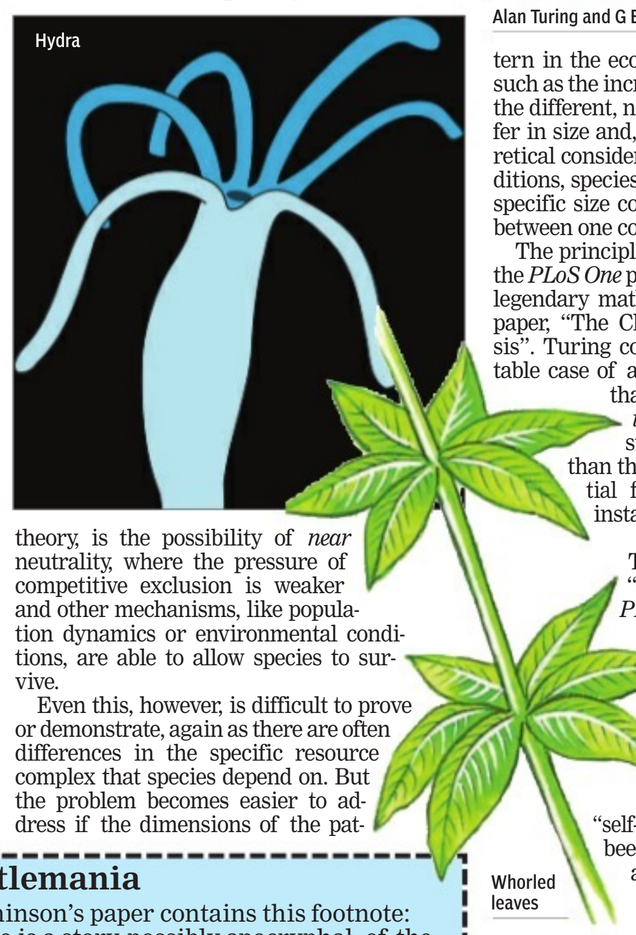
Security in being different

ALTERNATE ROUTES THAT LEAD TO THE SAME PLACE MAY BE NATURE'S WAY OF BEING SURE TO GET THERE, WRITES S ANANTHANARAYANAN

A problem with evolutionary biology is being able to explain how there could be such a large number of different species. While there are forces that limit the length of a food chain, many communities would affect each other by competing for the same resources. It is, hence, difficult to understand how one level in the food chain could maintain a large number of different communities. Marten Scheffer, Remi Vergnon, Egbert H van Nes, Jan GM Cuppen, Edwin THM Peeters, Remko Leijs and Anders N Nilsson of Wageningen University in The Netherlands, the University of Umeå in Sweden and institutes in Adelaide, Australia, report in the journal *PLoS One* their study of the 4,168 species of the *diving beetle*, which belongs to the group of animals that is the most species-rich in nature, and show nature's way of separating groups of species to occupy a habitat and still stay apart in their use of resources.

The foundation of the idea that there cannot be large numbers of species was described in a classic 1959 paper by American ecologist G Evelyn Hutchinson, where he suggests that there was a built-in limit, known as "limiting similarity", to how close — biologically, that is — species could get. This follows from the *competitive exclusion principle* that either one of two species that needs to use the same resource must overpower the other. When one species has even the slightest advantage or edge over the other then the one with the advantage will dominate in the long term and the other will either go extinct or, unless there are environmental changes, migrate to another ecological niche. But this did not seem to work in practice, as there were clearly not as many separate ecological "niches" as there were species. Further, the principle of competitive exclusion alone did not prove useful to explain evolution dynamics, as species depend on a brace of resources and it was found difficult to create conditions where the principle could be tested or demonstrated. In contrast is the neutral theory, which suggests that many species can, in fact, co-exist because their dependence on the same resources renders them equivalent and, hence, incapable of overcoming each other. But neutrality also has deficiencies and what is proving more fertile, as a theory, is the possibility of *near* neutrality, where the pressure of competitive exclusion is weaker and other mechanisms, like population dynamics or environmental conditions, are able to allow species to survive.

Alan Turing and G Evelyn Hutchinso



Even this, however, is difficult to prove or demonstrate, again as there are often differences in the specific resource complex that species depend on. But the problem becomes easier to address if the dimensions of the pattern in the ecological niche have a gradation, such as the increasing size of food items, where the different, near neutral, resident species differ in size and, hence, the items of food. Theoretical considerations predict that in such conditions, species would distribute themselves in specific size compartments, with a separation between one compartment and the next.

The principle behind such self-organisation, the *PLoS One* paper says, was first described by legendary mathematician Alan Turing in his paper, "The Chemical Basis of Morphogenesis". Turing considers a mathematically tractable case of a ring of living cells and shows that with the onset of any *instability* the ring may develop a wavy structure that is more persistent than the symmetric or homogenous, initial form. This might explain, for instance, the "tentacle pattern on *Hydra*, or for whorled leaves", Turing's paper says. Similar "self-organising" is in play, the *PLoS One* paper says, in the appearance of striped or dappled patterns in animal skin or in the distribution of desert vegetation, in the physical space, or, when there is competition, in the clubbing of species in the niche space. Robust "self-organisation" of this kind has been shown to exist analytically and mathematically and is also supported by some field data, the paper says. Nevertheless, the idea of "near neutrality" remains controversial as it seems to contradict the idea of a species occupying an exclusive "niche".

"Indeed, if self-organised similarity shaped much of nature's diversity we should fundamentally rethink the way we look at the ecological identity of species," the paper says.

Verification

Professor Marten Scheffer and others made use of massive global data of *Dytiscidae*, the *diving beetle* — beetles being the group of animals that have the greatest number of species found in nature. As the species share the environment, their distribution, both in local settings and in settings far removed, offers a view of how species evolve and an opportunity to test the theories that numerous competing species may organise themselves into regularly separated groups. The data was analysed in term of sizes of all known species of the diving beetle in different parts of the world, the pattern in size distribution of co-occurring species of beetles in 1,507 samples collected from ditches in Holland and also body size patterns in groups of species that have evolved independently while they were isolated for five million years in subterranean aquifers in Australia. Body size was the item of data considered because size would indicate the preferred dimensions of prey and, hence, tendency to compete. The data analyses then looked for similarities in the distributions in different groups, with the help of statistical data processing software.

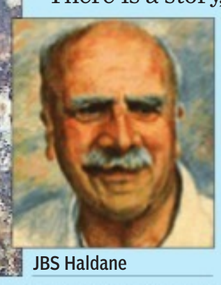
The result of the analysis shows with statistical rigour that body measurements are clumped around specific sizes so that there is reduced competition for resources between individuals in different groups. At the same time, there are large numbers of species that co-exist within each group. It is also found that species tend to be the same size or to be larger or smaller by a factor 1.3, a value that corresponds to the "limiting proximity" suggested by Hutchinson in his 1959 paper. The relationship is also found to be maintained in communities in different locations or with different evolution history.

The key finding, the *PLoS One* paper says, is that while similarity or dissimilarity, ie, the difference of 1.3 times, are common, intermediate size rations are rare. As the beetles are "generalist carnivores", the size ranges indicate, approximately, the "niche" that the groups of beetles occupy. The regularity of the size distribution, locally and long range, and their separation, hence shows that evolution drives species to either be sufficiently similar or sufficiently different, so that they do not compete. Size distribution was equally seen in the isolated communities that had evolved in cramped and resource-starved conditions. It was seen that fewer species survived, and their size-spacing was also more spaced out, which conforms to how it should be if there are fewer species to fit into the "niche-space".

In conclusion, the paper says, evolution seems to create "not only functional complementarity but also functional redundancy". While complementarity distributes the agents that carry forward the large interests of the community, the redundancy of several species occupying the same function makes the system resilient. Although the different species in a niche may be near neutral when it comes to competing for resources, they do have different resistance to stress, like from parasites or disease. An instance of diversity at the micro level!

THE WRITER CAN BE CONTACTED AT simplescience@gmail.com

Beetlemania



JBS Haldane

Hutchinson's paper contains this footnote: "There is a story, possibly apocryphal, of the distinguished British biologist, JBS Haldane, who found himself in the company of a group of theologians. On being asked what one could conclude as to the nature of the Creator from a study of his creations, Haldane is said to have answered, 'An inordinate fondness for beetles.'"

CELL CYCLE ARREST

THE P53 TUMOUR SUPPRESSOR GENE IS THE MOST FREQUENTLY MUTATED COMPONENT IN HUMAN CANCERS, WRITES TAPAN KUMAR MAITRA

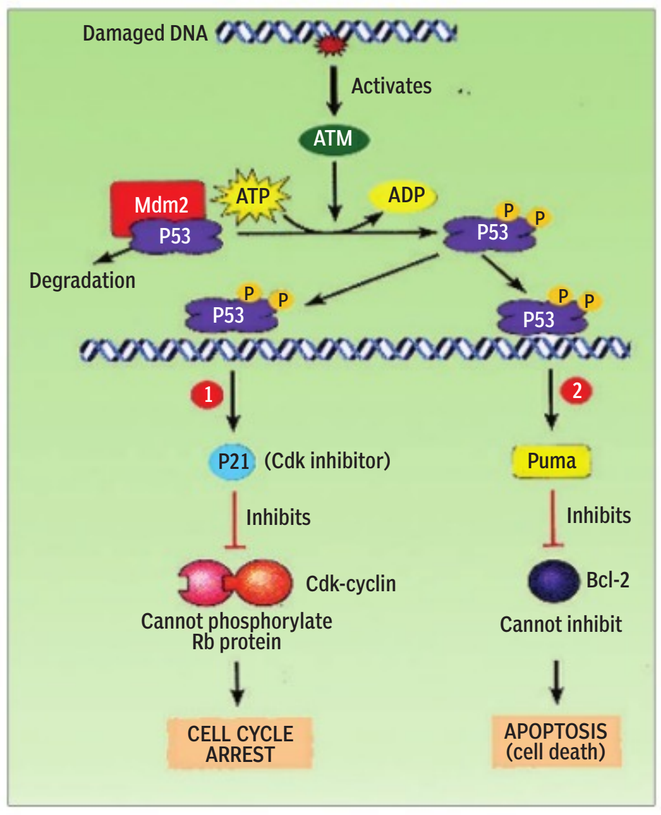
Since the discovery of the *RB* gene in the mid-1980s, dozens of additional tumour suppressor genes have been identified. One of the most important is the *p53* gene (also called *TP53* in humans), which is mutated in a broad spectrum of different tumour types. In fact, more than half the world's 10 million people diagnosed with cancer each year will have *p53* mutations, making it the most commonly mutated gene in human cancers.

The *p53* gene codes for the p53 protein, whose role is protecting cells from the effects of DNA damage. When cells are exposed to carcinogenic agents such as ionising radiation or toxic chemicals that cause extensive DNA damage, the altered DNA stimulates the p53 pathway, which then triggers cell cycle arrest and apoptosis to prevent the genetically damaged cells from proliferating. This protective mechanism is often missing in cancer cells because of mutations that disrupt the *p53* gene. The resulting inactivation of the p53 pathway contributes to cancer development by allowing the survival and reproduction of cells containing damaged DNA.

It is, therefore, not surprising that, as was the case for the *RB* gene, individuals who inherit a defective copy of the *p53* gene from one parent exhibit an increased risk of developing cancer. In this inherited condition, called the *Li-Fraumeni syndrome,* various types of cancer tend to arise by early adulthood as a result of mutations that inactivate the second, normal copy of the *p53* gene.

Mutations in *p53* are also common in non-hereditary cancers triggered by exposure to DNA-damaging chemicals and radiation. For example, carcinogens present in tobacco smoke cause several kinds of point mutations in the *p53* gene in lung cancers, and sunlight causes *p53* mutations in skin cancers. In some cases, mutation in one copy of the *p53* gene is enough to disable the p53 protein even when the other copy of the *p53* gene is normal. The apparent explanation is that the p53 protein consists of four polypeptide chains bound together to form a tetramer, and the presence of even one mutant chain in such a tetramer may be enough to prevent the protein from functioning properly.

Like the Rb protein, the p53 protein is a target for certain cancer viruses. For example, in addition to producing the E7 protein that inactivates Rb, the human papillomavirus has a second oncogene that produces the *E6 protein,* which binds to the p53 protein and targets it for destruction. This means that human papillomavirus can block the actions of the proteins produced by both the *RB* and *p53* tumor suppressor genes.



Damaged DNA activates the ATM protein kinase, leading to phosphorylation of the p53 protein. Phosphorylation stabilises p53 by blocking its interaction with Mdm2, a protein that would otherwise mark p53 for degradation. (The degradation mechanism is not shown but it involves Mdm2-catalysed attachment of p53 to ubiquitin, which targets molecules to the cell's main protein destruction machine, the proteasome.) When the interaction between p53 and Mdm2 is blocked by p53 phosphorylation, the phosphorylated p53 protein accumulates and triggers two events. The p53 protein binds to DNA and functions as a transcription factor that activates transcription of the gene coding for the p21 protein, a Cdk inhibitor. The resulting inhibition of Cdk-cyclin prevents phosphorylation of the Rb protein, leading to cell cycle arrest at the restriction point. When the DNA damage cannot be repaired, p53 then activates genes coding for a group of proteins that are involved in triggering cell death by apoptosis. A key protein is Puma, which promotes apoptosis by binding to, and blocking the action of, the apoptosis inhibitor, Bcl-2.

THE WRITER IS ASSOCIATE PROFESSOR, HEAD, DEPARTMENT OF BOTANY, ANANDA MOHAN COLLEGE, KOLKATA, AND ALSO FELLOW, BOTANICAL SOCIETY OF BENGAL, AND CAN BE CONTACTED AT tapanmaitra59@yahoo.co.in

Capturing actual motion

UNREALISTIC ANIMAL MOVEMENTS IN FILMS MAY SOON BE A THING OF THE PAST IF RESEARCHERS AT THE UNIVERSITY OF BRADFORD HAVE THEIR WAY, WRITES STEVE CONNOR

Actors are warned about working with animals but scientists have now found a way of working with cats, dogs and horses without the risk of unreliable results.

Researchers are fine-tuning the technology of motion capture for the film industry so that it can realistically portray the movements of four-legged creatures without the accompanying fears of being upstaged by unrealistic behaviour.

Although motion capture is used extensively to portray human movements in films and computer games, it is only recently being applied to the movement of animals, said Karl Abson, a motion-capture specialist at the University of Bradford. Actor Andy Serkis has portrayed the character of Caesar in the *Planet of the Apes* reboot series by using motion capture. Abson said that although films such as *War Horse* or *Life of Pi* used computer-generated animation to portray difficult scenes involving animal characters, it is expensive and often produces "stiff and unrealistic" animal movements.

Motion capture, however, is a way of building up a computer-generated image but based on the real movement of actors or animals that are filmed simultaneously in infrared light by several cameras positioned around the characters. Light reflected off a set of markers attached to an animal's coat or fur is used to generate a computerised image, with specialist software filling in the details or changing the overall look of the animal - a domestic cat for instance can be made to look like a tiger. Abson said, "We can't move a big cat because it will kill you, but maybe if we could motion capture a tame cat we can see differences that could alter the movement in a film's post-production, and it's surprising how well it worked.

"Motion capture on people has been done to death, animals not so much. The problem is that we've come really far in our ability to create skin textures and surfaces, such as the metal effect of Iron Man's suit. It can fool audiences but when it comes to motion we are still in the dark ages. When you come to animate something with motion, you always notice the slightest problem. The more you create something real, the harder it is". Abson and his colleagues at Bradford are working with post-production film companies in London, Hollywood and Moscow. The researchers are skilled in the movements of horses, and motion capture has been used to simulate jousting knights and racing chariots.

"We need a way of doing things that is not biased towards people. That is what we use motion capture for - we watch the data, we measure the data to see how animals actually move," Abson said.

He went on to say, "We have programmed the software to understand the quadruped and capture actual horse movement. For a film it is much more cost-effective and doable."

THE INDEPENDENT

Actor Andy Serkis has portrayed the character of Caesar in the *Planet of the Apes* series by using motion capture.

PLUS POINTS

Targeting dengue

Researchers at a Malaysian university have built an LED street lamp with a mosquito trap, both powered by wind and solar energy. The lamp can offer communities across the developing world clean power to light their streets and protection from mosquito-borne diseases such as dengue fever, they say.

Chong Wen Tong, a mechanical engineer at the University of Malaya in Kuala Lumpur and the project's principal investigator, says the integrated Eco-Greenery Lamp can fight mosquitoes while reducing greenhouse gases. According to him, the lamp's wind turbine is suitable even at the "low and unsteady" wind speeds sometimes found in the tropics.

The trap relies on the mosquitoes' natural attraction to carbon dioxide, which is breathed out by their human prey. The trap produces this gas to lure in the insects and then a fan prevents them from escaping. This feature is designed to reduce the prevalence of dengue fever, which is transmitted by mosquitoes and whose prevalence is growing in Malaysia. The lamp cost $30,000 to develop and pilot over 18 months and can be sold for use in "suitable locations all around the world" that have the infrastructure in place to set up and maintain the device, Chong says.

SCIDEV.NET

Protein dispute

A protein called Growth Differentiation Factor 11 has been at the centre of a tug of war: some scientists have provided evidence that it declines with age and that returning GDF11 to youthful levels can fix a heart problem in mice. Yet others have found no such relationship, and one group published evidence this year that GDF11 actually increaded with age.

To figure out why there have been discrepancies in the data, Richard lee of Harvard Medical School and his colleagues — the team that found GDF11 dropped with age — scrutinised a conflicting study and found the likely explanation to be a case of mistaken identity. Rather than measuring levels of GDF11, Lee's group found that the other researchers were likely measuring immunoglobulin light chain. "It's a misinterpretation," Lee said.

Novartis Research Institutes, which led the study that found GDF11 levels rising with age, did not respond to requests for comment. According to the Lee group's latest paper, the difference might be due to variability in the concentration of GDF11. Originally, Lee's team used a concentration of 0.1 mg/kg, but it turns out that the manufacturer had sent the group variable concentrations. Now, the Harvard researchers have reported that a concentration of 0.5 mg/kg is the effective dose.

THE SCIENTIST

Plague sequenced

Plague bacteria (*Yersinia pestis*) were recently discovered in Bronze Age skeletons, pushing back the recorded origin of plaque by about 3,000 years. The newfound ancient strains are genetically distinct from modern-day strains. An international team reported its findings on 22 October 22) in *Cell*.

Researchers led by Eske Willerslev of the University of Copenhagen were searching for the cause of Bronze Age human migrations in Europe and Asia, circa 3,000 BC-1,000 BC, suspecting that disease might have been a driving factor. So they sequenced 89 billion DNA sequences from 101 Bronze Age specimens obtained from museums and excavations. Seven of the specimens contained *Y. pestis* DNA. Most of the ancient bacterial genomes lacked a gene known to protect the pathogen while it was inside the flea gut, suggesting that the plague was not spread easily by fleas. The ancient *Y. pestis* bacteria also lacked a mutation that helps present-day strains avoid detection by the host's immune system. The authors suggest that *Y. pestis* did not evolve into its destructive flea-borne form until the beginning of 1,000 BC.

"It's really cool that they can pinpoint the acquisition of key genes that allow the movement of this bacteria into fleas," evolutionary geneticist Hendrik Poinar of McMaster University in Canada, who was not involved with the study, told *Science*.

"At that time we have a kind of intermediate plague," study co-author Simon Rasmussen from the Technical University of Denmark told *Smithsonian*. "These Bronze Age strains couldn't cause bubonic plague, but they caused septicemic plague in the blood and pneumonic plague in the lungs, which you can transmit through the air whenever you sneeze or cough."

KAREN ZUSI/THE SCIENTIST