temperatures assayed, splicing and aminoacylation activities paralleled each other, suggesting that the *cyt 18* gene product is involved in both processes.

One way in which a defect in a tRNA synthetase could interfere with splicing is indirectly, by way of a general block on mitochondrial protein synthesis and hence energy metabolism. This mechanism seems not to be the case in *cyt 18*. In a set of selected revertants to splicing proficiency, secondary mutations in the reading frame restore splicing activity, even though both aminoacylation and mitochondrial protein synthesis remain low. These results indicate that the link between the tRNA synthetase and splicing is a direct one: the synthetase is thus either a component of the splicing machinery, or is somehow involved in the regulation of its synthesis or stability. The results also show that splicing is not directly dependent on aminoacylation activity.

What is the role of the *cyt 18* product in splicing? There is as yet no clear answer to this question, although Akins and Lambowitz provide some intriguing clues. First, ribonucleoprotein particles isolated from *Neurospora* mitochondria are enriched in tyrosyl tRNA synthetase activity; partial purification of splicing activity from these separates tyrosyl tRNA synthetase activity into two main peaks, only one of which is associated with splicing. Thus activity may be attributable either to a specific quaternary form of the synthetase, or to a complex formed between it and other protein(s). It is to be hoped that further purification will resolve these alternatives.

Clues to a mechanism of action are also given by observations that point to the importance in splicing of the amino terminus of the protein. Most important of these is the finding that in one *cyt 18* mutant, splicing deficiency results from the change of the glycine at position 127 to glutamate. This residue corresponds to the conserved glycine at position 58 in both the *E. coli* and *Bacillus* enzymes, in which it forms part of the domain involved in adenylate formation (see ref. 5).

Akins and Lambowitz speculate that the *cyt 18* protein recognizes common tRNA-like structures in the introns whose excision it helps catalyse. So far, however, there is no indication that the protein makes direct contact with the intron RNA. Given that the functional part of the *cyt 18* protein is its amino-terminal adenylate-forming domain, rather than that involved in tRNA binding, other types of interaction may be more likely.

Surprising though the findings concerning *cyt 18* are, they are not the only instance of the implication of an aminoacyl-tRNA synthetase in mitochondrial splicing. In yeast, the *NAM2* mutation characterized by Labouesse *et al.*[6] is of particular interest. *NAM2* was isolated as a dominant suppressor of splicing deficiency arising as a result of mutations in the intronic reading frame of the fourth intron in the gene for cytochrome *b*. No other group I intron is affected by it, apparently because it functions through activation of the maturase encoded by the reading frame in the very similar fourth intron in the gene for subunit I of cytochrome *c* oxidase. The sequence of the *NAM2* gene displays similarity to bacterial aminoacyl-tRNA synthetases[6], and more recent sequence comparisons (C. Herbert *et al.*, personal communication) indicate that it encodes the yeast mitochondrial leucyl-tRNA synthetase. As in *cyt 18*, the mutation responsible for altered splicing activity is located in the amino-terminal region of the protein.

As Akins and Lambowitz point out, the involvement of tRNA synthetases in RNA splicing has some interesting ramifications. The finding that the *Tetrahymena* rRNA intron is spliced in *E. coli*, for example, although originally interpreted in terms of self-splicing, may in fact mean that splicing is promoted by *E. coli* tRNA synthetases and/or other proteins. Similarly, the splicing bacteriophage T4 introns in *E. coli* may also be mediated by host aminoacyl-tRNA synthetases.

From an evolutionary standpoint, it is possible that the relationship between aminoacyl-tRNA synthetases and splicing reflects a transition state in the devolvement of tasks originally carried out by RNA[3]. An initially self-splicing RNA may have recruited cellular RNA-binding proteins to its own ends. In many cases, gene duplication may have allowed such proteins to evolve separately. In other cases, like tyrosyl-tRNA synthetase, both functions may have been retained. □

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A.N. Kolmogorov (1903–1987)

ACADEMICIAN Andrei Nikolaevich Kolmogorov, one of the most respected mathematicians in the Soviet Union, died on 20 October at the age of 84. He gained world-wide recognition for his work in probability, topology, statistical mechanics, dynamics and an enormous range of other areas. In April 1983 the Presidium of the Supreme Soviet of the USSR conferred on him the Order of the October Revolution for "his great services in the development of the mathematical sciences".

He was born on 25 April 1903 in Tambov. His mother, Mariya Yakovlevna Kolmogorova, had been delayed there *en route* from the Crimea, and died in childbirth. He was brought up by his sister Vera Yakovlevna Kolmogorova. His father, the son of a clergyman, was an agronomist. In 1920 he enrolled at Moscow University, studying history and mathematics. He retained an interest in the humanities throughout his life. His first research, done in 1922 but not published for some years, marked a spectacular debut. He gave an example of an everywhere-divergent Fourier series, a highly surprising result.

In 1939 he was elected to the Academy of Sciences, becoming secretary of the Physics and Mathematics section. Between 1964 and 1966, and since 1976, he was President of the Moscow Mathematical Society. He held many positions at Moscow State University, including Dean of the Faculty of Mechanics and Mathematics, and from 1980 the Chair of Mathematical Logic.

He published more than 300 works, which range over many different areas of mathematics, both pure and applied. In 1933 he laid the foundation for the modern axiomatic treatment of probability theory. In the decade before 1939 he made an outstanding contribution to algebraic topology, with the invention of cohomology. In the 1940s he turned to fluid turbulence and derived his famous law on its statistical nature: the mean square difference of the velocities at two points of a fully turbulent fluid varies as the two-thirds power of the distance separating them. This result is fundamental to the idea of turbulence as a self-similar process. He advanced the understanding of the concept of entropy. In the 1950s he obtained the first result in a sequence leading to what is now known as the Kolmogorov–Arnold–Moser theorem, a cornerstone of modern nonlinear dynamics, with applications to the stability of the Solar System and to plasmas.

His interests included statistical mechanics, function theory, topology, logic, information theory, algorithms, celestial mechanics, biomathematics and mathematical linguistics. In all of them he made discoveries of lasting importance. Although his work ranged across an enormous area, it was all connected by the many links, usually surprising, which he found between apparently different topics.

His influence is best summed up in a statement made by N.N. Boglyubov, B.V. Gnedenko and S.L. Sobolev to mark his eightieth birthday: "The whole life of Andrei Nikolaevich Kolmogorov has been an unparalleled feat in the name of science. He is forever among great Russian scientists." Ian Stewart

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