ments in autosuggestion"[10].

The cyclical appearance of the varve sequence from Pichi Richi Pass is neither weak nor statistically marginal. Rather, it is visually obvious, and indeed dominates the total variance in the time series. The analysis by Williams and Sonett shows an impressive series of periodicities, including a basic cycle of approximately 12 'years', with alternating thick and thin cycles, and longer-term rhythmic fluctuations in the period every 13 cycles. Spectral analysis reveals a harmonic series with a fundamental period consisting of about 26.1 of the 12-'year' cycles and higher harmonics at 13.1, 8.8, 6.6 and 5.3 cycles.

Although the evidence for cycles in the varve sequence is overwhelming, there are problems concerning any interpretation of its physical significance. It is difficult to imagine any other explanation for the layered deposits than that of an annual meltwater cycle but the authors put 'years' in quotation marks, presumably to indicate that they have nothing more than circumstantial evidence for the annual origin of the layers. Similarly, the identification of the cyclical appearance with cycles of solar activity is largely circumstantial; we have nó direct evidence for the cyclic behaviour of solar activity some 680 million years ago, so correlation of varve cycles with solar activity can only be hypothetical. Indeed, the authors imply that in future papers they will use information derived from the varve sequence to help with modelling solar behaviour — a procedure that would introduce a strong element of circularity into the argument.

Williams and Sonett interpret the varve sequence as indicating that increases in solar activity caused corresponding increases in climatic temperature, resulting in greater annual meltwater discharge and the deposition of thicker varves. This is not consistent with a direct relationship between solar irradiance and surface temperature, since satellite measurements indicate that at times of increased solar activity, as indicated by sunspots, there is a slight decrease in solar irradiance[11]. Of course, temperature at a particular locality on the Earth may be influenced by topographic or other local effects and so may not vary directly with fluctuations in solar irradiance.

Perhaps the biggest question posed by the varve sequence is why cycles should be so obvious in rocks about 680 million years old, when they are not strongly in evidence in the modern record. Williams and Sonett cite the existence of "a relatively weak solar signal" in recent varves from Skilak Lake in Alaska[12] but if this is the best evidence for the influence of solar cycles that can be found in recent times it leaves much to be explained. □

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100 years ago

THE Government of Tasmania are making arrangments upon a large scale for naturalising lobsters, crabs, turbot, brill, and other European fishes in the waters of that country. The various consignments will be shipped at Plymouth, and transported through the medium of the steamship companies trading between London and Hobart. An exhaustive report has been published by the Government of Tasmania, setting forth the objects in view, and giving suggestions for carrying them into effect. The report adds that while the achievement of the acclimatisation of European fishes would lay the foundation of new and very valuable fishing industries in Tasmania, it might also prove a highly remunerative commercial enterprise to the shipping firms under whose auspices the operations will be conducted. Applications have been made for the supplies of fish, which have been satisfactorily responded to. Special tanks are being prepared in order to provide for the necessities of the fish *en route*.

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Oncogenes

Regulation and activation of c-*myc*

from Michael D. Cole

THE regulation of the c-*myc* oncogene and its role in the cancerous transformation of cells have been among the most widely studied and controversial subjects in the study of the molecular basis of cancer. This interest has been stimulated largely by the discovery that c-*myc* genes that have been activated by a variety of mechanisms (reviewed in ref. 1) are characteristic of a very wide range of tumours. Since the expression of c-*myc* is also characteristic of normal proliferating cells, however[2], it has become clear that in order to understand the nature of the abnormal activation of c-*myc* in tumour cells it is essential to understand how it is normally regulated. The paper by Adams *et al.* on page 533 of this issue[3] provides important insight into the mechanism of abnormal c-*myc* activation and a number of recent studies have revealed an unexpected complexity in the normal regulation of c-*myc* expression.

Two of the possible mechanisms of c-*myc* activation can now be dismissed. First, it is clear that the levels of c-*myc* messenger RNA are no higher in normal proliferating cells than in tumour cells, so that activation is not caused by overexpression[4]. Second, the expression of c-*myc* is constant throughout the cell cycle in normal cells[5], so earlier suggestions that the gene is cell-cycle regulated in normal but not in tumour cells are not borne out. This has led to the alternative suggestion that activation involves the loss or disruption of control elements that enable the normal gene to be switched off when the cell enters the quiescent, non-proliferating state. The question then becomes, how do the chromosomal rearrangements and retroviral insertions that are associated with the activation of c-*myc* disrupt its regulation? One possibility is that they place c-*myc* under the control of a constitutively active gene enhancer so that it is no longer responsive to the normal cellular controls. This is consistent with the finding that the normal c-*myc* allele in tumour cells containing one activated c-*myc* gene is almost always silent[6-9].

An alternative possibility is based on highly suggestive evidence implicating the 5′ exon of the cellular gene in its regulation. This exon, which is not translated, is missing from the v-*myc* oncogene that is found in some tumour viruses and is very often lost from the cellular gene as a result of the rearrangements in tumour cells. This has led to the proposal[10] that c-*myc* is under negative regulation acting on the 5′ end of the gene or its flanking sequences through the binding of a repressor protein. The experiments of Adams *et al.*, however, now show that the activation of c-*myc* is almost certainly independent of the presence or absence of 5′ sequences; while the most recent evidence on the normal regulation of the gene suggests that the 5′ exon may be important not in the control of c-*myc* transcription but in its post-transcriptional regulation.

The evidence that c-*myc* is normally transcriptionally regulated comes from measurements of c-*myc* mRNA. The induction of cell proliferation by mitogens dramatically increases c-*myc* mRNA levels[2], while the cessation of cell proliferation that accompanies differentiation leads to an equally dramatic reduction[11]. These changes were initially assumed to be in the transcription rate of the gene. However, although a rapid induction of c-*myc* transcription is found in serum-stimulated fibroblasts[12], the 3 – 4 fold increase is not sufficient to account for the 20 – 40 fold increase in mRNA level. Furthermore, after a transient increase, the rate of transcription returns to the pre-induction level while the cytoplasmic RNA level remains high.