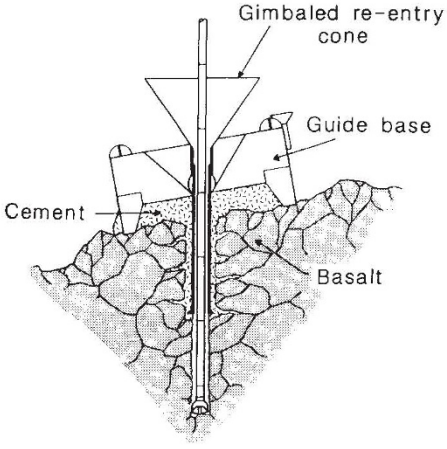
mid-ocean ridges. The requirement for significant thicknesses of sediment (>100 m) on the basement to spud-in to the crust also precluded any drilling within the narrow, zero-age crust of the accretionary zone itself.

The main objective of ODP Leg 106 was to test and evaluate new bare-rock drilling techniques at the site in the Mid-Atlantic Ridge Rift Valley and to establish a zero-age crustal hole that could be re-occupied and deepened on subsequent legs. Among the new engineering systems tested were a hard-rock guide base to confine the drill bit during initial spud-in on sediment-free rock (see figure); a low-light television camera system for imaging the sea floor and monitoring drilling operations; and



The hard rock guide base used on Leg 106, designed to provide the lateral support necessary for the drill bit to spud-in to hard, volcanic rock on slopes up to 20 degrees and water depths of up to 6,000 m. The 20-ton guide base stands on four legs and is 5.7 m square and 2.3 m high. It is lowered to the sea floor and cemented in place with 56 m^3 of cement. A cone 5.3 m in diameter and 2 m deep is built into the base and serves as a temporary re-entry cone. Once the hole is started and cased, a permanent, gimbaled re-entry cone is positioned in the base.

new downhole drilling and coring motors to facilitate bare-rock spud-in.

The site chosen for the first bare-rock drill hole is about 70 km south of the Kane Fracture Zone on the flat summit plateau of a small axial volcano. This site is characterized by bulbous and tubular pillow lavas up to 1–2 m in diameter with a light dusting of pelagic sediment. The hard-rock guide base was successfully lowered 3,344 m to the sea floor, released and cemented without incident. A positive-displacement, downhole drilling motor was used to spud-in to the basaltic crust. The drilling motor and conventional rotary drilling advanced the hole to a total depth of 33.3 m below the sea floor before drilling was terminated after 25 days.

The average recovery for the 26.7 m of cored hole was 23 per cent, comparable with that experienced during previous drilling in young volcanic rocks. All the recovered rocks are very fresh, plagioclase–olivine, sparsely phyric basalt. The texture of the groundmass ranges from glassy to subvariolitic to intersertal to intergranular, indicating that most samples are probably derived from parts of pillow lavas. The presence of plagioclase and olivine glomerocrysts and the absence of chromian spinel suggest that the basalts are typical, moderately evolved mid-ocean ridge basalts. The drill hole was left cased and open so that it can be deepened when the ship returns later this year. □

Adrenergic receptors

Some similarities and surprises

from Peter Newmark

Two particularly striking results have emerged from the sequence, reported by Richard Dixon *et al.* on page 75 of this issue, of the gene for one of the most studied of all receptors, the β-adrenergic receptor through which adrenaline and other catecholamines exert their physiological effects. The first is that there are some structural resemblances between the receptor and rhodopsin, the protein at the front end of the machinery in rod cells of the retina that converts light into the visual response. The second surprise is that the protein-coding sequence of the gene that encodes the hamster β-adrenergic receptor is not interrupted by non-coding sequences (introns).

Adrenergic receptors have been subject to the relentless prying of endless ranks of scientists. Physiologists have established the responses to catecholamines mediated by them. Pharmacologists have categorized them into types (α_1, α_2, β_1 and β_2) and established their behaviour (for example, desensitization and down-regulation, to use the operational terms beloved of pharmacologists) in response to various types of exposure to catecholamines. And biochemists have elucidated the initial events that follow the binding of catecholamine to the receptor.

What biochemists (biochemical pharmacologists?) have established is that catecholamine binding to β-adrenergic receptors induces their coupling to so-called G (or N) proteins (which come in several types, each of which has three subunits, α,β and γ). The coupling is followed by the binding of GTP to the α-subunit of the G protein, which then dissociates from its partners to activate adenylate cyclase, and thus the synthesis of cyclic AMP. This is a transient event, lasting only until the α-subunit has hydrolysed the GTP that is bound to it. The cyclic AMP produced during that time mediates the eventual physiological responses to the catecholamine.

Dixon *et al.* report two ways in which the structure of the β-adrenergic receptor protein, whose amino-acid sequence they deduce from the DNA sequence, resembles rhodopsin. First, there are three peptides within the receptor sequence that have a considerable number of amino acids in common with peptides in roughly equivalent positions within rhodopsin; and second, rhodopsin and the receptor have "remarkably similar" hydropathicity profiles (providing some measure of which parts of a protein lie within the cell membrane and thus how the protein is threaded through the membrane).

These structural resemblances make sense in the context of biochemical parallels between the two systems. When rhodopsin is stimulated by light, it couples to transducin, a G protein whose α-subunit binds GTP and, until the bound GTP is hydrolysed, activates a phosphodiesterase that in turn hydrolyses cyclic GMP. The resulting closure of ion channels on the rod cell membrane produces the visual response (see Altman, J. *Nature News and Views* **313**, 264; 1985).

It is because of the structural resemblances between the two proteins that it comes as a particular surprise to find that the gene for the β-adrenergic receptor contains no introns. In any case, it joins a very select band: histone and interferon genes are the only other mammalian genes to lack introns; and introns have so far turned up in each of the genes for cell-surface receptors. In the particular case of the rhodopsin gene and its relatives, there are four introns in the mammalian gene, four or five in the genes that encode the three human colour pigments (Nathans, J., Thompson, D. & Hogness, D.S. *Science* **232**, 193; 1986) and three or four in two different opsin genes of the fruitfly (see Cowman, A.F., Zucker, C.S. & Rubin, G.M. *Cell* **44**, 705; 1986). Dixon *et al.* are confident that they have sequenced the real gene for the receptor rather than a pseudogene, so their discovery will be interesting grist for the mill of those who try and explain the evolutionary goings (and comings?) of introns.

And for those with an interest in β-adrenergic receptors either as a prime example of a receptor or as a target for drugs that modify the physiological responses to catecholamines, the structure of the receptor itself, quite apart from its resemblance to rhodopsin, should prove immensely valuable. □

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