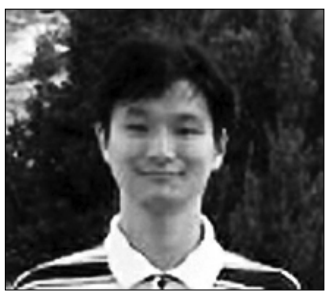
Genes save resources for real need

Plants do not call out the army when there is no enemy, says s ananthanarayan

PLANTS use ingenious devices to overcome their two major differences with animals. The first difference is that plants stay rooted to where they grow, unlike animals that can move about and seek prey and mates. The other is that plants do not have a circulatory system that can quickly transport chemical signals from one part of the plant to another.

Animals are mobile but plants depend on external agents, like butterflies, to transfer pollen for reproduction. The annual phlox, a flowering plant that is native of Texas, exists in two streams that breed separately. When grown together, both streams have light blue flowers. But when grown together where pollination across streams is possible, one stream turns its flowers to red, to guide butterflies!

Wei Wang and other scientists at Duke University, North Carolina, University of California at San Diego and the University of



Wei Wang

Worcester, UK, report in *Nature* an instance of plants that cannot transport antibodies to a site of infection through blood circulation locating their immunity forces at all sites but saving on maintenance costs by activating the forces only at the time when infection is likely.

Plant defence

How plants react to infection is complex and still not well understood. A first reaction, when a hostile organism attacks, is that molecular patterns associated with these *pathogens* get recognised by a "lock and key" matching with templates that the plant has. When there is a match, it switches on the immune reaction, by which the plant cells can react in different ways. Pathogens, to counter this defence, have developed ways to suppress this response of the plant, and the plants, in turn, have developed disease-resistant (*R*) genes that enable the plant to evade these immunity-suppressing components of pathogens.

There are two main kinds of pathogens – the *necrotrophs*, which kill the plant cell and live off the dead remains; and the *biotrophs*, which let the cell live and live off fruits of the living cells' metabolism. The chief defence of plants against *biotrophs* is the *R-gene mediated* defence, which usually induces the attacked cell to die and hence starve the enemy. Wei Wang and his group studied the way the *thale cress*, a small flowering plant found in Europe, Asia and northwest Africa reacts to a fungus-like biotrophe called ***Hpa***, which causes downy mildew disease.

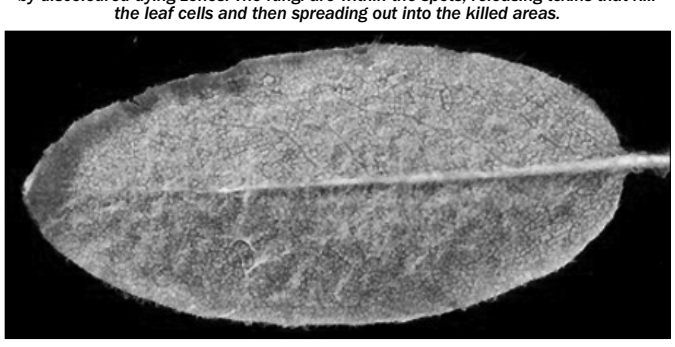
The particular pathogen-target pair was chosen because this pathogen brings about the infection in the *thale cress* leaves through clearly defined steps. It then became possible to collect samples at different stages of infection and identify the specific immune biochemistry initiated at each step. Hence, among the varieties of *thale cress*, a variety known to be resistant to *Hpa*, due to the presence of an *R-gene* called *RRP4*, was first studied. Examining the leaves as the infection progressed showed that a total of 106 genes got expressed, which is to say it is the proteins created with the help of these 106 genes that were activated in resisting the *Hpa* infection. The immune response of strains of *thale cress* which lacked *RRP4* was studied next. Each or groups of the 106 implicated genes were introduced, one by one, to reveal patterns in the effects of these genes as they were introduced, and the 106 genes could be grouped into heiracrchies of resistance effects.

Arabidopsis, a downy mildew pathogen.

Circadian clock

Living things do many things according to the daily rhythm or in cycles of 24 hours. Animals, plants, fungi and bacteria all display such rhythms. While the rhythms clearly arise through the environment being tuned to the rhythm of the sun, it is observed that the same rhythm

In this example the leaves are riddled with dead spots brought about by necrotrophic plant parasites. These range from small dark spots to larger dead areas, often ringed by discoloured dying zones. The fungi are within the spots, releasing toxins that kill the leaf cells and then spreading out into the killed areas.



Because they do not produce toxins, biotrophs may have longer and less damaging relationships with their hosts.

continues even when the animal or plant is kept away from sunlight or other cues. This shows the existence of a "biological clock" which allows the organism to anticipate and prepare for precise and regular environmental changes.

In plants, it is this rhythm that tells them what season it is, when it is best to flower and attract insects, when to grow, when to germinate and so on. The clock is set through signals such as light, temperature and nutrient availability so that the internal time matches the local time. The plant is thus able to optimise interaction with the environment, using timing as the instrument rather than chasing after prey or changing location to seek light or shade, the means adopted by animals.

It is found that the adaptation to follow rhythmic timing of functions is implemented through a pair of special genes called the *Circadian and Clock Associated 1 (or the CCA1)* gene and the *Late Elongated Hipocotyl* gene, which promote agents called *transcription factors* that permit other processes. *CCA1* and *LHY* expression peaks in the early hours of the morning and regulates the daily processes in plants. Similar genes also operate in animals to regulate rhythmic or periodic changes for optimal benefit and economy in biological processes.

Infection and timing

The work of Wei Wang and associates discovered that of the 106 genes that were identified as involved in disease resistance an important category was found to have ample molecular formations to act as "binding sites" for a "lock and key" fit with the *CCA1* factor. The implication follows that action of the *R-genes* mediated resistance against downy mildew in the *thale cress* is controlled by the *CCA1* transcription factor. The agent *Hpa*, which causes downy mildew disease, is also *CCA1* controlled and it sprays out its spores at dawn to benefit from the humidity at this time to better germinate and colonise hosts like *thale cress*.

This indicates that it is the circadian rhythm that sets off a rise in the expression of the defence gene when the expression of *CCA1* is high, anticipating an encounter with *Hpa*, which also peaks at this time. Wang and company also found that mutant varieties of *thale cress*, where the clock function was impaired, showed higher susceptibility to downy mildew infection.

The activation of defence mechanisms is not without cost to the plant, as growth process slows down to reserve maximum resources to defence. The timing and regulation of defence processes, to become effective only when needed, thus amounts to optimising deployment of resources to allow other business to proceed unimpeded at times when the likelihood of attack by pathogens is low.

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Earth's last great virgin habitat invaded

A borehole through Lake Vostok's polar ice could reveal hidden life forms, write alissa de carbonnel **and** david randall

FOR 15 million years a vast ice-bound lake has been sealed deep beneath Antarctica's frozen crust, possibly hiding prehistoric or other unknown life. Now Russian scientists are on the brink of piercing through to its secrets. They suspect its depths will reveal new life forms, show how the planet was before the Ice Age and how life evolved. It could even offer a glimpse at what conditions for life exist in the similar extremes of Mars and Jupiter's moon, Europa.

"There's only a bit left to go," said Alexei Turkeyev, chief of the Russian polar Vostok Station. Talking by satellite phone, he said his team had been drilling for weeks in a race to reach the lake – 12,000 feet beneath the Polar ice cap – before the end of the brief Antarctic summer. It was here scientists recorded the coldest temperature ever found on earth — -89.2° C.

With the rapid onset of winter, scientists were on Sunday set to take the last flight out for this season. "It's -40°. "But, whatever, we're working. We're feeling good. There's only five metres left until we get to the lake, so it'll all be very soon."

The Antarctica's subglacial lakes are the last great unexplored habitat on earth, and Lake Vostok is the largest of them. It is immense – 160 miles long and 31 miles across at its widest point – a similar size to Lake Ontario, the smallest of North America's five Great Lakes. Despite its position in Antarctica, the weight of the ice above it and the heat from the bedrocks beneath it means that scientists believe its waters may be as temperate as minus three degrees Celsius, but liquid and not frozen. Lake Vostok is the largest, deepest and most isolated of Antarctica's 150 subglacial lakes. It is supersaturated with oxygen, resembling no other known environment on the planet. Geoscientists say the ice sheet – believed to have been formed about 15 million years ago – acts like a duvet, trapping in the earth's geothermal heat. Sediment from the lake could take scientists back millions of years to tropical prehistoric times.

Some 65 million years ago, Antarctica had a climate akin to the more temperate interior of Australia, to which it was then connected. Valery Lukin of Russia's Arctic and Antarctic Research Institute in St Petersburg, which is overseeing the expedition, said, "It's like exploring an alien planet where no one has been before. We don't know what we'll find."

What all the scientists are hoping to find are whole ecosystems of hitherto unknown microbes and bacteria, trapped in a lightless world. One researcher, Brent Christner of Louisiana State University, has studied ice cores from the Lake Vostok operation and calculated that the quantity of living cells in Antarctica could exceed those in all the world's other fresh water. Chuck Kennicutt, an oceanographer who co-chaired a conference on these lakes last year, said then, "When it comes to understanding our planet, Antarctica is about the last frontier."

However, the discovery of what exactly are in these tantalising waters will have to wait until the next Antarctic summer, when the team can return to make the final breakthrough. A centenary since the first expeditions to the South Pole, the discovery of Antarctica's hidden network of subglacial lakes via satellite imagery in the late 1990s has sparked a new exploratory fervour among the world's scientists.

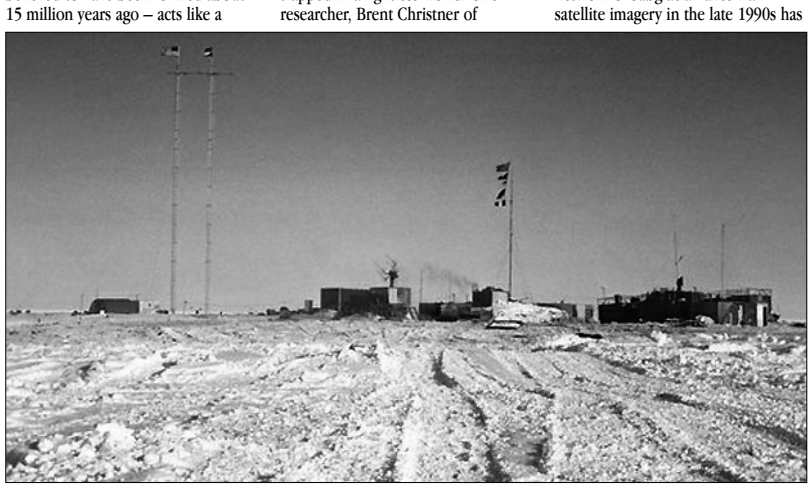
Two other teams, including a British one, are boring down on similar, if smaller, lakes. Martin Siegert, head of the University of Edinburgh's school of geosciences, who is leading a British expedition to a smaller polar lake, said, "It's an extreme environment but it is one that may be habitable. If it is, curiosity drives us to understand what's in it. How is it living? Is it flourishing?"

"The Russians are leading the way with a torch," said John Priscu of Montana State University, a chief scientist with the US programme to explore another Antarctic lake. Beneath the endless white landscape, Priscu suspects creatures may lurk, far from the sunlight, around thermal vents in the depths of Lake Vostok. "I think Lake Vostok is an oasis for life under the ice sheet. It would be really wild to thoroughly sample... But until we learn how to get into the system cleanly that's an issue," he said.

The low-lying, snowdrift buildings and radio towers of Vostok Station sit above the eponymous lake. The borehole, pumped full of kerosene and freon to keep it from freezing shut, hangs poised over the pristine lake. The explorers now face the question: "How do we go where no one has gone before without spoiling it or bringing back some unknown virus?"

"I feel very excited, but once we do it there is no going back," said Alexei Ekaikin, a scientist with the Vostok Station expedition. "Once you touch it, it will be touched forever."

The Independent, London



Lake Vostok has been sealed off from the rest of the world for 15 million years.

The split within

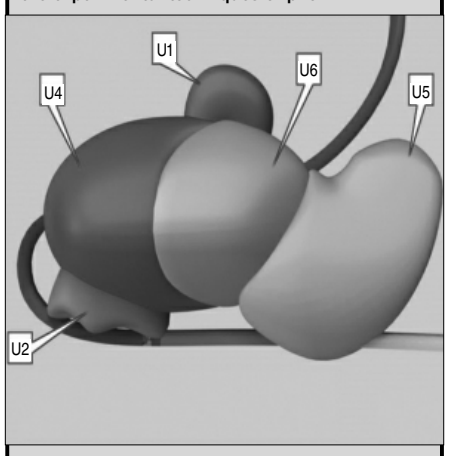
tapan kumar maitra **explores the function and evolution of introns**

EUKARYOTIC nuclear messenger RNAs have their introns removed by way of a lariat structure, just as in type H introns, but with the help of RNA-protein particles. Consensus sequences in nuclear messenger RNA for the majority of introns. At the left (5') side of the intron, the GU sequence is invariant, as is the AG at the right (3') side. The right-most A of the UACUAAC sequence is the branch point of the lariat and is also invariant.

Unlike the mitochondrial group H introns, however, nuclear messenger RNAs have their introns removed with the help of a protein-RNA complex called a spliceosome, named by J Abelson and E Brody. The splicing apparatus in eukaryotic messenger RNAs consists of several components called small nuclear ri-bonucleoproteins (discovered and named by J Steitz and colleagues), abbreviated as snRNPs and pronounced "snurps". Five of these particles take part in splicing, each composed of one or more proteins and a small RNA molecule; they are designated Ul, U2, U4, U5, and U6. The RNA molecules range in size from 100 to 215 bases.

The snRNPs and their associated proteins are located in 20 to 40 regions in the nucleus called speckles because of their appearance in the fluorescent microscope.

The RNAs of these particles have been sequenced, and sequencing shows they have regions of complementarity to either sites in the exons, sites in the introns, or sites in the other snRNP RNAs. These sequences, together with the experimental techniques of pho-



The assembled spliceosme: the U4 and U6 snRNPs bind together to form a complex independent of the pre-mRNA. The U5 snRNP associates with that complex forming another that further associates with the U1 and U2 already bound to the pre-mRNA.

tocrosslinking and the creation of selective mutations (using techniques of site-directed mutagenesis, have given us insight into the splicing mechanism. Photocrosslinking tells us which components are in contact. Mutations change pairings of components and may disrupt the structure.

The change can be rescued — the pairing restored — by making a second change in the complementary RNA. When this happens successfully, the presumed pairing is then confirmed. For example, if an A-U base pair occurs between two pieces of RNA, changing the A to a C disrupts the pairing. However, if the U is converted to a G, the pairing is restored (complementary A-U bases are converted to complementary C-G bases via a non-complementary C-U intermediate). From these techniques, we believe that the following sequence of events takes place.

First, the Ul snRNP binds at the 5' site of the intron and the U2 snRNP binds at the branch point. The U4, U5, and U6 snRNPs form a single particle. The U4 snRNP releases, freeing the U6 snRNP to bind to the 5' site displacing the U1 snRNP. (The Url snRNP, with the help of other proteins, may bind at the 5' site simply to mark it and initiate the process.) The U6 snRNP then also binds the U2 snRNP, allowing the lariat to form in the intron. The U5 snRNP binds the two exon ends together, allowing the splice to be completed as the lariat is removed.

The splicing machinery for the majority of introns also includes numerous other polypeptides called auxiliary and splicing factors; the entire splicing process requires about 50 polypeptides. A second, less common, intron, called the U12-dependent intron, with different consensus sequences, also exists. It is removed by a similar splicing process involving different snRNPs (U11, U12) as well as many components shared with the major spliceosome.

Currently, we believe the splicing out of the intron may be autocatalysed, just as in the type II self-splicing introns. The spliceosome may have evolved to ensure control over the process, allowing different introns to splice with differing efficiencies and allowing alternative splicing to take place. In many eukaryotic genes, alternative paths of splicing can take place — different splice sites may be chosen or splices may be avoided entirely. Thus, a single gene can produce several different proteins, depending on splicing choice. For example, in yeast, the gene RPL32 codes for a ribosomal protein.

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