where γ is the membrane tension for a single graphite layer. (This behaves as a surface tension). If we now assume that γ is curvature-independent and that the molecules are spherical with radius R, the hydrostatic tension P_t is given by the Laplace equation $P_t = 2\gamma/R$. If we treat the C_{60} molecule more correctly as a truncated icosahedron rather than a sphere, the right-hand side of this equation must be multiplied by 1.0881. Substituting for γ in equation (1),

$$P_t = \frac{h}{R}\,\frac{1.088}{(S_{11}+S_{12})}\,\frac{\Delta A}{A} \qquad (2)$$

or, in terms of the volume V,

$$P_t = \left[\frac{2}{3}\,\frac{h}{R}\,\frac{1.088}{(S_{11}+S_{12})}\right]\frac{\Delta V}{V} \qquad (3)$$

and so we obtain for the bulk modulus at zero pressure,

$$B_0 = \frac{2}{3}\,\frac{1.088}{(S_{11}+S_{12})}\,\frac{h}{R} \qquad (4)$$

As this argument is based on linear elasticity, hydrostatic pressure (instead of tension) would give the same result. As the molecule is treated here as an elastic continuum, under hydrostatic pressure the pressure everywhere within the molecule is the same: the pressure at the molecular radius R_{AC} is the same as the external pressure P. Hence we do the energy balance leading to equation (4) at the surface of the truncated icosahedron defined by R_{AC}. For a quantitative estimate of B_0 we use the experimental quantities[2] $S_{11} = 0.00098\ \text{GPa}^{-1}$, $S_{12} = -0.00016\ \text{GPa}^{-1}$ and $h = c/2 = 3.354$ Å (ref. 3). For the C_{60} ball, $R_{AC} = 3.52$ Å (this is the distance from the centre of the ball to the centre of the atoms on the ball's surface (T. Siegrist, personal communication)). This yields $B_0 = 843$ GPa, which is larger than the modulus of 441 GPa found experimentally for diamond[4].

The molecular radius obtained from the lattice parameter of the closest-packed f.c.c. crystal is $R_{CP} = 5.02$ Å (ref. 1). When C_{60} balls are placed on a f.c.c. lattice, they interact essentially via van der Waals attractive forces (as do Ne, Ar and Kr) and initially the crystal would be compliant (the bulk modulus would be relatively small). When the pressure is increased to the point where the hard spheres touch, however, the bulk modulus of the crystal will become similar to that for a single molecule: the filling factor for balls in an f.c.c. crystal is 0.74, so that using the volume-fraction rule we obtain $B = 843 \times 0.74 = 624$ GPa for such (presumably modest) pressures.

The hardness of materials is related to their modulus[5]. It is thus possible that at pressures of about 20 GPa or more (where the soft-sphere repulsion is overcome), f.c.c. C_{60} crystals will be harder than diamond, and even harder than the hypothetical C_3N_4 compound suggested by Cohen[6].

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Plastid genes and parasitic plants

SIR — Howe and Smith[1] have provided one explanation for the retention of a plastid genome in achlorophyllous *Epifagus virginiana*, as reported by dePamphilis and Palmer[2]. But porphyrin biosynthesis is not the only significant metabolic activity restricted to plastids; the biosynthetic pathways for glutamate, lysine, threonine, methionine, isoleucine, leucine, valine, tryptophan, phenylalanine, tyrosine, arginine, cysteine, serine and glycine are wholly or partly located within the plastid[3]. Thus the synthesis of most of the protein amino acids, and all the metabolites derived from them, is dependent on intact and functional plastids. Other metabolic pathways, not necessarily linked to photosynthesis, are also localized in plastids.

Even in a plant that has lost one aspect of plastid metabolism, namely photosynthetic carbon fixation, there would be little advantage in losing all plastid functions. The genes for all the biosynthetic enzymes for the pathways mentioned are encoded in the nucleus, and the polypeptides produced from these genes have leader sequences directing them to plastids. It is thus hard to envisage any evolutionary process in which all these enzymes could be redirected (simultaneously?) to other parts of the cell, and their functions integrated there.

The plastid is an essential component of the plant cell — the chloroplast is merely a specialized plastid restricted to a few cell types and tissues. So the retention of a plastid genome in *E. virginiana* is not remarkable, although its absence most surely would be.

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Muscle damage in mdx mice

SIR — Menke and Jockusch reported[1] that hypo-osmotic shock exposes a reduced stability of dystrophin-deficient mdx mouse muscle. However, this is not apparent with experimental models of damage to mdx mouse muscle induced by contractile activity[2] or as an enhanced damage response in people with Duchenne muscular dystrophy who exercise[3].

The isolated, intact mature muscle-fibre studies by Menke and Jockusch were virtually free of extracellular matrix as a result of the collagenase treatment used in the isolation procedure. Surrounding muscle cells and pericellular fibrous tissue may well exert a restraining effect on muscle cells *in vivo*, preventing the substantial distortions of the membrane and hypercontraction apparent in hypo-osmotic shock to isolated muscle fibres. Further, the nature of the mechanism of cell death is unknown in hypo-osmotic shock, but the stress on the membrane presumably involves a substantial increase in internal pressure in the myofibres as extrusions (blebs) on the cell membrane are formed, a type of stress not presented to the muscle fibre in the models of contractile activity induced by damage.

Interestingly, the progression of the damage to the isolated fibres described by Menke and Jockusch is not unique to hypo-osmotic shock, but is qualitatively similar to that which occurs in isolated fibres under various forms of stress, including trauma and deliberate elevation of intracellular calcium with the calcium ionophore[4]. It therefore seems necessary to determine whether the decreased stability of mdx muscle found by Menke and Jockusch is a consequence of the hypo-osmotic shock or of the nature of the model system used. Although the results of Menke and Jockusch's report are interesting and potentially important, the 'fragile membrane' theory is by no means proven; for an abnormality such as increased fragility to be important in the pathogenesis of Duchenne and Becker muscular dystrophy, it must be apparent under physiological conditions.

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