Table 1 Calculated π-electron ring-current magnetic properties

Fullerene	χ_{RC} (relative to benzene)	δ_{RC}* (p.p.m., centre)
C_{60} (I_h)	−0.5	1.2
C_{540} (I_h)	144.6	−13.3
C_{576} (C_{6v}, toroid)	657.2 (⊥)	−19.6
	22.4 (∥)	−1.9

*δ_{RC} is the NMR ring-current chemical shift of a central atom.

by scaling the quantum mechanical result with the free-electron value of the magnetic susceptibility. Using a modern parametrization we obtain an effective mass of $1.5m_e$ for the highest occupied molecular orbital (HOMO) of benzene, whereas a value for the toroid may be obtained from

$$\frac{m^*(C_{576})}{m^*(\text{benzene})} = \frac{\chi_{RC}(\text{benzene})}{\chi_{RC}(C_{576})} \times \frac{\rho^2(C_{576})}{\rho^2(\text{benzene})}$$

$$= \frac{99.8}{4 \times 130.5} = 0.2$$

where ρ^2 is the mean square of the distance of the electronic circulation from the axis of the magnetic field, Z. This is evaluated in the case of the toroid from the atomic coordinates of the carbon atoms:

$$\sum_i^{576} \frac{(x_i^2 + y_i^2)}{576}$$

and thus the area of the toroid in the x–y plane is about 100 times that of the benzene molecule. The ring-current magnetic susceptibility of the HOMO electrons in the toroid is 130 times larger than the total ring-current magnetic susceptibility of the benzene molecule. Thus $m^*(C_{576}\ \text{HOMO}) = 0.3m_e$, and because the mobility is usually taken to be inversely proportional to the effective mass we may expect that these carriers will show high conductivities. This is in agreement with the relatively high conductivities measured for linear nanotubes[10–14].

There has been interest in the change in electronic structure that would occur on passing a magnetic flux quantum through a benzene ring. The strong coupling of some of the toroid energy levels to the magnetic field and the concentration of levels near the energy gap (especially in other toroids), suggests that these objects will provide ideal candidates for magnetically induced changes in electronic structure[15]. At large size, of course, the level distinctions vanish as the π-electron states merge into energy bands and the flux jumps would be observable as the Aharonov–Bohm effect[1].

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Vision in dim light

On testing my own vision in very dim light I observed two phenomena associated with the lack of retinal rods (the receptors specialized for vision in dim light) in the fovea, the region corresponding to our centre of gaze. First, a bright (or dark) straight line passing through the fovea was seen as discontinuous, with a clear 1° gap. Second, after adapting to dim light conditions, when I blocked light to one eye as far as possible and viewed a brightly lit surface with the other eye, I perceived a swarm of colourless scintillations throughout the visual field of the occluded eye, except for an area about 1° in diameter at the centre of gaze. Each scintillation may represent the simultaneous capture of single quanta by several closely spaced rods.

Damage to a small area of the primary visual cortex produces a circumscribed area of blindness in the visual field, a scotoma, which is not usually apparent as the background fills in the blind area. A straight line crossing through the blind area is generally seen as uninterrupted. This completion phenomenon is also said to occur for lines crossing the normal blind spot produced by the absence of receptors where the optic nerve enters the retina.

The fovea contains cones but no rods. In light that is too dim to activate cones (scotopic conditions) we therefore have a blind spot at the centre of our visual field. I asked whether a line would show a gap when it crossed this foveal scotopic blind spot, or be completed as in the case of a cortical scotoma. On getting up at night I tested this on an abundance of lines, such as the edges of walls, wallpaper designs, and window lattices projected onto walls by street lights. Under these conditions only rods were active (no colours were visible, and any small spots vanished when looked at directly).

I could discern a clear gap when a line passed through the point of fixation. Fixation on a small object is not easy in dim light as the object disappears and gaze tends to wander, but with practice it is possible to fixate on a line so that the gap becomes obvious. Both light lines on a dark background and dark lines on a light background showed a gap as if invaded by the background. Edge boundaries between light and dark areas presented a notch of intermediate brightness. These gaps subtended about 1° of arc. The corners of a dark or light square were chopped off and similarly replaced by a region of intermediate brightness. In uniformly lit regions, no dark or light spots were seen wandering with shifting gaze. When I fixated on a 12-mm dark spot on patterned wallpaper it disappeared, filled in by the lighter background, and when I viewed a coarsely textured rug I saw a spot of intermediate brightness which wandered to follow my direction of gaze.

I conclude that under conditions of rod vision, line completion does not occur across the foveal scotoma (mentioned in passing in an earlier paper[1]), corners are not filled in, nor are patterns and textures completed. But filling-in does occur across diffusely lit surfaces, both dark and light.

When I get up at night, dark adapted over many hours, there is just enough light for me to make my way around but too little to allow me to find small objects. By cupping a hand lightly over one eye before turning on the lights this nuisance can be avoided, because when the lights are turned off again dark adaption is fully preserved in the covered eye, though the other eye is temporarily completely blind. In its thickest part my hand attenuates light by 7–8 log units (measured with a Pritchard photometer, Photo Research PR-1980, Chatsworth).

Recently I noticed a curious phenomenon. With the room light on, facing a bright, uniformly lit wall (luminance 1.8 log cd m^{-2}), I observed tiny speckles in the dark-adapted covered eye. These bright, colourless points were scattered evenly over the field of view except for an area roughly 1° wide in the centre, and each appeared for a very short time (less than 0.5 s). Their concentration increased dramatically as I let more light into the occluded eye (and tended to be replaced by a wavy, swirling texture), whereas placing two hands over the eye greatly reduced the speckles. They waxed and waned in vividness over a period of many seconds, out of phase with the view seen by the open, light-adapted eye. They were most apparent when I attended to them and faded when attention was switched to the open eye. The variation in brightness is presumably a manifestation of binocular rivalry.

It is well established that the absolute threshold for perception of light is reached when five or more closely spaced rods all capture a quantum of light within a brief period of time[2]. Absolute threshold is commonly estimated at 10^{-6} cd m^{-2}, a level of darkness that may plausibly be reached by my 7–8-log-unit filter. I suggest that the speckles could represent such coincident threshold events, because they appear at very low light levels, they increase in density as more light is admitted, they are absent in the rod-free part of the retina, and they are colourless. I have no idea why they should occur only when one eye is dark adapted

while the other views a bright scene, or what binocular rivalry has to do with the phenomenon.

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A polymerase I palm in adenylyl cyclase?

Zhang *et al.*[1] recently reported the long-awaited structure determination of one of the catalytic core domains of eukaryotic adenylyl cyclase, which promises a greater understanding of the regulatory mechanisms associated with the use of cyclic AMP as a second messenger. We have searched the Brookhaven Protein Data Bank[2] using the program PROTEP[3] and found that, far from having a completely novel fold, the fold of the domain bears an extraordinary resemblance to the 'palm' domains of the polymerase I family of prokaryotic DNA polymerases, including *Escherichia coli* DNA polymerase I[4,5] and *Thermus aquaticus* (*Taq*) polymerase[6,7]. The similarity has important implications for the function and evolution of eukaryotic adenylyl cyclases and related proteins.

The 'palm' domain of the polymerases consists of four β-strands and three helices (Fig. 1a). This structure is contained, identical in order and topology, within the adenylyl cyclase catalytic core (ACYc) domain (Fig. 1b). There is no significant sequence similarity between the domains, but 62 α-carbon atoms superpose with a root-mean-square deviation of 1.63 Å (Fig. 1c). An extra domain, the 'fingers' domain[4], lies between strand 1 and helix A of the polymerases (Fig. 1a). In ACYc the amino terminus of the domain and the loop between strand 4 and helix C form an auxiliary lower β-sheet (Fig. 1b) which occupies a similar position in three dimensions to the 'thumb' domain of the polymerases.

ACYc does not bind DNA and so the absence of 'fingers' and 'thumb' is unsurprising. Instead, the lower β-sheet acts as a spacer to create the 'wreath-like' dimeric structure of ACYc, in which the 'palm'-like domains face each other to form a ventral cavity[1]. The polymerase I palm domain terminates with helix C, but in ACYc helix C is extended and followed by an additional β-strand and helix (blue in Fig. 1b) and a disordered region. This region has been implicated in regulator binding[1].

The reactions catalysed by ACYc and the DNA polymerases are analogous. Both involve attack by the 3′ OH group of a ribose unit on the α-phosphate of a nucleotide 5′-triphosphate, with the elimination of pyrophosphate. However, in the polymerase reaction a deoxyribonucleotide is ligated to a DNA primer, whereas in adenylyl cyclase the reaction involves an intramolecular cyclization within one ATP molecule. The key catalytic residues in the polymerase I active site are three acidic groups (Asp 610, Asp 785 and Glu 786 in *Taq* polymerase) which bind Mg^{2+} and are positioned at the top end of the palm region (Fig. 1a). Although two of the equivalent residues are usually aspartates (Asp 891 and 942) in the C_1-region of ACYc, all three are hydroxyls in the C_2-region reported by Zhang *et al.*[1] (Fig. 1b: Ser 891, which hydrogen-bonds to the highly conserved Arg 977; Ser 942 which interacts with forskolin at the dimer interface; and Thr 943).

The ATP-binding site in ACYc is not known, but mutagenesis studies[1,8] show that residues from strand 4 and helix C are involved in ATP binding and catalysis. These residues are adjacent to the positions equivalent to the polymerase active site (Fig. 1a, b) on the same face of the β-sheet, so it seems likely that nucleotide triphosphate will bind in a broadly similar position in both classes of enzyme. Differences in the active site side-chains are to be expected because, in the polymerase, base recognition is in part provided by the complementary template DNA strand. It would be surprising if the detailed chemistry of intra- and intermolecular elimination of pyrophosphate were identical.

These molecules share the same unusual fold, their active sites seem to be in roughly

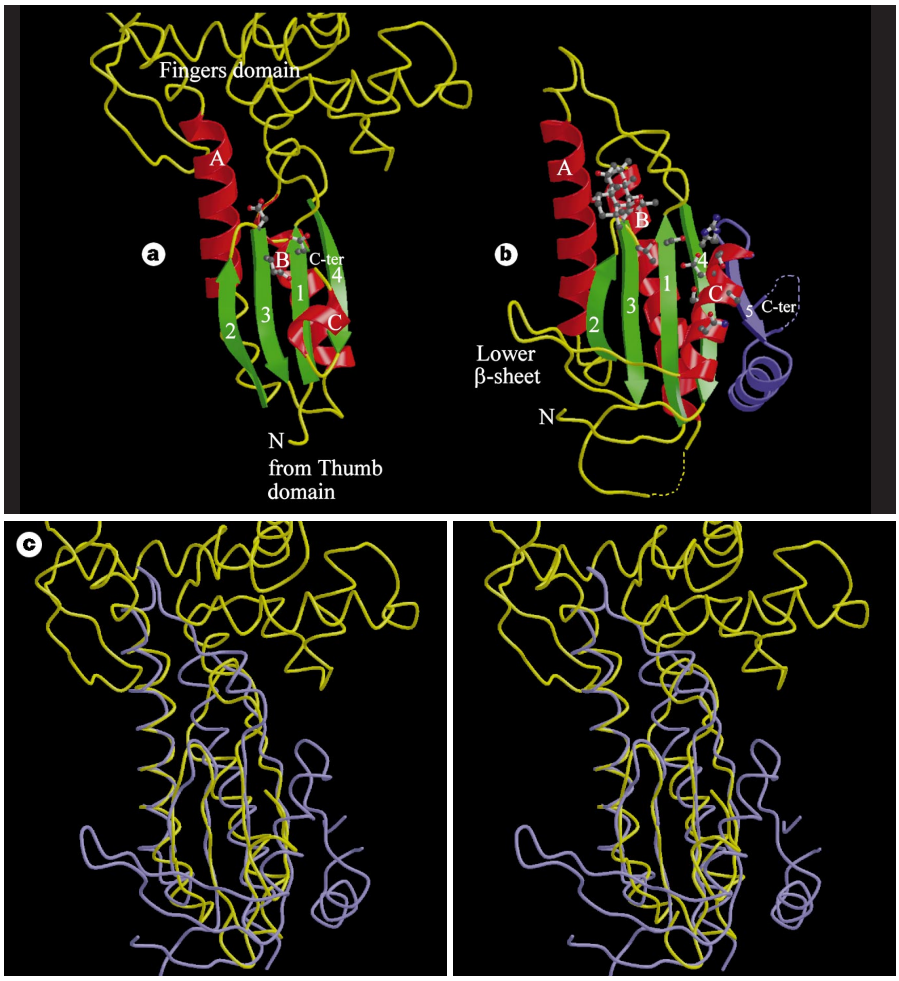

Figure 1 Chain traces[9,10] of **a,** the palm domain of *Taq* polymerase (ref. 6; PDB code, 1TAQ) and **b,** a monomer of ACYc (ref. 1; PDB code, 1AB8). Equivalent helices and strands, shown as red coiled ribbons and sequentially numbered green arrows, respectively, occur in the same order in both structures. The additional strand and helix in the C terminus of ACYc are in blue. Other non-equivalent parts of the structures are shown as yellow smoothed Cα traces, with dashes indicating disordered regions in ACYc. In the polymerase the thumb domain (not shown) is towards the N terminus of the palm domain, and the fingers domain is between strand 1 and helix A. Side chains implicated in the activity of both enzymes are shown in ball-and-stick representation: **a,** left to right, Asp 785, Glu 786 and Asp 610; **b,** top to bottom on helix C and strand 4, Arg 977, Ser 1,032, Asp 1,031, Arg 1,029 and Asn 1,025. For ACYc, a forskolin molecule is shown above Ser 942, Thr 943 and Ser 891 (left to right). **c,** Stereo diagrams[9] showing the superposition of α-carbons of ACYc catalytic domain (blue) and the palm domain of *Taq* polymerase (yellow) in the same orientation as in **a** and **b**.