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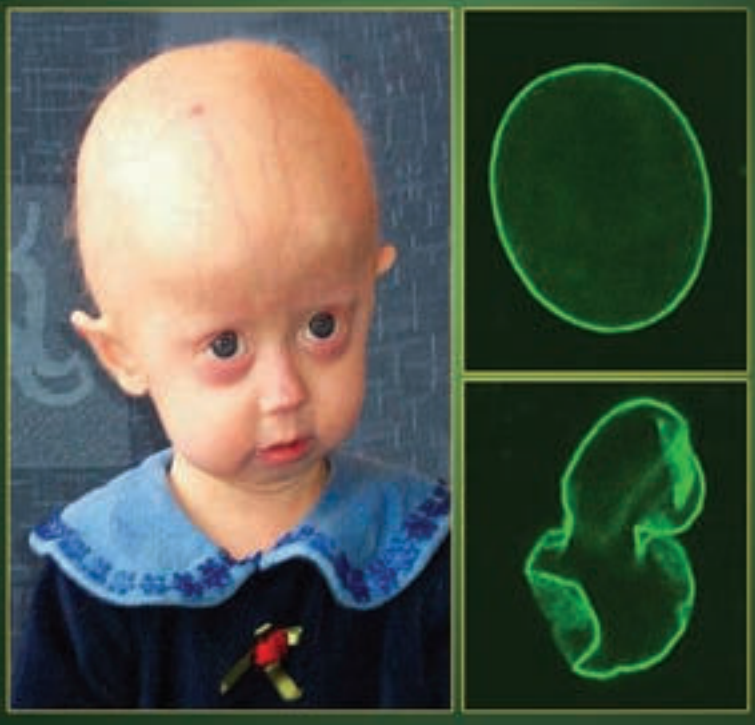
Progeria
Life in Fast - Forward Mode



Inside

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

A Worthy Challenge

E-mail: sanuj@vigyanprasar.gov.in

Over the past two decades the forces of economics unleashed because of liberalisation, privatisation and globalisation have seen large sections of the Indian society rapidly progress and prosper. Materialism has witnessed wide acceptance and symbols of progress due to technological inputs are evident both in rural and urban India.

At the same time, increasingly large sections of our society have been marginalised over the same period and disparity appears to have aggravated. Social justice appears wanting and this impacts societal, economic and democratic systems. Their exploitation is rooted in poverty, poor literacy levels, belief systems, and several inter-related compounding factors. Social and natural upheavals find these sections more vulnerable and tend to decimate their reserves of resources.

This divide first needs to be accepted and then addressed. Symptomatic measures without addressing the causative factors will only prove short-sighted. Societal development cannot be measured by gross factors of per capita domestic production or energy consumed. Use of human development indices based on access to essential services like health and education, right to livelihood, and justice are desirable for designing interventions.

For development of any society, understanding of art, culture, politics, and other faculties is important. Science and technology awareness is equally, if not more, important. This is not limited to knowledge of research in various disciplines and/or use of new technology and products. True understanding of science and method of science includes capacity for reasoning and logical thinking. This is the foundation for reaching decisions on developmental issues based on informed choices.

Nurturing curiosity is not only at the root and but also the fruit of good science. Looking for causes and effects, framing the right question and evaluating the worth of replies are elements of the methodology. The ability to reject that which does not stand established tests and the courage of accepting a hypothesis only after verifying it repeatedly - are facets of the method of science. This has established our understanding of natural forces and phenomena, observations at macro and micro level and developed a formidable body of knowledge. The progress in various fields is testimony to the effectiveness of the method.

Individual and societal perspectives of scientific outlook are required. One deals with inquisitiveness and curiosity and this leads to a spirit of enquiry. The other serves to encourage rational decision-making and acts as a 'watch dog.' Together the outlooks will result in balanced progress and will insure against exploitation of skills and resources for short-term material gain. Meaningful participation in debates and dialogue will strengthen democracy at many levels.

Using technology and science without the right touchstone of scientific temper will encourage materialistic tendencies and exploitative deployment of resources. Political and economic freedom in absence of scientific outlook leads to a culture of consumerism. Scientific outlook is useful in a different form for scientific research and technology application. The next decade will require such capabilities in large measure in a significant majority of the population.

The issues that concern our society today and are likely to engage our energies - both locally and regionally - will have acceptable solutions with higher levels of science literacy and a very wide spread of scientific awareness in the society. We need to distinguish between these two and will discuss this at another opportunity.

Environmental concerns, energy inadequacies, genetic manipulation, etc., are facets of development attributable to science that are in the public space for discussion and deliberation. The concerns for future of mankind and the choices to be made by our generation are certainly not easy. Immediate benefits, projected risks and probable costs need to be discussed with inadequate data, biased analysis and high pitched advocates on both sides.

Neither the captains of industry nor the executive should drive decisions on such issues. There could be obvious or veiled vested interests guiding the process. But what are the options available to the society? Judiciary has been active in considering several important science related issues and these are cases brought for litigation and *suo moto* in recent times. The judgments are influenced by the power and force of advocacy lined up by the parties. Progressive pronouncements have been followed up by the executive. Appeals in higher courts by the aggrieved parties are time and cost consuming.

We need practical solutions to critical issues of development. Leaders with high levels of scientific outlook are required at every level of governance. There is little comfort in following a path that will meander towards visible and desirable goals. Several well- meaning efforts, if sub critical, will dissipate energy without adequate impact. Meaningful approaches need to be developed quickly and a major programme has to be launched after adequate consultation. Are science communicators ready to accept this grand challenge?

Anuj Sinha

Editor : **Er Anuj Sinha**
Address for correspondence : Vigyan Prasar, C-24, Qutab Institutional Area, New Delhi-110 016
Tel : 011-26967532; Fax : 0120-2404437
e-mail : info@vigyanprasar.gov.in
website : http://www.vigyanprasar.gov.in

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Editor : Er Anuj Sinha

Henry Cavendish
Discoverer of Hydrogen

Subodh Mahanti
E-mail: smahanti@vigyanprasar.gov.in

"In his long lifetime this eccentric recluse (Henry Cavendish) achieved most in chemistry: notably in showing that gases could be weighed, the air is a mixture and that water is compound—all fundamental matters if chemistry was to advance. His work in physics was equally remarkable but was largely without influence because much was unpublished."

The Cambridge Dictionary of Scientists (2nd Edition), Cambridge University Press, 2002

"Much of Cavendish's work remained unpublished in his lifetime and he is now known to have anticipated or come very close to several major discoveries. His electrical studies, which were edited by Clerk Maxwell in 1879, following the discovery of his notebooks and manuscripts, included the clear distinction between electrical quantity and potential, the measurement of capacitance, and the anticipation of Ohm's law (1781)."

A Dictionary of Scientists, Oxford University Press, 1999.

"Henry Cavendish was without question the most eccentric scientist in the 18th century. Born to a famous and wealthy British family, he never had to worry about money. In fact, he once told his banker that if the banker ever bothered Cavendish about what to do with his money, the bank would lose his account, which was worth millions. Needless to say, the banker never bothered him again. But where other Cavendishes had hobnobbed with kings and participated in general political plots, Henry's one and only interest was the pursuit of pure science."

The History of Science in the Eighteenth Century by Ray Spangenburg and Diane K. Moser, Universities Press (India) Limited, 1999

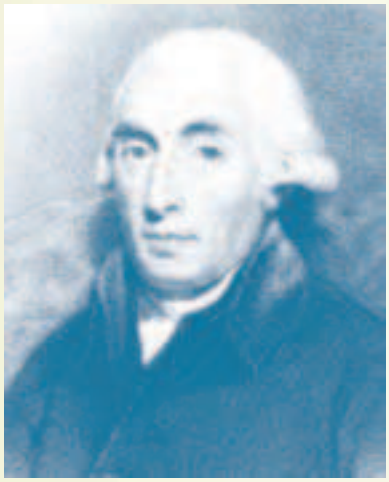
Henry Cavendish was one of those whose works paved the way for proper understanding of chemical elements, their nature, the way they react with one another and the processes taking place in chemical reactions. Cavendish contributed significantly towards better understanding of gases. Like British physician-chemist Joseph Black, Cavendish instituted the use of careful analysis in chemistry. He was known for his experimental care and precision. As we know the French chemist Antoine Laurent Lavoisier (1743-1794), who is usually regarded as father of modern chemistry, successfully convinced other chemists about the importance of the methods introduced by Cavendish and Black. Cavendish was the first to recognise hydrogen gas as a distinct substance and thus he was credited with the discovery of hydrogen. He showed that water was a composed of oxygen and hydrogen. He demonstrated that common air and also air brought by balloon from upper atmosphere was made up of nitrogen in a 4:1 ratio. It is reported that Cavendish had discovered argon, which was rediscovered by William Ramsay one hundred years later. Cavendish made notable electrical studies. One of his most celebrated investigations was the determination of the density of the Earth.

Cavendish was one of the wealthiest persons in England of his time. However he himself lived a frugal and solitary life. He was described as "the richest of the learned and the most learned of the rich".

Cavendish did not publish most of his work. This was perhaps largely due to his peculiar social and secretive behaviour. He did not even discuss about his findings with fellow scientists. So there is no wonder that the vast amount of his unpublished works had things for which others had been given credit. Among these were Richter's Law of Reciprocal Proportions, Ohm's Law, Dalton's Law of Partial Pressures, Coulomb's Law, and Charles' Law of Gases.

Cavendish was a highly eccentric man. He was an extremely shy person. He interacted only with his scientific friends, members of the Royal society, who used to gather together for the weekly meetings. Outside the sphere of his scientific friends he avoided conversation to an extreme degree, especially with women. According to one account, Cavendish added a back staircase to his house in order to avoid encountering his female housekeeper. He used to issue instructions to his housekeeper and other female servants through a system of notes. He had standing instruction to his female servants to keep away from him or

Henry Cavendish



Joseph Black

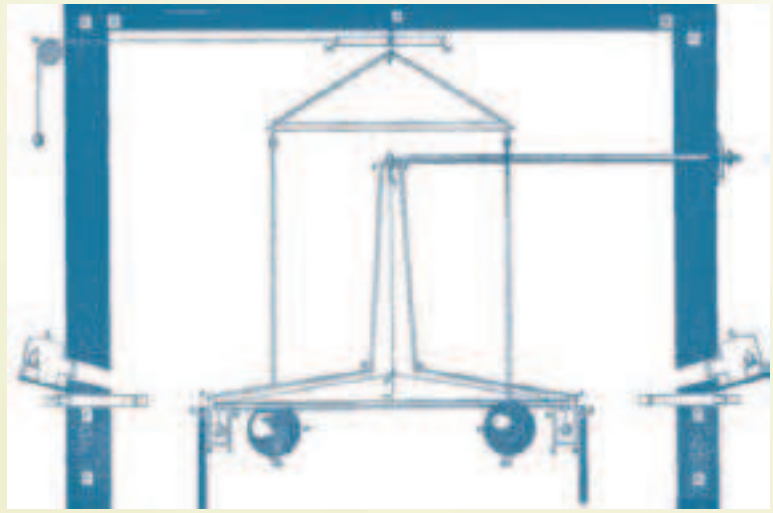
face dismissal. Some have gone to speculate that Cavendish suffered from Asperger syndrome (people with it show significant difficulties in social interaction). It is said "he uttered fewer words in the course of his life than any man who lived to four-score years." There is only one portrait of Cavendish in existence. This was painted by W. Alexander and is preserved in the Bettmann Archive of the British Museum.

Henry Cavendish was born on 10 October 1731 in Nice, France, where his family was living at the time. His father Lord Charles Cavendish, a Fellow of the Royal Society of London, was the son of the 2nd Duke of Devonshire. His family could trace its lineage across eight centuries to Norman times. Many aristocratic families of Great Britain had close ties with the Cavendish family. His mother Lady Anne Grey was the daughter of the Duke of Kent.

We do not know much about early life of Cavendish. He started studying at Peter Newcome's School at Hackney at the age of 11. On 24 November 1749 he joined the Peterhouse (then St Peter's College) of Cambridge University. He left the college without a degree after studying there for three years. He then undertook the grand tour of Europe along with his brother. In those days such a tour was obligatory for higher studies. After completion of the tour he returned to London to live with his father. Initially he helped his father in conducting experiments on heat, electricity and magnetism. He later built a laboratory and workshop of his own for pursuing his scientific studies. After the death of his father in 1783 he moved his laboratory to Clapham Common and started living there.

The first important scientific investigation undertaken by Cavendish was an attempt to produce a Newtonian theory of heat based on corpuscular motions and distance forces. For realising his goal he began studying airs (or gases). This was natural because the expansion of airs provided a simple and convenient model for the interaction of heat and matter. Cavendish described methods of handling and weighing gases. He investigated the products of fermentation. He showed that the gas generated by the fermentation of sugar was indistinguishable from the "fixed air" or today's carbon dioxide earlier identified by Joseph Black.

In 1766, Cavendish isolated and studied hydrogen gas. He produced hydrogen gas by reacting metals with strong acids. It may be noted that even before Cavendish, hydrogen gas was prepared by others, for example, Robert Boyle. However, it is Cavendish who is usually given the credit for recognising the elemental nature of hydrogen. Cavendish's careful studies involving specific gravities of gases established hydrogen gas as an individual substance. The name "hydrogen", derived from Greek words meaning "to give rise to water", was given by Antoine Lavoisier. Cavendish called it "inflammable air", which he considered as pure phlogiston. The name "phlogiston" was given by Georg Ernst Stahl, who described phlogiston as a "fluid that was released or lost by any substance when it burned, calcined or otherwise oxidised". It was an erroneous concept, which was finally overthrown by Lavoisier. However, it should be noted that the concept of phlogiston or the phlogiston theory helped to explain a large number of puzzling chemical phenomena and it guided chemical research for more than 90 years.



Cavendish' apparatus

Robert Boyle

Cavendish determined the compositions of water and of nitric acid. He found that volume ratio of oxygen and hydrogen in water was 2:1. In 1783, Cavendish determined the composition of Earth's atmosphere with great accuracy. He concluded that "common air consisted of one part of dephlogisticated air (oxygen), mixed with four of phlogisticated (nitrogen)".

In 1783-84, Cavendish demonstrated that hydrogen reacted with oxygen to produce water. In this way Cavendish demonstrated that water is a combination of two gases and not an element. It was a path-breaking discovery. Since the days of Aristotle water was regarded as one of the four elements of which all substances were made. James Watt and Lavoisier also made similar observations.

Georg Ernst Stahl

Cavendish worked on specific heat and latent heat. It is quite possible that he was aware of Joseph Black's work. He believed that heat was generated from "internal motion of the particles of bodies."

In 1798, Cavendish conducted an experiment to measure the force of gravity between masses in laboratory and to produce an accurate value for Earth's density. This experiment has come to be known as the Cavendish Experiment. The experiment was originally devised by John Michell and he had also a built a torsion balance apparatus for the purpose. However, Michell died in 1793 without completing the work. After the death of Michell, his apparatus was first sent to Francis John Hyde, who in turn sent it to Cavendish. While Cavendish rebuilt the apparatus, he did not much deviate from the original plan of Michell. He conducted a series of measurements with the apparatus. He published the results of his carefully conducted experiments in the *Philosophical Transactions of the Royal Society* in 1798. It is often reported that Cavendish's purpose for conducting the experiment was determining the gravitational constant (G). But it is not true. In his paper, which presented his experimental results, he did not mention the value of G. His chief goal was to measure the density of the Earth, what he called "weighing of the world". It may be noted that in those days the gravitational constant was not considered very significant. It was viewed simply as proportionality constant in Newton's law. But 18th century astronomers were much interested in the value of the density of the Earth. This was because once the Earth's density was known the densities of the Moon, the Sun and the other planets could be deduced from it.

Cavendish's apparatus consisted of a torsion balance made of a 1.8-metre wooden rod suspended from a wire with a 51 mm diameter 0.73-kg lead sphere attached to each end. Two 300-mm 158-kg lead balls were located near the smaller balls, about 230 mm away. Cavendish measured the force on a small ball caused by a large ball of known mass and compared it with the force on the small ball caused by the Earth. After this he could deduce Earth to be N times more massive than the large ball. Thus to measure the density of the Earth it was not necessary to find the numeric value for G. Cavendish reported the value of Earth's density as 5.48 g/cm^3. It may be noted that because of a simple arithmetic error the originally reported value of Cavendish was 5.448. The error was detected by F. Baily in 1821.

Most of Cavendish's electrical studies remained in unpublished. His notebooks and manuscripts containing his electrical studies discovered after almost 100 years were edited by James Clerk Maxwell. Among the major discoveries of Cavendish in the area of electricity are:

- The concept of electric potential, which Cavendish called the "degree of electrification".
- The formula for the capacitance of a plate capacitor.
- The concept of dielectric constant of a material.
- The relationship between electric current and electric potential, now called Ohm's Law.
- Laws for the division of current in parallel circuits, now attributed to Charles Wheatstone.
- Inverse square law of variation of electric force with distance, now called Coulomb's Law.

Cavendish had established a library at his house in Bedford Square. After the death of the librarian he himself acted as librarian. On a specific day of the week he used to lend books to men of letters who were either personally known to him or were recommended by his friends. Even when he took a book from the library for his own use he did not forget to enter it in the loan book.

Cavendish died on 24 February 1810. He left about 700,000 pounds in bank deposits and a landed estate with an annual income of 6,000 pounds. The famous Cavendish Laboratory of the Cambridge University was endowed by one of his relatives William Cavendish, the 7th Duke of Devonshire, who also served the University as its Chancellor from 1861 to 1891.

James Clerk Maxwell

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(The article is a popular presentation of the important points on the life and work of Henry Cavendish available in the existing literature. The idea is to persuade the younger generation to know more about Henry Cavendish. The author has given the sources consulted for writing this article. However, the sources on the Internet are numerous and so they have not been individually listed. The author is grateful to all those authors whose works have contributed to writing this article.)

Progeria

Life in Fast - Forward Mode

Sukanya Datta
E-mail: sukanya@csir.res.in

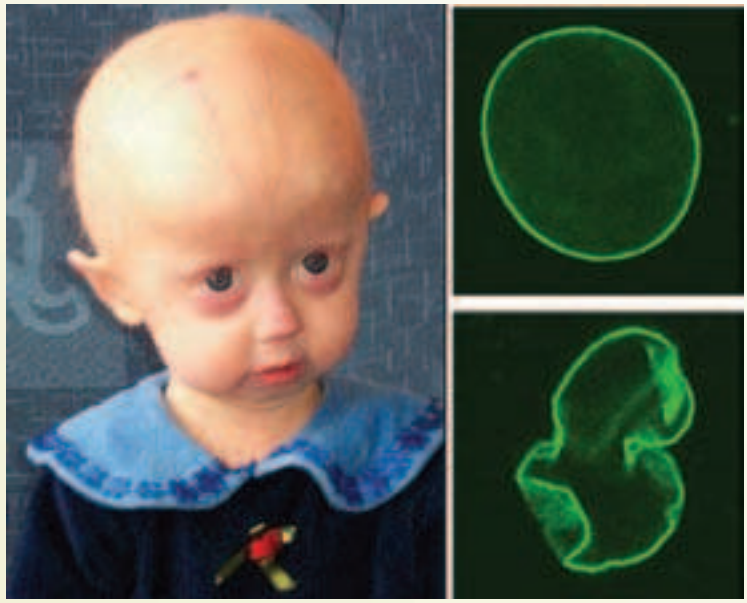
Young Ikramul died of old age. No, you did not read that wrong. The apparently contradictory statement is actually correct; irrespective of which word denoting age that you choose to focus on. Chronologically Ikramul was only twenty-two years of age when he died. Young, by any standard. Biologically he was eighty five. Old, you will agree. This is the tragedy of progeria—an extremely rare genetic condition that is characterized by premature ageing.

The word 'progeria' comes from Greek words *pro* and *geros*. *Pro* in Greek means 'before' and *geros* means 'old age' Put together, progeria therefore means 'prematurely old'. It sums up what this genetic condition does to the afflicted person—makes him/her old before his/her time. This is because in this syndrome, the rate of ageing is accelerated up to seven times that of normal. Human progeria manifests in two major forms, Werner's syndrome and Hutchinson-Gilford syndrome. The best known or classic type is Hutchinson-Gilford Progeria Syndrome (HGPS). Together, Werner's syndrome and HGPS are sometimes referred to as progeroid syndromes.

There are a number of other less well-known forms of progeria or accelerated-aging diseases. These present different aspects of aging but never all the aspects simultaneously. For example, in HGPS, patients though appearing to be 'old', do not show arthritis (which is actually a wear and tear problem) or cataracts. However, their skin and heart show signs of aging. Therefore these are often called "segmental" progerias.

History

Progeria was first described, almost simultaneously, by Dr. Jonathan Hutchinson in 1886 and Dr. Hastings Gilford in 1897. This is how the name Hutchinson-Gilford Progeria Syndrome came about. However, little research was done till the 1990s because this condition is so rare. It is reported to occur in just one out of 4-8 million newborns worldwide. Only about 130 cases have been reported in scientific literature since the condition was first described. As a result, very little was known about progeria, till recently. Much of the accelerated pace in progeria research in the past decade has been catalysed by a pair of parents whose son was diagnosed with the condition. Thus progeria research is not only driven by pure intellect. It is motivated by a mother's love too.



Public awareness about this disorder was long in coming, perhaps because this condition is so rare. Probably, progeria entered the consciousness of the general public in 1981, when the Pulitzer Prize-winning photographer Eddie Adams took a picture of Fransie Geringer of South Africa and Mickey Hays of Texas. The boys met in Disneyland, where Fransie had been sponsored for a trip by a wish-granting philanthropic organisation called the Sunshine Foundation. The photo was carried on Associated Press wires and appeared on the front page of the Style section of *The Washington Post* under the headline "Growing Up Old." Public awareness in India has received a similar fillip thanks to the recently released movie, Paa.

Tell-tale early signs

New-born children with progeria do not manifest the symptoms at birth. However, by the time they reach two years (or as early as 10 months) signs of accelerated aging start to appear. There may be:

- Growth failure
- Loss of body fat and hair
- Aged-looking skin
- Joint stiffness
- Hips locked in a position called a "horse-riding stance"
- Generalised atherosclerosis (hardening of arteries)
- Stroke

Progressive problems

Children with progeria all look remarkably similar. They have a small face

and jaw as compared to head size. They are bald and have a pinched nose with a beaked tip. They develop a characteristic facial appearance including prominent eyes, thin lips, and protruding ears. Loss of hair and body fat along with changes in the skin give rise to characteristic *"plucked bird"* appearance at about 6-12 months of age. Scalp hair and eyelashes are progressively lost. There is increased prominence of scalp veins. Delayed eruption of teeth and abnormal dentition are also common.

Thankfully, the development of motor skills such as sitting, standing, and walking develop normally. In addition, they also have normal intelligence, which perhaps in a way makes the condition more tragic, as they can fully understand what lies ahead.

Children with HGPS are prone to premature, progressive heart disease and experience severe hardening of the arteries beginning in childhood. This condition greatly increases the chances having a heart attack or stroke at a young age. Heart disease is the commonest cause of death. Such children also face high blood pressure, angina (chest pains), enlarged heart and heart failure – all conditions linked to aging. Death comes at around age thirteen for most (but may occur as early as eight or as late as twenty-one), usually by heart attack or stroke.

Joining hands to target progeria

In 1998, Dr. Leslie Gordon and Dr. Scott Berns' son Sam (then 22 months old) was diagnosed with HGPS. They soon realised that there was woefully little knowledge and/or resources available to parents battling the situation they were in. They then set up the Progeria Research Foundation (PRF) with the aim to discover the cure and effective treatment for the condition and its aging related disorders. PRF – a non-profit organisation funds medical research aimed at developing treatments and a cure for progeria. PRF also has its own Cell & Tissue Bank that provides the biological materials researchers need to conduct their experiments.

In 2001, PRF began working in partnership with major institutes of the National Institutes of Health (USA), including the National Institute on Aging and the Office of Rare Diseases, to co-host a joint workshop. PRF and NIH brought together leading scientists from across the globe to identify promising areas of research in progeria. This workshop led to funding for progeria research and the formation of the PRF Genetics Consortium, a group of twenty scientists whose common goal was to find the genetic cause, treatment and cure for progeria.

In April 2003, the Progeria Research Foundation and the National Institutes of Health, announced that the gene for Hutchinson-Gilford Progeria Syndrome had been found by a the researchers of PRF Genetics Consortium. The report was published, in May 2003 in *Nature*.

Unearthing genetic roots

HGPS is caused by a point mutation in the lamin A, or LMNA gene located on the long arm of Chromosome 1. This means HGPS is caused by a tiny change in the DNA of one single gene. The scientific study reported in May 2003 in *Nature* stated that 18 out of 20 classical cases of HGPS had an identical *de-novo* (newly arisen; not inherited) single-base substitution. The lamin-A gene in almost all of them was abnormal in one consistent way. Instead of a C nucleotide in one spot of Chromosome 1, progeria patients had a T. One additional case was identified with a different substitution within the same codon. Both mutations resulted in an aberrantly stunted protein that was short by 50 amino acids towards one end. The mutated protein was named progerin.

Role of the normal protein

The LMNA gene normally encodes a protein called Pre-lamin A. This protein is further processed and becomes lamin A. Lamin A is a protein that is an essential supporting component of the nuclear envelope, which is the membrane that surrounds the nucleus. Normally, lamin proteins polymerise to form an intra-nuclear supporting structure or scaffold known as the lamina, particularly around the edge of the nucleus. This lamina supports the nuclear architecture and helps to organise nuclear processes such as DNA and RNA synthesis. So, in a way lamin A helps to keep the integrity of the nucleus by preserving its boundaries in the correct way.

Effect of mutated protein

Quite a few degenerative human disorders are linked to different mutations in lamin A. Interestingly, so far, eleven so-called lamin-opathies are known and many of these involve premature aging.

The mutated LMNA gene generates an abnormal lamin A protein (progerin) that disrupts the cell's nuclear membrane. It is believed that the defective lamin A protein makes the nuclear membrane unstable. It also progressively damages the nucleus, making cells more likely to die prematurely. Under the microscope, nuclei of cells taken from progeria patients have dramatically different morphology as compared to the uniform shape typically found in healthy individuals.

The research article in *Nature* has major implications not only for the patients (not many, thankfully) but also for all of us. Francis Collins, Director, National Human Genome Research Institute rightly pointed out that, "Isolating the progeria gene is a major achievement for the medical research community,...The discovery not only gives hope to children and families

affected by progeria, but also may shed light on the phenomenon of aging and cardiovascular disease."

Werner's Syndrome

Werner's syndrome or adult progeria is named after Otto Werner, a German scientist, who described the syndrome as part of his doctoral thesis in 1904. It partly mimics the symptoms of progeria, although these patients are diagnosed only in early maturity as opposed to early childhood for HGPS. Patients with Werner's syndrome usually have an average life span of forty-seven years.

The gene associated with Werner syndrome lies on chromosome 8 in humans. It codes for a DNA helicase — an enzyme that unwinds DNA for replication, transcription, recombination, and repair. The inability to repair DNA may explain the features of premature aging. The mutation in the DNA helicase gene is not present in HGPS cells. It is thought that, unlike Werner's syndrome, HGPS is not caused by defective DNA repair.

Is progeria hereditary?

HGPS is a genetic condition—since its roots lie in altered structure of the gene. However, because HGPS almost always occurs in people with no history of the disorder in their family, it is not considered to be hereditary in the sense that the condition is passed down the generations. HGPS is considered an autosomal dominant condition. This means just one copy of the altered gene in each cell is sufficient to cause the disorder (We inherit two copies of genes; one from each parent. Just one copy of a dominant gene is needed for the encoded trait to be expressed; both copies of the recessive genes are needed for a recessive trait to be expressed.)

It is thought that HGPS cases arise because of new mutations in the LMNA gene, and it is believed that the mutation happens purely by chance. There is no "cause" leading to progeria—more's the pity! Since this is an autosomal (related to the 22 pairs of similar chromosomes that men and women have in common) condition, both males and females run equal risk of having it. A non-twin sibling runs the same risk of having progeria as any other child from another family. Progeria is not seen in siblings of affected children; but in extremely rare cases more than one child in the same family may have the condition. There are just two or so such families in the world and one of them is from India.

The Indian Chapter

Classical Hutchinson-Gilford Progeria Syndrome is almost never passed on from parent to child. The word to notice is "almost" because in very rare instances, about once every 100 cases of HGPS (or frequency of about 1 in 400 million births), HGPS can be passed down within a family.

It is usually caused by a new (sporadic) mutation during the early division of the cells in the developing infant. It is usually genetically dominant; therefore, parents who are healthy can be assumed NOT to have a copy of the mutated gene to pass on to their children. Children affected with progeria rarely live long enough to have children of their own, so they almost never pass on the affliction.

However, sometimes tragedy strikes the same family multiple times belying the odds. This is the case with Bisul and Razia Khan who had seven children in all; five of them with progeria. Their daughter Rehana died in 2008, chronologically aged just 23, but biologically, a woman of 90. Their son Ikramul passed away recently. Another son, Ali Hussein is about eleven years old but with a body of a person in his sixties. Their two surviving daughters are normal. The family in India is therefore extremely unusual in having five members who are/were afflicted. This family is now under monitoring and it is believed that their progeria is because of recessive genes. Researchers are hopeful that they will be able to add vital pieces to the jigsaw puzzle that is progeria. It is interesting also that there has been, at least, one other report which says that a certain form of progeria is an autosomal recessive condition. This is the report in 2008 by Rajul Rastogi and SM Chander Mohan, who presented radiological features of a rare case of progeria in the *Indian Journal of Orthopedics*.

Why study such a rare disorder?

According to resources on the Internet, there are only 53 known cases of progeria around the world. Over the last 15 years, the cases have been reported from Algeria, Argentina, Australia, Austria, Canada, China, Cuba, England, France, Germany,

India, Israel, Italy, Mexico, the Netherlands, Poland, Puerto Rico, South Africa, South America, South Korea, Switzerland, Turkey, US, Venezuela, Vietnam and Yugoslavia. So, no population can hope to be forever safe and never ever to be subject to the ravages of progeria. Because it is a random mutation, it can strike any foetal genome; anywhere. Studying progeria has tremendous implications for all because it will definitely uncover more facts about heart disease and the normal aging process that affects us all.

Mouse model for HGPS

Researchers from the National Cancer Institute (NCI), USA have developed a mouse model for studying HGPS, which may facilitate a better understanding of the syndrome, as well as provide clues to the normal senescence. Colin Stewart (NCI's Center for Cancer Research) said that, "The similarities between mice with this particular mutation and patients with progeria are remarkable...Now that we've identified the critical gene and have an animal model that mimics progeria, we have powerful tools for studying both the aging process and this devastating disease."

NCI researchers reported that the mouse model (with mutation in the LMNA gene) present symptoms very similar to progeria patients. These mutated mice are indistinguishable from their littermates at birth, but show severe growth retardation and signs of premature aging such as thin skin and hair loss. They also have degenerated heart and skeletal muscles; incomplete development of the skeleton or a premature loss of bone mass; abnormal teeth, and incomplete sexual maturation. They die within five weeks. Normal mice, on the other hand have a life span of up to two years.

A faint ray of hope

Just about a decade ago, there were practically no resources for patients of progeria. However, today, the Progeria Research Foundation has created a diagnostic testing program that can confirm progeria through a genetic test. This is the first time ever, that a definitive and scientific way to diagnose the children has been devised. In addition to having a potential gene target that can be spotted when a patient's genome is decoded, there are clinical trials on progeria that are under way.

Clinical trials

The progeria clinical drug trial began on 7 May 2007 with two children arriving in Boston, USA for seven visits over a two-year period. The first-ever progeria clinical drug trial was completed on 14 August 2009. PRF and Children's Hospital, Boston are once again partnering to conduct a second clinical trial for children with progeria. This much larger trial will include up to 45 children from 19 different countries!

Potential treatment regimes?

Researchers have identified a potential drug treatment for progeria. It involves a group called Farnesyl-Transferase Inhibitors or FTIs. For the first time, there is hope for a possible treatment for children with progeria.

To understand how FTIs are expected to act, it is important to know how the mutated protein progerin acts. A molecule called a "farnesyl group" must be attached to the progerin protein for progeria to occur. FTIs short-circuit this 'attachment' by blocking (inhibiting) the attachment of the farnesyl group onto the progerin protein. Scientists hope that by blocking the farnesyl group attachment in progeria patients, FTIs can paralyse the action of progerin and lead to an improvement in the condition. Additionally, the researchers have identified two other drugs (pravastatin and zoledronate) that, when used in combination with the current FTI drug being tested, may provide an even more effective treatment for children with progeria than FTI's alone. All three drugs will target different points along the pathway leading to production of the disease-causing progerin. In laboratory studies presented by Dr. Carlos Lopez-Otin of Spain at the 2007 PRF Scientific Workshop, the two new drugs extended life span in mouse models of progeria.

While currently, there is no treatment for progeria, it is likely that in the not too distant future, those with progeria will have viable medical options that may extend the life span that the cruel throw of genetic dices had ordained for them. ■

Dry Eyes
Causes and Cures

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

Dryness of the eye is a common problem. It can occur in people of all ages and can relate to a variety of factors. Those of us who work long hours on personal computers frequently face this problem, while those who live in congested towns and put up with environmental irritants suffer equally from its wrath. Although the dryness does not produce any permanent damage in the eye, irritation within the eye and diminished vision may prompt the sufferer to seek medical attention.

Symptoms

The condition occurs when the tear-producing apparatus of the eye suffers a break down or delivers tears of poor quality. This causes the cornea, or parts of it, to dry out. Both the eyes are usually affected. The symptoms include :

- A stinging, burning, or scratchy sensation,
- Stringy mucus in or around the eyes,
- Increased eye irritation from smoke or wind,
- Eye fatigue after short periods of reading, and
- Difficulty wearing contact lenses.

In some people, dryness of the eyes is also associated with excessive watering. This may sound like a contradiction, but there is a good reason for this. Tears are produced in two ways. "Basic tearing" produces tears at a slow, steady rate and keeps the eyes lubricated. In contrast, "reflex tearing" produces large quantities of tears in response to eye irritation or emotions. Reflex tears contain much more water than do basic tears, and they are low in mucus and oils. When the eyes become irritated from dryness, the lachryimal gland floods the eye with reflex tears. Fluid overwhelms the tear duct and overflows the eyelids. Yet, because these tears are of poor quality, they do not help the dryness.

Common culprits

A variety of conditions can lead to dryness in the eyes. This can happen due to insufficient tear production, if the tears are of poor quality or if the environment is hostile to the eyes.

Insufficient production of tears

In some people, the tear-producing apparatus of the eye does not produce enough tears. As a result, the eyes are not comfortably lubricated. This can happen due to the effects of ageing, medications, menopause, autoimmune disorders, chemical burns or deformities of the eyelid.

Disruption of the blink reflex

Basic tearing produces tears at a slow, steady rate and keeps the eyes lubricated. The natural blinking process helps spread the tears more evenly. However, people who spend long hours on computer stations, tend to concentrate so hard on the monitor that their natural blinking process gets adversely affected. That's why eyestrain and dry eyes

are a common complaint among those people who work on laptops, PCs or other video display terminals as a part of their job.

Damage to the tear glands

The lacrimal glands can suffer damage from inflammation or radiation. This can hamper tear production. Dry eye is also associated with certain diseases such as rheumatoid arthritis, systemic lupus erythematosus (SLE, an inflammatory disease of connective tissue), scleroderma (an autoimmune disease that affects the blood vessels and connective tissue), and Sjogren's syndrome.

Medications and dry eyes

A wide variety of common medications, both prescription and over-the-counter (OTC), can cause dry eyes. These include:

- Allergic reaction to eye drops or ointment
- Diuretics or water pills
- Antihistamines and decongestants
- Sleeping pills
- Tricyclic antidepressants
- Accutane-type drugs for treatment of acne
- Opiate-based pain relievers such as morphine

The ageing effect

As one gets older, the tear production tends to dry up. When you're unable to produce enough tears, your eyes become easily irritated. Although dry eyes can affect both men and women at any age, it is more common among women, especially after menopause. This may be due to hormonal changes brought on by this phase.

Deformities of the eyelids

The eyelid — usually the lower lid — may turn in toward the eye, allowing the skin of the eyelid and the eyelashes to rub against the conjunctiva. This condition is called 'entropion'. Ectropion is a condition quite the reverse of an entropion. The eyelid turns out, rather than in, and sags; it can no longer close properly, and without the protection of the lid, the inside of the eyelid and the surface of the eye become dry, irritated

and inflamed. Both entropion and ectropion can cause a dry eye.

Poor tear quality

Some people produce a normal amount of tears, but the composition of the tears is of poor quality. The tears lack certain components, such as oil, which is essential for lubrication.

If you analyse tears more closely, they are much more than just water. In actuality, they are a complex mixture of water, fatty oils, proteins, electrolytes, bacteria-fighting substances and growth factors that regulate various cell processes. This mixture helps make the eye surface smooth and clear. Without it good vision is impossible. The eyelids spread tears across the surface of the eye in a thin film. The tear film has three basic layers:

Mucus. The inner layer consists of mucus produced by the conjunctiva. This layer allows tears to spread evenly over the surface of the eye.

Water. The middle layer, which makes up about 90 per cent of the tear, is mostly water with a little bit of salt. This layer, produced by the lachrymal glands, cleanses the eye and washes away foreign particles or irritants.

Oil. The outer layer, produced by glands on the edge of the eyelid, contains fatty oils called lipids. These smooth the tear surface and slow evaporation of the watery layer.

Considering this complicated mix of ingredients, it is not surprising that the balance is sometimes off. An imbalance causes the tears to evaporate faster and your eyes to become dry. Certain diseases or chemical burns can cause changes in the oily and mucous layers of your tears.

Blepharitis, acne rosacea and other skin disorders also can disrupt production of the oily layer. Blepharitis is the inflammation of the eyelids. A chronic condition, it involves the edges of the eyelids. The signs and symptoms include a gritty, burning sensation in the eyes, watery or red eyes, swollen eyelids, and flaking of the skin around the eyes. The eyelids may appear greasy and crusted with scales that cling to the lashes. This debris can cause the eyelids to stick together at night.

Environmental factors

Several environmental irritants such as high pollution, excessive smoke, strong Sun, high velocity wind, and indoor heating may also cause dryness and scratchy feeling in the eyes. These environmental factors are a common cause of dry eyes in the large cities and metros of this country.

What You Can Do

Like any liquid, tears will evaporate when exposed to air. These are simple steps you can take to help slow evaporation:

- Remember to blink. Conscious blinking repeatedly helps spread the tears more evenly and keeps the eye moist.
- Do not direct hair dryer, air conditioner, fan or car heater toward your eyes.
- Wear glasses on windy days. Use protective goggles while swimming.
- Keep your home humidity between 30 per cent and 50 per cent. In winter a humidifier can add moisture to dry indoor air.

Medical help

If your eyes feel dry and irritated, your eye doctor can help. Regardless of the cause of dry eyes, the goal of treatment is to keep your eyes moist. This can be done either by replacing the tears or conserving them.

Artificial tears

A mild case of dry eyes usually can be treated with artificial tears. You can use the lubricating drops as often as needed, even several times an hour, to provide relief. If you use drops frequently, preservative-free eye drops might be the best choice to avoid an allergic or toxic reaction to preservatives.

Ointments can be used to ensure lubrication. These ointments can blur vision, so it is best to use them only at night.

Conserving tears

Your eye doctor may also suggest methods to keep your tears from draining. The tear drainage ducts can be plugged either temporarily or permanently with tiny collagen or silicone plugs. The closure conserves both your own tears and artificial tears you may have added.

Collagen plugs will slowly dissolve over a few days. Silicone plugs can be removed or left in. A more permanent option is thermal cautery. In this procedure the doctor numbs the area with an anesthetic and then applies a hot wire that shrinks the tissues of the drainage area and causes scarring, which closes the tear duct.

Correcting eyelid deformities

Both entropion and ectropion can be corrected through simple surgery. You can discuss the option with your eye doctor or a plastic surgeon. ■

Recent Developments in Science and Technology

Biman Basu
e-mail: bimanbasu@gmail.com

Bollworm develops resistance against Bt cotton

Seven year after genetically modified Bt cotton was introduced for cultivation in India, the bollworm moth, one of the most destructive pests of cotton crops, appears to have developed immunity to Bt cotton, a GM crop variety that contains a protein toxin (originally found in a bacterium called *Bacillus thuringiensis*) that is poisonous to some insects but normally harmless to animals. A bollworm species resistant to the Bt toxin has recently come to light from Gujarat. According to a report published in *Science* (19 March 2010), Monsanto scientists have detected "unusual survival" of pink bollworms that fed on cotton containing the Cry1Ac gene from the bacterium *B. thuringiensis* (Bt). The type of Bt toxin to which these bollworms have become resistant is called Cry1Ac. The Cry1Ac gene codes for a protein that is toxic to many insect pests including cotton bollworm. According to the *Science* report, Monsanto scientists collected "large numbers" of pink bollworms from Bollgard I hybrid cotton and fed them Bt toxins at normally lethal concentrations in the lab, and the worms survived. Bt cotton was developed by the multinational chemical company Monsanto primarily to fight bollworm infestation, which is a major pest of cotton.

This is, however, not the first instance of bollworm developing resistance to Bt toxin. In a study in 2008 by University of Arizona entomologists, which looked at data from six experiments to monitor pests in fields sown with transgenic cotton and corn in Australia, China, Spain and the United States, researchers found evidence of genetic mutation among bollworms (*Helicoverpa zea*) in a dozen cotton fields sown in Mississippi and Arkansas in USA between 2003 and 2006. The mutation entailed a slight change in the bollworm's DNA to help it resist a toxin that the Bt cotton plant exudes. The researchers had found that resistance among the bollworms developed faster in places where there was little or no "refuges," the term for areas where there are non-Bt crops. The idea behind refuges is to provide a haven for pests that do not have the genetic mutations. This boosts the probability that a resistant pest will mate with a non-resistant pest, creating a hybrid that would still be susceptible to the toxin. In most pests, offspring are resistant to the novel toxins only if both parents are resistant. However, a new variety of Bt cotton, which combined a second toxin, Cry2Ab, with Cry1Ac, was found to successfully combat the resistant bollworms.

Cotton boll damaged by bollworm

(In India, Bt cotton variety with two Bt genes is marketed as more expensive Bollgard II.)

Ironically, since Indian farmers adopted Bt cotton for cultivation in 2002, cotton production in the country has gone up substantially. Today India is the second-largest cotton producer in the world, after China. According to an estimate of the Central Institute for Cotton Research (CICR) in Nagpur, a total of 8.3 million hectares, or 83% of the country's total cotton crop, is the Bt variety and most of it was Bollgard II cotton. According to Monsanto, Bt resistance was seen only in farms that planted the Bollgard I variety, with only the Cry2Ab gene; Bollgard II variety is safe. More than 65% of cotton farmers in Gujarat state chose Bollgard II in 2009, and indications are that more than 90% farmers may go for it this year too. Nevertheless, the emergence of resistance against Bt toxin in bollworm does raise doubts about the long-term implications of Bt crops as a viable alternative to conventional pesticide use.

Banana chemical fights HIV

A potent new inhibitor of HIV, derived from bananas, may open the door to new treatments to prevent sexual transmission of HIV, according to a University of Michigan Medical School study. In laboratory tests, scientists have found that

a key ingredient of banana called BanLec was as potent as two existing anti-HIV drugs (*Journal of Biological Chemistry*, 19 March 2010). BanLec is a lectin – a naturally occurring chemical found in plants which fights infection. Lectins are glycoproteins that act like specific antibodies but are not antibodies in that they are not evoked by an antigenic stimulus.

Scientists have long been interested in lectins because of their ability to halt the chain of reactions that leads to a variety of infections. Lectins are sugar-binding proteins. They can identify foreign invaders, like a virus, and attach themselves to the pathogen. The Michigan scientists found that banana lectin acted as a potent HIV inhibitor by blocking the virus's entry into the body. According to the lead author Michael Swanson of the University of Michigan, "The problem with some HIV drugs is that the virus can mutate and become resistant, but that's much harder to do in the presence of lectins". This is because lectins can bind to the sugars found on different spots of the HIV-1 envelope, and presumably it would take multiple mutations for the virus to get around them.

The finding is significant because HIV is still widespread globally, and especially in poorer countries it continues to be a grave problem because of tremendous human suffering and the cost of treating it. The rate of new infections of HIV is outpacing the rate of new individuals getting anti-retroviral drugs by 2.5 to1, and at present it appears that an effective vaccine is years away. So there was urgent need find new ways of stopping the spread of the HIV. According to the researchers, therapies using BanLec could be cheaper to create than current anti-retroviral medications, which use synthetically produced components, plus BanLec may provide a wider range of protection.

The Michigan researchers are developing a process to alter the BanLec molecule to enhance its potential clinical utility. Clinical use is considered years away but researchers believe it could be used alone or with other anti-HIV drugs as a vaginal microbicide (an agent that destroys microbes) to prevent HIV infection. According to the researchers, even modest success could save millions of lives. Other investigators have estimated that 20 percent coverage with a microbicide that is only 60 percent effective against HIV may prevent up to 2.5 million HIV infections in three years. The researchers are hopeful that of all the available therapies, the banana lectin may become one of the least expensive treatments to prevent the sexual transmission of the lethal virus and save millions of lives.

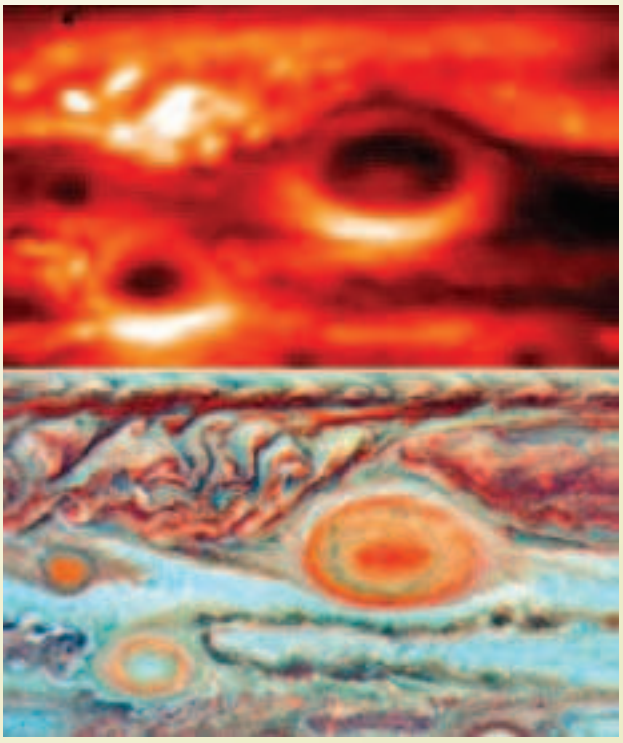
First look at weather inside Jupiter

The best known feature of Jupiter is the Great Red Spot, a persistent oval-shaped storm system, located 22° south of the equator. The oval spot is at least hundreds of years old and has been observed by astronomers on Earth since the 19th century. The storm is massive, and is large enough to fit three entire Earths inside. It rotates counter-clockwise, with a period of about six Earth days. The spot is large enough to be visible through Earth-based telescopes with an aperture of 12 cm or larger. Mathematical models suggest that the storm is stable and may be a permanent feature of the planet.

Despite observations spanning several decades the real structure of the Great Red Spot was unknown to astronomers, although it was known that the spot could not be associated with any deeper feature on the planet's surface, as it revolves along with the rest of the cloud bands of the planet, but at a slower speed. Over the past few decades, astronomers had begun to understand the weather patterns around the Great Red Spot, but not inside of it.

Astronomers had also noticed that the colour of the spot changes considerably, but what drives the changes is unclear. A leading theory was that sulphurous molecules from deep in the Jovian atmosphere were being lofted by the storm, exposing them to ultraviolet radiation that would break them apart. The newly freed sulphur atoms would then change colour and lend the area its distinctive tinge. But, as is known now, that might not be the case. Previous measurements had also indicated that the spot towered over the surrounding cloud cover, much like supercells on Earth. (Supercells are long-lived thunderstorms which generally produce severe weather on Earth.)

Now, stunning new thermal images of the Red Spot derived from ground-based telescopes reveal the first detailed weather maps of Jupiter's giant storm system. They also show a clear correlation between the environmental conditions



Jupiter's Great Red Spot as never seen before. The top image was obtained with the VLT in the infrared wavelength range of 10.8 microns. The bottom image in visible light was obtained by the Hubble Space Telescope. (Credit: ESO/NASA/ JPL/ESA/L. Fletcher)

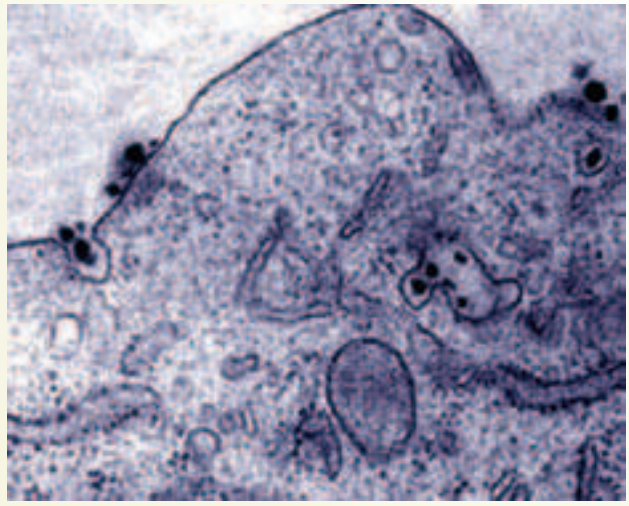
and colour. The images reveal surprising weather and temperature variation within the solar system's most famous storm. They show swirls of warmer air and cooler regions never seen before within the Red Spot (*Icarus*, 6 February2010, doi:10.1016 j.icarus.2010.01.005).

The new images were taken by the VLT Imager and Spectrometer for mid Infrared (VISIR) instrument on ESO's Very Large Telescope (VLT), the Gemini South telescope, and the National Astronomical Observatory of Japan's Subaru Telescope. The images have enabled scientists to link the spot's temperature, winds, pressure and composition to its colour. By combining the thermal data with observations of the deep cloud structure by the 3-metre NASA Infrared Telescope Facility in Hawaii, the details seen in the image is comparable to visible light images from the space-based Hubble Space Telescope.

In the new images, the reddest colour of the spot has been found to correspond to a warm core within the otherwise cold storm system that averages -160°C, with dark lanes at the edge of the storm marking the locations where gases are sinking deeper into the planet. According to Leigh Fletcher, an Oxford astronomer and the lead author of the paper, "One of the most intriguing findings shows the most intense orange-red central part of the spot is about 3-4°C warmer than the environment around it." According to the researchers, the new images do not tell us anything about the chemicals or processes that are causing that deep red colour, but they do tell us that it is related to changes in the environmental conditions right in the heart of the storm.

RNA-Loaded Nanoparticles Fight Cancer

Researchers had postulated for years that nanoparticles offer a treatment to many forms of cancer. Now it appears that indeed it is possible. Recently researchers at the California Institute of Technology (Caltech) in the US have developed a nanoparticle carrying a molecular marker that binds to the surface of cancer cells, triggering the cells to absorb it. When these nanoparticles carrying RNA were injected directly into a cancer patient's bloodstream in a clinical trial, the nanoparticles successfully reached cancer cells and silenced the target gene. When the researchers later analysed biopsy samples from three melanoma patients in the trial who had received different doses of the therapy, they found that the amounts of RNA they could see in the tumour cells were proportional to the doses the patients received; that is, a higher number of nanoparticles sent into the body leads to a higher number of nanoparticles in the tumour cells. (*Nature*, 21 March 2010| doi:10.1038/nature08956). This is the first time that this type of tissue targeting and gene-silencing in humans has been demonstrated.



Nanoparticles (black dots) carry a molecular marker on their surface that engages receptors on a cancer cell's surface, allowing the nanoparticles to be taken into the cell. Here, the nanoparticles can be seen both entering and inside the cell. (Credit: Swaroop Mishra)

Since the discovery 12 years ago that double-stranded RNA can silence genes in a targeted manner, researchers have hailed the technique, known as RNA interference (RNAi), as a powerful approach to creating new and potent medicines. (RNAi is a system within living cells that helps to control which genes are active and how active they are.) Indeed, the discoverers of RNAi, Stanford University's Andrew Fire and Craig Mello of the University of Massachusetts Medical School, were awarded the 2006 Nobel Prize in medicine. But the trouble was, getting the therapeutic RNA to the right cells proved to be a sticky challenge. When not packaged with nanoparticles and injected on their own, the so-called 'small interfering RNA's are quickly filtered out by the kidneys, and researchers have struggled to design particles that carry their contents to target cells with enough specificity.

In the Caltech clinical trial, the nanoparticles delivered double-stranded small interfering RNAs, and turned off an important cancer gene using RNAi. The RNA carried within the nanoparticles was designed to silence a gene called ribonucleotide reductase M2 (RRM2), which regulates DNA synthesis and repair and is known to be an anti-cancer target.

According to some oncologists, the trial is a promising start, but much remains to be done before such therapies are truly ready for clinical use. The study does not discuss the clinical effects of the treatment on the melanoma patients in the trial, but at least it has demonstrated the feasibility of a new route of drug delivery using targeted nanoparticles.

Breeding flightless mosquitoes for dengue control

Dengue fever causes severe flu-like symptoms and is among the world's most pressing public health issues. There are 50 million to 100 million cases per year, and nearly 40 percent of the global population is at risk. It is caused by a virus spread through the bite of infected female *Aedes aegypti* mosquitoes. There is no vaccine or specific therapeutic drug for dengue fever, and control efforts are complicated by the fact that the female mosquitoes (which bite) are active during the entire day so that bed nets are ineffective. Now a team of researchers from University of California,

Irvine, California, USA, and colleagues from Oxford Insect Technology (Oxitec) Ltd, UK, and the University of Oxford, UK, led by Guoliang Fu, report the creation of a new breed of *Aedes aegypti* in which the females cannot fly. This, the researchers hope, may help curb the transmission of dengue fever. Besides, the flightless females should be more susceptible to predators and unable to attract males with their wing beating sounds for mating. Consequently, the flightless females are expected to die quickly in the wild, curtailing the number of mosquitoes and reducing – or even eliminating – dengue transmission. The report appears in the online edition of the *Proceedings of the National Academy of Sciences*, 22 February 2010 (doi/10.1073/pnas.1000251107).

Aedes aegypti is an invasive species spread inadvertently around the world by human trade and travel and is now distributed widely in tropical and subtropical regions, most notably in urban environments where it has adapted to breed in artificial containers and refuse. This increase in the geographic distribution of the vector is accompanied by the emergence of the virus and disease in new areas. There is currently no vaccine or specific therapeutic drug available for dengue; therefore, control mainly focusses on the mosquito.

Using concepts developed by Oxitec, researchers made a genetic alteration in the mosquitoes that disrupts wing muscle development only in female offspring, rendering them incapable of flight. Males' ability to fly is unaffected, and they show no ill effects from carrying the gene. When genetically altered male mosquitoes mate with wild females and pass on their genes, females of the next generation are unable to fly. Scientists estimate that if released, the new breed could sustainably suppress the native mosquito population in six to nine months. The approach offers a safe, efficient alternative to harmful insecticides. According to the researchers, the technology is completely species-specific, as the released males will mate only with females of the same species. Field trials are expected to start within two years. ■

NOMINATION FORM FOR TRAINING PROGRAMME ON INNOVATIVE EXPERIMENTS IN PHYSICS

Vigyan Prasar is organizing six regional Training programmes in East, West, South, North, North-East, and Central India during August to October 2010. The objective of this training programme is to illustrate and demonstrate a series of novel activities that may help enhance interest in physics amongst students and teachers. Activities aim at conceptual understanding of Physical phenomena rather than verification of experiments described in a text book. The experiments were jointly developed by Department of Physics, Indian Institute of Technology, Kanpur and Vigyan Prasar.

If you are a physics teacher/science communicator and willing to attend this workshop, kindly send us information as per the details below.

INNOVATIVE EXPERIMENTS WORKSHOP 2010

1. NAME : .....
2. DATE OF BIRTH : .....
3. SEX : .....
4. PROFESSION : .....

Photograph

5. ADDRESS (O) : .....

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PIN .....

6. PHONE : .....
7. E-MAIL : .....
8. Have you developed any low cost or otherwise Innovative experiments/teaching aid in physics? If yes, brief description with photograph. Use additional page, if required.
9. Have you attended any workshop based on innovative experiments in Physics? If yes, specify date, organizer and your contribution, if any?
10. If you are a working teacher, kindly give your School address, with phone/fax number and your nomination should be endorsed by School Principal)

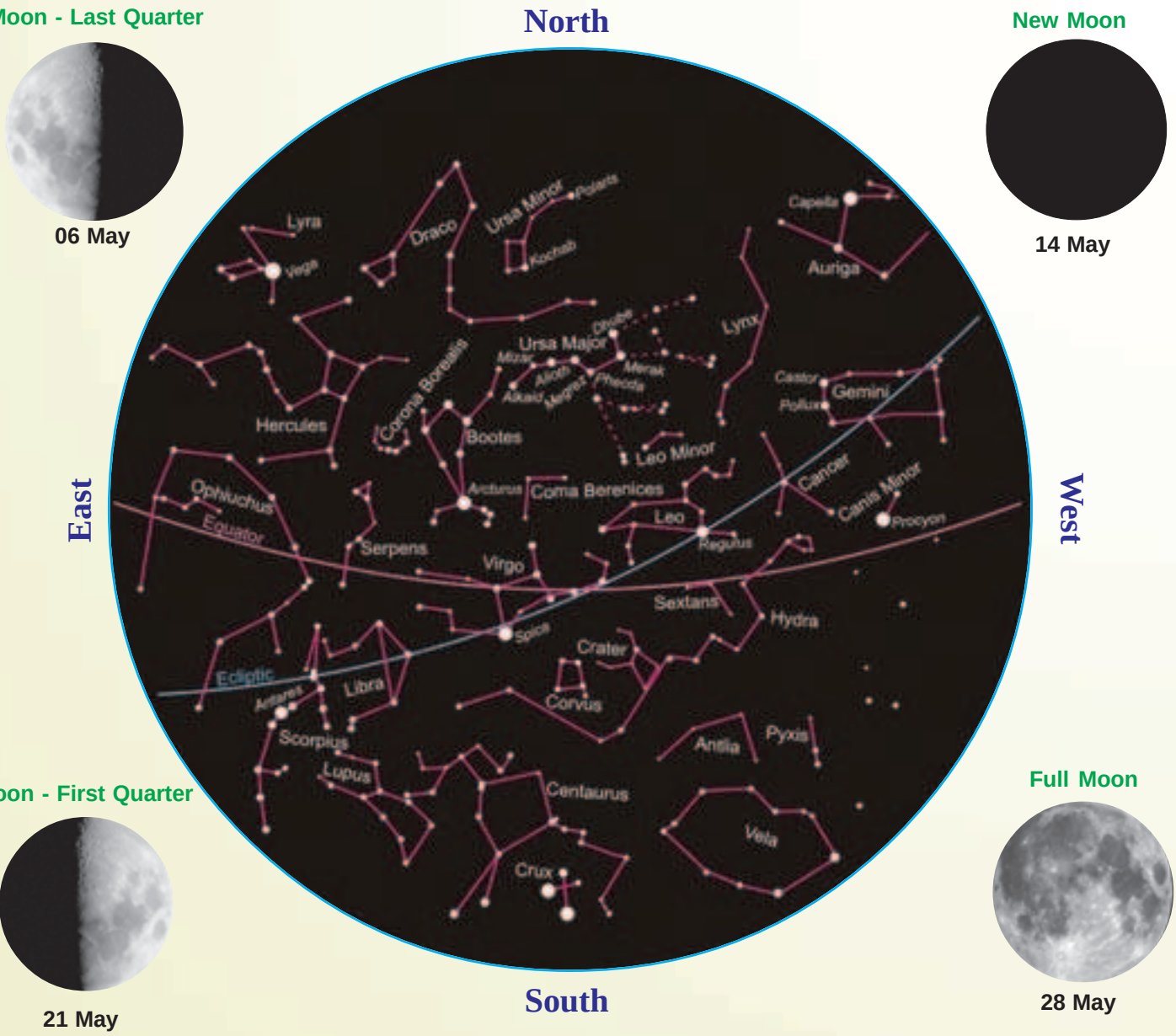
(Signature)

(The workshops will be organized in Chandigarh, Mumbai, Hyderabad, Kolkata, Shillong & Bhopal. After the selection, each nominee will be informed by Vigyan Prasar individually about the exact dates/venue etc.)

Send nomination to : **Desk, Innovative Experiments Workshop 2010**
Vigyan Prasar
A-50, Institutional Area, Sector-62
Noida – 201307 (U.P.)

(You can also fill nomination form online. Visit www.vigyanprasar.gov.in for online submission.

Sky Map for May 2010



The sky map is prepared for viewers in Nagpur (21.090 N, 79.090 E). It includes constellations and bright stars. For viewers south of Nagpur, constellations of the southern sky will appear higher up in the sky , and those of the northern sky will appear nearer the northen horizon. Similarly, for viewer north of Nagpur, constellations of northern sky will appear higher up in the sky, and those of the southern sky will appear nearer the southern horizon. The map can be used at 10 PM on 1 May, at 9 PM on 15 May and at 8 PM on 31 May.

Tips to use sky map:

(1) Choose a place away from city lights/street lights.
(2) Hold the sky-map overhead with North in the direction of Polaris.
(3) Use a pencil torch for reading the sky map.
(4) Try to identify constellation as shown in the map one by one.

Visibility of Planets (IST)

	Rising	Setting	In the Zodiac
Mercury	04:38	17:06	Aries-Pisces-Aries
Venus	07:43	20:55	Taurus-Gemini
Mars	11:47	00:36	Cancer-Leo
Jupiter	02:32	14:29	Aquarius-Pisces
Saturn	14:34	02:49	Virgo
Uranus*	02:40	14:41	Pisces
Neptune*	00:59	12:31	Aquarius

Time shown is subject to vary (± 1 hr) from place to place.
*Not naked eye object

Sky Event

Date IST		Event
01		Venus: 26.6° E
05	11:20	Aquarid Shower
06	09:45	Last Quarter
07	03:23	Moon Apogee
14	06:34	New Moon
16	15:46	Moon-Venus: 0.1° S
20	14:08	Moon Perigee
21	05:13	First Quarter
26	07:29	Mercury Elongation: 25.1° W
28	04:37	Full Moon

Arvind C. Ranade
E-mail : rac@vigyanprasar.gov.in

YOUR OPINION

Dream 2047 **has been inviting your opinion on a specific topic every month. The reader sending the best comments will receive a popular science book published by VP. Selected comments received will also be published in *Dream 2047*. The comments should be limited to 400 words.**

This month's topic:

"Will increased use of CFL increase the risk of mercury pollution of the environment?"

Response should contain full name; postal address with pincode and email ID, if any; and should be accompanied by a recent passport size photograph. Response may be sent by email (**opinion@vigyanprasar.gov.in**) or by post to the address given below. If sent by post, "**Response: *Dream 2047* May 2010**" should be clearly written on the envolope.

Vigyan Prasar
A-50, Institutional Area, Sector-62, NOIDA 201 307 (U.P.)
Phone: 91-120-240 4430/35 Fax: 91-120-240 4437
Email: info@vigyanprasar.gov.in Website: www.vigyanprasar.gov.in

Winners of "Your Opinion" contest for February 2010

Topic: "Will the Jawaharlal Nehru National Solar Mission be able to bring electricity to every village in the country?"

Kum. Bhawana Huidrom
Std. – IX /C
Kendriya Vidyalaya, Ganeshkhind,
Pune – 411007.

Electrification to every village in the country under the Jawaharlal Nehru National Solar Mission is a debate among the citizens although it is one of the main targets promised by the government. The mission, on paper, have a very focussed research and development program which seeks to address the twin objective across India-specific challenges to contribute to India's long term energy security as well as its ecological security. Keeping in mind the changing climate scenario, the launch of this mission is a commendable approach that could enable India to help change the destinies of people around the world. Solar energy is an environmentally as well as eco-friendly as it has zero emissions while generating electricity or heat. It has also the advantage of permitting the decentralised distribution of energy, thereby empowering people at the local level. For a large proportion of poor and energy un-served population in remote and far-flung areas across the country where grid penetration is neither feasible nor cost effective, the solar imperative is both urgent and feasible to enable the country to meet its long-term energy needs.

*****

Amit Kumar Verma
c/o Sri Ashok Kumar
529/180 Rahim Nagar,
Lucknow (U.P.)-226006

If everything goes well, the JNN Solar Mission may become a reality. However, for proper implementation of this mission, the concerned authorities have to be sincere and acquire technical expertise. Prime focus should be on the best utilisation of natural resources and to spread awareness about its benefits by choosing at least one village as model. The present Government seems to be serious about the mission by providing adequate funds from time to time. No doubt, it will prove to be a boon for rural India.

*****

The winners will receive a copy of VP Publication

Exhibition on "Global Warming"

An international conference on "Global Warming: Agriculture, Sustainable Development and Public Leadership" was held at Gujarat Vidyapith, Ahmedabad from11 to 13 March 2010. The Conference was organised jointly by the International School for Public Leadership, Indian Society for Community Education, and Manthan Educational Programme Society, India. The Forest & Environment Department, Govt. of Gujarat, and Gujarat Vidyapith, Ahmedabad were also partners in this conference. Around 200 papers were presented during the conference in parallel sessions. Distinguished scientists, communicators, and science journalists from India and abroad chaired the sessions.

Inaugural session of the Conference in progress

Exhibitions and VP Stall in the Conference

Vigyan Prasar put up an exhibition on global warming. Agriculture, Sustainable Development and Public Leadership' to create awareness amongst general public. Some of the panels were on 'Planet Earth', 'Humans & Planet Earth', 'Human Development', 'Disturbances Created', 'What is Global Warming?', 'Causes of Global Warming', 'Impact of Global Warming', 'Threats', 'The fight against Global Warming', 'A new perspective against Global Warming', 'Greenhouse gases', 'Agriculture as a key' in the fight against Global Warming', 'Sustainable Development', 'Public Leadership', and 'Communication methodologies.'

A stall was put up to display our publications and other software. This was appreciated by both Indian and foreign participants. Navneet Kumar Gupta coordinated the activity. ■

Exhibition in Dilli Haat at Pitampura

Department of Environment, Govt. of Delhi organised a three-day National Science Day Exhibition from 26 to 28 February 2010 at Dilli Haat in Pitampura. In this exhibition clubs, schools, scientific departments and NGOs participated. A stall-cum-activity corner was put up by Vigyan Prasar to display its publications and other software.

Vigyan Prasar stall-cum-activity corner in Delhi Hatt