VATIS UPDATE
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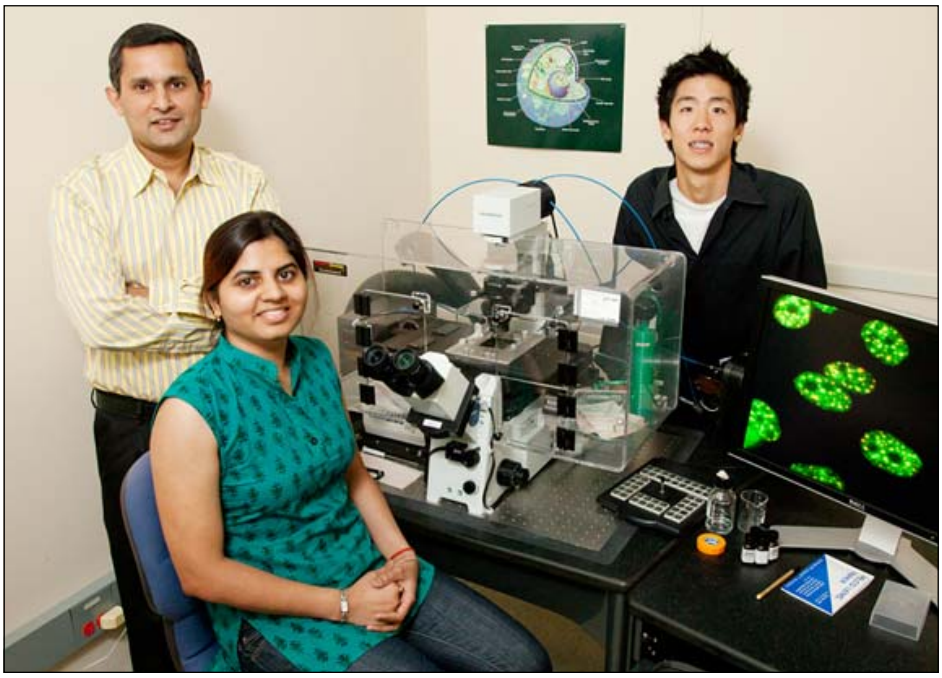
Highlights

- Possible new genetic risk for Alzheimer's disease
- Scientists discover a new DNA repair mechanism
- Enzyme in saliva shapes perception of food texture
- Novel antimalarial drug candidate
- Curcumin may be an effective therapy for liver fibrosis
- GM corn helps non-modified crop by killing off pests



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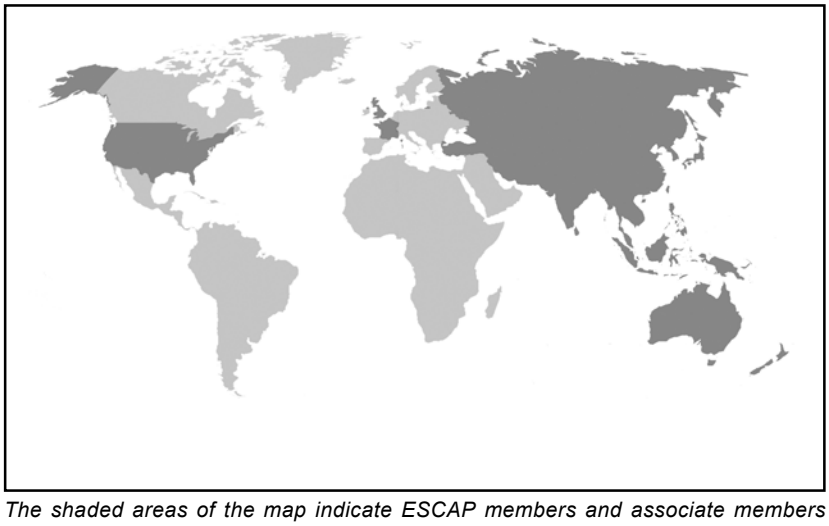

The shaded areas of the map indicate ESCAP members and associate members

Cover Photo

Prof. Kannanganattu Prasanth (left), Dr. Vidisha Tripathi (seated), Mr. David Song (right) and their colleagues found that MALAT1, a long non-coding RNA, plays a key role in pre-mRNA processing.
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(Credit: L. Brian Stauffer/University of Illinois, the United States)

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IN THE NEWS

Third generation map of human genetic variation published

An international consortium has published a third-generation map of human genetic variation, called the haplotype map or HapMap, which includes data from an additional seven global populations, increasing the total number to 11 populations. The improved resolution will help researchers interpret current genome studies aimed at finding common and rarer genetic variants associated with complex diseases.

The most common genetic differences among people are single-nucleotide polymorphisms, or SNPs, that serve as landmarks across the genome. The first version of the HapMap contained approximately 1 million SNPs, and the second-generation map brought that total to more than 3.1 million SNPs. Those two versions of HapMap resulted from the analysis of DNA collected from 270 volunteers from four geographically diverse populations: Yoruba in Ibadan, Nigeria; Japanese in Tokyo; Han Chinese in Beijing; and Utah residents with ancestry from northern and western Europe. The latest HapMap is the largest survey of human genetic variation performed thus far. It has data on 1,184 people, including the initial HapMap samples. Additional human samples were collected from the original populations and from seven new populations – individuals of African ancestry from the Southwestern United States; Chinese individuals from metropolitan Denver; Gujarati Indians from Houston; Luhya people from Webuye, Kenya; Maasai people from Kinyawa, Kenya; individuals of Mexican ancestry from Los Angeles; and individuals from Tuscany, Italy.

Researchers analysed approximately 1.6 million SNPs in about 500 samples from the four original populations and more than 650 samples from the seven new populations. In addition, the consortium sequenced 10 regions totalling about 1 million base pairs in 692 samples from this set in 10 of the 11 populations. More than 800 copy-number variants, where people have different numbers of copies of genomic regions, were also added to the resource. As expected, the increased number of samples allows detection of variants that are much rarer than could be found by the earlier HapMaps. Of the detected SNPs, 77 per cent were new, revealing that many more variants remain to be found, especially rare variants. (Source: www.genengnews.com)

Scientists investigate evolution of new polio virus

As a result of a global health campaign, polio viruses have almost been eradicated in many areas of the world. However, enterovirus 71 – closely related to poliovirus – has caused major outbreaks of hand, foot and mouth disease, with a high number of cases occurring in the Asia-Pacific region. In the first major review of diagnostic and treatment measures for the disease, a team of scientists from University of Liverpool, the United Kingdom, in collaboration with Universiti Malaysia Sarawak, has revealed that the virus evolves fast and is transmitted among family members more easily than previously thought.

Dr. Mong How Ooi, who led the study from Malaysia, says: "We have produced predicative tests (for brain infection) that include peak temperature measurements and monitoring the duration of fever, as well as looking for signs of lethargy. These assessments allow medics to decide which patients are most at risk from brain infection." The team analysed current preventive measures, which rely on guidance to families for improving personal and domestic hygiene. It is thought that the virus is most likely spread through faeces, although scientists have now found evidence to suggest it can also be spread through coughing and sneezing. (Source: www.genengnews.com)

Help to WHO to build global flu vaccine manufacturing capacity

The Biomedical Advanced Research and Development Authority (BARDA) of the United States Department of Health and Human Services is providing three sets of grants totalling US$10.4 million to the World Health Organization (WHO) to strengthen the ability of developing countries to produce influenza (flu) vaccine, potentially reducing the global threat from flu. The grants will be used to assist developing countries with pan-

demic flu vaccine manufacturing infrastructure, training on flu vaccine manufacturing, and development and distribution of certain technologies for pandemic flu vaccines.

BARDA awarded a US$6.4 million grant to WHO as part of an ongoing international cooperative agreement in the Initiative for Vaccine Research. This funding will support requests to WHO from developing countries to expand regulatory systems, construct and validate facilities for vaccine manufacturing, and transfer new technology for recombinant or cell-based flu vaccines.

The second set of grants supports training on cutting-edge manufacturing techniques for WHO grantees. In the United States, North Carolina State University's Biotechnology and Education Centre received US$861,000, and Utah State's Centre for Integrated Biosystems received US$ 322,000 to train selected personnel from manufacturers in developing countries in the latest good manufacturing practices in vaccine production.

The third set of grants supports development of adjuvant and the transfer of technology to produce adjuvants. The use of adjuvants has been shown to reduce the amount of protein needed for flu vaccine to be effective, so the adjuvanted vaccine made in these countries could serve a greater number of people. To develop adjuvants and other technologies that can be transferred without intellectual property right restrictions, the Infectious Disease Research Institute in Seattle, the United States, received US$790,000, and the University of Lausanne in Switzerland received US$1.8 million. (Source: www.medicalnewstoday.com)

China tipped to be life sciences major by 2020

Monitor Group, a global management consulting firm based in the United States, has released a new report that finds China poised to become the global leader in life sciences discovery and innovation within the next decade. At a time when the global life sciences and pharma industries are beset by major challenges, China has developed a strategy of targeted government investments. Through a variety of national and regional programmes, China is investing on a new health care "safety net," encouraging the growth of life science parks and start-ups, financing the development of a high-quality research infrastructure and luring back tens of thousands of Chinese researchers educated in the West.

"In just a decade's time – a short-term horizon in the life sciences field – China will not only be a significant engine of innovation, but has the potential to create a new model for advanced drug discovery," said Mr. George Baeder, co-author of the report titled *China, the Life Sciences Leader of 2020*. Based on research and interviews with dozens of life sciences professionals both in the United States and China, the report finds that China's life sciences industry is today gathering a critical mass of highly skilled talent, savvy and focused venture investors, and growing government support as its market for medical devices and drugs escalates. China's domestic market is expected to overtake Japan and become the world's second drug market by 2015. Many now see China as a compelling destination to conduct new cutting-edge research in life science-related areas. (Source: www.biospectrumasia.com)

Funding for development of 3G DNA sequencing technologies

The National Human Genome Research Institute (NHGRI) of the United States has announced more than US$18 million in grants to spur the development of third generation (3G) DNA sequencing technologies. The new technologies will sequence a person's DNA quickly and cost-effectively so that it can be used routinely by biomedical researchers and health-care workers to improve the prevention, diagnosis as well as treatment of human disease. NHGRI's vision is to cut the cost of whole-genome sequencing of an individual's genome to US$1,000 or less (from the current US$40,000), which will enable sequencing to be a part of routine medical care.

The new grants will fund ten investigator teams to develop revolutionary technologies that may make it possible to sequence a genome for US$ 1,000. The collective approaches incorporate many complementary elements that integrate chemistry, biochemistry and physics with engineering to enhance the efforts to develop the next generation of DNA sequencing and analysis technologies. (Source: www.genome.gov)

MARKET NEWS

Pioneer Hi-Bred to set up second seed processing unit in India

Pioneer Hi-Bred International, the global seed major based in the United States, has decided to set up its second seed processing-cum-production unit in India with significant investments. The unit will be set up during 2011, according to its global President, Mr. Paul Schickler. The first seed processing and production centre was established in 1999 on the outskirts of Hyderabad in Andhra Pradesh. It is involved in corn drying, seed conditioning and packaging for all crops and has cold storage facilities.

"India is a growing market and our focus will be on corn, millet, cotton, rice and mustard. In cotton, Pioneer Hi-Bred made two acquisitions during 2009 in Andhra Pradesh state. It bought the cotton seed business of Nandi Seeds in Mahboobnagar, and the germplasm and distribution network of Nagarjuna Seeds in Secunderabad. This has given us a good start in cotton markets," Mr. Schickler said. Pioneer Hi-Bred India, the Indian subsidiary, at present offers corn, rice, pearl millet, sunflower and mustard in the domestic market, with revenues of around Rs 4 billion (US$88.1 million). It has also established a corn research centre in Bangalore, Karnataka, which will develop high-yielding hybrids adapted to local growing conditions. The investments and research in cotton and rice being made at the DuPont Knowledge Centre in Hyderabad will be leveraged for the global markets and overall growth globally. Pioneer Hi-Bred is part of the US$26 billion DuPont's Agriculture business. More than half of DuPont's R&D budget of US$1.4 billion goes into agri and nutrition sectors. (Source: www.thehindubusinessline.com)

Geron to develop embryonic stem cell-derived chondrocytes

Geron Corp., the United States, has inked deals to develop chondrocytes derived from human embryonic stem cells for the treatment of cartilage damage and joint disease. The firm entered into a worldwide, exclusive license agreement with the University of Edinburgh, the United Kingdom, covering technology that allows the efficient production of chondrocytes from human embryonic stem cells.

The technology was developed in the laboratory of Professor Brendon Noble, as part of research collaboration between Geron and the University. The agreement gives Geron the right to develop therapeutic applications of the technology. Additionally, it is expected to enhance the company's programme to develop cell-replacement therapies for orthopaedic indications such as osteoarthritis. Pre-clinical studies conducted by Professor Noble and his team have shown that injection of human embryonic stem cell-derived chondrocytes into damaged cartilage of the knee joint of immunocompetent rats produced well-integrated cartilage. Full repair of the lesion for at least nine months was demonstrated. (Source: www.genengnews.com)

Genzyme divests genetics business

In the United States, Biotechnology giant Genzyme Corp. has sold its genetics testing business for US$925 million in cash to Laboratory Corporation of America. Genzyme will likely use the cash generated from the sale to buy back stock to fend off takeover pressure from rivals, and also boost the company's value in the eyes of any potential suitor. Recently, the pharmaceutical giant Sanofi-Aventis SA of France had made a takeover bid on the company.

Genzyme says that the sale is part of a "five-point plan" vision laid out by Chief Executive Mr. Henri Termeer earlier this year to increase shareholder value. "The completion of this sale allows us to focus our resources on core growth areas and create stronger returns on invested capital," Mr. Termeer said. Genzyme Genetics specializes in cancer testing and "esoteric" reproductive research. (Source: www.theepochtimes.com)

Biocon and Pfizer in global commercialization pact on insulin

Asia's premier biotechnology company, Biocon of India, and the world's leading biopharmaceutical company, Pfizer Inc. based in the United States, have entered into a strategic global agreement for the worldwide commercialization of Biocon's biosimilar versions of insulin and insulin analogue

products – recombinant human insulin, Glargine, Aspart and Lispro. Pfizer will have exclusive rights to commercialize these products globally, with certain exceptions, including co-exclusive rights for all of the products with Biocon in Germany, India and Malaysia. It will also have co-exclusive rights with existing Biocon licensees with respect to some of the products, primarily in developing markets.

Biocon will remain responsible for the clinical development, manufacture and supply of these biosimilar insulin products, and for regulatory activities to secure approval for these products in various geographies. According to the agreement, Pfizer will make upfront payments totalling US$ 200 million. Biocon is eligible to receive additional development and regulatory milestone payments of up to US$150 million and will also receive additional payments linked to Pfizer's sales of its four insulin biosimilar products across global markets. (Source: www.worldpharmanews.com)

MorphoSys acquires Sloning

In Germany, MorphoSys AG has acquired Sloning BioTechnology GmbH, a biotechnology company developing new methods of synthetic biology. The transaction will make MorphoSys the sole source of Sloning's state-of-the-art Slonomics technology, which dramatically improves the assembly and quality of protein libraries. With the integration of Slonomics into its existing antibody technology platform, MorphoSys expects to improve the generation of drug candidates such that one in every two projects started reaches clinical development.

The technology will also be used to accelerate the generation of both therapeutic and diagnostic antibodies. For MorphoSys, Sloning's patented core technology opens the way to a new and flexible approach to generate optimized proteins, such as antibodies. "This acquisition secures our position at the forefront of antibody technology," said CEO of MorphoSys, Dr. Simon Moroney. (www.biotechnology-europe.com)

Biomerix and Synthecon announce strategic distribution agreement

In the United States, Biomerix Corp., a medical technologies company developing and manufacturing innovative regenerative medicine biomaterials, has entered into a strategic distribution agreement with Synthecon, a leading biotechnology company specializing in the design and manufacture of rotating cell culture systems, for three-dimensional (3D) cell culture scaffolds developed using Biomerix's unique biomaterial technology.

The Biomerix 3D Scaffold is a medical grade biomaterial that features a novel micro-architecture, consisting of an interconnected 3D network of cells and pores designed to support tissue growth and biointegration. Used together with Synthecon's bioreactors, it would serve as an ideal technology to create an environment conducive to the growth of cells and tissues. Under the new agreement, Biomerix will develop and manufacture its 3D Scaffold, which Synthecon will market and sell nationwide for research applications. (Source: www.wten.com)

MethylGene issued key patents for lead oncology programme

MethylGene Inc., Canada, has been granted two 'composition of matter' patents by the United States Patent and Trademark Office for MGCD265, a multi-targeted kinase inhibitor for oncology, which is currently in clinical development. The patent on "Substituted Thieno (3,2-D) Pyridines as Inhibitors of the VEGF Receptor and HGF Receptor" covers composition of matter encompassing chemical structures of MGCD265 and its analogues. The second patent "Inhibitors of VEGF Receptor and HGF Receptor Signalling" covers composition of matter, including the compound MGCD265 specifically, and methods of treating angiogenesis-mediated cell proliferative disease and inhibiting solid tumour growth. (Source: www.genengnews.com)

GENOMICS

Genomic sequencing finds world's smallest genome

Until recently, *Encephalitozoon cuniculi*, a parasitic fungus commonly found in rabbits that can also be fatal to immuno-compromised humans, has been widely regarded as having the smallest known nuclear genome. At 2.9 million base pairs (Mbp) and approximately 2,000 genes, the genome of *E. cuniculi* is less than two-thousandth the size of the human genome. Recently, a team of researchers from Switzerland, Canada and the United States – led by Prof. Patrick Keeling from University of British Columbia, Canada – has sequenced the genome of another parasite that makes the *E. cuniculi* genome seem positively king-sized.

The genome of *E. intestinalis*, a sister species of *E. cuniculi* that infects human intestines, is 20 per cent smaller at only 2.3 Mbp. Dr. Keeling and his team compared the genome of *E. cuniculi* and *E. intestinalis* and found little difference between the chromosome "cores" but that the ends were all "trimmed" in *E. intestinalis*. They say the discovery provides insights into how genomes evolve, especially in extreme conditions. (Source: www.biotechnology-europe.com)

Possible new genetic risk for Alzheimer's disease

A team of researchers in the United States has identified a gene that seems to increase a person's risk of developing late-onset Alzheimer's disease, the most common form of the disease. The gene, MTHFD1L, is on chromosome six and was identified in a genome-wide association study. The researchers were able to identify small differences in the genetic sequences of the MTHFD1L in people with and without Alzheimer's disease. The team found that those with the variation may be nearly twice as likely to develop Alzheimer's disease as people without the variation.

The researchers were led by: Dr. Margaret A. Pericak-Vance, Director of the John P. Hussman Institute for Human Genomics at the Miller School of Medicine, University of Miami; Dr. Joseph D. Buxbaum, Department of Psychiatry, Mount Sinai School of Medicine; and Dr. Jonathan L. Haines, Vanderbilt Centre for Human Genetics Research, Vanderbilt University.

"Identifying this gene is important because the gene is known to be involved in influencing the body's levels of homocysteine, and high levels of homocysteine are a strong risk factor for late-onset Alzheimer disease," said Dr. Pericak-Vance. "In addition, variations of the MTHFD1L gene have been reported to possibly increase the risk of coronary artery disease. Since the function of blood vessels in the brain may affect Alzheimer's disease, this finding may help us understand how homocysteine levels and blood vessel function in the brain affect Alzheimer's disease," she added. (Source: www.sciencecodex.com)

Diving deeper into the gene pool

Scientists from Tel Aviv University (TAU), Israel, have developed innovative software to analyse and manipulate diseased cells. The software, dubbed miRNAkey, was developed by Mr. Roy Ronen as part of a research team headed by Dr. Noam Shomron of the Sackler Faculty of Medicine. Dr. Shomron states that miRNAkey searches for microRNA patterns in both healthy and diseased tissues, improving scientists' understanding of the data collected from deep sequencing technology, which is used to determine the ultimate sequence and expression of cellular DNA or RNA.

Until now there were very few unified codes that could interpret what information the microRNA held, and none that could run on a local computer or explain ambiguous microRNA behaviours. The miRNAkey program is designed to identify the relevant microRNA molecule, determine its level, and generate statistically valuable information from it. "Such identification of microRNAs allows us to manipulate them," Dr. Shomron says. With his software, data obtained from deep sequencing can be quickly and correctly analysed, allowing scientists to take a more deep look into disease behaviour and potentially build specialized treatments with this knowledge, explains Dr. Shomron. It may also encourage the creation of "smart drugs" which target individual damaged cells. (Source: www.aftau.org)

Gene activity in the brain depends on genetic background

Scientists at the Allen Institute for Brain Science, the United States, have found that the same genes have different activity patterns in the brain in individuals with different genetic backgrounds. These findings may help to explain individual differences in the effectiveness and side-effect profiles of therapeutic drugs and thus have implications for personalized medicine.

In this study, the researchers compared where in the brain each of 49 different pharmaceutically related genes is turned on (expressed) in seven genetically distinct groups of mice with known genealogical relationships. They precisely mapped where these genes are active, down to the level of individual cells, by analysing 203 distinct brain areas over 15,000 thin sections of tissue. The genes all encode molecular targets of well-known pharmaceuticals, such as antidepressants, antipsychotics and pain relievers. More than half of the genes examined showed striking, localized differences in expression patterns between the different genetic groups, or strains, of mice. For example, the dopamine D2 receptor gene, which encodes a target of action of Zyprexa, a drug used for schizophrenia and bipolar disorder, is active in a memory-related area called the entorhinal cortex in one strain of mice, but not in two others. Because different parts of the brain have different functions, variations in the localization of gene activity likely have functional implications.

"Our results show that genetic background, the specific blend of gene variants comprising an individual genome, can influence how the activity of a given gene is regulated and where it is expressed," said Mr. Allan Jones, Chief Executive Officer of the Institute. Taken together, the data from the study demonstrate that closer genetic relatives exhibit fewer differences in gene expression patterns, whereas more distant relatives show greater variation. Interestingly, the study found that the expression variations between genetic strains were more likely to be found in areas of the brain that evolved more recently. These areas are those that are more commonly linked to functions of higher order such as cognition, social behaviour, learning and memory. (Source: www.genengnews.com)

Scientists discover a new DNA repair mechanism

In the United States, researchers at Vanderbilt University, Pennsylvania State University and the University of Pittsburgh have discovered a fundamentally new way that DNA-repair enzymes detect and fix damage to the chemical bases that form the letters in the genetic code. "There is a general belief that DNA is rock solid – extremely stable," says Dr. Brandt Eichman, Associate Professor of Biological Sciences at Vanderbilt, who directed the project. "Actually DNA is highly reactive."

The newly discovered mechanism detects and repairs a common form of DNA damage called alkylation. A number of environmental toxins and chemotherapy drugs are alkylation agents that can attack DNA. When a DNA base becomes alkylated, it forms a lesion that distorts the shape of the molecule enough to prevent successful replication. If the lesion occurs within a gene, the gene may cease functioning. To make matters worse, there are dozens of different types of alkylated DNA bases, each of which has a different effect on replication.

Human cells contain a single glycosylase, named AAG, that repairs alkylated bases. It detects and deletes "ethenoadenine" bases, which have been deformed by combining with highly reactive, oxidized lipids in the body. AAG also handles many other forms of akylation damage. Many bacteria, however, have several types of glycosylases that handle different types of damage. AlkD is one such glycosylase. AlkD has a molecular structure that is very different from that of other known DNA-binding proteins or enzymes. AlkD uses several rod-like helical structures called HEAT repeats to grab hold of DNA. Similar structures have been found in the portion of DNA-dependent kinases with no known function, raising the possibility that they play an unrecognized role in DNA repair.

The newly found repair mechanism may also prove to be the key to understanding the differences in the way that the repair enzymes identify and repair toxic and mutagenic lesions. That is important, as mutagenic lesions that the repair mechanisms miss are copied to daughter cells and so can spread whereas the deleterious effects of toxic lesions are limited to the original cell. Knowledge

of these differences could lead to more effective chemotherapy agents, Dr. Eichman points out. (Source: news.vanderbilt.edu)

Solution to the mystery of selective silencing of genes

Cells in our body use very complex, sophisticated regulatory mechanisms to make sure that not all genes are read (that is, creating RNA copies of individual genes, which will then be translated into proteins) simultaneously. Particular gene switches need to be activated and, in addition, there are particular chemical labels in the DNA determining which genes are transcribed into RNA and which others will be inaccessible. The process is called epigenetic gene regulation.

Silencing of genes by methyl groups is one of the well-studied epigenetic mechanisms. This is done by specialized enzymes called methyltransferases, which attach methyl labels to particular 'letters' of a gene whereby access to the whole gene is blocked. "One of the great mysteries of modern molecular biology is: How do methyltransferases know where to attach their labels in order to selectively inactivate an individual gene?" says Professor Ingrid Grummt of the German Cancer Research Centre (DKFZ). She has come much closer towards unravelling this mystery by studying those text passages in the genetic material that do not contain any recipes. Nevertheless, these texts are transcribed into RNA molecules in a controlled manner. More than half of our genetic material is transcribed into non-coding RNA. This has prompted Prof. Grummt to propose: "It is very well possible that there are exactly matching non-coding RNA molecules for all genes that are temporarily silenced. This would explain how such a large number of genes can be selectively turned on and off." (Source: www.sciencedaily.com)

Cancer-associated RNA regulates pre-mRNA splicing

Researchers from University of Illinois at Urbana-Champaign, the United States, have reported that MALAT1, a long non-coding RNA (lncRNA) that is implicated in certain cancers, regulates pre-mRNA splicing – a critical step in the earliest stage of protein production. Researchers are only beginning to understand the functions of a few lncRNAs, said Cell and Developmental Biology Professor Dr. Kannanganattu Prasanth, who led the study. Dr. Prasanth's laboratory focuses on understanding the role of lncRNAs, which normally are distributed in mammalian cell nucleus.

Preliminary studies suggest that lncRNAs carry out vital regulatory functions in cells. When those functions go awry, serious consequences can result. Abnormal expression of the MALAT1 gene, for example, is implicated in many cancers, such as breast, lung and liver cancers. Dr. Prasanth had participated in another study, which found that MALAT1 plays a role in recruiting important proteins, called pre-mRNA splicing factors, to the gene transcription site in the nucleus. In Pre-mRNA splicing, unneeded sequences are cut out and mRNAs pieced together before they are exported from the nucleus and translated into proteins.

In the new study, Dr. Prasanth's team tested the hypothesis that MALAT1 interacts with and modulates a group of pre-mRNA splicing factors known as the SR-family splicing factors. They found that MALAT1 sequence contains multiple regions that can bind SR-splicing proteins, and that MALAT1 does bind to several members of SR-proteins. The data obtained strongly suggested that MALAT1 act as a regulator of splicing by modulating the levels of the splicing factors in the cell. This study verifies that MALAT1 plays a key role in pre-mRNA processing, with broad implications for human health. (Source: esciencenews.com)

Researchers publish turkey genome sequence

An international consortium of researchers has published the majority of the genome sequence of the domesticated turkey. It is reported to have sequenced 90 per cent of the turkey genome, using 'next-generation' sequencing technology. The majority of data pertain to the 10 largest chromosomes, called macrochromosomes, and researchers in the consortium are searching for the best route to sequence the remaining microchromosomes. The work promises new data for avian researchers and, ultimately, a better quality product for the producers as well as consumers of turkey, which is a major source of meat in some countries. (Source: www.physorg.com)

PROTEOMICS

Structural details of a key protein for cellular signal transduction

In Germany, scientists of the Institute of Structural Biology of Helmholtz Zentrum München and the Technische Universität München elucidated the structure of an important region of the Sam68 protein, a key protein for cellular signal transduction. Professor Michael Sattler and colleagues elucidated the spatial structure of the Qua1 region of Sam68, which is responsible for the dimerization of the protein. In collaboration with the research group of Professor Ruth Brack-Werner of the Institute of Virology, the scientists showed that this region is essential for the biological function of Sam68.

The image from nuclear magnetic resonance (NMR) spectroscopy reveals an unusual spatial structure, in which two helices of Qua1 region interact with each other and mediate the dimerization of Qua1 and thus of Sam68. Sam68 belongs to the family of STAR proteins that carry out key tasks in the signal-regulated processing of genetic information and its translation into protein. Among others, Sam68 regulates specific processes linked to the cell cycle and apoptosis and plays a key role in the pathogenesis of cancer. (Source: www.sciencedaily.com)

New look at multitalented protein sheds light on HIV mysteries

New insights into the human immunodeficiency virus (HIV) infection process, which leads to acquired immunodeficiency syndrome (AIDS), may now be possible through a method developed in part at the National Institute of Standards and Technology (NIST), the United States, where an important protein molecule's behaviour was revealed with unprecedented clarity. The HIV protein, known as Gag, plays several critical roles in the assembly of HIV in a host cell, but persistent difficulties with imaging Gag in a lab setting have stymied researchers' efforts to study how it functions. "Our method might reveal how to inhibit new viruses as they grow," says Dr. Hirsh Nanda at the NIST Centre for Neutron Research (NCNR).

The Gag molecule is a microscopic gymnast: at different stages during HIV assembly, the protein twists itself into several different shapes inside a host cell. One shape, or conformation, helps it to drag a piece of HIV genetic material towards the cell membrane, where the viral particles grow. Gag's opposite end becomes anchored there, stretching the protein into a rod-like conformation that eventually helps form a barrier surrounding the infectious genes in the finished virus. But while scientists have been aware for years that Gag appears to play several roles in HIV assembly, the specifics have remained mysterious.

The research team potentially solved this problem by creating an artificial cell membrane where Gag can show off its gymnastic prowess for the neutron probes at the NCNR. "We were able to mimic the different stages of the virus' development, and look at what Gag's conformation was at these various stages," Dr. Nanda says. "We saw conformations that had never been seen before." (Source: www.proteomicsnews.com)

Novel protein communication mechanism that cause apoptosis

Various cellular components involved in apoptosis have been identified over the past two decades. Nevertheless, there are still important unresolved questions about the functioning of certain key elements in this great riddle of programmed cell death. A recent international study by investigators at five research centres provides new clues for understanding the complex process of apoptosis. The new study has revealed that three essential components of the apoptotic process – the BAX and DRP-1 proteins and cardiolipin – act in a joint manner to produce a large hole in the external membrane of the mitochondria, proving to be lethal for the cell. Taking part in this research, led by Professor Jean-Claude Martinou of the Department of Cell Biology at University of Geneva, Switzerland, were Biophysics Unit of Spanish Scientific Research Council (CSIC)-University of the Basque Country (UPV-EHU), the University of Salzburg, Germany, University of Hanover, Germany, and University of Florida, the United States.

Probably the most surprising aspect of the new research is that the researchers have managed to decipher a novel 'language' used by BAX and

DRP-1 for communicating: these two proteins do not interact with each other physically, as usually happens, but they do so through the lipids of the membrane. "More specifically, what one of the proteins (DRP-1) does is to deform the lipid bilayer of the membrane and the resulting structure is what apparently enables the activation of the second protein (BAX)", explained Mr. Gorka Basañez, from the CSIC-UPV/EHU Biophysics Unit. These findings can open new ways to the rational development of anti-tumour drugs, which specifically target these components of the apoptotic cell machinery. (Source: www.sciencedaily.com)

Cell survival protein discovery rewrites immune system story

A discovery by Walter and Eliza Hall Institute researchers in Melbourne, Australia, could rewrite a long-held belief about how the body's immune system establishes its memory. The findings of Dr. Ingela Vikstrom and Dr. David Tarlinton, from the Institute's Immunology Division, centre on immune cells called B cells, which produce the antibodies that fight infections. "B cells and antibody production are key to the success of all currently used vaccines for immunity in humans," said Dr. Tarlinton. Memory B cells are essential for the long-lived immunity that arises after immunization. To develop into memory cells, B cells have to survive the natural process of apoptosis, or programmed cell death, that occurs following a large immune response.

According to Dr. Vikstrom, B cell memory arises in temporary cellular structures called germinal centres that develop in response to activation of the immune system. The researchers studied two well-known pro-survival proteins, called Bcl-xL and Mcl-1, which regulate B cell survival and are therefore responsible for instructing these cells whether to live or die. Bcl-xL and Mcl-1 were known to be involved in the process of instructing B cells to establish germinal centres, as well as instructing activated B cells to proliferate and differentiate into memory B cells.

"It surprised us to find that, contrary to popular belief, Mcl-1 is the essential pro-survival protein required for the creation and maintenance of B cell memory," said Dr. Vikstrom. The finding contradicts the widely accepted theory in immunology circles that Bcl-xL is the major pro-survival protein responsible for sustaining the development of memory B cells. (Source: www.eurekalert.org)

Protein sets stage for exchanges of DNA code in germ cells

A research team led by a scientist at the University of Pittsburgh School of Medicine, the United States, has discovered a regulatory protein that influences where genetic material gets swapped between maternal and paternal chromosomes during the process of creating eggs and sperm. Most cells contain 46 chromosomes, half coming from each parent. Eggs and sperm, known as germ cells, have 23 each so that when they combine to form an embryo, the correct number of chromosome is maintained, said senior author Dr. Judith Yanowitz, an assistant professor of obstetrics, gynaecology and reproductive sciences.

"When germ cells form, segments of DNA are exchanged, or recombined, between maternal and paternal chromosomes, leading to greater diversity in the daughter cells," she said. "Our research reveals a protein that plays a key role in choosing where those cross-overs occur." Crossing over is essential for the correct movement, or segregation, of chromosomes into the germ cells. Failure to exchange DNA properly can lead to offspring with the wrong number of chromosomes and, in humans, defects in this process are a leading cause of infertility, Dr. Yanowitz noted.

In the genome of *Caenorhabditis elegans,* the tiny round worm that the researchers studied, gene recombination typically occurs towards the ends of the chromosomes, which contains fewer genes. This is the first gene that is specifically required for the segregation of single chromosomes. "The fact that this is the X chromosome is interesting because the sex chromosomes play a unique role both in germ line and general development," Dr. Yanowitz said. The researchers suggest that xnd-1 affects the way chromosomes are packaged into the cell nucleus as chromatin, a DNA protein complex. They showed that xnd-1 alters a chromatin component that has remained through the species evolution and that this packaging is directly responsible for the effects on cross-over formation. (Source: www.scientificcomputing.com)

Non-stick protein coating found in semen reduces HIV infection

A non-stick coating for a protein found in semen dramatically lowers the infection rate of immune cells by human immunodeficiency virus (HIV), a new study in the United States has found. The new material is a potential ingredient for microbicides designed to reduce HIV transmission. The coating clings to the fibrous strings and mats of an amyloid protein called semen-derived enhancer of viral infection (SEVI). SEVI seems to attract the virus and deposit it onto the surface of T-cells, components of the immune system that are the primary target of HIV infection, and may play a major role in sexual transmission of HIV.

To test whether the coating strategy might interfere with SEVI's role in promoting HIV infection, Dr. Jerry Yang, Associate Professor of chemistry at University of California, San Diego, and his group teamed up with researchers led by Dr. Stephen Dewhurst, Chair of the Microbiology and Immunology Department at University of Rochester Medical Centre. When the researchers added the molecule that forms non-stick coatings to a mix of SEVI, virus and cells, infection rates fell to levels seen in the absence of SEVI. They saw a similar effect with semen as well, evidence that this potential microbicide supplement works to inhibit infection within a mixture of proteins and other molecules found in semen. The coating molecule is a modified form of thioflavin-T, a dye that stains amyloid proteins. It fits in between the individual small proteins that cluster to form SEVI, and blocks SEVI's interactions with both the virus and the target immune cells. Further, unlike many current microbicide candidates aimed at reducing HIV infection, this one does not cause inflammation in cervical cells. (Source: www.sciencedaily.com)

Too much protein turns stem cells into 'evil twin' cancer cells

Researchers at North Carolina State University, the United States, have found that overproduction of a key protein in stem cells causes those stem cells to form cancerous tumours. Their work may lead to new treatments for a variety of cancers. Dr. Jon Horowitz, Associate Professor of molecular biomedical sciences, and colleagues looked at the protein SP2, which regulates the activity of other genes. They knew that elevated amounts of SP2 had been observed in human prostate-cancer patients, and that these levels increased as the tumours became more dangerous. They then showed that precisely the same thing occurs in mouse skin tumours.

Dr. Horowitz and his team investigated SP2 as a possible cause of tumour formation in epithelial cell-derived tumours, which comprise about 80 per cent of all human tumours. They found that overproduction of SP2 protein in epithelial stem cells stopped them from spawning mature descendants. The affected stem cells, unable to produce mature cells, kept proliferating, resulting in tumours. "SP2 basically hijacks the stem cell, and turns it into its evil twin – a cancer cell," said Dr. Horowitz. "Our hope is that we can find an 'antidote' to SP2, to restore normal cell proliferation to those cancer stem cells and reverse the process." (Source: www.genengnews.com)

Enzyme in saliva shapes perception of food texture

A new study from the Monell Chemical Senses Centre, the United States, reports that individuals' perception of starch texture is shaped by variability in the activity of the oral enzyme, salivary amylase. "Differences in starch perception likely affect people's nutritional status by influencing their liking for and intake of starchy and starch-thickened foods," said the study's lead author Dr. Abigail Mandel, a nutritional scientist. Amylase enzymes in saliva help break down starches into simpler sugar molecules that are absorbed into the bloodstream and thus influence blood glucose levels.

In the study, analyses revealed that changes of starch consistency in the mouth were directly related to salivary amylase activity. Foods with different starch levels will be perceived very differently by people as a function of how much salivary amylase they produce, said senior author Dr. Paul A.S. Breslin, a sensory geneticist. The findings may also extend to starch digestion and metabolism. Individuals with more salivary amylase may break starchy foods down more quickly, leading to a more rapid increase of post-meal blood glucose levels. (Source: news.biocompare.com)

MEDICAL BIOTECH

Novel antimalarial drug candidate

Scientists are developing a new antimalarial drug with a novel mechanism of action that holds promise for clearing malarial infection after a single dose. The antimalarial candidate, spiroindolone NITD609, will most likely be the next generation for drug-resistant malaria. The researchers say that spiroindolone NITD609 is effective against both *Plasmodium falciparum* and *P. vivax* – two malaria parasite strains, as shown in a mouse model. Spiroindolone NITD609 has resulted from a collaboration between the Genomics Institute of the Novartis Research Foundation (GNF), the United States, the Swiss Tropical & Public Health Institute, Switzerland, and The Scripps Research Institute, the United States, with support from The Wellcome Trust of the United Kingdom, Medicines for Malaria Venture of Switzerland, A*STAR of Singapore, and the United States government.

The authors write that if ongoing regulatory pharmacological and safety studies go according to plan, spiroindolone NITD609 will most likely progress to Phase I human trials. The identification and validation of new antimalarials drugs has proven challenging, despite considerable progress in *Plasmodium* genome biology, the researchers explain. With spiroindolone NITD609, they say, they have found a potential target by identifying mutations that undermine the parasite's sensitivity to this new class of compound. (Source: www.medicalnewstoday.com)

'Humanized' mice may provide clues to address typhoid fever

Better treatment and prevention for typhoid fever may emerge from a laboratory model that has just been developed for the disease. The model is based on transplanting human immune stem cells from umbilical cord blood into mice that are susceptible to infections. The transplanted cells live alongside the mouse's own immune system. While mice are normally resistant to *Salmonella typhimurium* (*S. typhi*) that causes typhoid fever, the bacteria are able to reproduce in the mice that have received transplanted human cells.

Progress in creating more effective vaccines and medications has been limited because typhoid fever affects only humans, notes Dr. Ferric C. Fang, Laboratory Medicine and Microbiology Professor at University of Washington, the United States, and the senior scientist on the project. The new model enables scientists to study innovative approaches against the disease in a living system, before testing them on people.

S. typhi is highly adapted to people. It has evolved many ways to evade infection-fighting defences inside humans. It can also enter and kill disease-fighting cells. The researchers demonstrated that human blood-forming cells engrafted into immune-deficient mice allowed the mice to be infected with the organism that causes human typhoid fever, and that the typhoid bacteria appeared to reproduce inside those human cells. The scientists were also able to use this model to look for genetic factors that the typhoid bacteria need to cause severe illness. Their research also shows how mice engrafted with human stem cells can allow researchers to better understand human infections. (Source: www.eurekalert.org)

A drug against AIDS could be effective against the herpes virus

Scientists at the Institute for Research in Biomedicine, Spain, headed by the Coordinator of the Structural and Computational Biology Programme, Dr. Miquel Coll, have demonstrated that raltegravir, the drug approved for the treatment of acquired immunodeficiency syndrome (AIDS) and sold by Merck under the name Isentress, cancels the function of an essential protein for the replication of one kind of herpes virus. This study is seen as the first step towards the development of a drug against the entire herpes virus family.

Herpes viruses include herpes simplex 1 and 2, zoster virus (the virus that causes chickenpox), Epstein-Barr virus (associated with several types of cancer), roseola virus, cytomegalovirus and the virus associated with Kaposi sarcoma. The study was performed on human cytomegalovirus (HCMV), which causes many medical conditions. Although 90 per cent of adults carry HCMV, this virus acts in individuals with weakened immune systems such as in cancer and AIDS patients, recipients of organ transplants and neonates.

To replicate, the herpes virus enters the nucleus of a cell where it uses the cell machinery to copy its DNA several times into a single large chain. Once this copy has been made, a complex called terminase, formed by three protein subunits, cuts the new DNA into small fragments, the size of a single viral genome, and introduces these into empty shells (capsids) that have developed in the cell nucleus. Then, the new viruses leave the cell to continue infection. The researchers resolved the three-dimensional structure of one part of the terminase and when they noted that it resembled the integrase of the AIDS virus, for which drugs are available, they tested it against the herpes virus protein. Thus, they discovered that raltegravir acts on the subunit UL89 of the terminase and cancels the scissor function, which is required for viral replication. (Source: esciencenews.com)

Yeast holds some clues to Parkinson's disease

Dr. Tiago Fleming Outeiro from the Instituto de Medicina Molecular, Portugal, and his colleagues are revealing the molecular basis of Parkinson's disease by studying the associated human protein in yeast cells and how yeast could be a powerful ally in the discovery of new therapeutic drugs to treat the disease. Baker's yeast, *Saccharomyces cerevisiae,* is helping researchers learn how alpha-synuclein might lead to Parkinson's disease. Dr. Outeiro explains that many of the biochemical pathways involved are similar between yeast and humans. Together with researchers in the United States, Dr. Outeiro screened a library of 115,000 small compounds to identify those that are able to block the toxic effects of alpha-synuclein. Several of these molecules have proved effective in preventing Parkinson's disease in worms and blocking alpha-synuclein toxicity in rat neurons. If developed further, they could form the basis of future drugs to treat the disease in humans as well. (Source: www.disabled-world.com)

Curcumin may be an effective therapy for liver fibrosis

Curcumin, a chemical that gives curry its zing, holds promise in preventing a well as treating liver damage from an advanced form of a condition known as fatty liver disease, suggests research by Saint Louis University (SLU), the United States. Curcumin is contained in turmeric, a plant used in traditional Chinese medicine and Indian cuisine. SLU's recent research highlights its potential in countering an increasingly common kind of fatty liver disease called non-alcoholic steatohepatitis (NASH), linked to obesity and weight gain. NASH can lead to a type of liver damage called liver fibrosis, and possibly cirrhosis and liver cancer.

High levels of blood leptin, glucose and insulin are commonly found in human patients with obesity and type 2 diabetes, which might contribute to NASH-associated liver fibrosis. The SLU research tested the effect of curcumin on the role of high levels of leptin in causing liver fibrosis in vitro, or in a controlled lab setting. "Leptin plays a critical role in the development of liver fibrosis," said Dr. Anping Chen, Director of Research in SLU Pathology Department, and corresponding author. High levels of leptin activate hepatic stellate cells that cause overproduction of the collagen protein, a major feature of liver fibrosis. The researchers found that among other activities, curcumin eliminated the effects of leptin on activating hepatic stellate cells, short-circuiting the development of liver damage. (Source: www.news-medical.net)

Black rice bran may help fight disease related inflammation

Scientists from the Republic of Korea and the United States are reporting evidence that black rice – a little-known variety of the grain that is the staple food for one-third of the world population – may help soothe the inflammation involved in allergies, asthma and some other diseases. The researchers – from Ajou University and Kyungpook National University, the Republic of Korea, and Western Regional Research Centre of the Agricultural Research Service (ARS), the United States Department of Agriculture (USDA) – point out that their previous research showed several potential health benefits of eating black rice bran. Those experiments, which were done in cell cultures, hinted that black rice bran suppressed the release of histamine, which causes inflammation.

In the new study, they tested the effects of black rice bran extract on skin inflammation in laboratory mice. When they injected the extract into the mice, it reduced skin inflammation by about 32

per cent compared with control animals and also decreased production of certain substances known to promote inflammation. Brown rice bran extract did not have these effects, they say. When the scientists fed the mice a diet containing 10 per cent black rice bran, it reduced swelling associated with allergic contact dermatitis, a common type of skin irritation. The findings "demonstrate the potential value of black rice bran as an anti-inflammatory and anti-allergic food ingredient and possibly also as a therapeutic agent for the treatment and prevention of diseases associated with chronic inflammation," the study notes. (Source: esciencenews.com)

New approach for early diagnosis of pancreatic cancer

A new study in the United States that peers deeply into the genetics of pancreatic cancer presents an opportunity for early diagnosis of pancreatic tumours. "There have been two competing theories explaining why pancreatic cancers are so lethal," says Dr. Bert Vogelstein, the Howard Hughes Medical Institute investigator who helped lead the study. "The first is that pancreatic tumours are aggressive right from the get-go and spread to other organs very quickly. The second theory is that pancreatic tumours are, in fact, not more aggressive than other tumours, but that symptoms appear so late in the process that patients have little chance of surviving. We were surprised and pleased to discover that this second theory is correct, at least for a major fraction of tumours. It means that there is a window of opportunity for early detection of pancreatic cancer."

Working with Dr. Christine Iacobuzio-Donahue, a pathologist at Johns Hopkins University School of Medicine, Dr. Vogelstein obtained samples of primary pancreatic tumours from seven autopsied patients, as well as metastatic lesions from their lungs, liver and other organs. They sequenced the DNA of every gene in each metastatic tumour as well as in the primary tumour. These genetic read-outs provided data to compare the genetic mutations found in patient's metastatic lesions with the mutations found in the primary tumour.

The scientists found that each metastatic lesion had, on average across all patients, 61 cancer-related genetic mutations. Further, the majority of these mutations – 64 per cent on average – were also present in the primary tumour. The researchers then worked with Dr. Martin Nowak, an evolutionary biologist at Harvard University, to estimate how long it took these mutations to accumulate. They found that it took a long time – more than 20 years – from the first mutated pancreatic cell and the death of a patient.

Unlike other cancers, though, pancreatic tumours usually produce no symptoms until they have spread. Jaundice is often the first symptom, but that arrives only after a pancreatic tumour has metastasized to the liver. But Vogelstein says the new data suggest that a blood or stool test might be able to pick up early cancer-causing mutations. His team is already examining the efficacy of such tests for detecting early signs of colorectal cancer. (Source: www.news-medical.net)

Scientists discover inner workings of potent cancer drug

Scientists at University of California Santa Barbara, the United States, in cooperation with scientists in the pharmaceutical industry, have discovered the mechanism by which a potent drug, derived from an evergreen tree, may soon save the lives of some patients with the deadliest form of breast cancer. The team has isolated the drug's action in the test tube as well as in cancer cells.

"This anticancer drug, called maytansine, when linked to a tumour-targeting antibody, shows promising early results in clinical trials on patients with metastatic breast cancer," said Dr. Mary Ann Jordan, a professor in the Department of Cellular, Molecular and Developmental Biology. Early clinical trials show that the drug shrank the tumours of one-third of the patients in the breast cancer study – a strong result, according to Dr. Jordan. The drug works by targeting the microtubules of cancer cells. It was previously considered too dangerous to use, because of its toxicity to non-cancer cells. However, the team was able to show that modifying the anticancer drug by adding an antibody caused the drug to target only cancer cells, greatly reducing its toxicity. The new drug is named trastuzumab-DM1. DM1 is a synthetic derivative of maytansine, a molecule found in an evergreen tree in the genera *Maytenus,* which grows in many continents. (Source: www.physorg.com)

AGRI BIOTECH

Biofuel production from inedible plant material made easier

In the United Kingdom, a research team funded by the Biotechnology and Biological Sciences Research Council (BBSRC) has discovered key plant enzymes that normally make the energy stored in wood, straw and other non-edible parts of plants difficult to extract. The team, based at the University of Cambridge and now part of the BBSRC Sustainable Bioenergy Centre (BSBEC), has identified and studied the genes for two enzymes that toughen wood, straw and stalks and make it hard to extract sugars to make bioethanol or other plant-derived products.

This knowledge can now be used in crop breeding programmes to make non-edible plant material that requires less processing, less energy and fewer chemicals for conversion to biofuels or other renewable products and therefore have an even lower overall impact on atmospheric carbon. The research also increases the economic viability of producing sustainable biofuels from the inedible by-products of crops by increasing our understanding of plant structures. (Source: www.sciencedaily.com)

Indian transgenic super-spuds with more protein and higher yield

A genetically modified (GM) potato has 60 per cent more protein per gram than ordinary potatoes. But even with that help spuds don't contain much protein, so that is not the most interesting part: in a surprise result, the GM crop also yielded more potato per hectare. This is the first time that a simple genetic modification has increased yield.

Dr. Subra Chakraborty and colleagues at India's Central Potato Research Institute created the high-protein GM "protato" in 2003 by giving potatoes a gene from the grain amaranth, a South American plant widely eaten across the tropics. The gene codes for a "storage" protein in amaranth seeds, but in the protato it was linked to a DNA code that turns on production of the storage protein. The team has now spliced this gene into seven commercial potato varieties, and field-tested them for several seasons. This is crucial, as GM crops often behave differently in the lab and the field. Some tubers contained almost twice as much extra protein as the prototype, with increases in several essential amino acids. Tests in rats and rabbits revealed no toxic or allergic effects. The plants also photosynthesised more, and produced 15 to 25 per cent more potatoes per hectare by weight – the first time this has been reported for a plant with just one additional gene. (Source: www.newscientist.com)

GM corn helps non-modified crop by killing off pests

Pest resistance of transgenic corn (maize) has benefited even non-transgenic corn, according to agricultural researchers and entomologists in the United States. The researchers found that widespread planting of genetically modified (GM) Bt corn throughout the Upper Midwest has lowered populations of European corn borer, historically one of corn's primary pests. This area-wide suppression has dramatically reduced losses caused by the pest even on non-GM corn.

Corn borer moths cannot distinguish between Bt and non-Bt corn, so females lay eggs in both kinds of fields, said Dr. William Hutchison, an entomology professor at University of Minnesota. Once eggs hatch in Bt corn, young borer larvae feed and die within 24 to 48 hours. This decline in pest population has helped reduce corn borer numbers also in neighbouring non-Bt fields by 28-73 per cent in Minnesota, Illinois and Wisconsin. This is the first work to show a direct association between Bt corn use and an area-wide reduction in corn borer abundance. Bt corn is engineered to express insecticidal proteins from the bacterium *Bacillus thuringiensis*.

Dr. Paul Mitchell, an agricultural economist at the University of Wisconsin-Madison and a co-author of the study, emphasized: "In this case, the value of the indirect yield benefits for non-Bt corn acres exceeded the net value of direct benefits to the Bt corn acres." The analysis does not consider benefits for other important Midwestern crops affected by the corn borer, such as sweet corn, potatoes and green beans. (Source: www.seedquest.com)

RECENT PUBLICATIONS

Genome Profiling for Genetic Marker Discovery

Recent advancement in genome technology, especially high-throughput low-cost sequencing and microarrays, revolutionizes the way for discovering genetic markers and enables their new applications. This book is designed to build a genetic tool box with these new and emerging techniques for molecular biologists, geneticists, clinical scientists and breeders. While theses, methods and protocols were developed in genomics and bioinformatics laboratories in the forefront of the fields, the format of each method or protocol is easy to grasp and practical to address different challenging issues in genetic marker discovery.

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Regenerative Medicine: From Protocol to Patient

Regenerative medicine is a fast-emerging interdisciplinary field of research and clinical therapies on the repair, replacement or regeneration of cells, tissues or organs in disease. This new textbook aims to explain the science, technology and the clinical application of regenerative medicine in different organ systems and diseases. The process of translating science of laboratory protocols into therapies is explained in sections on basic science, clinical translation, regulatory, ethical and industrial issues. The textbook is aiming to give the student, the researcher, the health care professional, the physician, as well as the patient a complete survey on the current scientific basis, therapeutic protocols, clinical translation and practised therapies in regenerative medicine.

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