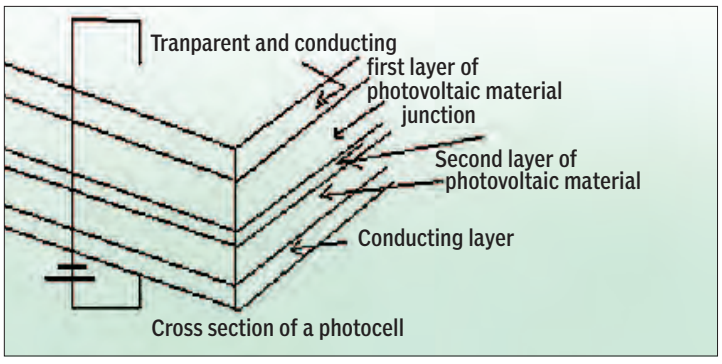
Ready to cut prices

KITCHEN SALT MAY HELP MAKE SOLAR CELLS BECOME CHEAPER TO BUILD, SAYS **S ANANTHANARAYAN**

Solar cell technology has seen great strides and the cost has been steadily falling. Use of these cells has become commonplace, moving from remote locations to rooftops, handheld devices, traffic signals and parking meters. The cost of installation is now down to less than Rs 40 a watt and solar power is being seen on a par with other sources as feeders to the power grid.

Apart from crystalline silicon, there are now other materials being used in solar cells. And cadmium telluride, which can match and surpass silicon in the cost per unit electrical output, is set to be the market leader in solar cell materials. But the problem with cadmium telluride (CdTe) is that cadmium is toxic and the process uses cadmium as cadmium chloride ($CdCl_2$), a form of cadmium that can leak into the environment, which calls for expensive precautions. Aq B r o l P `fbk `b L m b k C l o r j /-.1) @l m b k e ^ d b k) G Major, R Treharne, LJ Phil* lips and K Durose) Ptephenson Institute of Renewable Energy, l f Liverpool, p^fa qebv abp`of_ba fk ^ m^mbo fk the journal *Nature*, an alternative to cadmium chloride, which makes CdTe solar cells much safer.

Solar cells depend on the ability of some materials to throw out electrons from their atomic structure and create an electric charge when light falls on them. Now, when there is an arrangement to prevent the electrons so freed from falling back to the atoms that released them, the electrons can be drawn off as an electric current, which is the solar cell. The arrangement to control the movement of electrons is through *doping* or modifying the nature of the crystals of the photo-material and creating a junction that allows electrons to flow only one way. This enables one side of the junction to collect electrons that get pumped out by the action of light and, hence, drive an electric current.



Cross section of a photocell


John Major

While solar cells were first made of blocks of silicon, they are now used as thin sheets of silicon, or the other materials developed, and the sheets, known as thin film solar cells, can be laid on flexible substrates. The sheets can then be used on roofs of building, small devices or even as a film on windowpanes, which cuts the glare and also generates electricity.

In the use of CdTe as the material, the junction is with cadmium sulphide (CdS). The efficiency of the junction is greatly increased in a process known as *activation* with the use of cadmium chloride, which allows chlorine and oxygen atoms to take part in the mechanism of the junction.

Although cadmium is toxic, the forms of CdTe and CdS are stable and it has been shown that when used in thin film photovoltaic cells, as in sheets laid on rooftops, the materials do not escape the environment even if there is a fire. But this is not true of $CdCl_2$, which is used in the manufacturing process. $CdCl_2$ powder is water-soluble and a positive hazard, both for workers in the industry as well as to the environment. The control and retrieval of $CdCl_2$ is, thus, a major element of cost in the manufacture of the CdTe solar cell. $CdCl_2$ is also an expensive raw material, in any case.

But the use of $CdCl_2$ in annealing the junction has made for raising its efficiency of converting solar energy to power from less than two per cent to more than 10 per cent, which makes it commercial.

Laboratory trials have even touched 20 per cent and there is every interest in cutting cost and risk in the use of $CdCl_2$. The only alternative that was tried is a chlorofluorocarbon gas, which is related to CFC, the material of aerosols and refrigerants that leads to damage to the ozone layer. As use of CFCs is internationally controlled, this alternative of $CdCl_2$ is not available and $CdCl_2$ is still there in the commercial high-efficiency CdTe solar cell programme.

New kid

The Liverpool team experimented with other low cost chlorides as possible replacements for $CdCl_2$, namely magnesium, sodium, potassium and manganese. Others, like chlorides of copper or zinc, either have environment issues or involve high cost. Of the chlorides tried out, it was found that magnesium chloride, $MgCl_2$, gave results almost the same as with $CdCl_2$. Magnesium was found to be suitable, because even with $CdCl_2$, there is magnesium that enters through the soda-lime glass substrate, and magnesium does not affect performance. This is found to be because of the magnesium ion being electrically inactive in CdTe, unlike ions of sodium, potassium or manganese, whose use led to device performance being compromised.

In the case of magnesium chloride, the efficiency, in fact, was 13.5 per cent compared to only 13.2 per cent even with $CdCl_2$, the traditional activation agent. The long-term stability was also found to be the same, the degradation over six months being only that due to oxidation at the gold-CdTe contacts that are used. Magnesium chloride is also a low cost and commonly available salt. As some news reporters observe, it is an ingredient salt that is used in the bean curd) QLCR.

The discovery of the Liverpool team is thus that magnesium chloride can immediately replace cadmium chloride in the manufacture process and eliminate both the risk and the co l stly toxin control measures involved. The stage is, hence, set for development for industrial use and wider deployment of CdTe devices.

One factor that affected CdTe was the scarcity and limited reserves of tellurium and this factor was considered as a serious limitation. But new reserves have been discovered in China, Mexico and Sweden. Interestingly, astrophysicists have also found that tellurium is the most abundant of the heavier elements in the universe!

Nearer home, rich reserves, more than we will ever need, have been found in undersea ridges and feasibility of recovery is being looked into) as also l q e b o possible reserves. CdTe technology) k l t b^pfbo t fqe J d@i/) appears to have found a place in major increase in the role of solar power.

THE WRITER CAN BE CONTACTED AT simplescience@gmail.com

ALL IN THE COLON

TAPAN KUMAR MAITRA EXPLAINS HOW THE APC TUMOR-SUPPRESSOR GENE CODES FOR A PROTEIN THAT INHIBITS THE WNT SIGNALLING PATHWAY

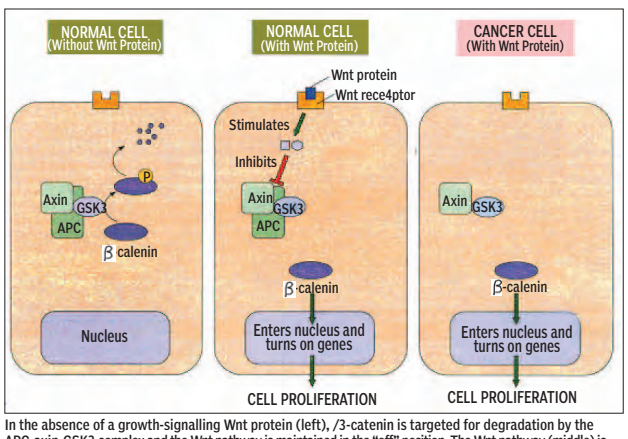
Like the p53 gene, the APC gene is also a common target for cancer-causing mutations, although in this case cancers arise mainly in one organ — the colon. Mutations affecting this gene are associated with an inherited disease called *familial adenomatous polyposis*. Individuals inherit a defective APC gene that causes thousands of polyps (benign tumors) to grow in the colon and creates a nearly 100 per cent risk that cancer will develop by the age of 60. Although familial adenomatous polyposis is a rare disease, accounting for less than one per cent of all colon cancers, APC mutations are also triggered by environmental mutagens and occur in roughly two-thirds of the more common non-hereditary forms of colon cancer.

The APC gene's cancer risk is through its involvement in the *Wnt pathway*, which plays a prominent role in controlling cell proliferation and differentiation during embryonic development. The central component of this pathway is a protein called β-*catenin*, which is normally prevented from functioning by a multiprotein complex that consists of the protein produced by the APC gene combined with the proteins *axin* and *glycogen synthase kinase 3* (GSK3). When assembled in this APC-axin-GSK3 complex, GSK3 catalyses the phosphorylation of β catenin, which is then linked to *ubiquitin*, which targets β-catenin for destruction by proteasomes. The net result is a low concentration of βcatenin that renders the Wnt pathway inactive.

The Wnt pathway is turned on by extracellular signalling molecules called Wnt proteins, which bind to and activate cell-surface Wnt receptors. The activated receptors stimulate a group of proteins that deactivates the APC-axin-GSK3 complex and thereby prevents the degradation of β:atenin. Theβ catenin then enters the nucleus and interacts with transcription factors that activate a variety of genes, including some that stimulate cell proliferation.

Mutations causing abnormal activation of the Wnt pathway have been detected in numerous cancers, most of which are loss-of-function mutations in the APC gene that are either inherited or, more commonly, triggered by environmental mutagens. The resulting absence of functional APC protein prevents the APC-axin-GSK3 complex from assembling and β-catenin therefore accumulates, locking the Wnt pathway in the on position and sending the cell an ongoing signal to divide.

Current estimates suggest that there are more than 100 different proto-oncogenes and several dozen tumor-suppressor genes in the human genome. For cancer to arise, it is rarely sufficient to have a defect in just one of these genes, nor is it necessary for a large number to be involved. Instead, each type of cancer tends to be characterised by a small handful of mutations involving the inactivation of tumor-suppressor genes as well as the conversion of proto-oncogenes into oncogenes. In other words, creating a cancer cell usually requires that the brakes on cell growth (tumor-suppressor genes) be released and the accelerators for cell growth (oncogenes) be activated.



In the absence of a growth-signalling Wnt protein (left), /3-catenin is targeted for degradation by the APC-axin-GSK3 complex and the Wnt pathway is maintained in the "off" position. The Wnt pathway (middle) is normally turned "on" by Wnt proteins, which bind to and activate cell-surface Wnt receptors. The activated receptors stimulate proteins that inactivate the APC-axin-GSK3 complex, thereby protecting /3-catenin from degradation. The /3-catenin then enters the nucleus and activates a variety of target genes, including those that control cell proliferation. Some cancer cells (right) have loss-of-function mutations in the APC gene. In the absence of functional APC protein, the APC-axin-GSK3 complex cannot be formed. As a result, /3-catenin accumulates and locks the Wnt pathway in the "on" position.

This principle is nicely illustrated by the stepwise progression toward malignancy observed in colon cancer.

Scientists have isolated DNA from a large number of colon cancer patients and examined it for the presence of mutations. The most common pattern to be detected is the presence of a KRAS oncogene (a member of the RAS gene family) accompanied by loss-of function mutations in the tumor-suppressor genes APC, SMAD4 and p53. Rapidly growing colon cancers tend to exhibit all four genetic alterations whereas benign tumors have only one or two. This suggests that mutations in the four genes occur in a stepwise fashion that correlates with increasingly aggressive behaviour.

The earliest mutation to be routinely detected is loss of function of the APC gene, which frequently occurs in small polyps before cancer has even arisen. Mutations in KRAS tend to be seen when the polyps get larger and mutations in SMAD4 and p53 usually appear when cancer finally develops. However, these mutations do not always occur in the same sequence or with the same exact set of genes. For example, APC mutations are found in about two-thirds of all colon cancers, but this means that the APC gene is normal in one out of every three cases. Analysis of tumors containing normal APC genes has revealed that many of these possess oncogenes that produce an abnormal, hyperactive form of b-catenin, a protein that — like the APC protein — is involved in Wnt signaling. Because APC inhibits the Wnt pathway and β-catenin stimulates it, mutations leading to the loss of APC and mutations that create hyperactive forms of β-catenin have the same basic effect. Both enhance cell proliferation by increasing the activity of the Wnt pathway.

Another pathway frequently disrupted in colon cancer is the TGFb-Smad pathway, which inhibits rather than stimulates epithelial cell proliferation. Loss-of-function mutations in genes coding for components of this pathway, such as the TGF/J receptor or Smad4, are commonly detected in colon cancers. Such mutations disrupt the growth-inhibiting activity of the TGF/ 3-Smad pathway and thereby contribute to enhanced cell proliferation.

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

Learning chimpanzee

RESEARCHERS AT ST ANDREWS UNIVERSITY STUDIED 80 WILD APES IN A UGANDAN RAINFOREST AND IN A 'WORLD FIRST' DECODED FOOT STOMPS AND HAND FLINGS. **NATASHA CULZAC** REPORTS

Scientists have deciphered as many as 66 chimpanzee gestures, which they say are used intentionally by the apes and which provide a potential link with the early development of the human language. From simple requests to complex social negotiation, the chimps were found to use a number of hand and body gestures to get their intentions across.

Eighty wild chimpanzees in the Budongo rainforest in Uganda were analysed in an attempt to interpret what the ground slaps, foot stomps and arm raises really meant. Researchers from St Andrew's University say that they have shown in unprecedented detail what our closest living relatives are trying to convey, after looking at over 4,500 gestures using secret recordings.

"Although it has been known for over 30 years that apes use gestures to communicate, until now no one has worked out what they are actually trying to say," the team, led by primatologists Dr Catherine Hobaiter and Professor Richard Byrne, said.

They have now created a sort of dictionary or lexicon of 19 different meanings, publishing the results in the journal *Current Biology* recently after concentrating on the apes' non-playful uses of their gestures.

Said Professor Byrne, "There is abundant evidence that chimpanzees and other apes gesture with purpose. Apes target their gestures to particular individuals, choosing appropriate gestures according to whether the other is looking or not; they stop gesturing when they get the result they want; and otherwise they keep going, trying out alternative gestures or other tactics altogether."

A tap by a chimpanzee was found to mean "stop that", a hand fling or slapping an object meant "move away," and an arm raise meant "I want that" or "give me that".

"Just as with human words, some gestures have several senses, but importantly the meanings of chimpanzee gestures are the same irrespective of who uses them," Dr Hobaiter said.

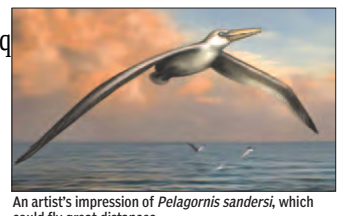
"Now that the basic chimpanzee gesture 'dictionary' is known, we can start to tackle other interesting questions. Do some gestures have very general meanings, where their intended sense is understood from the context? Or do subtle variations in how a gesture is made determine which sense was meant?" she added.

THE INDEPENDENT

PLUS POINTS

Largest wingspan

The largest flying bird in history had a wingspan of 24 feet — more than twice that of the biggest living albatross — and dined on fish probably caught in mid-flight over an open ocean, a study has found. Fossilised remains of the extinct species were first unearthed in 1983 during the building excavations for a new terminal at Charleston airport in South Carolina, but the latest analysis of the paper-thin bones of its skeleton


An artist's impression of *Pelagornis sandersi*, which could fly great distances.

revealed the giant wingspan, scientists said. The bird, named *Pelagornis sandersi*, lived about 25 million or 28 million years ago. Its long, slender wings suggest it could glide for long periods with minimal energy, although its huge size suggests it may have had trouble taking off and landing without the help of air currents or a downhill slope to run, they said.

Its wingspan would have been greater than that of the giant condor and royal albatross combined. Computer models of the wing bones and flight feathers of living birds suggest that Pelagornis was a very efficient glider and was capable of spending longer periods of time at sea without landing.

"Pelagornis sandersi could have travelled for extreme distances while crossing ocean waters in search of prey. That's important in the ocean where food is patchy," said Dan Ksepka, formerly of the US National Evolutionary Synthesis Centre in Durham, North Carolina, and now curator of science at the Bruce Museum in Greenwich, Connecticut.

The bird's bony, teeth-like projections and other anatomical details of its well preserved skeleton indicate that it belonged to the *Pelagornithidae*: an extinct group of giant seabird that lived during this period 25 million years ago, long after the dinosaurs had died out but long before the rise of humans.

"Pelagornithids were like creatures out of a fantasy novel. There is simply nothing like them around today… The upper wing bone alone was longer than my arm," Dr Ksepka said.

The latest study naming and describing *Pelagornis sandersi* is published in the journal *Proceedings* of the National Academy of Sciences. The species may eventually prove to have had the longest wingspan in the 140-million year history of the bird, being at the theoretical upper size limits for powered flight.

STEVE CONNOR/THE INDEPENDENT

Patenting virus

A new coronavirus called the Middle East Respiratory Syndrome has evoked fear and loathing, the fear on account of its deadly nature. Globally, 701 laboratory-confirmed cases of infection with Mers-cov, with at least 249 related deaths, have officially been reported to the World Health Organisation so far. That's a high rate of fatalities from a disease closely allied to the Severe Acute Respiratory Syndrome which infected more than 8,000 people in 2002-03 and killed several hundred.

The loathing has been evoked by a Dutch institute, the Erasmus Medical Centre in Rotterdam, which had taken out a patent application on the pathogen and its derivatives. It must be admitted here that the loathing is limited to analysts who are familiar with the complex interplay between public health and Intellectual Property Rights, primarily patents and the costs these impose. Western media reports, predictably, have focused almost entirely on the Saudi kingdom's inept handling of the Mers outbreak and failed, by and large, to question the legitimacy or the morality of Erasmus' patent claims. The Rotterdam institute had filed its application in September 2012 soon after it received the virus sample from an Egyptian doctor working in a Jeddah hospital who had extracted the sample from a patient but was unable to identify it. Erasmus isolated the gene sequence and identified it as a new strain of coronavirus on which it has applied for several US and international patents.

Unfortunately, details of the patent application were not known till recently. Thanks to the digging done by Edward Hammond, director of Prickly Research, a US consultancy on policies related to biodiversity, agriculture, infectious diseases and Intellectual Property, we know have some idea of the sweeping patent claims made by Erasmus. According to Hammond, the application claims "the virus as a whole, and its genetic material, particularly the unique variations that differentiate Mers from related viruses and appear to enable it to infect humans". In addition, the claims cover any diagnostics and vaccines derived from it. Those opposed to such patents worry that Erasmus' move will impede research to control Mers, which has already spread beyond the Arabian peninsula.

LALITHA JISHNU/CSE-DOWN TO EARTH FEATURE SERVICE