

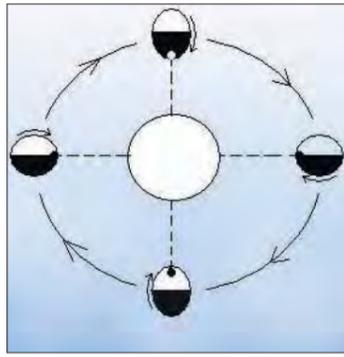
Rotation keeps in step

A NEAR SATELLITE IS NOT FREE TO SPIN AS IT WISHES WHILE IT GOES ROUND THE MOTHER PLANET, SAYS S ANANTHANARAYAN

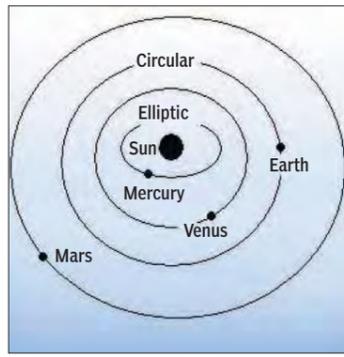
A day on the moon lasts as long as a lunar month. This is because the moon spins round only once as it circles the earth. But Mercury spins round thrice in the time it takes to go twice round the sun. Near satellites tend to slow their speed of rotation to match their period of orbit. But the way Mercury has settled in seems to be a little different.

Dr Benoit Noyelles of the University of Namur, Belgium, in collaboration with Drs Julien Frouard of the University of São Paulo, Rio Claro, Brazil, and Valeri Makarov and Michael Efroimsky of the US Naval Observatory, Washington, DC, have presented before the *American Astronomical Society* their model of how this end-state of the rotation-orbit relationship came about, where they draw inferences of the early state of the interior of the planet.

When a satellite goes around a parent body, the latter exerts greater gravitational force on the nearer side of the satellite than on the farther side. The gravitational stress causes a bulge, albeit small, along the line connecting the two objects. But as the satellite is spinning, and deformation of a satellite is slow to adjust, this bulge moves away,



The moon keeps the same face towards the earth



Planets and the orbits

while the greater gravitational pull is at the part of the satellite that now comes in line. The parent body thus tries to draw out the part that is now nearest and also exerts a force on the bulge that is moving ahead, which tends to slow the rotation of the satellite.

The result, over millennia, is to slow down the rotation and finally keep the same part of the satellite always facing the parent body, with a permanent though minute bulge in the shape of the satellite. This is the case with our own moon, which always shows the same face to the earth. But the moon does turn around, as it goes round the earth and, over the length of a lunar month, all parts of the moon experience night and day, in respect of light from the sun. The time the moon takes to spin once around is hence the same as the time to go once round the earth.

This is also the case with satellites of other planets. It is not the case with the planets themselves, because they are at large distances from the sun and the gravitational effects of the sun to cause deformation of the planets is not appreciable. But we know that the effect is large with respect to the earth and the moon, as ocean tides are largely because of the moon. In fact, ocean tides should gradually slow down the speed of rotation of the earth, while the speed of orbit, and hence of rotation, of the moon should slightly

increase in reaction.

But in the case of Mercury, which is the innermost planet of the solar system, the tidal effect of the sun is not small and negligible. The speed of rotation has thus been slowed down, over the ages and, compared to the 365 rotations of the earth for each orbit of the sun, Mercury goes round only one and a half times during each orbit. The question is, why one and a half times — why not only once?

Elliptical orbit

The answer lies in the shape of the orbit. All the planets of the solar system, except Mercury and Pluto, move in almost circular orbits. The orbits of Pluto and Mercury are like distinct ovals, Pluto's is more so, but that of Mercury is also nearly the same, with the length of the oval one and a half times the breadth. But as Pluto is so far away from the sun, there is no meaning of any tidal force of the sun on Pluto, in any case.

Mercury, in this elongated orbit, moves fast when it is near the sun, but slows down to a crawl when it is at a distance. When it is quickly moving past, when nearer the sun, it feels strong tidal forces and its speed of rotation during that time would be very nearly the same as the short time it takes to move around the sun.

But once it has moved round the sun, it sets out on its long journey through the re-

maining part of the oval, and as it moves away its speed of orbit reduces. But with reduced gravitational pull, the speed of spin, which was fixed while it was near the sun, does not change and the planet continues to turn at nearly the same speed as it slowly proceeds along the orbit and ends up turning round, or rotating, more than once. It would appear that it would take a specific composition of the material of the planet for deformation to induce a single rotation for each orbit, in a case of a non-circular orbit.

The settling in of the speed of rotation to be the same as the speed of orbit is known as resonance, after the same word which indicates two musical notes of the same, or mutually multiple, pitch. In the case of Mercury, where the planet turns one and a half times round in an orbit, or three times in two orbits, so that the same side as before would be facing the sun after the second orbit, there is asymmetry and this is also resonance. Understanding how Mercury came to stop at this resonance and not go on to the state of one rotation for each orbit, which is a more stable condition, would help understand how molten and deformable the planet was, at different times after it first fell into orbit around the sun.

This is the work that the group which has presented its results to the *American Astronomical Society* has done. Given the ovality of Mercury's orbit, the fact that the planet has been trapped in the 3:2 spin-orbit resonance has come about because it reached a not fully molten — which is a relatively cold — state quite early in its life and also that the segregation of matter, as a solid crust and a liquid core, happened after the resonance had been established.

The study also shows that such a 3:2 resonance may occur in exoplanets, or planetary systems of other suns, where the orbits are even more markedly oval. This insight would be useful in estimating how far such exoplanets may have conditions to support life.

The National Aeronautics and Space Administration's *Messenger* mission and the European/Japanese mission in the next decade, to study the surface of Mercury, would help refine the tidal models of Mercury and exo-planets.

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PLUS POINTS

Calming influence

Even though the concentration of greenhouse gases has been steadily increasing in the atmosphere, the global mean temperature has remained in check



for the past decade. The reason behind this compelling phenomenon has largely remained elusive. Now two scientists from the Scripps

Institution of Oceanography and the University of California, San Diego, claim to have found the answer: the tropical Pacific Ocean.

The study, published in *Nature* on 28 August, says the cooling effect of the eastern Pacific Ocean waters has kept a check on global warming since 1998. Prior to this, the temperature had been increasing globally at the rate of 0.13° Celsius per decade since 1950. "We used an advanced climate model that takes radiative forcing (the difference between radiant energy received by the earth and the energy radiated back to space) and tropical Pacific Ocean surface temperature as inputs. The simulated global mean temperature is in excellent agreement with the observations, showing that the decadal cooling of the tropical Pacific causes the current hiatus," the scientists report in their study.

"Climate models consider anthropogenic forcings like greenhouse gases and tiny atmospheric particles known as aerosols, but they cannot study a specific climate event like the current hiatus," says Yu Kosaka, one of the authors of the study and a project scientist at the Scripps Institution of Oceanography. "We devised a new method for climate models to take tropical Pacific Ocean temperatures as an additional input. Then amazingly our model could simulate the hiatus well."

"Specifically, the model reproduced the seasonal variation of the hiatus, including a slight cooling trend in global temperature during the northern winter season," says co-author Shang-Ping Xie.

They however, also predict that sooner or later the cooling of the Pacific will stop and the world will experience full-blown global warming. "When the Pacific swings into a warm phase, this natural warming will be added on top of the greenhouse gas warming. The last time this happened, global temperature shot up by 0.4° Celsius over a 20-year-period of 1975-1994," says Xie.

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Corridors of uncertainty

Schemes and projects on tiger and leopard conservation in India are primarily focused on protected areas. Much less attention has been paid to the protection and restoration of corridors that connect fragmented habitats where the remaining tiger and leopard populations survive. Researchers are now finding mounting evidence to suggest that protecting the corridors



Corridors help big cats migrate, which is essential to maintain genetic diversity.

might be the best way to ensure the survival of big cats. Naturally existing corridors help in their migration, thereby maintaining genetic diversity crucial for survival.

In two large sample analyses, researchers from the USA collected about 1,400 faecal samples representing 273 tigers and 217 leopards from central India. The samples belonged to four tiger and leopard populations from five tiger reserves — Kanha, Melghat, Satpura, Pench (Maharashtra) and Pench (Madhya Pradesh) — spanning an area of about 45,000 square kilometres. The researchers calculated the gene flow between these reserves in the past 20-25 years and simulated historical data for about 2,500 years. They found that the gene flow between populations had reduced in areas that had lost forest cover and connectivity, but had a high rate in areas where corridors were conserved.

Trishna Dutta, a researcher at the Smithsonian Conservation Biology Institute in the USA, said, "We should definitely protect corridors if we want tigers (and leopards) to persist for a longer duration of time."

The study on tigers was published in *Proceedings of the Royal Society B: Biological Sciences* and the one on leopards in *Evolutionary Applications* this September. The studies come at a time when the Union ministry of environment and forests has given clearance to coal mining projects in a functional corridor under study (Pench-Satpura) and is considering the expansion of National Highway-7 that passes via the Kanha-Pench corridor. "This is going to be detrimental to tiger and leopard movement between the reserves," says Sandeep Sharma, author of both the studies.

RICHA MALHOTRA/CSE-DOWN-TO-EARTH FEATURE SERVICE

POST-TRANSCRIPTIONAL CONTROL

TAPAN KUMAR MAITRA EXPLAINS EUKARYOTIC GENE REGULATION

The first two levels of regulation for eukaryotic gene expression involve genomic controls and transcriptional controls. After transcription has taken place, the flow of genetic information contains a complex series of post-transcriptional events, any or all of which can turn out to be regulatory points. Post-transcriptional regulation may be especially useful in providing ways to fine-tune the pattern of gene expression rapidly allowing cells to respond to transient changes in the intracellular or extracellular environment without changing their overall transcription patterns.

Post-transcriptional control begins with the processing of primary RNA transcripts, which provides many opportunities for the regulation of gene expression. Virtually all RNA transcripts in eukaryotic nuclei undergo substantial processing, including addition of a five-inch cap and a three-inch tail, chemical modifications such as methylation, a splicing together of exons accompanied by the removal of introns and RNA editing. Among these processing events, RNA splicing is an especially important control site because its regulation allows cells to create a variety of different mRNAs from the same pre-mRNA, thereby permitting a gene to generate more than one protein product.

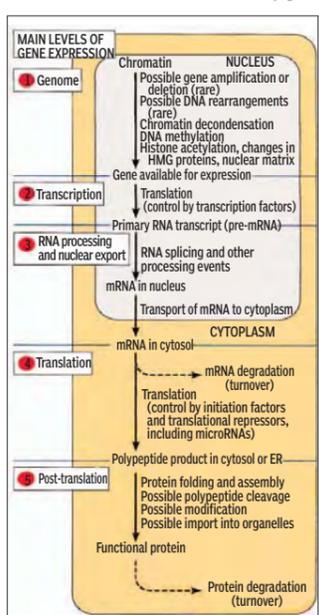
This phenomenon, called alternative RNA splicing, is controlled by proteins that bind to pre-mRNA molecules and cause some splice sites to be skipped and others to become activated. By increasing the ways in which a given pre-mRNA can be spliced, such mechanisms make it possible for a single gene to yield dozens or even hundreds of different mRNAs. A remarkable example has been discovered in the inner ear of birds, where 576 alternatively spliced forms of mRNA are produced from a single potassium channel gene. Expression of varying subsets of these mRNAs in different cells of the inner ear facilitates perception of different sound frequencies.

It illustrates another well-documented example of alternative RNA splicing, in this case involving the mRNA coding for a type of antibody called immunoglobulin M (IgM). The IgM protein exists in two forms, a secreted version and a version that becomes incorporated into the plasma membrane of the cell that makes it. Like all antibodies, IgM consists of four polypeptide sub-units, two heavy chains and two light chains. It is the sequence of the heavy chain that determines whether IgM is secreted or membrane-bound.

The gene coding for the heavy chain has two alternative poly sites where RNA transcripts can terminate, yielding two kinds of pre-mRNA molecules that differ at their three-inch ends. The exons within these pre-mRNAs are then spliced together in two different ways, producing mRNAs that code for either the secreted version of the heavy chain or the plasma membrane-bound form. Only the splicing pattern for the

membrane-bound version includes the exons encoding the hydrophobic amino acid domain that anchors the heavy chain to the plasma membrane.

After RNA splicing, the next post-transcriptional step subject to control is the export of mRNA through nuclear pore complexes and into the cytoplasm. We know that export is controlled because RNAs exhibiting defects in capping or splicing have markedly decreased rates of export from the nucleus. Evidence that the export process is closely coupled to RNA splicing has come from experiments in which introns were artificially removed from purified genes prior to the insertion of the genes back into intact cells. Although the altered genes were found to be actively transcribed, their transcripts were not exported from the nucleus unless at least one intron was initially present.



Gene expression can be regulated by influencing any of the events that occur within any of these levels. (1) The genome level, including rarely occurring amplification or rearrangement of DNA segments, chromatin decondensation (and condensation), and DNA methylation. (2) Transcription, where critical control mechanisms determine which genes are active at a given time. (3) Processing of RNA and its export from the nucleus. (4) Translation, the synthesis of polypeptides. This level includes targeting of some newly forming polypeptides to the ER. (5) Post-translational events, including polypeptide folding and assembly, polypeptide cleavage, modifications of polypeptides by the addition of chemical groups, and the import of proteins into organelles (and secretion of some from the cell). Degradation of completed protein molecules is also subject to regulation.

sent. Experiments involving HIV, the virus that causes Aids, have provided even stronger evidence that the export of individual RNAs can be selectively controlled. One type of RNA molecule encoded by HIV is synthesised in the nucleus and remains there until a viral protein called Rev is produced. The amino acid sequence of the Rev protein includes a nuclear export signal, which allows the protein to guide the viral RNA out through the nuclear pores and into the cytoplasm.

For the export of material out of the nucleus, comparable mechanisms operate. The main difference is that transport out of the nucleus is used mainly for RNA molecules that are synthesised in the nucleus but function in the cytoplasm, whereas nuclear import is devoted largely to the import of proteins that are synthesised in the cytoplasm but function in the nucleus.

Although the main cargo for nuclear export is RNA rather than protein, RNA export is mediated by proteins that bind to the RNA. These adaptor proteins contain amino acid sequences called Nuclear Export Signals, which target the protein and hence its bound RNA, for export through the nuclear pores. Nes sequences are recognised by nuclear transport receptor proteins called exportins, which bind to molecules containing Nes sequences and mediate their transport out through the nuclear pores by a mechanism that resembles the way in which importins transport cytoplasmic molecules into the nucleus.

The difference in direction between importin- and exportin-mediated transport is governed by the interaction between Ran-GTP and these two classes of molecules, accompanied by a concentration gradient of Ran-GTP across the nuclear envelope. The Ran-GTP concentration is maintained at high levels inside the nucleus by a Guanine-nucleotide Exchange Factor that promotes the binding of GTP to Ran in exchange for GDP. In contrast, the cytosol contains a GTPase activating protein (GAP) that promotes the hydrolysis of GTP by Ran, thereby lowering the Ran-GTP concentration outside the nucleus. The relatively high concentration of Ran-GTP inside the nucleus has two effects: first, nuclear Ran-GTP promotes the release of NLS-containing cargo from importin; and, second, nuclear Ran-GTP promotes the binding of NES-containing cargo to exportin.

The net result is that the direction of transport for any given cargo molecule is determined by the type of targeting sequence it contains (NLS or NES), which dictates whether importins will release the cargo in the nucleus and bind it in the cytoplasm, or exportins will bind the cargo in the nucleus and release it in the cytoplasm.

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'Printing' alien life from Mars

THE NEXT REVOLUTION IN GENETICS WILL COME FROM SYNTHETIC BIOLOGY, WRITES JAMES VINCENT

Scientists will soon be able to design and print simple organisms using biological 3D printers, says J Craig Venter, the scientist who led the private-sector's mapping of the human genome. He predicts that new methods of digital design and manufacture will provide the next revolution in genetic with synthetic cells and organism tailor-made to tackle humanity's problems: a toolkit of sequenced genes will be used to create disease-resistant animals, higher yielding crops and drugs that extend human life and boost our brain power.

These ideas have been outlined in Venter's latest book, *Life at the Speed of Light: From the Double Helix to the Dawn of Digital Life*, in which the geneticist asks the age-old question, "What is life?" before detailing the history — and future — of creating the stuff from scratch.

For Venter, life can be reduced to "protein robots" and "DNA machines" but he also believes that technology

Venter's ideas may sound like science fiction but he has achieved comparable feats in the past. Frustrated by what he viewed as slow government-led efforts to sequence the human genome in the '90s, he raised private capital to create a rival effort under the company name of Celera. Fears that Venter and his backers would attempt to patent the genome spurred the US-led effort into action and the global genes-race was sparked, with both sides eventually agreeing to announce their result one day apart in February 2001.

Venter parted ways with Celera in 2002 and founded the J Craig Venter Institute in 2006. In 2010, he and his colleagues at the institute announced that they had created the world's first synthetic organism. The team created a bacterium genome from scratch and "watermarked" it with custom DNA strings (these included



Venter poses for a photo after the 2010 announcement of his lab's creation of the first synthetic organism.

will unlock far more exotic opportunities for creating life. The title of the publication refers to the idea that we may be able to transmit DNA sequences found on Mars back to earth (at the speed of light) to be replicated at home by biological printers.

"I am confident that life once thrived on Mars and may well still exist there today," he writes. "The day is not far off when we will be able to send a robotically controlled genome-sequencing unit in a probe to other planets to read the DNA sequence of any alien microbe life that may be there."

an encoded email address) before transplanting it into another cell. The cell then began to reproduce, making it the first living species created by humanity.

Although such pioneering work frequently raises ethical questions over the danger of humanity "playing God", Venter writes that he is not concerned with such concerns. In *Life at the Speed of Light*, he writes, "My greatest fear is not the abuse of technology but that we will not use it at all."

THE INDEPENDENT