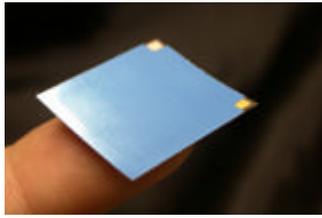


Energy



Safer, Longer-Lasting Batteries



Long-lasting thin-film batteries

A new type of rechargeable battery will soon be available commercially that overcomes these problems. But at a cost. These new batteries replace the liquid or gel electrolyte with thin layers of solid glass-like or polymer materials, which are more stable. "Nothing can leak, nothing can freeze, nothing can boil, rupture, or explode," says Tim Bradow, at Infinite Power Solutions of Golden, CO, a leading developer of thin-film batteries. In a battery, the electrolyte allows positive ions to move from one electrode to the other, while forcing electrons to travel through an external circuit, providing power. Bradow's company and a handful of others are using a solid glassy electrolyte, which they deposit as one of a series of flat layers that make up the battery. In addition to being safer, this solid material allows developers to use electrodes of pure lithium metal, which has the potential to significantly increase storage capacity. The batteries can survive extremes of cold and heat, which means, for example, they could be built into rubber tires to power air pressure sensors. Thin-film cells also can be stored for decades and retain almost all their charge, developers say--and deliver a powerful burst of energy when finally needed. And, in many applications, they can be actively used for decades, since they can be charged and discharged tens of thousands of times. These characteristics make thin-film batteries ideal for some new technologies. Remote sensors that scavenge tiny amounts of energy from vibrations, radio transmissions, or light, require batteries that can store this micro-supply of energy without leaking it away over time. And remote sensors need the high-power bursts many of these cells can deliver, to send data via radio signals to a central station. The ability to power radio transmission is also important for future medical implants that will deliver drugs or measure glucose levels. And these applications will also benefit from the batteries' long lifetimes; they can be recharged and discharged over many years, eliminating the need for surgery to replace them.

Bradow's company plans to start mass-producing its batteries next year. Nonetheless, thin-film batteries may not be the next-generation choice for most laptops. That's because the processes used to make them, such as physical vapor deposition, are still too expensive for producing large batteries. Also, these batteries, which can be a mere 0.1mm thick, each hold only micro-amounts of energy--as little as 1/1000 the amount in today's laptop batteries. While they could be stacked to provide adequate storage capacity, the layers of packaging separating the active materials in each battery would cancel out their capacity advantages. That is, they'd likely cost more, but not necessarily be smaller. The first applications, such as in industrial sensor packages in high-temperature equipment or oil wells, will be ones in which buyers are willing to pay \$100 apiece for batteries that meet their needs. Bradow says their batteries could be made for much less in high volumes, however, eventually making them practical for distributed sensor networks.

In spite of the current drawbacks to thin-film batteries, Donald Sadoway, at MIT, says some versions of them will power laptops--and electric vehicles--in the future. In contrast to the glass-like electrolyte used by Infinite Power Solutions and others, Sadoway has developed a solid-polymer electrolyte (today's lithium-ion polymer batteries use a gel) for use in thin-film batteries. This electrolyte, he says, could be processed in rolls like newspaper, or some other high-throughput process. Such a process for thin-film batteries, although not now being developed by industry, could bring down costs, he says, while innovative ways of packaging electrodes could reduce size. "We've made batteries in the laboratory that are 300 Whs/kg," he says. "That's two times the best lithium-ion [battery] on the market today."

Engine on a chip promises to best the battery

MIT researchers are putting a tiny gas-turbine engine inside a silicon chip about the size of a quarter. The resulting device could run 10 times longer than a battery of the same weight can, powering laptops, cell phones, radios and other electronic devices. It could also dramatically lighten the load for people who can't connect to a power grid, including soldiers who now must carry many pounds of batteries for a three-day mission -- all at a reasonable price. The researchers say that in the long term, mass-production could bring the per-unit cost of power from microengines close to that for power from today's large gas-turbine power plants. "

How can one make a tiny fuel-burning engine? An engine needs a compressor, a combustion chamber, a spinning turbine and so on. Making millimeter-scale versions of those components from welded and riveted pieces of metal isn't feasible. So, like computer-chip makers, the MIT researchers turned to etched silicon wafers. Their microengine is made of six silicon wafers, piled up like pancakes and bonded together. Each wafer is a single crystal with its atoms perfectly aligned, so it is extremely strong. To achieve the necessary components, the wafers are individually prepared using an advanced etching process to eat away selected material. When the wafers are piled up, the surfaces and the spaces in between produce the needed features and functions. Making microengines one at a time would be prohibitively expensive, so the researchers again followed the lead of computer-chip makers. They make 60 to 100 components on a large wafer that they then (very carefully) cut apart into single units. The MIT team has now used this process to make all the components needed for their engine, and each part works. Inside a tiny combustion chamber, fuel and air quickly mix and burn at the melting point of steel. Turbine blades, made of low-defect, high-strength microfabricated materials, spin at 20,000 revolutions per second -- 100 times faster than those in jet engines. A mini-generator produces 10 watts of power. A little compressor raises the pressure of air in preparation for combustion. And cooling (always a challenge in hot microdevices) appears manageable by sending the compression air around the outside of the combustor. "We're now trying to get them all to work on the same day on the same lab bench," Epstein, chief author, said. Ultimately, of course, hot gases from the combustion chamber need to turn the turbine blades, which must then power the generator, and so on. "That turns out to be a hard thing to do," he said. Their goal is to have it done by the end of this year. Predicting how quickly they can move ahead is itself a bit of a challenge. If the bonding process is done well, each microengine is a monolithic piece of silicon, atomically perfect and inseparable. As a result, even a tiny mistake in a single component will necessitate starting from scratch. And if one component needs changing -- say, the compressor should be a micron smaller -- the microfabrication team will have to rethink the entire design process.

New fuels from bacteria

A breakthrough in the production of biofuels has been developed by scientists in Germany. Research published in the September 2006 issue of *Microbiology*, a Society for General Microbiology journal, describes how specially engineered bacteria could be used to make fuel completely from food crops.

“Biodiesel is an alternative energy source and a substitute for petroleum-based diesel fuel,” explains Professor Steinbüchel of the Westfälische Wilhelms-Universität in Münster. “A growing number of countries are already making biodiesel on a large scale, but the current method of production is still costly”. “Biodiesel production depends on plant oils obtained from seeds of oilseed crops like rapeseed or soy”, explains Professor Steinbüchel. “However, production of plant oils has a huge demand of acreage which is one of the main factors limiting a more widespread use of biodiesel today. In addition, biodiesel production must compete with the production of food, which also raises some ethical concerns”.

Microdiesel, as the scientists have named it, is different from other production methods because it not only uses the same plant oils, but can also use readily available bulk plant materials or even recycled waste paper if engineering of the production strain is more advanced. Also, it does not rely on the addition of toxic methanol from fossil resources, like many other biodiesels. The bacteria developed for use in the Microdiesel process make their own ethanol instead. This could help to keep the costs of production down and means that the fuel is made from 100% renewable resources. “Due to the much lower price of the raw materials used in this new process, as well as their great abundance, the Microdiesel process can result in a more widespread production of biofuel at a competitive price in the future”, says Professor Steinbüchel.

There is a growing number of fuels used in cars and homes that are produced with the help of microbes. UK ministers are considering doubling the targets for the amount of biofuels sold in Britain by 2015.

Power from Not-So-Hot Geothermal

This power system could make it feasible to generate cheap electricity from lukewarm geothermal sources.

A large share of the geothermal resources suitable for power generation--those with temperatures higher than 300°F--are deep underground, beyond the reach of current technology. Lower-temperature resources are generally used for heating, but could be a bountiful source of power as well, if researchers were able to find an economical way to convert them into electricity.

Engineers at the United Technologies Research Center (UTRC), in East Hartford, CT, say they have developed a low-cost system that can utilize low-temperature geothermal resources. The technology could be particularly useful in generating electricity from waste hot water generated at oil and gas wells.

The modular, 200-kilowatt power plant from UTRC can convert temperatures as low as 165°F into electricity. The technology is similar to steam engines, except that steam or hot water vaporizes a hydrofluorocarbon refrigerant that drives the turbine. And the refrigerant has a lower boiling point than water. "It's hard to run a steam engine at 165 degrees [Fahrenheit]," says Bruce Biederman, who leads the project at UTRC. "The size of the equipment would be enormous and your turbine would be very poor in efficiency." The UTRC power plant can be thought of as a reverse cooling system, and the new turbine is essentially a refrigerator compressor running backwards, Biederman says. Instead of using power to create a temperature difference, like a refrigerator does, it converts a temperature difference into electricity.

The company is now testing a unit at a remote hot springs resort 60 miles northeast of Fairbanks, Alaska. Biederman expects a commercial power plant to be ready by early next year, after they've tested the reliability of the demonstration system.

According to him, the system could utilize the large amount of hot water pumped out of the ground at oil and gas wells. In Texas alone, more than 12 billion barrels of water are produced from wells. Oil companies usually discard the waste water by re-injecting it into the earth; but they could use it to generate electricity. Biederman is planning to set up demonstration projects at oil and gas wells in Texas and Nevada next year.

This reverse cooling concept isn't new; but until now no one has made an efficient turbine at a reasonable cost, he says. UTRC has kept down costs by modifying refrigeration units that its sister company, Carrier Corp., makes, and using its production line in Charlotte, NC.

The system's small size also keeps costs down, and makes it more usable. "The fact that it can fit on the back of a flatbed truck and be driven to a well site makes it much more convenient and less expensive," she says. "It's [like comparing] a mainframe computer and a laptop." And, as with other renewables, increasing fuel costs are spurring interest in geothermal power units.

Conversion in California

Chevron Corporation and the University of California, Davis (UC Davis) have formed a research collaboration to pursue advanced technology aimed at converting cellulosic biomass into transportation fuels. The joint research effort will coordinate with the California Biomass Collaborative to focus on renewable feedstocks available in California, including agricultural waste such as rice straw. Chevron Technology Ventures, a subsidiary of Chevron Corporation, plans to support a broad range of UC Davis scientists and engineers with funding of up to \$25m over five years for research into and development of these emerging energy technologies.

According to a statement, the objective of the Chevron-UC Davis research is to develop commercially viable processes for the production of transportation fuels from renewable resources such as new energy crops, forest and agricultural residues, and municipal solid waste. The collaboration calls for research in biochemical and thermochemical conversion, as well as a demonstration facility to test the commercial readiness of these technologies.

‘We think it's important to pursue research that could accelerate the use of biofuels since we believe they may play an integral role in diversifying the world's energy sources. Developing next-generation processing technology will help broaden the choice of feedstocks, including cellulosic materials,’ said Don Paul, vice president and chief technology officer, Chevron Corporation.

‘Once developed, next-generation processing technology will allow locally grown biomass to be harvested, processed into transportation fuels and distributed to consumers,’ said Rick Zalesky, vice president of Biofuels and Hydrogen, Chevron Technology Ventures.

The collaboration is expected to focus its research on understanding the characteristics of current California biofuel feedstocks; developing additional feedstocks optimised for features such as drought tolerance, minimal land requirements and harvesting technology; production of cellulosic biofuels; and the design and construction of a demonstration facility for biochemical and thermochemical production processes.

Ceramic microreactors developed for on-site hydrogen production

Scientists at the University of Illinois at Urbana-Champaign have designed and built ceramic microreactors for the on-site reforming of hydrocarbon fuels, such as propane, into hydrogen for use in fuel cells and other portable power sources. Applications include power supplies for small appliances and laptop computers, and on-site rechargers for battery packs used by the military.

"The catalytic reforming of hydrocarbon fuels offers a nice solution to supplying hydrogen to fuel cells while avoiding safety and storage issues related to gaseous hydrogen," said Paul Kenis, author of the paper on the work (it will appear on the journal *Lab on a Chip*)

In previous work, Kenis and colleagues developed an integrated catalyst structure and placed it inside a stainless steel housing, where it successfully stripped hydrogen from ammonia at temperatures up to 500 °C. In their latest work, the researchers incorporated the catalyst structure within a ceramic housing, which enabled the steam reforming of propane at operating temperatures up to 1,000 °C. Using the new ceramic housing, the researchers also demonstrated the successful decomposition of ammonia at temperatures up to 1,000 °C. High-temperature operation is essential for peak performance in microreactors, said Kenis. When reforming hydrocarbons such as propane, temperatures above 800 °C prevent the formation of soot that can foul the catalyst surface and reduce performance. "The performance of our integrated, high-temperature microreactors surpasses that of other fuel reformer systems," Kenis said. "Our microreactors are superior in both hydrogen production and in long-term stability." Kenis and his group are now attempting to reform other, higher hydrocarbon fuels, such as gasoline and diesel, which have well-developed distribution networks around the world.

Automobili a microonde

Un ingegnere inglese sta sviluppando un nuovo tipo di motore elettromagnetico per aerei, razzi e altre applicazioni

La fine delle ruote e delle ali. È questo lo slogan che un ingegnere inglese, Roger Shawyer, usa per pubblicizzare il suo prototipo di un nuovo motore, assolutamente rivoluzionario. La spinta infatti arriverebbe dalla radiazione elettromagnetica, e più specificatamente dalle microonde. Il nuovo motore non ha parti mobili e non emette sostanze inquinanti o pericolose per il clima.

L'ingegnere inglese sta pensando di costruire un prototipo che garantirebbe una spinta paragonabile a quella necessaria per le sonde spaziali. Un giorno si potrebbero poi realizzare automobili più simili agli hovercraft che alle auto di oggi. L'idea di Shawyer è semplice: si prende un generatore di microonde (magnetron) e si sparano le microonde prodotte all'interno di tubi vuoti all'interno e chiusi in fondo. L'urto delle microonde dovrebbe trasferire parte dell'energia ai tubi stessi e spingerli in una direzione.

In realtà, nonostante Shawyer abbia costruito un primo prototipo efficace, ci sono ancora molti ostacoli da superare. I tubi devono essere costruiti con materiale superconduttivo che riesca a condurre elettricità senza resistenza a temperature più alte rispetto a quelle usate per gli acceleratori di particelle, che generalmente si trovano a pochi gradi al di sopra dello zero assoluto, una temperatura chiaramente poco pratica per un motore di uso comune.

L'idea sembra comunque interessare americani e cinesi, ma non ha ancora convinto tutti gli scienziati. Tanto che il governo inglese, che ha finanziato l'ingegnere con 250 mila sterline, ha chiesto la revisione del progetto da parte di un gruppo indipendente di esperti.

Life Sciences

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New Target For Cancer Therapy Identified

A new target for cancer therapy has been identified by Monash University scientists investigating the cell signalling pathways that turn on a gene involved in cancer development. A team led by Jun-Ping Liu has identified two proteins that are involved in stopping the gene from producing a protein called telomerase that is essential if cancer cells are to proliferate.

Telomerase plays a key role in controlling the life span of cells by modifying structures called telomeres that are found at the end of chromosomes. Although it is involved in tumour development, telomerase is also found in modest quantities in most cells. It is plentiful in stem cells where it keeps the telomeres long, allowing the cells to keep dividing without limit which is necessary for the repair of damaged and worn out tissues throughout the human body.

However, studies have shown that telomerase also plays a key role in the formation of cancerous tumours. "It's the best indicator of cancer -- 85 per cent better than any other tumour marker," Professor Liu said. "What's more, telomerase is not associated with benign tumours; it's a marker for malignant tumours only." "If we can control the production of telomerase we can prevent the immortality of cancer cells and therefore cancer formation."

Professor Liu and his colleagues have been investigating breast cancer cells to identify the molecular signalling that is required to turn on, and also inhibit, the gene that produces telomerase. They have found two proteins - Smad3 and c-Myc - that are involved in turning off telomerase production. Their findings are published in the current issue of the Journal of Biological Chemistry. "It's significant to find inhibitors of telomerase and we have found, for the first time, the pathway that inhibits telomerase in human cells," Professor Liu said. "This reveals an important mechanism for developing anti-cancer agents that mimic these proteins and thereby inhibit the production of telomerase."

Two-faced protein can stop metastasis or promote it, researchers say

A protein known to be a key component of the glue that holds cells together also is involved in breaking them apart and promoting their movement when tumors begin to spread to other parts of the body, researchers at Mayo Clinic have found.

The study, published on the *Journal of Cell Biology*, helps illuminate the very first steps involved in metastasis, the spread of cancer that makes the disease difficult to treat, and suggests that a future designer drug might be able to block the beginning of this dangerous process, or stop it once it starts. "Our data show that this one protein, p120 catenin, is a key player in both suppressing invasion and promoting it," says the study's senior author, Panos Anastasiadis, "This is very exciting, because the findings open up a whole new field of discovery for novel therapeutics that should be applicable to most types of tumors." Their laboratory study looks at how p120 catenin interacts with different cadherin cell adhesion proteins in cancer cells. Cadherin proteins go through a cell membrane, and on the outside, they act like Velcro, sticking to other cadherin proteins on adjacent cells. On the inside of the cell membrane, cadherins bind, chain-like, to catenins, and catenins, in turn, regulate a cell's shape and function. The best understood cadherin is E-cadherin, which provides tight connections between epithelial cells, forming a strong barrier-like layer covering the inside of organs and body cavities and the outside skin of humans. "E-cadherin holds a human's cells and tissues together," Anastasiadis says. The other cadherins featured in this study belong to a group that collectively is called "mesenchymal" cadherins, which provide a looser bond between the cells that sparsely populate the connective tissue. "Collagen usually provides the strength to the connective tissue, so tight cell-cell adhesion is not that important," he says.

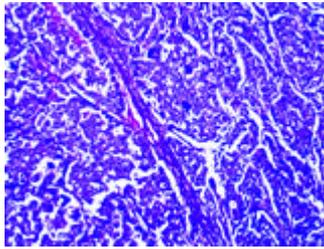
Sometimes, such as during human development or wound repair, epithelial cells need to travel to other areas, and to do this, they undergo a process known as "epithelial-mesenchymal transition" (EMT). The cell reduces its production of E-cadherin proteins and increases expression of mesenchymal cadherins, thus effectively loosening the anchors that keep the cell bound to its neighbors. Cancer, unfortunately, has adopted this strategy in order to spread, Anastasiadis says. "When the function of E-cadherin is lost in a cell, it can break free from its neighbors and travel to settle elsewhere," he says. "This means that E-cadherin normally helps suppress invasion." But researchers have noted that the p120 catenin protein seems mysteriously two-faced: while it normally strengthens cell-cell bonding, in some cases it can also negatively affect cell adhesion. They also have found that over production of p120 increases a cell's ability to move. But the significance of these observations had eluded scientists. This study provides an answer as to why p120 acts this way, which helps explain how the EMT shift between E-cadherin and mesenchymal cadherins allows cancer cells to break away from tissue and spread. The researchers that p120 "prefers" to bind to E-cadherin, rather than to mesenchymal cadherins. So in normal epithelial cells p120 always associates with the more abundant E-cadherins. But when E-cadherin production is lost during the progression of cancer, p120 catenins begin binding to mesenchymal cadherins. And when that happens, the researchers found that p120 unexpectedly switches on a cascade of events that promote cell movement.

"We show that E-cadherin suppresses invasion, at least in part, by binding to p120 protein in the cell," Anastasiadis says. "If E-cadherin is missing, p120 is free to bind to mesenchymal cadherins, setting off a process that leads to metastasis." Thus, p120 acts as a "rheostat" that promotes either stability when associated with E-cadherin or motility when it interacts with mesenchymal cadherins, he says. The investigators say that further research is needed to see if p120 functions the same way in living tissue as it does in laboratory cell culture, and they add that other "pathways" are likely involved in the transition to metastasis. But if the results continue to hold up, "it might be therapeutically possible to selectively shut down the pro-invasive function of p120 on mesenchymal cadherins while keeping the pro-adhesion function of p120 in normal epithelial cells.

"We have provided a better understanding of the processes involved in the initiation of tumor spread, and it is this process that we all seek to shut down," Anastasiadis says.

Who Really Needs Chemotherapy?

New diagnostic tests can predict which patients are most likely to benefit from chemotherapy.



In the Oncotype DX test, tissue samples from tumor biopsies are analyzed for their gene expression profile

The vast majority of breast cancer patients who get chemotherapy don't actually need it. But since it's difficult to pick out the few who do, almost all patients receive chemotherapy--and with it the fatigue, nausea, and pain that often accompany the treatment. But that could be about to change. A number of new diagnostic tests that predict who is most likely to benefit from chemotherapy are now under development or being tested in clinical trials. "If these tests catch on, physicians will start to prescribe chemotherapy more sparingly and save women the toxicity of it," says A. Raymond Frackelton, at Brown University, who is developing one such test. Thanks to new genomics techniques, scientists can rapidly test the DNA of tumor tissues collected during biopsies. By searching for genetic and molecular markers that correlate with a particular patient's outcome, they have identified hundreds of potential markers that signal when one patient's tumor will be more aggressive than another's, or more responsive to a certain type of therapy.

Collections of such markers are now being turned into diagnostic tests. One example is the Oncotype DX test, marketed by Genomic Health, which measures the expression of 21 genes in breast cancer tumors. Originally developed to determine a patient's prognosis--how likely she is to have a recurrence of the cancer--the test was recently shown to predict which patients will benefit from chemotherapy. According to Sheila Taube, at the National Cancer Institute, women classified by the test as having a high risk of recurrence showed a clear benefit from chemotherapy--their recurrence rate dropped by 27 %. Women with a good prognosis showed little benefit.

Such tests could solve a huge problem in breast cancer treatment. Using the current guidelines for treatment of women with a certain type of early-stage breast cancer, about 90% of women would be prescribed chemo and hormone therapy. "But 70 % of these women would still be alive 15 years later with surgery alone. Add hormone therapy, and it's about 85 %," says Taube. "Clearly, we are over-treating patients. But because there hasn't been a test that would predict who would recur and die from their disease, the community felt it was better to give chemo to everyone to benefit the few."

The Oncotype test is already commercially available. However, some questions remain about how best to use the results of such tests. For example, treatment choice is clear for patients who fall on the high or low end of the testing spectrum. Scientists eventually hope to develop tests indicating the appropriateness of chemotherapy for other cancers, as well as tests that predict exactly what type of therapy is best. While many potential markers have been identified--a meeting of the American Association of Cancer Research in Chicago listed presentation after presentation of potential diagnostic markers--the real challenge is turning these markers into a truly predictive test. Says Taube, "the process of evaluating a test is much more difficult than finding some potentially interesting markers."

How to follow proteins' interaction inside the cells; a major step for the study of Parkinson's and Alzheimer's disease

For the first time scientists have succeeded in developing a method that enables them to follow protein interactions directly inside cells. The discovery, now published in the "FASEB Journal" has crucial implications for the study and treatment of those neurodegenerative illnesses - such as Parkinson's, prion or Alzheimer's diseases - which are known to result from aberrant protein interactions and deposits. In fact, the new technique, which the researchers test by studying the protein believed to be behind Parkinson's disease, shows important potential not only to understand the mechanisms behind this type of diseases but also allows to observe, directly in the cells affected, the action of potential new treatments.

Parkinson's disease (PD) is a progressive neurodegenerative disease characterised by increasing motor problems that can render the patient totally dependent of others for everyday tasks. The illness is believed to result from the loss of specific neural cells in an area in the brain - called substantia nigra - involved in motor function. These specific neuronal cells produce dopamine, a neurotransmitter used for the communication between the different parts of the brain involved in coordination and movement, and their death leads to interruption of the nervous signal and, ultimately, to the motor problems observed in PD patients. Many neurodegenerative diseases, including PD, result from incorrectly folded proteins - all proteins have a specific shape/folding associated with their normal function - that either by becoming toxic, or by getting clumped together into insoluble aggregates, provoke the death of the brain cells in their surroundings. In the case of PD, the protein responsible for the pathology is believed to be alpha-synuclein, a brain protein of unknown function found in high quantities around the brain lesions of the substantia nigra. Recently it has also been found that mutations or multiplications of the alpha-synuclein gene are responsible for some forms of Parkinson's disease. However, until now the understanding of this type of neurodegenerative diseases has been a very slow process due also to the lack of processes that could allow the observation of proteins directly inside the cell. But the development, by Jochen Klucke and colleagues from the Massachusetts General Hospital and the University of Regensburg, Germany, of a technique called "Fluorescence Lifetime Imaging Microscopy" might change radically this. The new method consists in tagging the two ends of a protein with coloured dyes, which emit different energy specific wavelengths that can be read by a machine. The logic behind the technique is that the closer the two ends of the protein are (and so the two dyes), the higher is the interference between the two emitted wavelengths, allowing to infer if the protein has an open shape, is enrolled within itself or clotted together with other tagged proteins. This is crucial information as the same protein folded differently can have totally different effects/functions within the cell (it can even become toxic).

Using the new technique Klucke and colleagues studied alpha-synuclein in human cells and discovered a new aberrant interaction between different molecules of alpha-synuclein, which is probably involved in some forms of PD. Even more interesting, the researchers were able to see inside the cell how the chaperone protein Hsp70 - chaperone proteins are molecules whose function is to assist other proteins achieving a proper folding - reverts alpha-synuclein toxicity.

In fact, although no cure for PD has been found so far, very promising work in animals and cells in laboratory has found that Hsp70 is capable of eliminating alpha-synuclein toxicity and so (theoretically at least) might help control the disease. What the researchers now found was that Hsp70 opens alpha-synuclein misfolded structure allowing it to then revert to its normal (benign) shape. This result not only explains the mechanism behind Hsp70 protective effect, but proves that Fluorescence Lifetime Imaging Microscopy can be used to test new therapies in neurodegenerative diseases by directly accessing their effect inside the cells what is exciting news for both researchers and patients.

Research breakthrough for the protein factories of tomorrow

Using a kind of molecular ‘hip joint operation,’ researchers at Uppsala University have succeeded in replacing a natural amino acid in a protein with an artificial one. This step forward opens the possibility of creating proteins with entirely new properties that can be tailored to biotechnological applications. The study is presented in the latest issue of the journal *Chemistry and Biology*. All proteins are made out of twenty amino acids. These natural building blocks determine the structure and function of the protein.

Bengt Mannervik’s research team at Uppsala University has now demonstrated that artificial amino acids can be exchanged for a natural one that is critical to the stability and catalytic properties of the protein. The study opens the possibility of a new chemical biology where entirely new properties can be custom made for biotechnological applications. Their research work has focused on an important enzyme, glutathione transferase, which participates in the detoxification of the body from carcinogenic substances. The enzyme is made up of two identical protein structures that are joined by a contact similar to a key that fits a lock. The key is an amino acid that fits a cavity in the neighboring protein structure. In their work, the key has been replaced by artificial amino acids. Some exchanges yielded a fully active enzyme, while others did not.

The current study is a molecular equivalent to a hip joint operation, where the natural joint is replaced by an artificial part that is more robust. With the same methodology it is also possible not only to replace natural structures and functions but also to give proteins entirely new properties. Using simple chemistry, the twenty existent amino acids can be exchanged for hundreds of new chemical structures. In this way new proteins can be created with building blocks far beyond the limits of the genetic code.

International Initiative to Understand the Function of All Genes Has Started

The Conditional Mouse Mutagenesis Program EUCOMM, funded by the European Union with €13 million, and its Canadian partner project NorCOMM are getting support from the US: today, 7 September, the Knockout Mouse Project (KOMP) of the American National Institutes of Health (NIH) was launched. In close cooperation the three big projects want to mutate practically all genes of the mouse genome, in order to be able to understand the function of all genes with the help of mouse models.

European scientists have been pioneers in this initiative, the structure of which is going to be similar to that of the International Human Genome Project carried out earlier. Since the beginning of the year EUCOMM has already been producing mutated mouse genes in embryonic stem cells of the mouse, which will soon be made freely available to the scientific community. From these cells mouse models can be generated for all human diseases with genetic causes.

The European program is coordinated by Prof. Dr. Wolfgang Wurst, Director of the Institute of Developmental Genetics at the GSF – National Research Center for Environment and Health, Munich/Neuherberg, Germany. Wurst, his team, and the German Gene Trap Consortium (GGTC), funded by the German Federal Ministry of Education and Research, in the past developed part of the methods applied in the new projects and, thereby, made EUCOMM possible in the first place.

The methods used for the mutation of the mouse genes are conditional gene trapping and gene targeting technologies, which allow a particularly precise modelling of genetic diseases in the mouse, i.e. of Morbus Parkinson, cancer, cardiovascular diseases, and others.

EUCOMM raises the production of mouse models to a new level, because the models can now be generated in a coordinated manner as well as faster, more easily and at lower cost than in the past. This means that the decipherment of the mechanisms of all genetically caused diseases is becoming tangible.