



Vigyan Prasar

DREAM 2047

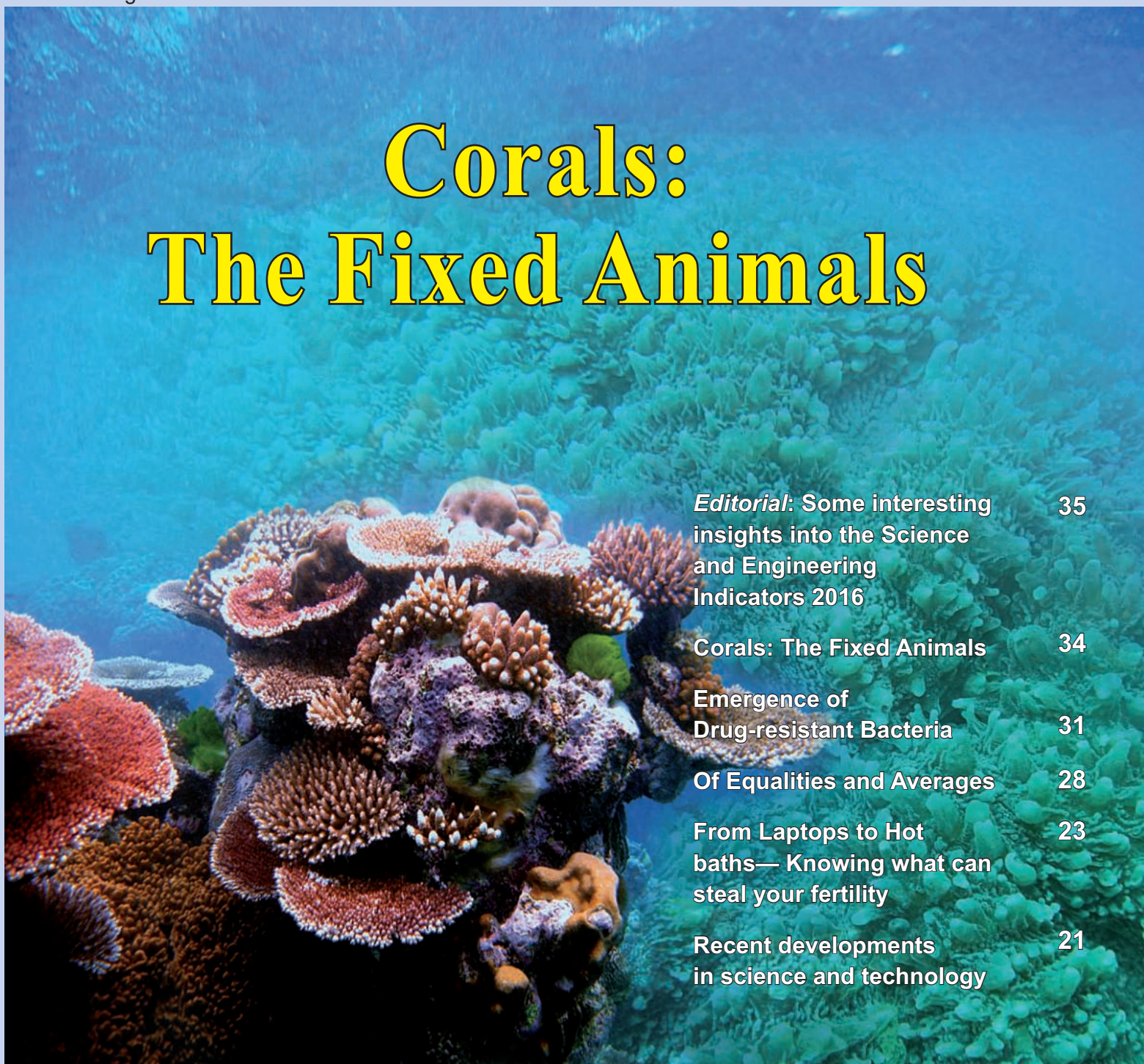
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Corals: The Fixed Animals



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... think scientifically, act scientifically... think scientifically, act scientifically... think scientifically, act...

Some interesting insights into the Science and Engineering Indicators 2016



Dr. R. Gopichandran

The National Science Board, United States of America, has recently published the Science & Engineering Indicators 2016¹. Chapter 7 of the publication is about Science and Technology: Public Attitudes and Understanding. It discusses such indicators as interest in discoveries, access to and use of internet, newspapers, television, factual knowledge and ability to consistently comprehend several layers of information, belief on benefits of science and related confidence, priorities, choice and preparedness to use alternatives and response to contentious issues. Confidence on the scientific community and willingness to allow investments/public funding for scientific pursuits are two other indicators. Important inferences are derived through comparisons that span over three decades. They are comparable also with trends/insights from Eurobarometer and Canada reported two years ago, on science culture.

The report however categorically states, and rightly so, the limitations that permeate documentation and analyses of empirical evidences. These include spread and depth of information presented, articulated, documented and interpreted and the influence of socio-cultural characteristics and economic constraints that determine quality of life that influence perceptions at both ends. Based on these learnings, it will be useful to examine inferences derived through studies, by the CSIR - National Institute for Science Communication and Allied Information Resources in India on related aspects.

The most important take-away for researchers in this field of study is the need to exercise highest levels of caution in interpreting and communicating inferences. Such aspects as prior knowledge about topics and the limits and limitations of evidences that can influence decisions are critical. These include the ability of respondents to even comprehend the relevance of questions and more importantly the consequences of their

responses on inferences. The academies of science, engineering and medicine in the United States of America rightly asked about the 'limits of scientific evidence in policy areas'.

This takes us to the next level of complexity including policy intent, plans to transform intent to real benefits and programmes/projects that sustain delivery where rubber meets the road and hence the quality of life. Perceptions about benefits and therefore interests on science and technology can be justified through correlations on actual benefits delivered and accessed. Such qualitative aspects as beliefs and superstitions are probably less amenable to quantitative dynamics and therefore should feature much lower in the order of preferences to research.

Researchers in the field of science journalism and communication will like to take note an interesting paper published recently. The paper was authored recently (2016) by Désirée Motta Roth and Anelise Scotti Scherer². They indicate, access to scientific knowledge by common citizens and their interest in science are not linearly aligned. They on the other hand indicate that interests could be directly influenced through science education.

References gathered on 13 July 2016: Sources indicated.

1. Science and Technology: Public Attitudes and Understanding, National Science Board, Science & Engineering Indicators 2016, 101p <http://www.nsf.gov/statistics/2016/nsb20161/uploads/1/10/chapter-7.pdf>
2. Désirée Motta Roth & Anelise Scotti Scherer 2016 Science Popularization: Interdiscursivity among Science, Pedagogy, and Journalism [http://dx.doi.org/10.1590/2176-457323671Bakhtiniana, São Paulo, 11 \(2\): 171-194, May/Aug. 2016](http://dx.doi.org/10.1590/2176-457323671Bakhtiniana, São Paulo, 11 (2): 171-194, May/Aug. 2016).

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Corals: The Fixed Animals



Ratnadeep Banerji

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What kind of organisms are corals? You are likely to think of corals as plants because they remain fixed to rocks, are colourful to look at, and many of the branched ones resemble plants. Till the 18th century, people largely believed corals to be plants. However, much earlier in the 11th century the Persian polymath, Al Biruni had recognised corals to be animals by noticing their reaction to tactile sensation.

India hosts more than 60% species of corals in global scenario with the majority of the species being recorded from Andaman and Nicobar Islands. A total of 464 species corals belonging to 72 genera and 17 families have been reported from Indian seas.

Classification

Corals are essentially marine invertebrates belonging to the class Anthozoa of phylum Cnidaria. The simplest tissue grade of organisation can be seen in the cnidarians (radially symmetrical animals having saclike bodies with only one opening and tentacles with stinging structures). Anthozoa is the most primitive group among the cnidarians and poses difficulty in systematics due to their diverse form and simple morphology that poses difficulty in comparing taxa. Corals are completely fixed and hence differ from most other cnidarians in not having a medusa (free-swimming sexual phase) in their life cycle.

Corals are divided into three subclasses – hexacorallia, octocorallia and ceriantheria. The Hexacorallia include polyps generally with 6-fold symmetry and include the stony corals. Octocorallia has polyps with 8-fold symmetry, each polyp having eight tentacles and includes blue corals and soft corals. These are rather primitive members. Ceriantheria are the tube-dwelling anemones, are not anemones but are a type of corals. Fire corals are not true corals as they belong to the class hydrozoa.

Structure

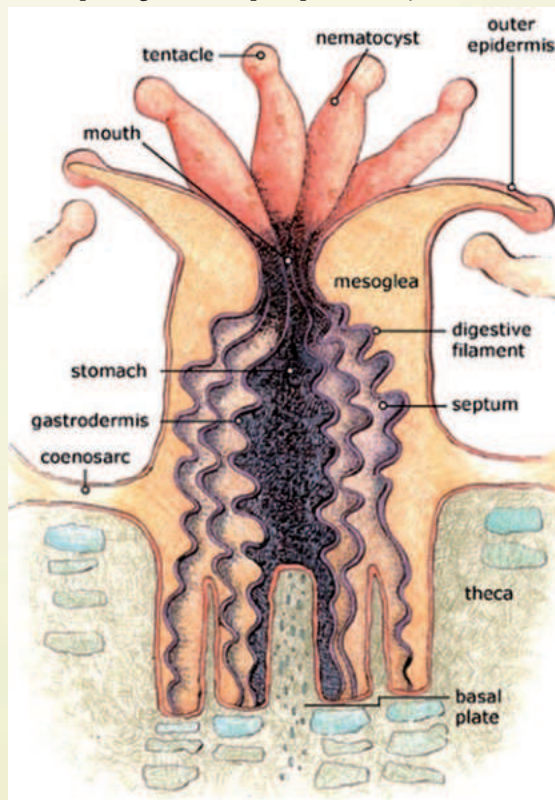
Polyps are the individual coral animal entities. They are sac-like animals measuring a few millimetres in diameter and a few centimetres



Lobophyllia dentatus was first recorded of its existence in India in 2014 by Tamal Mondal, C. Raghunathan and K. Venkataraman. (Courtesy – Zoological Survey of India)

in length. They give rise to densely built colonies consisting of genetically identical polyps.

Each polyp is a hollow cylinder of tissues with tentacles and a central mouth opening at the top. Open at only one end,



Cross-section of stony coral polyp

the polyp takes in food and expels waste through its mouth. Their bag-like body is composed of two cell layers, outer ectoderm or epidermis and inner endoderm or gastrodermis which is further subdivided into partitions. There is an intermediate layer of translucent jelly-like substance called 'mesoglea' made up of scattered cells and collagen fibres. The central part of the cylinder has filaments called mesenteries. They serve like the stomach to absorb the food and also have gonads that produce male and female gametes. All corals have stinging cells called nematocysts in their tentacles near

the mouth.

Coral skeleton

The stony corals secrete calcium carbonate to form a hard exoskeleton. They are the reef builders. The soft corals do not produce any stony skeleton and are not reef builders in a major way. However, their tissues are toughened by the presence of tiny skeletal elements of calcium carbonate called sclerites. Soft corals have chemicals called terpenoids in their tissues as the toxic substance for defence.

The term coral usually denotes the skeleton. A coral animal, also called a polyp, constructs a skeleton around its body for protection. Calicoblasts are special cells on the external surface of the polyp that can assimilate dissolved calcium from seawater to construct the aragonite (a form of crystalline calcium carbonate) skeleton. The skeleton thus looks like a cup with side walls, a bottom and open at the top. Inside the skeleton, the polyps sit this cup-shaped depression known as corallite.

Coral colony

Most corals happen to be colonial. The corals that you see are colonies of polyps and are not a single coral. A cursory inspection would reveal numerous pores



on it. Each pore shows where one individual polyp lives. Most corals are colonial, the initial polyp budding to produce another and the colony gradually develops from this small start. In a coral colony, a thin layer of living tissue overlying the stony skeletal material known as coenosarc joins the adjacent polyps to each other.

Generally the shapes of corals are massive stone-like, leaf-like, branching tree-like, flower-like or encrusting coating-like. The water depth and sunlight often control their shapes. At greater depths, higher pressure flattens the coral skeleton.

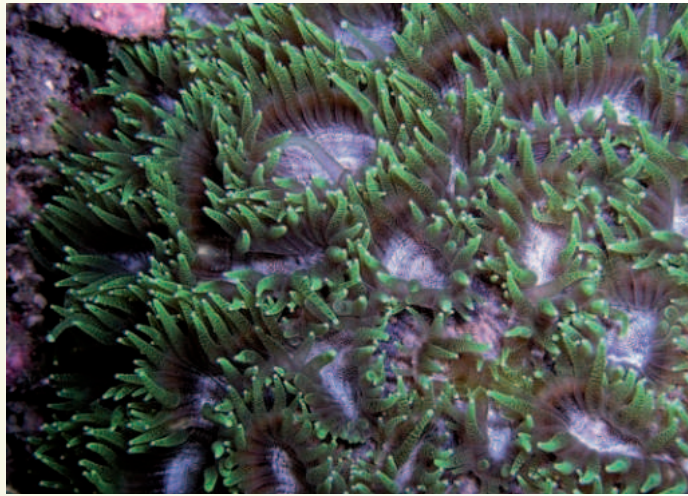
All reef-forming corals have microscopic photosynthetic algae called zooxanthellae living inside their cells and are called hermatypic corals seen only in tropical and subtropical seas. These algae, zooxanthellae have chlorophyll pigments for photosynthesis. So the coral has to remain near the surface where the sunlight is abundant. The colour of the corals, in fact, comes from these algal pigments.

Other corals, called ahermatypes, do not have zooxanthellae. Therefore they do not need light and can live in deep waters up to the depths of 1,000 or 2,000 metres. These corals generally lead a solitary life or form a very small colony. These corals occur in all latitudes.

Nutrition and feeding habits

All corals are nocturnal, becoming active feeders at night, when planktons and small organisms come to the surface layer from deep waters where they remain during daytime. During night, the corals are highly active with spread out tentacles unlike daytime when they are withdrawn into their skeletal cavities. Corals feed on microscopic zooplankton which drifts along with currents. When near, the corals catch the drifting zooplankton with their tentacles, sting them with their stinging cells called nematocysts, paralyse them by injecting venom and swallow them.

Besides this form of eating, hermatypic corals derive nutrition from zooxanthellae algae which



Favites flexuosa, hard coral with extended polyps at night to feed
(Courtesy – Nhbogood Nick Hobgood)

live symbiotically with polyps. These photosynthetic unicellular dinoflagellate algae of the genus Symbiodinium living within the tissue of the polyps produce food matter by photosynthesis part of which is passed on to corals through a complex and well-developed system of gastrovascular canals. The coenosarc (the living tissue overlying the stony skeletal material of the coral) contains the gastrovascular canal system that stands interconnected among polyps and allow them to share nutrients with symbiotic zooxanthellae. These zooxanthellate corals, like animals, excrete nitrogen and phosphorous salts and carbon dioxide needed for photosynthesis by algae and are readily taken up by them. Such corals require sunlight and grow in clear, shallow water less than 60 m deep. Thus the corals supplement their plankton diet with the products of photosynthesis produced by zooxanthellae algae.



Aerial view of coral spawning slick-Wildlife Trust of India

The presence of zooxanthellae also enhances coral growth. When they photosynthesise, they remove carbon dioxide. This reduces the acidic conditions at the sites where calcium is deposited by the corals. This in turn retards calcium dissolution, thus enhancing precipitation of calcium and coral skeletal growth.

Reproduction

Polyps reproduce both asexually and sexually. Budding or 'gemmation' is the asexual method of reproduction. Each polyp is a hermaphrodite and can produce both male and female gametes.

Generally around full-moon nights, the corals simultaneously release both male and female gametes in the ocean. Though there are no sexual manifestations, this synchronous spawning is very typical on coral reefs, and often, even when multiple species are present, all corals spawn on the same night. The spawning event is visually spectacular phenomenon of sexual reproduction with mass expulsion of colourful eggs and sperm clouding of the usually clear water, creating a colourful slick with gametes.

Spawning normally happens when the water is warm, and when the tide is changing from high to low. Different species spawn at different times and follow different patterns.

The gametes unite and produce a larva called planula, typically pink and elliptical in shape. A typical coral colony gives rise to several thousand larvae in a year to survive the odds. The planulae swim around for a few days, looking for a hard substratum to finally grow by budding into a colony.

Incredulous enough, planulae exhibit positive phototaxis – the tendency of swimming towards light to reach surface waters, where they drift and grow before descending till it anchors on a hard surface. They also exhibit positive sonotaxis – the property of moving towards sounds that emanate from the reef and away from open water.

Reef building

Only hermatypic corals form coral reefs. The formations can be fringing reefs, barrier reefs or atolls.

The greatest number of corals is found in the tropical belt, with a decrease towards the subtropical seas. The reef building corals require warm temperatures and sunlight and thus are found in the shallow waters. They grow well at temperatures greater than 20°C and can thrive even at 35°C, as in the Red Sea, Persian Gulf or in our Gulf of Kutch. Corals also need a requisite amount of salinity of 35 grams of salts per litre of water.

Survival conditions

When waters remains turbid, inorganic particles keep falling upon corals although while they try to clean themselves by ciliary movements. But at high turbid conditions, the cleaning mechanism proves insufficient and the corals eventually die of smothering. The sedimentation also has an indirect effect of reducing light penetration in the sea, thus reducing photosynthesis by zooxanthellae and the coral growth, although it does not completely kill the corals.

Water temperature changes of more than 1 to 2°C or salinity changes can kill some species of corals. Under such environmental stresses, corals expel the algae Symbiodinium from their body. This is called coral bleaching wherein the coral tissues reveal the white of their skeletons.

Coral growth depends on favourable climate; hence they act as potent indicators of climate change. However, it has been seen that in many cases, corals are adapting to climate change. These are usually due to a shift in coral and zooxanthellae genotypes. These shifts in allelic frequencies have progressed toward more tolerant types of zooxanthellae. Scientists have found that a certain zooxanthella associated with stony corals is becoming more common where sea temperature is high. Symbiodiniums are able to tolerate warmer water seem to photosynthesise more slowly, implying an evolutionary strategy.

Medical use

A substance for use as bone transplant should have nearly the same chemical composition as the bone, to give mechanical strength and at the same time remain porous enough to allow fine blood vessels to pass through.



Caulastrea echinulata was first recorded of Indian presence in 2012 by Tamal Mondal, C. Raghunathan and K. Venkataraman. (Courtesy – ZSI)

Among the several bio-materials, some corals like the massive Goniopora and Isididae offer befitting choices. Either a bone-shaped structure from the coral is cut and used as a transplant or better still; the skeletal material is converted to hydroxyapatite, which is the

mineral component of the bone. This forms a bond directly with the bone and hence can be used as a bone-replacing material which upon implantation promotes new bone growth. Coral skeletons also find use in filling of dental cavities.

There's a misbelief in India over the origin of red coral (red opal, *moonga* in Hindi *pala* in Bengali) widely used in jewellery. Many people tend to think it to be unique to India. The red coral, *Corallium rubrum*, is not a reef coral at all. It is a stony coral without zooxanthellae. The red coral occurs only in the Mediterranean Sea and around Japan. The red colour comes from natural pigmentation of the skeleton that remains even after the death of the polyp. Again, unlike the reef corals that are porous with cavities in the skeleton, the red coral is dense and compact. So, it can be shaped or machined without breakage and are shaped for jewellery.

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Workshop on Writing Science



3rd – 15th October 2016, IISER Thiruvananthapuram
Catalysed by Vigyan Prasar and
organised by IISER-Thiruvananthapuram

Eligibility: Post graduates in any branch of science OR Post graduates in Mass Communication/Journalism, aspiring to contribute scientific content to print media in English and other languages of India. (Those who are in the second year of PG can also apply).

Minimum requirements for participation: ability to understand and speak English, the language of the workshop, ability to write using word processors and prior exposure to internet browsers and search engines. Demonstrable ability to write in any Indian language would be an advantage. The workshop, compulsorily residential, will have lectures, presentations, discussions, role-play, demonstrations, hands-on practice, feedback, mentoring and highly interactive sessions. The focus will be on work done as individuals and in teams.

Maximum number of participants for the workshop is limited to 14. Boarding and lodging free for selected candidates. Re-imbursement of the cost of travel by 2nd AC train fare only for deserving candidates. No registration fee.

To get the application form, send an e-mail request to scienceandmediaworkshops@gmail.com

Last date for receipt of application: 29th August 2016.

Emergence of Drug-resistant Bacteria



M.S.S. Murthy

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Since the discovery of the first antibiotic penicillin by Alexander Fleming in 1927, scores of antibiotics have been developed to treat almost any type of bacterial infection. Health experts estimate that antibiotic drugs have added an average of 20 years to our lives. Yet, today “antibiotic resistance will turn common infection into incurable killers and make routine surgeries a high-risk gamble” warns the World Health Organization (WHO). Antibiotic resistance is the resistance that bacteria develop to a drug that was originally effective against it. Resistance helps the bacteria to remain unaffected by the drug, rendering the standard treatments ineffective. So, antibiotic resistance means that certain bacteria become resistant and help spread the illness.

When a class of bacteria becomes resistant to a particular drug, the pharmacologist develops a new kind of antibiotic. In due course the bacteria may become resistant to even the new drug. Thus, over a period of time multi-drug-resistant bacteria develop, making it a serious public health problem. The evolution of multi-drug-resistant tuberculosis is a classic example.

Extent of the problem

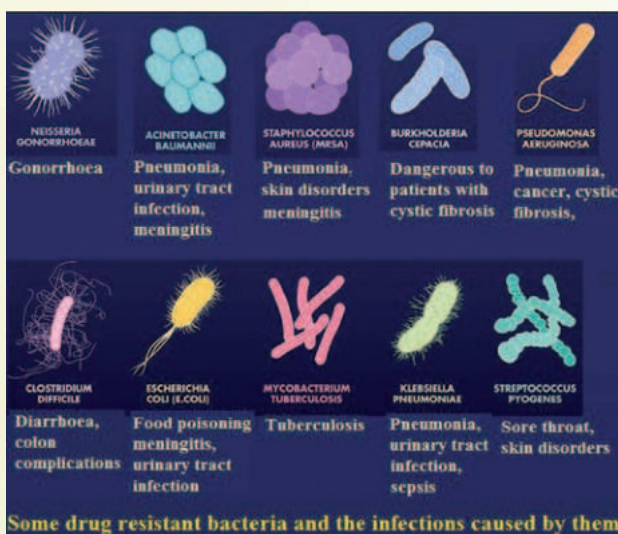
The *Indian Journal of Cancer* reported in 2013 (Vol. 50, pp 71-73) that Indian hospitals experience a high prevalence of gram-negative bacteria resistant to the broad spectrum beta-lactam antibiotics.

More recently, the national daily *The Hindu* (29 November 2015) carried a front page report about the emergence of drug-resistant bacteria in hospitals across the country. Doctors reported that a pathogen called *Klebsiella pneumoniae*, which causes urinary tract infection, pneumonia and sepsis, has become resistant to carbapenems and colistin – considered to be the antibiotics of “last resort”. The report pegs the resistance to as high as 50 percent in some hospitals, leading to high mortality rates. The situation is particularly bad for lymphoma

and transplant patients with compromised immunity.

Mycobacterium tuberculosis is another drug-resistant bacterium which is bothering public health authorities, particularly in the Third World countries. It is reported that tuberculosis kills about 17 lakh people around the world, of which three to four lakh deaths occur in India due to the presence of resistant strains like multi-drug-resistant TB (MDR-TB) and extremely drug-resistant TB (XDR-TB). Now, Hinduja Hospital, Mumbai has reported the isolation of yet another resistant strain known as totally drug-resistant TB (TDR-TB), which is found to be resistant to 12 drugs

while investigating the epidemiology of antimicrobial resistance (resistance not only to bacteria, but also to other microorganisms like viruses, fungi, etc.) found that the biological samples collected in 1966 from a patient of a North Carolina hospital, was infected with *Klebsiella pneumoniae* carrying a gene called KPC (*Klebsiella pneumoniae* carbapenemase), which rendered the bacteria resistant to carbapenems and a number of other antibiotics. In 2003 the KPC-positive strains were found spreading rapidly through hospitals across the New York City. By 2007, twenty-one percent of the *Klebsiella pneumoniae* in the city carried the resistance gene. By 2008 the KPC-positive bacteria had made their way from New York to many other countries including Israel, Italy, Colombia, the UK, and Sweden. Three years later doctors in Sweden found a new carbapenems resistance gene named NDM (New Delhi-metallo-beta-lactamase-1, which also inactivates a broad range of beta-lactam antibiotics), which was traced back to New Delhi. (That India protested against linking New Delhi with the gene is another matter). From then on NDM-positive strains have moved out quickly to other places.



How does antibiotic resistance arise?

Emergence of antibiotic resistance has become a global problem, increasing every year at an accelerating rate. At least 20 lakh people are infected with antibiotic resistant bacteria each year in USA and nearly 23,000 die of the infection. In the European Union, an estimated 4,00,000 are infected by drug-resistant bacterial strains, of which about 25,000 die. According to a report published in *Nature* (8 May 2014), the problem is more acute in BRIC countries – Brazil, Russia, India and China.

The global nature of antibiotic resistance is very well illustrated by the case of *Klebsiella pneumoniae*. In 2000, researchers at the Centers for Disease Control, USA,

To understand how bacteria acquire resistance to antibiotic drugs, we may first review how antibiotics work. They are designed to block some essential steps in the life cycle of the bacteria and prevent their further growth and survival. These include cell wall synthesis, folic acid synthesis, DNA replication, RNA and protein synthesis. Since many of these steps are common to the host cells also (human and animal cells), the targets chosen are specific to the bacteria, so that the drug may not harm the host cells. For example, unlike human and animal cells, bacterial cells have a thicker cell wall. Antibiotics of the class beta-lactams (which includes penicillin) bind and inactivate an enzyme (called ‘penicillin binding protein’-

PBP) which is essential for the synthesis of cell wall. Without a robust cell wall a bacterial cell cannot survive.

Folic acid biosynthesis is another example. Folic acid is required by both bacteria and humans for the synthesis of nucleic acids and proteins. While humans can use preformed folic acid, bacteria cannot. They synthesise their own folic acid. An important starting compound for the synthesis of folic acid is para-aminobenzoic acid (PABA). Sulfonamides and other sulfa drugs are analogous to PABA and bacteria cannot distinguish between the two. These compounds compete with PABA in the biochemical reactions and block the synthesis of folic acid. When that happens the bacteria cannot survive.

One of the essential steps in DNA replication prior to cell division is to unwind the double stranded DNA. This is carried out by an enzyme called DNA gyrase. Fluoroquinolones, another class of antibiotic, bind to bacterial DNA gyrase and inhibit DNA replication, preventing bacterial growth. Similarly, Rifamycins inhibit RNA synthesis with the same end result. Ribosomes are structures on which protein synthesis takes place. Tetracycline, Erythromycin and similar group of antibiotics bind to ribosomes to prevent protein synthesis.

Scientists point out that the evolution of antibiotic resistance in bacteria is a natural process. When bacteria replicate by billions, some may undergo mutations – changes in their genes. Some of these spontaneous mutations may confer resistance traits to the bacteria, so that they can thrive even in the presence of antibiotics. The resistance itself comes in many forms. For example, a resistant bacterium may produce an enzyme that can bind to the drug and make it ineffective.

Some penicillin resistant bacteria produce an enzyme called beta-lactamase, which deactivates penicillin. Alternatively, the target itself may be altered so that the drug may no longer be able to bind to it. This can be seen in another type of penicillin-resistant bacteria where the structure of the binding site is altered. Resistance to sulfonamide arises when bacteria develop the ability to utilise the pre-existing folic acid rather than synthesising it.

There are other mechanisms through which bacteria may develop drug resistance. When a bacterium dies, it breaks up and releases its DNA to the surrounding environment. Other bacteria may scavenge these free-floating DNA pieces and incorporate them into their own genome. If the incorporated DNA contained an antibiotic-resistance gene, the recipient bacteria too acquires that property.

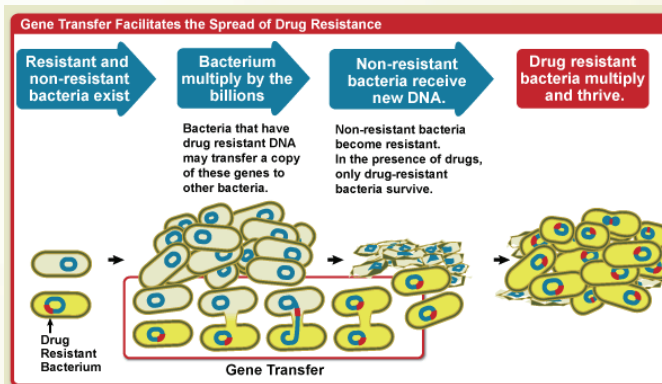
so that the progeny will also be resistant to the drug.

Recent research has shown that in a mixed population of resistant and susceptible bacteria, the highly resistant mutants can, at some cost to themselves, provide protection to the vulnerable cells by releasing a signalling molecule called indole. It appears that indole turns on efflux pumps and oxidative-stress protection mechanism in the vulnerable ones, thus enhancing their survival capacity in the presence of an antibiotic drug.

Thus in a large population of bacteria there will always be a few that have developed resistance to antibiotics. When an infected person is treated with antibiotics, the susceptible ones perish, leaving behind the resistant ones, which will multiply at an opportunistic moment. Next time when the same antibiotic is given to the patient, it may not be effective in controlling the infection. What worries the doctors is the rapidity with which drug-resistant bacteria can spread through the contaminating equipment or hands of care givers in the hospital environment or outside.

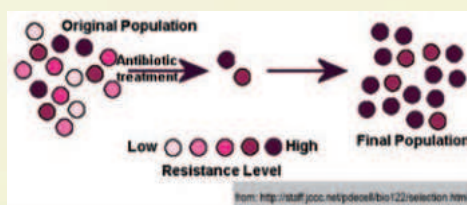
Though antibacterial resistance is a natural phenomenon, there are several human activities which accelerate the process. One of the most cited examples is the overuse or misuse of antibiotics. In many countries (including India), these drugs are sold over-the-counter without prescription. Even in countries where prescription is required, many patients think that when they are ill, antibiotics is the solution and pressurise their physicians to prescribe them. But antibiotics work only for infections caused by bacteria and not other microorganisms like virus, fungi, etc. At other times, even when prescribed with proper evaluation, patients may not complete the course because they start feeling better. All these lead to antibiotic resistance.

When a person is treated with antibiotics only about 30 percent of the drug is absorbed and the rest goes to the sewage system. In addition, antibacterial soaps and disinfectants used in homes and hospitals are also washed into the sewer. They are not readily degradable. Ultimately the sewage enters a treatment plant, which encourages the growth of bacteria to digest the sewage. During this process, in the presence of low



The process is known as “horizontal gene-transfer”. Penicillin-resistant *Niesseria gonorrhoea* results from such a process.

Another example of horizontal gene transfer is resistance acquired through plasmids. In addition to their own chromosomal DNA, bacteria contain another DNA entity called ‘plasmids’. Plasmids are circular DNA strands capable of replication, independent of the bacterial DNA. A unique property of plasmids is that they can flit from one bacterium to another, sometimes even across species. Plasmids contain many genes, some of which may render the bacteria resistant to specific antibiotics. In such a case, the recipient bacteria also become resistant to that antibiotic. A single plasmid can provide a slew of different resistance genes. Bacterial species *Klebsiella pneumonia*



are known to have picked up the resistant KPC gene through a plasmid. The gene produces the enzyme *Klebsiella pneumoniae* carbapenemase, which breaks down the drug carbapenems. Similarly, viruses may act as a vehicle for horizontal transfer of genes. The interesting aspect is that, all these resistance mechanisms involve genetic modifications

BOX: What you can do

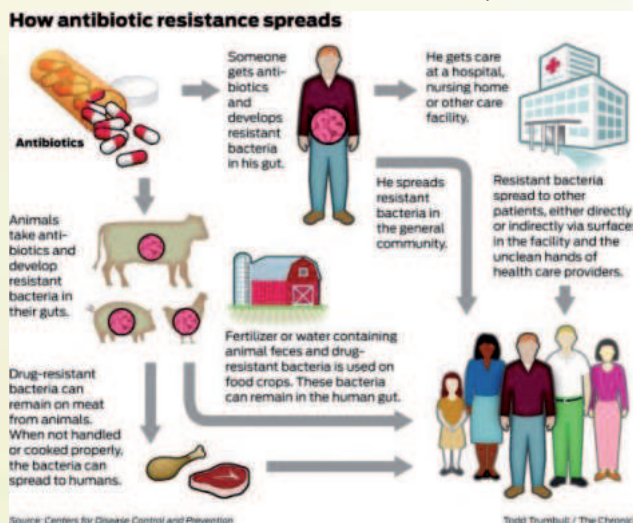
As an individual	As a health worker /care giver
1) Only use antibiotics when prescribed by a certified health professional.	1) Prevent spreading of infection by ensuring your hands, instruments and environment are clean.
2) Always take full prescription, even if you feel better half way.	2) Keep patients' vaccine up-to-date.
3) Never use leftover antibiotics.	3) If you think a patient may need antibiotics, where possible, test to confirm and try to find out which one.
4) Never share antibiotics with others.	4) Only prescribe and dispense antibiotics when they are truly needed.
5) Prevent infections by regularly washing your hands, avoiding close contact with sick people and keeping your vaccinations up-to-date.	5) Prescribe and dispense the right antibiotics at the right dose for the right duration.

levels of antibiotics, some bacteria may develop resistance. When the digested sludge is dried and used as manure, some of the farm products may get contaminated with bacteria and enter the food chain. In addition sewers may directly contaminate drinking water system, as often happens in our country. Both help the spread of resistant bacteria in the community.

Another important area is the rearing of livestock where antibiotics are used (and misused) on healthy animals, not only to prevent diseases but also make them gain weight at a rapid rate. The residues of these may reach humans through improper handling and cooking of meat. Even high milk-yielding cows like Holstein-Friesian and Jersey are fed with antibiotics to keep them from falling sick in the Indian environment. According to a recent survey conducted by the Tamil Nadu Veterinary and Animal Sciences University, milk from these cows showed much higher levels of antibiotics than permitted.

Recognising the seriousness of the problem, medical societies in India organised a joint meeting in August 2012 in Chennai to address the problem of antibiotic resistance. It looked at all aspects of the problem and suggested solutions in what is called "The Chennai Declaration: A road map to tackle the challenge of antimicrobial resistance". However, the Government of India is yet to come out with a national policy to contain the antimicrobial resistance.

At the international level, the World Health Organization has developed a draft action plan to combat antimicrobial resistance which has been recently submitted to the 68th World Health Assembly. It also



observed World Antibiotics Awareness Week from 16 to 22 November 2015 to increase public awareness and suggests what everyone can do to help contain the problem of antibiotic resistance (See Box).

Newer approaches to treat bacterial infection

As long as the effectiveness of the drug is based on chemical processes, there will always be bacteria that develop resistance to the process. Hence, departing from the conventional approach, some researchers are trying to develop next generation antibiotics that may attack bacteria through physical or mechanical means. Scientists at the University of Pennsylvania in USA are trying

to design protein molecules which, by the virtue of the electric charge on them can bind to bacterial cell walls and bust them in ways similar to 'defensins' (small cysteine-rich cationic proteins found in both vertebrates and invertebrates) – a component of our immune system – works.

Scientists at the IBM research centre are developing organic nanoparticles that function in a similar way. These particles are so designed that they are physically attracted to the bacteria like a magnet, break through the cell wall and destroy them. Clinical trials are under way in China. Researchers hope that they can be incorporated into soaps, deodorants, hand sensitizers and lotions.

Researchers at the Gamaleya Institute of Epidemiology and Microbiology, Moscow have developed cold plasma torch that is found effective against two bacterial types – *Pseudomonas aeruginosa* and *S. aureus*, which show up frequently in infected wounds. In preliminary experiments on rats with infected

wounds, exposure for ten minutes killed 90 percent of the bacteria. The plasma, interacting with the tissue releases reactive oxygen species which are lethal to the bacteria. Not only that; it also accelerated wound healing. The other encouraging factor is, these methods do not distinguish between resistant and non-resistant bacteria, but prove equally lethal to both.

Meanwhile, Longitude – a charitable organisation based in London has instituted a ten million Pound prize to whoever can develop "a point-of-care diagnostic test that can conserve antibiotics for future generations and revolutionise the

delivery of global health care. The test must be accurate, rapid, affordable, easy to use and available to anyone in the world. It will identify when antibiotics are needed and, if they are, which one to use". The entry is open until 31 December 2019.

Drug-resistant bacteria have evolved into a serious community health problem. Will man be able to conquer the *Bhasmasura* he has himself created through the indiscriminate use of antibiotics? Time only will tell.

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Of Equalities and Averages



Bhupati Chakrabarti

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Cook books often come up with recipes which state, “take equal amounts of say, sooji and sugar”, and in television shows on cooking that are quite popular we often



School children from the same grade vary in heights and weights

see how these so-called ‘equal’ amounts are taken. The question we normally do not ask is “do we need to take equal amounts by mass (weight) or by volume?” People often feel this is not a pertinent question as the equality is considered to be sacrosanct. However, if we do not mention whether the amounts were selected by volume or by mass then the two approaches of making the things equal or taking them in a particular proportion actually may not lead to the same result.

Sometimes taste of the final product may not differ much, but if you put this situation to test you will find that things do differ. For example, one litre of edible oil weighs between 900 grams and 920 grams whereas one litre of water weighs one kilogram. So if we suggest we are taking equal amounts of water and oil in two vessels one very legitimate question would be: are they equal by volume or by mass? In fact there are more issues. We know temperature plays a role if we are talking about volumes, particularly for gases and to some extent for liquids. As density varies with temperature and strictly speaking we need to mention the temperature while making statements involving the volumes of gases and, may be, for liquids also. If you are asking for equal amounts of water and oil you will have to

mention whether they are equal by weight or by volume and the temperature also needs to be mentioned for a rigorous statement. In our everyday life we come across large number of equalities where the inequalities exist side by side. Two boys studying at the same grade may be equal in height but likely to differ in weight or may be in age; two books may have equal number of pages but may differ in total number of words.

Sometimes, while taking the so-called equal amounts, people feel it may be important to mention if that equality is on the basis of volume or mass for the liquids

and gases, but it does not matter when dealing with solids. However, that is not really correct. Let us take a look at an example. Suppose we want to mix three types of metals with densities 4 g/mL, 5 g/mL and 6 g/mL in equal amounts to form an alloy. We assume that no change of volume occurs due to mixing. If we take equal volume V of each of the metals and mix them up by melting, the total volume becomes $3V$ while the total mass becomes $15V$ ($4V+5V+6V$) grams. Hence the resultant density of the alloy becomes $15/3 = 5$ g/mL, which is the average as expected. But would the result be the same if we start with equal masses ‘ m ’ of the three metals? In that case the total mass would be $3m$, but the total volume would be $(m/4 + m/5 + m/6)$ or $(37m/60)$ mL. One can see, here the resultant density would be $180/37$ g/mL. If we round up the result, it comes close to 4.86 g/mL and not 5 g/mL as in previous case. So equality is not an easy goal to achieve.

Equality in voting

Interestingly, in a democratic process we always stress that every voter is equal as each one has one single vote. In fact the strength

of a democratic process lies there. Then how come during the election of the President of this country where our elected representatives from different state assemblies and the members of parliament vote, the voters have different values for their votes?

Members of the Legislative Assemblies (MLA) of various states have ‘values’ of their votes depending on the population of a state. That gives a value of 173 to an MLA from Bihar, but a value of 176 to an MLA from Jharkhand in the election of the President. An MLA from Uttar Pradesh is worth 208 and the value of vote for an MLA from Uttarakhand is only 64. Does that make these voters unequal? No. The value of the vote of MLAs is in a way is related to the number of people they represent and follows the simple principle that a member elected



Our parliament

by larger number of people has got more value for his or her vote. The formula is:

$$\text{Value of an MLA vote} = \frac{\text{Total population of the state}}{\text{Total no. elected members} \times 1000}$$

It needs to be mentioned that once again, average gets into the scenario. For example, an MLA from Puducherry has a much lesser value (16) of his vote than an MLA from Tamil Nadu (176) as the later is elected by a larger number of voters. This is being represented in the formula. If both the total population and the number of elected members in the Legislative Assembly are proportionately reduced then the value of

the vote remains the same. However this does not happen for the smaller states or Union Territories where normally lesser members of voters elect an MLA.

Every MLA in a particular state has the same value of his or her vote. This is because it is assumed that in a particular state all the assembly segments have equal number of voters. We know from our experience all the constituencies actually do not have same number of voters and the largest one may have as many as twice the number of voters than the smallest one. However, the value of the vote of an MLA is not related to the number of voters of his or her constituency, but to the total population and the number of Assembly seats in the state. It needs to be noted in this connection that the value of the vote of an MLA from a particular state is also not linked to the number of votes he or she has actually polled or the number of people who actually cast their votes.

Although the Lok Sabha MPs get directly elected by the people while Rajya Sabha members get elected by the votes of MLAs of the respective states, the value of an MP vote for the Presidential election is calculated to be the same. Total value of the votes of all MLAs from all the states and the Union Territories are added together and is divided by the total number of MPs from both the houses. Thus an MP from Puducherry has the same value of vote as that of an MP from Tamil Nadu or Rajasthan.

The enigma of averages

Average is a common word we come across in our everyday life. An average student or an average performance or an average salary does not make one comfortable. Average refers to the arithmetic mean, which is also simply called the 'mean'. The use of average to establish something or to contest some other things is really quite effective. And that is also fine-tuned by statisticians to make things quite interesting.

Let us take an example from the field of cricket. We know that the average implies the arithmetic mean and the mean cannot be greater than the highest number or lesser than the lowest number involved in this consideration. However, in cricket the batting average of different batsmen is calculated in an entirely different way, which

is not the arithmetic mean. The way term 'average' is used in cricket is rather confusing because sometimes the average of a batsman may be more than his highest score! This happens because while calculating the batting average the runs scored by the batsman in different innings are added but the sum is then divided not by the number of innings he has played but by the number of innings in which he was out. So if a batsman has scored 20, 12 not out, 35, and 24 not out in four innings then his batting average would be 45.5, which is more than the highest runs he has scored in a particular innings, namely 35 in this case. This is because his total runs 91 has been divided by 2 as he has been out in two innings out of the four he has played. Nobody can claim this to be the arithmetic mean or the average in the conventional sense, but this figure goes as the 'batting average'. There is however a cricketering argument in its favour. It is said if you want to judge the performance of a batsman do not only look at the number of runs he has scored but also see how many times he has remained not out in the process. A batsman



Operation in progress in a hospital

may be considered dependable not only for scoring runs but also if he has been proved to be a difficult batsman to dislodge.

Since all these things started in an era when there was only the longer variety of cricket, namely test cricket, scoring of runs was not considered the only quality of a batsman. If a batsman played, say, only two innings in a series and remained not out on both the occasions, his average would remain undefined as the division by zero; that is, the number of innings in which he got out is zero. In such a case, irrespective of total runs the batsman has scored in the two innings, the average is not mentioned.

Today, apart from test cricket the limited over cricket exists side by side and they are in fact more popular. Now the batting average is still calculated but a new parameter known as 'strike rate' has come in. It is simply the number of runs a batsman has scored on an average by facing 100 balls. People do not bother to know how many times he was out but are interested in his ability to hard hit the ball and quickly score runs. A bowler is also not assessed by his bowling average, i.e., runs conceded for one wicket. But in limited over cricket, the bowler's performance is evaluated in terms of the 'economy rate', i.e., runs given away by a bowler in an over on average. One is not interested in the number of wickets taken by the bowler but in his ability to restrict the batsman from scoring runs in limited over cricket.

Handling data

Now we shall look beyond the averages and try to see why statisticians need to handle data carefully. Sometimes the combined data may give us different information compared

to that obtained from the data taken in a piecemeal manner. In a particular town the municipality was running two hospitals and the Mayor felt that one of them may be closed down as the municipality was not in a position to run both of them. Naturally he felt that one with better performance will continue while the other will be closed down. He took a look at the data of the surgical operations carried out in the two hospitals one of which may be referred to as the 'large' one while the other is the 'small' one. In the first hospital, the large one, 4,200 surgeries were carried

out in the previous year. Out of the operated patients 126 died after the surgery, meaning that 3% of the patients did not survive after the surgery. In the small hospital, during the same period 1,800 surgeries were performed, out of which 36 did not survive. So in this case the failure rate, if look at it in this way, was only 2%. So the choice seemed obvious; the small hospital should run while the large one should be closed down simply on the basis of their comparative performance. Does like this look an inference correct?

This prompted the people in the large hospital to take a different look at the data. They broke up the data into the fate of the

male and female patients operated upon. It was found that out of the 4,200 patients who had undergone surgery in the large hospital there were 1,200 female patients and only 12 of them, i.e., 1% passed away after the operation. There were 3,000 male patients and 114 of them died after the surgery, making the percentage of the unfortunate patients 3.8% in this case. On the other hand the small hospital had 1,400 female patients out of which 18 died after the surgery making this percentage 1.3%, which is higher than the corresponding figure for the large hospital. Out of the 400 male patients operated upon in the small hospital 18 died, making the ill-fated patients 4.5% of the total male patients. This is also higher than the corresponding figure for the large hospital.

With this data in hand the Mayor got confused. He first tried to read between the lines, but the data looked perfect. So he could not decide but deferred his decisions. In this way: He suggested that the Municipality should run both the hospitals for another year and then he would take a look at the fresh data. Well, for him this was possibly the best way to handle the situation. But should we blame the Mayor or something else?

Simpson's paradox

So the same data, when looked at after segregation into two groups, were found to give opposite indications compared to the total data taken together. The large hospital showed better performance when the data were segregated into male and female patients, while its performance was considered to be inferior to that of the small hospital when the data were not segregated. This is not a mathematical trick and there is no twist in the language or calculations. Rather this happens because of the way the data is handled. The phenomenon is known as 'Simpson's paradox'. This was pointed out and analysed by the British statistician Edward H. Simpson in 1950s though the situation was known from an earlier time. So now it is sometimes referred to as 'reversal paradox' or 'amalgamation paradox'. Simpson made a detailed mathematical analysis to show that this phenomenon may occur if data is handled in two different ways – once by taking all together and then by taking them separately. Actually some sort of weighted



A university campus: www.topuniversitiesindia.wordpress.com

average comes into play in such cases.

Simpson's paradox came in the limelight through a case that occurred in 1975 where a university in the USA was accused of showing bias towards men against the women candidates who had applied for academic positions in two departments in the university. The data looked like this. The lecturers were appointed for the History and Geography departments. Both the departments had 13 candidates each. Moreover, out of these 26 candidates there were 13 male and 13 female candidates. It was finally found that a total of 6 female candidates out of 13 were offered appointment in the two departments while this number was 7 for the male candidates, once again from amongst 13 candidates in the two departments. Apparently female candidates were less preferred than males. When the case came before the Court of Law, the university came out with the following table:

Subject	Female candidates selected	Male candidates selected
History	2 out of 8 (25% selected)	1 out of 5 (20% selected)
Geography	4 out of 5 (80% selected)	6 out of 8 (75% selected)
Total	6 out of 13 (-46% selected)	7 out of 13 (-54% selected)

From this table one can easily see, as the university pointed out, that the percentage of selection in both the subjects is higher for the female candidates (25% in comparison to 20% in History, and 80% compared to 75% in Geography) but indicated a different

trend in the total. Here the sample size is very small. However, if the total numbers and the selections are multiplied say by 100 and suppose we make this selection for the students' admission and not for the selection of the faculty then the sample will be quite large but the selection rate will be similar and the Simpson Reversal will continue to take place. Same set of data may lead to two contrasting conclusions.

So it appears, average may be a simple arithmetical exercise, its consequences could be quite significant. And we have to admit that a set of data or some simple set of numbers can be quite interesting, but average should not be looked upon as an average issue. All sorts of averages from all spheres of life actually demand more attention from all of us.

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Dr Bhupati Chakrabarti is the General Secretary of Indian Association of Physics Teachers (IAPT) and a former Head of Dept. of Physics, City College, Kolkata 700009.

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Vidyarthi Vigyan Manthan (VVM) is a national program jointly organized by Vijnana Bharati, NCERT and Vigyan Prasar for educating and popularizing science among the school students of 6th to 11th standard. The main objective of the program is to identify keen knowledge seekers in science and then nurture them for higher level of science education. VVM is a multiple level process in which each student will pass through such testing procedures as objective type question answering, comprehensive writing, project proposal writing, group discussion, role play, practical examination & oral viva.

ELIGIBILITY CRITERIA:

Students in classes VI to XI of the CBSE, ICSE & State boards are eligible to appear for this examination.

SELECTION PROCEDURE:

1. School Level Examination (Prathama and Dwitiya)-

Two papers on same day with 30 minutes break between them will be organized at the school level. Separate examination will be conducted for junior group (class VI to VIII) and senior group (class IX to XI). The structure of questions paper is as follows:

Content	Contribution						Curriculum
	Prathama (Level-I) Multiple Choice Questions			Dwitiya (Level-II) Subjective Questions with MCQ			
	Questions	Marks	Duration	Questions	Marks	Duration	
Science from text book	40	40	60 minutes	25	50	90 minutes	NCERT
Indian contribution to Science	30	30		10	20		VVM study material *
Life of Dr APJ Abdul Kalam	20	20		5	10		VVM study material *
Logic/ Reasoning/ General Knowledge	10	10		10	20		General Reading
Total	100	100		50	100		

2. State Level Camp-

Top 20 students from each class will be selected i.e. total of 120 students will be selected at the State level. Selected students will be invited for one/two day State Level Camp. The camp will consist of hands on training, student - scientist interaction and the third stage including group activities, debate competition, role play etc. Based on the performance ranking of the students will be done at the state level and top three students from each class will be declared as a State winner.

3. National Level Camp-

Top two students from each class i.e. 12 students from each State will be invited to a two-days National Level Camp. These students will go through the fourth evaluation process involving presentations, viva and leadership aspects. Based on performance top 3 students from each class will be declared as National Level winners of VVM for the year 2016.

Language of Exam:

Students can take examinations either in English/Hindi or in their own mother tongue.

Exam Centres:

Exams will be conducted at various centres across India. Schools that can enroll more than 50 students will be designated as Examination Centre.

HOW TO APPLY:**1. Registration-**

Student : All students who want to appear in VVM should register by paying Rs. 100/-

School : Only government and government aided school can register their students with 50% concession (Rs. 50/-per students) only if school assure for minimum of 50 students. For other schools, they can register with Rs. 100/- per student.

2. Mode of payment-

The registration fees can be paid either through cheque or Demand Draft in favour of "VVM, Delhi" posted to Vijnana Bharati office mentioned at bottom of the page. Online registration can also be done through our website www.vvm.org.in

AWARDS TO WINNERS:

Prathama Level	:	Certificates	
Dwitiya Level	:	Memento & Certificate	
State level (3 per class/state)	:	1st Prize- Rs 5,000/-, Memento & Certificate 2nd Prize- Rs.3,000/-, Memento & Certificate 3rd Prize- Rs. 2,000/-, Memento & Certificate	
National Level (3 per class)	:	1st Prize- Rs. 10,000/-, Memento, Certificate & National Educational Visit. 2nd Prize- Rs. 7,000/-, Memento, Certificate & National Educational Visit. 3rd Prize- Rs. 5,000/-, Memento, Certificate & National Educational Visit.	

IMPORTANT DATES TO REMEMBER:

Registration Open	01 st July 2016	
Registration Closes	30 th September 2016	
Issue of hall tickets	Before 1 st November 2016	
Date of Prathama & Dwitiya	13 th November 2016	
Timing of examination	Prathama	10:00am to 11:00am
	Dwitiya	11:30am to 01:00 pm
Declaration of result of Prathama & Dwitiya	10 th December 2016	
One/Two day state camp	Between 1 st January 2017 to 30 th January 2017	
Two day national camp	On 27 th & 28 th May 2017	

For more information you can log on to website www.vvm.org.in



Vidyarthi Vigyan Manthan (VVM) is initiated by Vijnana Bharati (VIBHA) in collaboration with National Council of Education Research and Training – an institution under the Ministry of Human Resources and Development and Vigyan Prasar, an autonomous organization under the Department of Science and Technology, Government of India.

Vijnana Bharati :- C-486, Defence Colony, New Delhi-110024

From Laptops to Hot baths— Knowing what can steal your fertility



Dr. Yatish Agarwal
E-mail: dryatish@yahoo.com

Would you know that despite a teeming human population on planet Earth, some 15 per cent bisexual couples fail to make babies? Of them, 4 in 10 fall short because of the poor fertility of the male partner; an equal number because of the female fertility factor; about 10 per cent because both partners have a faulty procreation apparatus; and in the other 10 per cent, no amount of tests can identify why the conception is failing them.

The count of infertility in bisexual couples has been growing over the years. The newfangled life style, late age of marriage, especially in women, tight work schedules which reduce marriage to being at best a weekend romance, and a host of environmental factors seem to be in play. In this climate, it is no mean wonder that fertility clinics have mushroomed across towns and cities, and making babies has become a billion dollar industry!

Understanding some of the basic issues is therefore pivotal from the perspective of taking a preventive approach.

A number game

Despite that all it takes is a mating of one female egg with one male sperm for procreation to occur, the underlying process is far more complex. That is why even if a man has fewer than 15 million sperm per millilitre of semen, his sperm count is considered abnormally low.

Doctors call this condition of low sperm count by the name of oligospermia. The condition can sometimes be worse still, with a complete absence of sperm in the ejaculate and is called azoospermia.

Having a low sperm count decreases the odds that a sperm will fertilise the partner's egg, and that the wish of a pregnancy would be fulfilled. Nonetheless, some men who have a low sperm count are still able to father a child. The male infertility factor may simply relate to problems of quantity, abnormal sperm shape (morphology), movement (motility), or function.

A 1992 World Health Organization report described normal human semen as having a volume of 2 mL or greater, pH of 7.2 to 8.0, sperm concentration of 20×10^6 sperm/mL or more, sperm count of 40×10^6 spermatozoa per ejaculate or more, and motility of 50% or more with rapid forward progression of 25% or more within 60 minutes of ejaculation.

A complex process

Few people may know, but the production of sperm is a complex process. It requires a healthy functioning of the testicles (testes), besides the hypothalamus and pituitary glands — organs in the brain that produce hormones that trigger sperm production.

The buck doesn't stop here. Once sperm are produced in the testicles, delicate tubes transport them until they mix with semen and are ejaculated out of the penis. Problems with any of these systems can affect sperm production. Any of the units can go wrong and cause a problem of numbers, of abnormal sperm shape, of poor movement or low function.

Causes of low sperm counts

A number of factors related to lifestyle, environment, health and reproductive system have been found culpable for causing low sperm counts in men.

Lifestyle and environmental causes

Overheating the testicles

Health clubs may be in fashion, but frequent use of saunas or hot tubs may temporarily lower a man's sperm count. Likewise, sitting for long periods, wearing tight clothing or using a laptop on the lap



for long periods of time also may increase the temperature in the scrotum and reduce sperm production. This may be a significant contributory factor for infertility affecting modern youth.

Drug abuse

Men using anabolic steroids at the instance of their trainers in gyms and fitness centres to stimulate muscle growth and strength can suffer a shrinkage of the testicles and become infertile with a decrease in sperm production.

Likewise, the use of recreation drugs like cocaine or marijuana has also been found to temporarily reduce the number and quality of sperm.

Prolonged bicycling

Much though this fact may not appeal to the environmentalists, prolonged bicycling, especially on a hard seat or poorly adjusted bicycle, is another possible cause of reduced fertility due to overheating of the testicles. Likewise, prolonged horseback riding can also affect male fertility.



Alcohol use

Drinking alcohol can lower testosterone levels and cause decreased sperm production. In chronic alcoholics, due to a number of mechanisms at play, the testicles may shrink to the detriment of sexual health.

Occupation

Certain occupations also tend to increase a man's risk of infertility. For instance, those associated with extended use of computers or video display monitors, shift work and work-related stress, have been reported to carry higher rates of infertility.

Tobacco smoking

Smoking causes many ills. Among them, infertility. Men who smoke may have a lower sperm count than do those who don't smoke. The quality of sperm may also suffer.

Emotional stress

Severe or prolonged emotional stress, including stress about fertility itself, may interfere with certain hormones needed to produce sperm.

Radiation or X-rays

Exposure to radiation can reduce sperm production. It can take several years for sperm production to return to normal. With high doses of radiation, sperm production can be permanently reduced.

Weight

Obesity can cause hormone changes that reduce male fertility.

Heavy metal exposure

Overexposure to certain environmental elements can affect sperm production or function. For instance, exposure to lead or other heavy metals may cause infertility.

Industrial chemicals

Extended exposure to benzenes, toluene, xylene, herbicides, pesticides, organic solvents, painting materials and lead may contribute to low sperm counts.

Health-related causes

Low sperm count can be caused by a number of health issues and medical treatments. Some of these include:

Undescended testicles

During foetal development one or both testicles may sometimes fail to descend from the abdomen into the sac — the scrotum — that normally contains the testicles. The high temperatures in the abdomen damage the testicles and impair sperm production. Hence, decreased fertility is a frequent accompaniment in men with this condition.

Hormone imbalances

The hypothalamus, pituitary and testicles produce hormones that are necessary to create sperm. Alterations in these hormones, as well as from other systems such as the thyroid and adrenal, may impair sperm production.

Sperm duct defects

The tubes that carry sperm can be damaged by illness or injury. Some men are born with a blockage in the part of the testicle that stores sperm (epididymis) or a blockage of one of the tubes that carry sperm out of the testicles (vas deferens).

Men with some inherited conditions may be born without sperm ducts. Such men suffer with azoospermia.

Varicocele

A varicocele is a swelling of the veins that drain the testicle. It is a common cause of male infertility. This may prevent normal cooling of the testicle, leading to reduced sperm count and fewer moving sperm. The treatment of a varicocele often improves sperm quality and overall fertility.

Infection

Some infections can interfere with sperm production and sperm health or can cause scarring that blocks the passage of sperm. These include some sexually transmitted infections, such as chlamydia and gonorrhea; inflammation of the prostate (prostatitis); inflamed testicles; and other infections of the urinary tract or reproductive organs.

Ejaculation problems

Retrograde ejaculation occurs when semen enters the bladder during orgasm instead of emerging out of the tip of the penis. Various health conditions can cause retrograde ejaculation, including diabetes, spinal injuries, and surgery of the bladder, prostate or urethra.

Certain medications also may result in retrograde ejaculation, such as blood pressure medications known as alpha blockers. Some men with spinal cord injuries or certain diseases cannot ejaculate semen at all, though they still can produce sperm.

Anti-sperm antibodies

Antibodies that attack sperm are immune system cells that mistakenly identify sperm as harmful invaders and attempt to destroy them.

Chromosome defects

Inherited disorders such as Klinefelter's syndrome — in which a male is born with two X chromosomes and one Y chromosome instead of one X and one Y — cause abnormal development of the male reproductive organs.

Other genetic syndromes associated with infertility include cystic fibrosis, Kallmann's syndrome and Kartagener syndrome.

Tumours

Cancers and non-malignant tumours can affect the male reproductive organs directly, or can affect the glands that release hormones related to reproduction such as the pituitary gland. Surgery, radiation or chemotherapy to treat tumours can also affect male fertility.

Coeliac disease

A digestive disorder caused by sensitivity to gluten, coeliac disease can cause male infertility. Fertility may improve after adopting a gluten-free diet.

Certain medications

Testosterone replacement therapy, long-term anabolic steroid use, cancer medications (chemotherapy), certain antifungal and antibiotic medications, some ulcer medications and anti-psychotics and anti-hypertensives can also affect male sexual prowess and fertility.

Male infertility can be an absolutely silent condition. Oligospermia and azoospermia may pass completely unnoticed until a couple fails to conceive despite repeated tries and undergoes testing for infertility.

Prof Yatish Agarwal is a physician and teacher at New Delhi's Safdarjung Hospital. He has authored 47 popular health-books.

Next month: Male infertility — tests and treatments)

Recent Developments in Science and Technology



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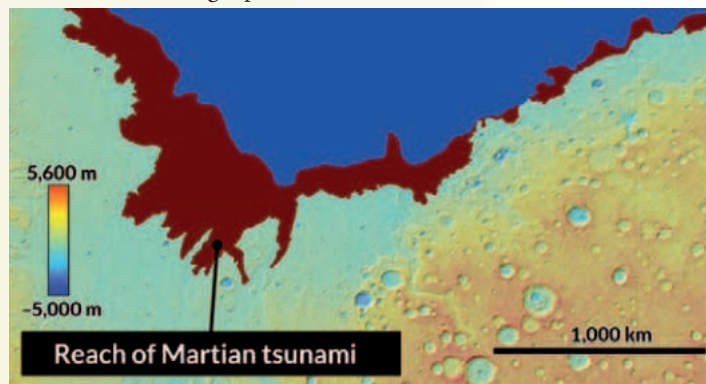
Ancient tsunamis on Mars reshaped its landscape

Mars may look dry and dead today, but it seems to have been much more Earth-like in the distant past, with a thick atmosphere and large bodies of standing water. Some scientists speculate that as much as a third of the planet's surface was once covered in standing bodies of water and ice. Water-carved channels hint that groundwater outbursts flooded Mars' northern lowlands, forming a colossal ocean larger in area than North America. Sedimentary deposits discovered in the Martian north by radar in 2012 support this view. According to planetary scientists, around 3.4 billion years ago, this ocean covered most of the Northern Martian lowlands.

A new study suggests that two large meteorites hit Mars billions of years ago and triggered mega-tsunamis in Martian water oceans. After carefully studying the Martian geography, Alexis Rodriguez of Planetary Science Institute, Tucson, USA and colleagues have identified evidence for at least two sizable tsunamis that occurred a few million years apart. These gigantic waves forever scarred the Martian landscape and yielded evidence of cold, salty oceans that the researchers believe could be conducive to sustaining life.

The first meteorite slammed into the ancient Martian ocean around 3.4 billion years ago, generating the first massive tsunami up to 120 metres high that reshaped the shoreline of the Martian ocean, leaving behind fields of sediments and boulders. By analysing satellite images of the Martian surface, Rodriguez and colleagues conclude that the tsunami could have sent water gushing hundreds of kilometres inland and carved the region's odd geography (*Scientific Reports*, 19 May 2016

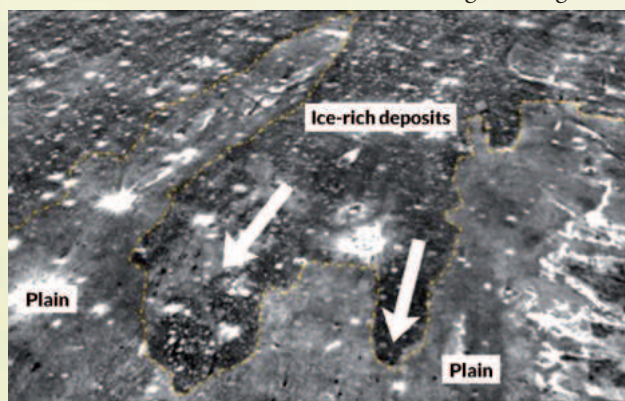
| doi: 10.1038/srep25106). According to the scientists, "The first tsunami pushed boulders as large as double-decker buses into strange places and shifted sediments that



Meteorite impacts may have generated supersized tsunamis on Mars that reshaped the coastlines of an ocean around 3.4 billion years ago. Red-shaded region shows the reach of the more recent of two tsunamis.

obscured the ocean coastlines. The giant wave's receding waters also carved backwash channels in the Martian surface".

The scientists found evidence for another big meteorite impact, which triggered a second tsunami wave that occurred a few million years after the first. During the intervening period conditions on Mars had changed, with temperatures dropping and glaciers marching across the landscape, gouging out deep valleys on the planet's surface. When the second tsunami hit the shore, its effect was different; its ice-rich waters froze before retreating, leaving



Ice Surge. This 250-km-long lobe of dark material on the surface of Mars was left behind when a tsunami of icy flush washed over the Martian coastline, freezing in place before it could wash back into the sea. (Credit: Alexis Rodriguez)

behind 'rounded lobes of ice'. "These lobes froze on the land as they reached their maximum extent and the ice never went back

to the ocean – which implies the ocean was at least partially frozen at that time," says Rodriguez. These lobes had been spotted before, but their geological significance had remained unsettled. Now scientists should be able to glean the chemistry of the ancient ocean by sampling these formations. These icy lobes retained their well-defined boundaries and their flow-related shapes. According to the researchers, the cold, salty waters might have offered a refuge for life in extreme environments, as the salts could help keep the water liquid ... If

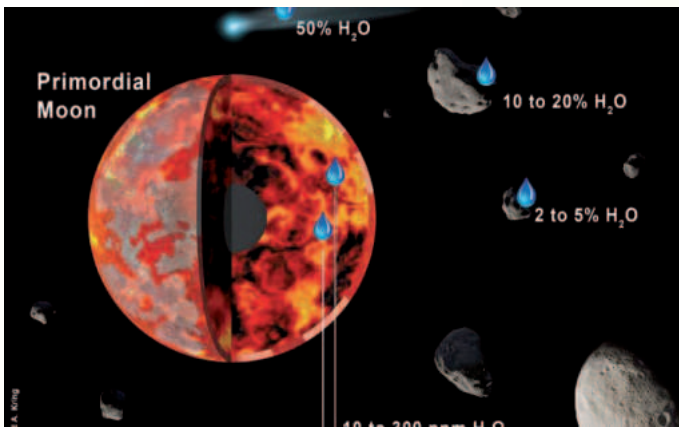
life existed on Mars, these icy tsunami lobes are very good candidates to search for bio-signatures.

Moon has plenty of water

Till India's *Chandrayaan-1* first detected presence of water on Moon in 2008, Earth's closest neighbour was believed to be bone dry, based on rocks brought back by NASA's Apollo lunar missions starting in the late 1960s. Soon NASA also corroborated the findings of *Chandrayaan-1* indicating that the Moon indeed has water. In recent years,

more advanced techniques have actually picked out significant signs of water in those lunar samples and scientists say, "Though the surface is parched, the lunar interior might actually have about 10,000 to 10 million times more water than the surface seems to hold". A recent study suggests that most of the water inside the Moon must have been delivered by asteroids some 4.5 to 4.3 billion years ago, when its molten oceans were hit by asteroids carrying water (*Nature Communications*, 31 May 2016 | DOI: 10.1038/ncomms11684).

For the study, an international team of researchers led by Jessica J. Barnes of The Open University, in the UK compared data from a range



New research finds that asteroids delivered as much 80 percent of the Moon's water. (Credit: LPI/David A. Kring)

of different studies that had analysed lunar samples brought back from the Moon or meteorites (which are thought to be chunks of asteroids that fell to Earth). The researchers studied the composition of certain elements in the space rocks, especially the ratio of hydrogen-to-deuterium (a heavier isotope of hydrogen), which allows them to figure out the origin of Moon's water. They found the hydrogen-to-deuterium ratio to be similar to that of a certain type of asteroids known as chondrites.

After carefully collecting and modelling the data, the researchers found that the Moon's water probably came mostly from asteroids – even though comets have the reputation for being rich in water ice. From about 4.5 to 4.3 billion years ago, the researchers say, more than 80% of the Moon's water likely came from various types of asteroids and less than 20% of it came from comets. Back then, the young, hot Moon was covered in a magma ocean and the asteroids would have sunk into the fluid mix. In addition to asteroids and comets, the researchers say, it is also likely that some of the water inside the Moon may be derived from the early Earth during the Moon-forming impact event.

According to the researchers, after the Moon was born of a collision between Earth and a Mars-sized planet some 4.5 billion years ago, it was bombarded with water-rich asteroids known as carbonaceous chondrites for tens of millions of years, maybe even longer, which delivered a lot of water to the Moon. Incidentally, Earth also got most of its water from asteroid bombardment.

The researchers estimate that the lunar interior could contain “of the order of 1,000 trillion tonnes” of water, probably locked

inside minerals in the form of hydroxyl (OH) ions. On the surface, they estimate, “up to a billion tonnes of frozen water – enough to fill a million Olympic pools – is probably lodged as ice inside deep craters around the north and south lunar poles, where the Sun's rays never penetrate”. It is one of the reasons several space agencies – including the European

Space Agency and NASA – are currently developing robotic missions to explore new regions on the Moon to better estimate the quantity of ice. They conclude that the water “has been trapped there for three or four billion years”.

How the giraffe got its long neck revealed

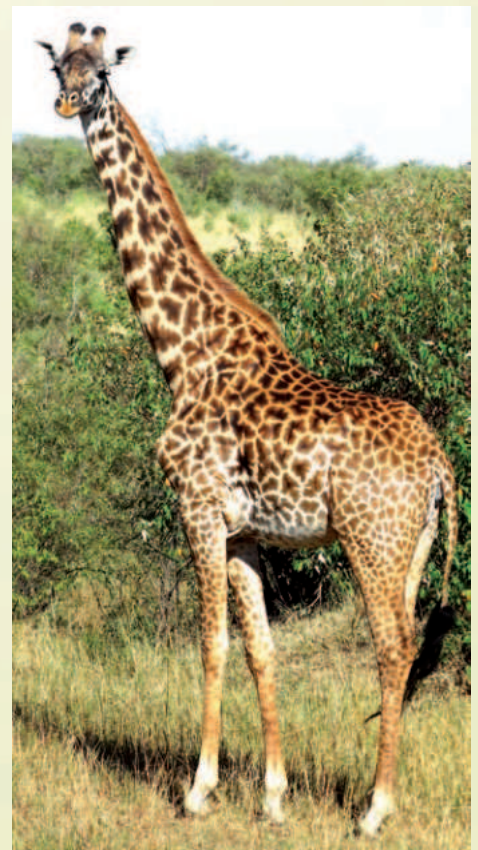
One of the most distinguishing features of the giraffe is its extraordinary long neck, which accounts for almost half of its height. Interestingly, unlike some species of long-necked birds, which have up to 25 vertebrae in their cervical spine, the giraffe has only seven of these bones – the same as we humans have. To compensate for the extra height, the giraffe has a blood pressure level two times higher than humans, powered by an extra-large heart, to reach blood to its brain.

There have been many hypotheses to explain the extra-long neck of the giraffe. The 18th century French naturalist Jean Baptiste Lamarck believed that the long necks of giraffes evolved as generations of giraffes reached for ever higher leaves on trees. He suggested that if a giraffe stretched its neck for leaves, for example, a “nervous fluid” would flow into its neck and make it longer. Its offspring would inherit the longer neck, and continued stretching would make it longer still over several generations. Of course, today we know that it does not happen like that. Recently, scientists seem to have found a genetic clue.

To figure out just how the giraffe got its long neck, researchers from the Giraffe Genome Project – a joint venture of Penn State University in USA and the Nelson Mandela African Institute for Science and Technology in Tanzania – sequenced and

compared the giraffe genome to that of the okapi, their nearest relative and the only other surviving member of the family Giraffidae. The analysis revealed the first clues about the genetic changes that led to the evolution of the giraffe's exceptionally long neck. The okapi is a herbivore found in tropical mountain forests of Central Africa. Despite its deer-like appearance the okapi is actually one of the last remaining ancestors of the giraffe, which is the tallest animal on Earth. The researchers say that giraffes and okapi split around 11.5 million years ago, after which the giraffe underwent a tremendous growth spurt.

Using a battery of comparative tests the researchers shortlisted 70 genes that showed multiple signs of adaptations. According to Douglas Cavener of Penn State University, “These adaptations include unique amino-acid-sequence substitutions that are predicted to alter protein function, protein-sequence divergence, and positive natural selection”. Over half of the 70 genes code for proteins that are known to “regulate development and physiology of the skeletal, cardiovascular, and nervous system” – just



Scientists have found a genetic link to giraffe's long neck.



The okapi is the nearest relative and the only other surviving member of the family Giraffidae.

the type of genes predicted to be necessary for driving the development of the giraffe's unique characteristics.

Among the genes showing multiple signs of adaptation in the giraffe the research team also discovered several genes known to either regulate the development of the cardiovascular system or to control blood pressure. Some of these genes control both cardiovascular development and skeletal development, suggesting the intriguing possibility that the giraffe's stature and powerful cardiovascular system evolved as a result of changes in a small number of genes.

According to the researchers, for the giraffe's necks to grow so long, two crucial things need to happen: First, the genes that tell the neck when to stop growing needed to be turned off; second, genes that promote growth need to be upregulated; that is, need to increase the response to a stimulus. They singled out one gene in particular, FGFR1, as playing a key role in promoting neck growth. The gene is responsible for adaptations unique to giraffes, especially for regulating embryo development and particularly the skeletal and cardiovascular systems. Also of importance are four so-called "homeobox" genes (a class of closely similar sequences that occur in various genes and are involved in regulating embryonic development in a wide range of species) that come into play during the giraffe's development from an embryo into adolescence. These four genes also have unique adaptations that the researchers think promote rapid growth in the giraffe's cervical vertebrae.

Eukaryotes without mitochondria!

Mitochondria, also known as the 'powerhouse' of the cell, have long been

considered as essential components for life in eukaryotes, the group including plants, fungi, and animals. Recently scientists have discovered a eukaryote that completely lacks mitochondria, making it necessary to rewrite biology textbooks. The surprising discovery came when researchers Anna Karnkowska and Vladimir Hampl at Charles University in Prague and BIOCEV (Biotechnology and Biomedicine Center of the Academy of Sciences and Charles University in Vestec), along with colleagues from the Czech Republic and Canada sequenced the genome of *Monocercomonoides*, a genus of flagellated protozoa belonging to the order Oxymonadida. They were surprised to find that this organism lacks all mitochondrial proteins (*Current Biology*, 23 May 2016 | DOI: 10.1016/j.cub.2016.03.053).



Light micrograph of Monocercomonoides sp. (PA203). (Credit: Dr Naoji Yubuki)

Organisms from the genus *Monocercomonoides* have been recognised for more than 80 years. They are related to the human pathogens *Giardia* and *Trichomonas*, all of which belong to a

group known as Metamonada, which lives exclusively in low-oxygen environments. It has been known that in low-oxygen environments, eukaryotes often possess a reduced form of the mitochondrion, but it was believed that some of the mitochondrial functions are so essential that these organelles are indispensable for their life. But the new discovery proves this belief wrong.

According to the researchers, *Monocercomonoides* seems to have managed without mitochondria because it acquired, by lateral gene transfer, a cytosolic sulphur mobilisation system to provide essential clusters of iron and sulphur required for protein synthesis. "Through a unique combination of events including the loss of many mitochondrial functions and the acquisition of this essential machinery from prokaryotes, this organism has evolved beyond the known limits that biologists circumscribed," says Karnkowska. In fact, researchers have been looking for organisms lacking mitochondria for decades, but were unsuccessful till now.

The new discovery will enable scientists to learn more about how these organisms function without mitochondria. Karnkowska says, "This amazing organism is a striking example of a cell which refuses to adhere to the standard cell biology text book, and we believe there may be many more similar examples in the so far hidden diversity in the world of microbial eukaryotes—the protists".

Biman Basu is a former editor of the popular science monthly *Science Reporter*, published by CSIR. He is a winner of the 1994 'NCSTC National Award for Science Popularisation'. He is the author of more than 45 popular science books. ■

Articles invited

Dream 2047

Vigyan Prasar invites original popular science articles for publication in its monthly science magazine *Dream 2047*. At present the magazine has 50,000 subscribers. The article may be limited to 3,000 words and can be written in English or Hindi. Regular columns on i) Health ii) Recent developments in science and technology are also welcome. Honorarium, as per Vigyan Prasar norm, is paid to the author(s) if the article is accepted for publication. For details please log-on to www.vigyanprasar.gov.in or e-mail to dream@vigyanprasar.gov.in