100 years ago

ANOTHER contribution to the subject of photography in colours is published by Mr. Carey Lea in the November number of the *American Journal of Science*. Mr. Lea finds that silver chloride combines with small quantities of many other chlorides, besides its own subchloride, to form coloured salts, comparatively stable and remarkably less sensitive to light. Thus if silver nitrate be added to a solution of ferric chloride in presence of free hydrochloric acid, the precipitate obtained is buff-coloured, and the ferric chloride carried down by the silver chloride cannot be washed out even by hydrochloric acid. The most remarkable property of this silver-ferric chloride is that it is almost unacted upon by light.

From *Nature* **37**, 88; 24 November 1887.

pattern and amplitude than of phase.

Or could the experience of wheel change have no direct impact on clock dynamics at all, but only heighten the animal's alertness to timing cues, perhaps specifically to visual cues, by which to reschedule sleep and waking?

And what about human jet-lag? Generalization from the activity rhythm to diverse unobserved physiological rhythms, or from rodents to humans, seldom turns out well when specifics are involved[5], but Mrosovsky and Salmon legitimately raise a general question for empirical resolution: could suitably chosen voluntary behavioural experience assist re-entrainment in humans as strikingly as in the hamster? Would getting angry or making love (if phase-shifting is involved, then perhaps necessarily at the right time in relation to the circadian clock's phase at first dawn or dusk in the new time zone) put our neurotransmitters and suprachiasmatic nucleus on the optimum trajectory? My own habit is to go running in bright sunlight at hours estimated to nudge my shifting clock's phase in the intended direction, and to avoid such exposure at hours when the effect would be opposite. Superstitious or not, it seems to work for me; but is it the sunlight, the running, the timing or the belief that is important? With so many aeroplanes continually crossing time zones, opportunities to find out are being missed every day. □

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Oceanic crust

Slow waves in young basalts

Peter Shearer

A UNIQUE machine that detonates explosive charges just above the sea floor helps to provide some of the best data to date about the seismic velocity structure at shallow depths in very young oceanic crust. The results, discussed in a recent paper by Purdy[1], indicate that the velocity of compressional waves (P waves) at the surface of young crust on the Mid-Atlantic Ridge is only 2.1 km s^{-1}. This is much closer to the velocity of the overlying ocean (1.5 km s^{-1}) than to that of sea-floor basalt samples (5.7 km s^{-1}), implying that the large-scale porosity of fresh basalts exposed at the sea floor is up to 30–50 per cent, much higher than previously thought.

Conventional seismic refraction experiments, in which the explosive sources are detonated near the sea surface, are poor for resolving seismic velocities within the top few hundred metres of the oceanic crust. A more direct measurement is possible if both the source and receiver are located near the sea floor. Ocean bottom receivers (seismometers or hydrophones) have been used for years, but until recently seismic sources were nearly always located near the sea surface because of technical difficulties in using sources at great depth, such as timing accurately the instant of the explosion and the unreliability of most explosives at high pressures. Recent attempts to solve these problems include free-falling 'pipe bombs' with timers[2] and devices towed deep beneath ships that can sequentially ignite a series of charges[3,4].

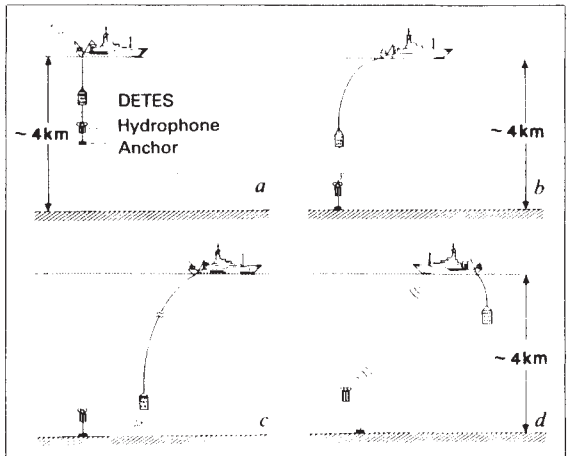

Fig. 1 Cartoon showing the operation of a DETES experiment. a, The deep-towed explosive source (DETES) and an ocean-bottom hydrophone are lowered to the sea floor. *b*, Close to the ocean floor, the hydrophone is released to free fall to the bottom. *c*, The vessel moves slowly away with the DETES firing charges at regular intervals, generating seismic waves which are recorded by the hydrophone. *d*, The DETES is hauled in and the hydrophone is released from its anchor to be recovered on the surface. (From ref. 1.)

Purdy and co-workers at the Woods Hole Oceanographic Institution have taken the latter approach, and have developed a deep-towed explosive source (DETES) capable of firing up to 48 individual explosive charges within 100 m of the sea floor (see figures)[3]. On command from the overlying ship, 2.3-kg charges are first released to hang 30 m below the device and then detonated, using an explosive that is reliable at high pressures. A hydrophone on the device records the explosions to ensure that the timing of the shot instant is known precisely. A further ocean-bottom hydrophone is anchored to the sea floor nearby. In a typical experiment, this ocean-bottom hydrophone is first released onto the sea floor by the DETES, which is then towed away slowly, firing a series of charges at increasing ranges from the receiver.

The first DETES experiment was conducted in 1985 above a small volcano within the inner floor of the Mid-Atlantic Ridge near latitude 22 °N. The results indicate a P-wave velocity of 2.1 km s^{-1} at the surface of the crust and a near-surface velocity gradient of approximately 4 s^{-1} within the top 200 m of the crust. Low seismic velocities and steep velocity gradients near the surface of the oceanic crust have been observed before, but these new results are better constrained than those from earlier experiments which did not use ocean-bottom sources, and the velocity is much lower than that indicated by most previous work. In contrast to these results for new crust, additional DETES experiments which were conducted 14 km away on 7 million-year-old crust indicate a surface P-wave velocity of 4.1 km s^{-1} and a velocity gradient of less than 0.5 s^{-1}.

Low P-wave velocities in the shallow oceanic crust have generally been explained as an effect of large-scale pores and cracks within rock of higher velocity[5–7]. The steep increase in velocity with depth in the top kilometre of the crust is thus related to the observed decrease in porosity in this layer[8]. In a similar way, the seismic velocity at the surface of the crust is thought to increase with age as a result of the slow cementing of cracks within the rock[9]. Purdy's results are consistent with this general model of the structure and evolution of the uppermost oceanic crust,

Fig. 2 The explosives platform of the DETES seismology apparatus used by Purdy and co-workers for high-resolution studies of young oceanic crust. (From ref. 1.)

but the observed velocity is so anomalously low that it implies the new crust is extremely porous, because laboratory analyses of basalt samples from the Mid-Atlantic Ridge consistently indicate velocities of 5.7–6.0 km s^{-1}.

Theoretical relationships between seismic velocities and porosity depend heavily on the shape of the pores and cracks in the host rock[5,6]. For a given velocity decrease, however, it is possible to determine the minimum porosity that is required, regardless of the shape of the cracks[7]. Assuming a host-rock velocity of 5.7 km s^{-1} and water-filled cracks, a minimum porosity of 22 per cent is required to reduce the velocities to the observed 2.1 km s^{-1}. But the predicted shear modulus for such a material is zero, which is incompatible with the observed shear waves from earthquakes recorded by ocean-bottom seismometers on the Mid-Atlantic Ridge[10]. The porosity must be higher than 22 per cent for the shear modulus not to go to zero, and Purdy speculates that it is as high as 30–50 per cent. No one has yet measured directly the porosity of new crust, but borehole measurements in 7 million-year-old crust indicate porosities of only 10–15 per cent. If younger crust is more porous, then there must be a process that reduces significantly near-surface porosity soon after the formation of oceanic crust. □

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RNA splicing

Helping RNA catalysis along

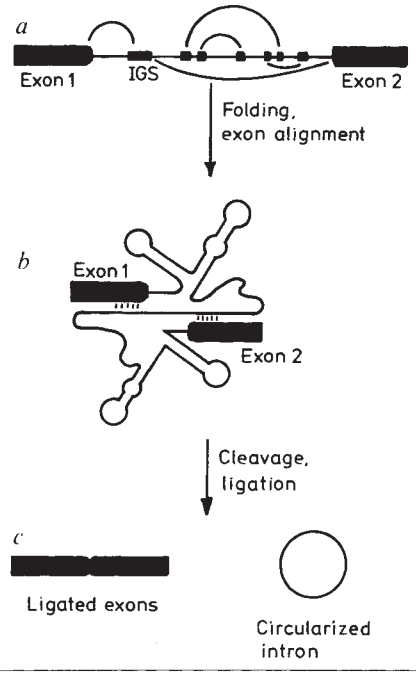
L.A. Grivell

SINCE the pioneering work of Thomas Cech and his colleagues on the self-splicing reaction carried out by the precursor to the large ribosomal (r)RNA in the protozoan *Tetrahymena*, the principle of RNA catalysis has become a familiar one (see ref. 1 for a review). When incubated in a simple salt medium, in the presence of a guanosine nucleotide, the intron in this RNA can catalyse many reactions involving the breakage and rejoining of phosphodiester bonds in RNA with an accuracy and efficiency that was once considered to be the exclusive prerogative of proteins. Despite this versatility *in vitro*, however, there is good reason to believe that *in vivo*, the reactivity of this RNA and others like it is tempered by the action of proteins. Nowhere is this more clearly apparent than in the splicing of introns in fungal mitochondria, where a decade of genetic and biochemical analysis has uncovered requirements for many proteins, some of them intron-encoded, in intron excision (see ref. 2 for a review). The latest instalment in this fascinating story is described in a recent paper[3] by Akins and Lambowitz.

The authors have isolated and sequenced a nuclear gene required for splicing of several introns in *Neurospora crassa* mitochondrial precursor RNAs, some of which have self-splicing activity *in vitro*. The encoded protein turns out to be the mitochondrial tyrosyl transfer (t)RNA synthetase. This protein, or a derivative of it, possesses both splicing and amino-acylation activities.

The intron studied by Akins and Lambowitz[3] lies in the gene encoding the large rRNA in *N. crassa* mitochondria. Its predicted secondary structure and conserved sequences within it label it as a group I intron, together with several others in the *Neurospora* mitochondrial genome. Although classification as such means that it in principle possesses all features thought to be necessary for self-splicing *in vitro* (see figure), excision *in vivo* and *in vitro* is dependent on proteins. Previous work from the same laboratory has identified temperature-sensitive nuclear mutations responsible for a deficiency in splicing at the non-permissive temperature of 37 °C; mitochondrial lysates prepared from one of these mutations, *cyt 18*, are deficient in a soluble activity that functions in splicing *in vitro* and this mutation has been characterized further[4].

Initial gene cloning by complementation of the *cyt 18* mutant, followed by judicious sub-cloning, shows that the gene corresponds to a 531 amino-acid reading frame, interrupted by a short intron close to its amino terminus. The sequence resembles those of tyrosyl tRNA synthetases from both *Escherichia coli* and *Bacillus stearothermophilus*, with 28.4 and 25.3 per cent identical residues, respectively. Armed with this knowledge, Akins and Lambowitz[3] checked tyrosyl tRNA synthetase activity in the *cyt 18* mutant and found it to be severely reduced in cells grown at 37 °C. At all



Stages in the splicing of a group I intron. *a*, Black boxes, exons; solid line, intron; boxed areas within the intron, short, conserved, complementary sequences involved in intron folding. One of these, the internal guide sequence (IGS), is capable of base pairing with regions of both exons and is responsible for exon alignment before splicing. *b*, Intron folding results in exon alignment along the IGS (base pairs shown as short vertical lines) and creation of a catalytic centre, to which a guanosine nucleotide (not shown) can bind. Binding triggers a series of transesterification events which result in cleavage and ligation reactions (see ref. 1). *c*, Transesterification results not only in exon–exon ligation, but also in intron circularization. In this latter step, a small, 5′ terminal segment of the intron (not shown) is split off. It is not yet known which stage of splicing is dependent on the product of the *cyt 18* gene.