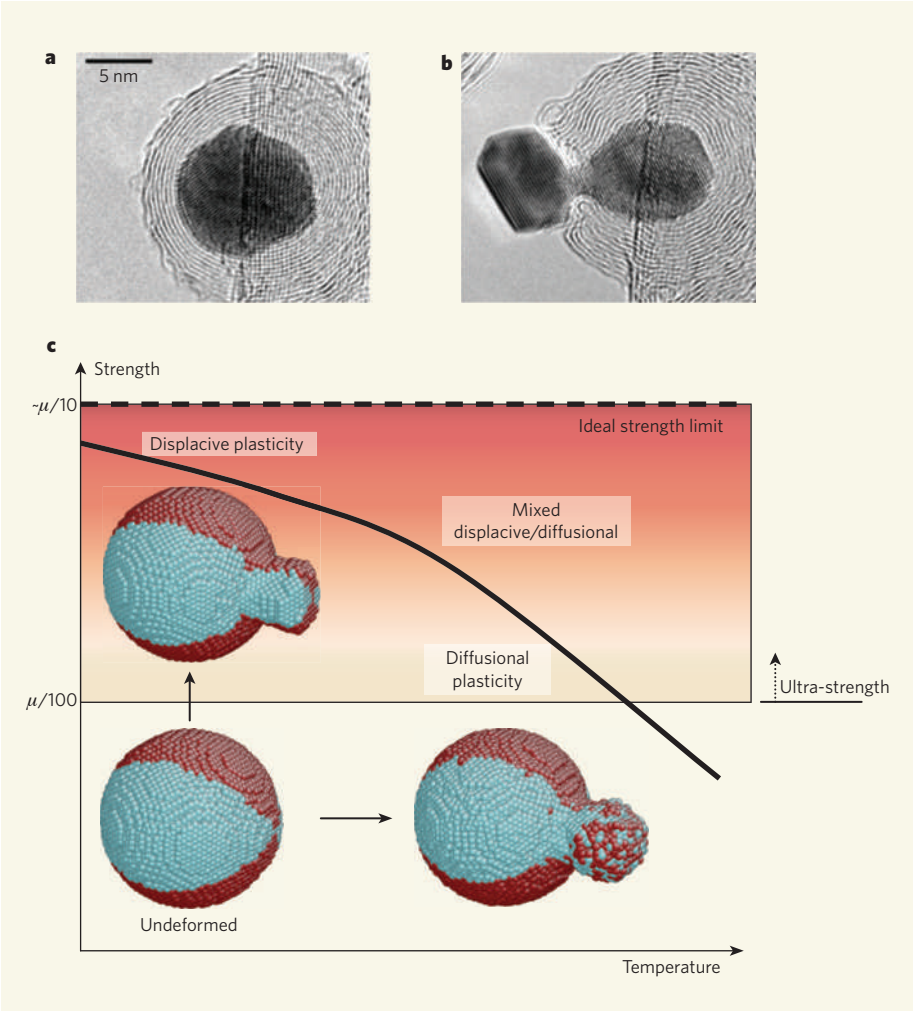


**Figure 1 | Crystal plasticity.** Sun *et al.*[2] performed *in situ* transmission electron microscopy observations of the extrusion of single gold nanocrystals from graphitic capsules under electron irradiation at 300 °C. **a**, Before irradiation. **b**, After irradiation for 540 seconds. **c**, Deformation mechanisms. The black curve shows the typical dependence of the strengths of crystalline materials — expressed as a fraction of their shear modulus, $\mu$ — on temperature. As the temperature increases, one of three competing mechanisms operates: displacive, mixed or diffusional plasticity. Superimposed are illustrative simulations, which we carried out, of the plasticity of copper nanospheres at a temperature of 300 K (sphere at the top) and 900 K (sphere on the right), in which deformation is thought to be controlled by displacive and diffusional plasticity, respectively. The copper atoms are shown in two colours (red and cyan) to make it easier to track their motions from the undeformed crystal state (bottom left sphere) to the deformed state. At 900 K, the random mixing of red- and cyan-coloured atoms in the extrusion-neck region (where the stress gradient is largest) indicates that extensive surface diffusion is taking place. (**a**, **b**, Courtesy American Physical Society.)

a TEM, Sun *et al.* observed a highly perfect atomic structure with occasional grain boundaries and planar stacking faults. But dislocations were not visible. On the basis of molecular-dynamics simulations, Sun *et al.* conclude that deformation originates from individual, transient dislocations that are freshly nucleated and vanish so fast that they cannot be seen with a TEM. Although diffusive atomic processes could be active at 300 °C in gold, the authors argue that diffusion does not contribute to plastic strain, and that the observed strength and deformation can be accounted for solely by the nucleation and motion of short-lived dislocations.

One of three competing mechanisms, all dependent on temperature and mechanical strain rate, induces plastic deformation: displacive, diffusional or mixed plasticity. Displacive plasticity[5,7,8] is produced by the collective shearing of atoms, that is, the glide of dislocations. Diffusional plasticity[9] is governed by many, almost random, individual atom or vacancy hops. In conventional coarse-grained metals, typically below about $T_M/3$, where $T_M$ is the absolute temperature at melting, deformation is dominated by displacive mechanisms, whereas above about $2T_M/3$ diffusional mechanisms control the process. A mixture of these two mechanisms occurs at in-between temperatures; in such cases the inelastic strain is still mainly produced by dislocation glide but its rate is controlled by diffusion (Fig. 1c).

Lack of understanding of the deformation mechanisms that can operate in ultra-strong materials severely limits our ability to create nanometre-scale systems with the desired mechanical properties. Information about deformation mechanisms is often gathered from molecular-dynamics simulations, but these are limited to unrealistically high strain rates. Recently, progress has been made through the use of computational methods that elucidate mechanisms of displacive plasticity at low temperatures through direct calculations of the activation volume, which characterizes the sensitivity of plastic-yield stress (the stress at which the material deforms permanently) to strain rate. Such computational studies reveal that low-temperature deformation of ultra-strong systems, such as the nanocrystals studied by Sun *et al.*, become highly sensitive to strain rate and temperature[7,8]. The underlying mechanism involves the nucleation, absorption and desorption of dislocations from interfaces and free surfaces, with a resultant reduction in activation volume, typically 2–20 times the volume of a single atom ($\Omega_0$). This activation volume is much smaller than those observed for traditional displacive-plasticity mechanisms (about $10^3\,\Omega_0$) that operate in coarse-grained polycrystals. It is, however, still larger than those of typical vacancy processes, for which the activation volume is less than about $\Omega_0$.

But at higher temperatures, such as 300 °C in gold, the way deformation changes with strain rate and the scale of nanostructures is unknown. In particular, the temperature and stress boundaries that separate the displacive processes from the diffusional and mixed processes will shift from those of the corresponding coarse-grained materials. Further experiments and modelling at higher temperatures[9–11] will inevitably be needed to understand deformation in nanostructured materials. Meanwhile, Sun *et al.*[2] have developed an innovative *in situ* experimental method that could provide insight into the process. ■

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