RÉSUMÉ

Deep heat

A LITTLE water goes a long way, if D. Deming's explanation for the formation of economically important Mississippi Valley-type lead–zinc deposits proves correct (*Geology* **20,** 83–86; 1992). Chemical alteration by hot fluids is the accepted origin of such deposits, but the source of the fluids has defied description. The coincidence of the age of deposits in the American mid-west with a period of growth of the Appalachian mountains has led some geologists to suggest that water squeezed out from under the newly elevated range could have made the 1,000-km journey to Missouri, but that still doesn't account for its temperature. Deming argues that the plate movement that built the Appalachians would also have fractured the crust to depths of 15 km or more. Fluids here could start to convect, resulting in a massive but geologically brief increase in heat flux from the Earth's interior.

Net loss

NOT only are dolphins the victims of drift-net fishing, they are also caught in netting deployed to protect human bathers from sharks. Many are young calves, some with lactating females in attendance, as revealed in a survey of 250 bottlenose dolphins (*Tursiops truncatus*) caught off Natal, South Africa, between 1980 and 1988 (V. G. Cockroft, *J. Zool., Lond.* **226,** 123–134; 1992). Devices to warn dolphins away from shark nets are ineffective, and the only sure way to reduce the toll is to remove the nets, at least during the peak period of capture. But as Cockcroft points out, that coincides with the peak period of shark capture, and a winning strategy for the dolphins could be a losing one for the tourist industry in Natal.

Six of one

COMPOUNDS in which a carbon atom is surrounded by six rather than the usual four atoms have been described. But only now, say H. Schmidbauer *et al.*, has the central carbon actually been seen (*Angew. Chem. Int. Edn Engl.* **30**, 1488–1490; 1991). The convention-defying atom is found in gold–carbon complexes; six gold atoms, each stabilized by a large aromatic ligand, form an octahedral cage around the central carbon. By using the isotopic variant ^{13}C in place of normal ^{12}C, the authors make the central atom visible to NMR. They find that the resonance is strongly overlapped by the signal from carbons in the ligands, which explains why it was not apparent when they used natural carbon (containing 1 per cent ^{13}C). Uncharacteristically moderate screening of the atom by the surrounding ions is needed to place the resonance at this frequency.

MOLECULAR BIOLOGY

recA: from locus to lattice

Kevin McEntee

FEW proteins rival the biochemical versatility of the recA enzyme found in *Escherichia coli*. Despite its modest size (352 amino acids), it possesses an extraordinary portfolio of activities that range from homologous recombination to mutagenesis and control of gene expression during periods of cellular stress. In two papers on pages 318 and 374 of this issue[1,2], Steitz and colleagues provide the first detailed view of the structure of this unusual protein. By combining the structural data with genetic information, the authors offer some intriguing suggestions as to how the protein uses its structural forms to modulate its functional diversity.

Long before the biochemical properties of recA protein were known, the genetic complexities of this locus were well documented. Many mutations in the gene completely abolished recombination and conferred upon cells a profound sensitivity to killing by radiation and chemical agents that damage DNA. Moreover, the mutants were unable to mount a cellular stress response, the SOS response, following exposure to these same agents. Although most mutants showed these wide-ranging defects, another class of mutation left cells partially disabled by blocking only a subset of these functions. A third class, represented by the *Tif*-1 allele, caused cells constitutively to express the SOS response in the absence of any cellular insult[3].

How could a single protein be involved in such disparate cell functions? Biochemical analysis[4] suggested a solution to the puzzle, while, at the same time, raising several new questions. *In vitro*, purified recA protein promotes the swapping of DNA strands between homologous partners. The best-studied model system involves three strands: a circular single-strand and a linear duplex. This reaction has been separated into several distinct steps which involve binding of recA protein to the single-stranded component to produce a nucleoprotein complex followed by binding of a duplex DNA molecule to the complex. Presumably, this second binding event is initially stabilized exclusively by protein–DNA interactions, with the duplex being held close to the location of the single-stranded component in the filament. The DNA molecules are brought into sequence register by a mechanism that is poorly understood but which involves extensive unwinding of the duplex recipient and possible formation of a triple-stranded DNA helix. By using DNAs substituted with 7-deazaguanine, Jain *et al.*[5] have cleverly shown that the structure of this intermediate differs from molecular models of triplex DNA in not requiring base pairing through the N7 position of this purine. Once homologous alignment has been achieved, recA protein must resolve the topological problems associated with unwinding the old partners and interwinding the new.

This amount of enzymatic dexterity would satisfy most proteins, but recA possesses an obviously distinct, yet equally remarkable activity that accounts for its gene regulatory function. Eschewing the vast majority of cellular proteins, it recognizes a select group of phage and cellular repressors (and at least one protein needed for damage-induced mutagenesis) and promotes their autodigestion[6]. Like its strand-exchange function, this coprotease activity resides in a recA filament and requires ATP. Although recA protein hydrolyses ATP in the presence of single-stranded DNA, hydrolysis is not mandatory for its coprotease activity. Substantial strand exchange proceeds in the presence of the poorly cleaved analogue, ATP-γ-S (ref. 7), but hydrolysis clearly stimulates the extent of this reaction and may be necessary for driving strand exchange through regions of sequence heterology[8].

There is considerable evidence that ATP binding results in a conformational change in the polypeptide, enhancing its DNA binding. Likewise, DNA binding seems to stabilize a form of recA protein with a higher binding affinity for nucleotides. These structural changes appear not to involve large scale rearrangements of the protein, but are confined to smaller domains or regions.

At first glance, the 2.3-Å crystalline structure of recA protein[1] resembles the recA DNA filaments mentioned earlier. Monomers of recA form an extended helical polymer that is stabilized in large part by extensive contacts on two separate surfaces of each subunit. Each monomer consists of a large central domain, comprising nearly two-thirds of the mass of the protein, flanked by distinct N- and C-terminal subdomains. Not surprisingly, this central core contains the critical residues for nucleotide binding and hydrolysis as well as two disordered loops located near the polymer axis that are implicated in DNA binding. By diffusing ADP into crystals of recA protein, Story and Steitz[2] are able to position the nucleotide within the large central domain. Not unexpectedly, the α- and β- phosphates bind a loop region containing the highly conserved