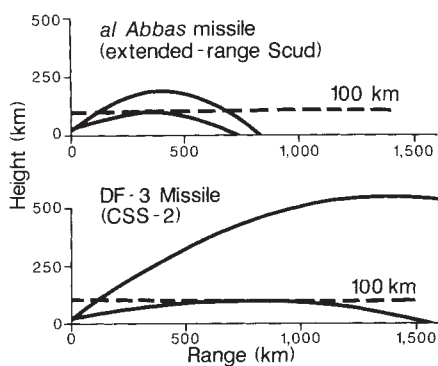


Underflying Brilliant Pebbles

SIR — Following the use of the Patriot missile in the Gulf War, the US Strategic Defense Initiative Office (SDIO) claims that its proposed space-based antimissile system Brilliant Pebbles could be used to destroy theatre ballistic missiles such as the extended-range Scud (the *al Abbas*), but its claim ignores a simple and effective countermeasure. SDIO has acknowledged that Brilliant Pebbles can intercept missiles only above about 100 km. Accordingly, if a missile's trajectory is adjusted so that it stays below this altitude throughout its flight, the missile becomes immune to Brilliant Pebbles. Our calculations show that flying theatre missiles on such depressed trajectories is not technically demanding; missiles



Minimum-energy maximum-range trajectories, and depressed trajectories with apogees of 100 km for the *al Abbas* and DF-3 missiles. The *al Abbas* has a maximum range of 830 km with a 250-kg warhead, and reaches an apogee of 190 km; on the depressed trajectory, it still has a range of 720 km. With a 2,000-kg payload, the DF-3 has a maximum range of 2,780 km with an apogee of 550 km; on the depressed trajectory, its range would be 1,550 km.

with ranges considerably greater than those used by Iraq can underfly Brilliant Pebbles on trajectories with apogees of 100 km or less.

The Brilliant Pebbles altitude limit results from the high atmospheric densities at low altitudes; flying through regions of dense atmosphere at high speeds would blind the interceptors by heating their sensors. On 12 February 1991, SDIO director Henry Cooper stated that Brilliant Pebbles could not attack targets below roughly 60 miles (100 km). Lowering this altitude limit is difficult because of the exponential increase in atmospheric density with decreasing altitude. For example, in going from an altitude of 100 to 80 km, the density and resulting heating increase by a factor of 40.

To determine whether a missile could be flown on a depressed trajectory, we considered whether doing so would lead to unacceptable increases of stress ('loading') on the missile or heating on the re-entry vehicle and how it would affect the range and accuracy. We calculated the missile trajectories by numerically integrating the equations of

motion¹, including forces due to gravity and atmospheric drag (lift was ignored). We compared the loading and heating for the depressed trajectory to that of the maximum-range minimum-energy trajectory for the same payload. The loading is roughly proportional to ρv^2 , where ρ is the atmospheric density and v is the missile velocity; for the coefficient, we used measured values for the V2 missile². The aerodynamic heating on re-entry is roughly proportional to ρv^3 , and was calculated using equations for laminar and turbulent hypersonic flow (assuming a boundary layer transition at 30–35 km)³.

To be specific, we considered two theatre-range missiles deployed in the Middle East: the 800-km Iraqi *al Abbas* and the Chinese-built 2,800-km DF-3 (CSS-2) missile deployed by Saudi Arabia. For the *al Abbas*, we assumed a total mass of 6,900 kg, fuel fraction of 80%, payload mass of 250 kg, specific impulse of 245 s, burntime of 115 s and booster diameter of 0.9 m (refs 4, 5 and S. Fetter, personal communication). For the DF-3, these parameters were taken to be 65,500 kg, 93.7%, 2,000 kg, 241 s, 142 s and 2.25 m (ref. 6). The weight-to-drag ratio for the DF-3 re-entry vehicle was taken as 48,000 N m⁻².

Calculations were done for depressed trajectories with apogees of 100 and 80 km. The loading was found to increase by 5–7% for the *al Abbas*, and 10–11% for the DF-3, which should be within the tolerances of both missiles. The heating was found to increase by 25–30% for the *al Abbas*, and 20–25% for the DF-3. This increase should not present a problem for the relatively crude *al Abbas*, for which the entire missile acts as the re-entry vehicle. If the increase in heat absorbed by the DF-3 RV exceeds its design tolerances, the heat shielding could be increased slightly, or the heating could be reduced to that of the standard trajectory by reducing the payload by 25%.

Flying missiles on depressed trajectories decreases their range; the percentage change increases with range (see figure). However, missiles with ranges of less than 1,500 km are sufficient to threaten most targets in the Middle East.

Our estimates suggest that the accuracy of these theatre ballistic missiles, which are very inaccurate, would not change significantly. Although errors due to atmospheric effects would increase on low-altitude trajectories, the dominant errors for these missiles are caused by the guidance system and would not be substantially affected. The inaccuracy of the *al Abbas*, estimated at 3–5 km (ref. 7), is not expected to change. The DF-3 inaccuracy of roughly 2.4 km (ref. 8) might increase by a factor of 2 to 3. As the original inaccuracy is so large that the only targets would be cities, such an increase would not remove its utility as a terror weapon.

Thus, theatre missiles like the *al Abbas*

and DF-3 missiles would be able to fly on low-apogee depressed trajectories, thereby rendering them invulnerable to space-based defences.

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Is C₆₀ stiffer than diamond?

SIR — The availability of single crystals of C₆₀ (ref. 1) should allow measurements of the stiffness of these molecules. By using a simple elasticity argument, we estimate the bulk modulus of individual C₆₀ molecules to be 843 GPa, which is greater than that of diamond (441 GPa). We expect that the modulus of single crystals of C₆₀ will reach 642 GPa at modest pressures and that crystalline C₆₀ will then be stiffer than diamond at the same pressure.

Consider a single crystal of graphite with equal orthogonal tensile (or compressive) stress components applied to areas normal to the basal plane. Let x_3 be the direction along the c axis and x_1 and x_2 lie in the basal plane. The stress state is

$$\begin{bmatrix} \sigma_1 \\ \sigma_2 \\ 0 \end{bmatrix} = \begin{bmatrix} \sigma \\ \sigma \\ 0 \end{bmatrix}$$

Hence the longitudinal strains ϵ_1 and ϵ_2 are given by

$$\epsilon_1 = \epsilon_2 = S_{11}\sigma_1 + S_{12}\sigma_2 = (S_{11} + S_{12})\sigma$$

where the S_{ij} are elastic compliances. The dilation ΔA of the basal area A is

$$\frac{\Delta A}{A} = \epsilon_1 + \epsilon_2 = 2(S_{11} + S_{12})\sigma$$

If h is the height of a graphite basal plane (one half of the lattice spacing c because of the ABAB stacking sequence) then

$$\frac{\Delta A}{A} h = 2(S_{11} + S_{12})\sigma h = 2(S_{11} + S_{12})\gamma \quad (1)$$

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),

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

or, in terms of the volume V ,

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

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

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

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 quantities² $S_{11} = 0.00098 \text{ GPa}^{-1}$, $S_{12} = -0.00016 \text{ GPa}^{-1}$ and $h = c/2 = 3.354 \text{ \AA}$ (ref. 3). For the C_{60} ball, $R_{AC} = 3.52 \text{ \AA}$ (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 $B_0 = 843 \text{ GPa}$, which is larger than the modulus of 441 GPa found experimentally for diamond⁴.

The molecular radius obtained from the lattice parameter of the closest-packed f.c.c. crystal is $R_{CP} = 5.02 \text{ \AA}$ (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 $B = 843 \times 0.74 = 624 \text{ GPa}$ for such

(presumably modest) pressures.

The hardness of materials is related to their modulus⁵. 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 Cohen⁶.

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Plastid genes and parasitic plants

SIR — Howe and Smith¹ have provided one explanation for the retention of a plastid genome in achlorophyllous *Epifagus virginiana*, as reported by dePamphilis and Palmer². 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 plastid³. 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 reported¹ 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 activity² or as an enhanced damage response in people with Duchenne muscular dystrophy who exercise³.

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 ionophore⁴. 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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