

Energy



Creating Ethanol from Trash

A new system for converting trash into ethanol and methanol could help reduce the amount of waste piling up in landfills while displacing a large fraction of the fossil fuels used to power vehicles. The technology, developed originally by researchers at MIT and at Batelle Pacific Northwest National Labs (PNNL), in Richland, WA, doesn't incinerate refuse, so it doesn't produce the pollutants that have historically plagued efforts to convert waste into energy. Instead, the technology vaporizes organic materials to produce hydrogen and carbon monoxide, a mixture called synthesis gas, or syngas, that can be used to synthesize a wide variety of fuels and chemicals. The technology has been further developed and commercialized by a spinoff called Integrated Environmental Technologies (IET). In addition to processing municipal waste, the technology can be used to create ethanol out of agricultural biomass waste, providing a potentially less expensive way to make ethanol than current corn-based plants.

The new system makes syngas in two stages. In the first, waste is heated in a 1,200 °C chamber into which a small amount of oxygen is added--just enough to partially oxidize carbon and free hydrogen. In this stage, not all of the organic material is converted: some becomes a charcoal-like material. This char is then gasified when researchers pass it through arcs of plasma. The remaining inorganic materials, including toxic substances, are oxidized and incorporated into a pool of molten glass, made using PNNL technology. The molten glass hardens into a material that can be used for building roads or discarded as a safe material in landfills. The next step is a catalyst-based process for converting syngas into equal parts ethanol and methanol.

Ethanol is now widely used as a fuel additive, and it can also be used as a substitute for gasoline in some vehicles. Methanol is important for producing biodiesel and is currently made from methane in natural gas.

There is enough municipal and industrial waste produced in the United States for the system to replace as much as a quarter of the gasoline used in this country, says Daniel Cohn, a cofounder of IET. According to Jeff Surma, another cofounder and the CEO and president of IET, the multistage system makes it possible to produce fuels from waste at competitive costs. The economics look even better when including the fact that cities and manufacturers will pay to have waste removed, he says. This makes possible costs of between 10 and 95 cents per gallon of fuel, depending on the size of IET's system and how much it is paid to take waste. IET is currently in talks with a major Midwest utility and several municipalities interested in employing its technology, Surma says.

At this stage, multiple new approaches for transforming waste into biofuels are being explored, and the winner is not yet clear. IET's success will depend in large part on how it scales up its technology and develops a complete system, from getting the waste in the first place to distributing the fuel that it makes.

Life Sciences

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Novel Approach To Cancer Drug Given Major Boost

Scientists at ProXara Biotechnology Limited, a spin-out company at the University of Bristol, have identified a way of switching off one of the key mechanisms that leads to the development and growth of a tumour. The researchers hope to use their findings to develop a drug which could be used to fight cancer.

All cells in the body contain protein kinase B (PKB), a naturally-occurring enzyme that if active prevents cells from committing suicide. Programmed cell death, or apoptosis, is an important process in the body's development, but when this process goes wrong, unregulated cell growth occurs, leading to the development of tumour cells.

Recent research has shown that certain types of genetic damage, common to many cancer cells, lead to the movement of PKB from the interior of the cell to its surface membrane. When PKB attaches to the surface membrane, it becomes active, triggering a signal that tells the cell not to commit suicide. Professor Jeremy Tavaré at ProXara believes that by preventing PKB binding to the cell's surface membrane, he can ensure that apoptosis occurs, thus killing the cancer cells. "There has been a lot of interest in targeting PKB as a way of preventing tumour growth," says Tavaré. "Most of the interest so far has been in developing drugs that block the enzyme's signal. However, such drugs are very non-specific and can have many adverse side effects. We are working on a novel approach to prevent PKB actually binding to the cell membrane." Professor Tavaré and his team have discovered a drug-like compound, which prevents PKB binding to the cell membrane and makes the tumour cells commit suicide. They now wish to develop this compound to a point at which it could be used in clinical trials. The drug would be used initially to target lung cancer. If the approach works it could be adapted to treat other types of cancer or even inflammatory diseases such as arthritis or asthma.

The research has now been given a major boost by way of a £2.8 million award to the University of Bristol under the Wellcome Trust's Seeding Drug Discovery initiative. The initiative aims to bridge the funding gap in early-stage drug discovery, assisting researchers to take forward projects in small molecule therapeutics that will be the springboard for further R&D by the biotech and pharmaceutical industry.

Anticancer Nanoparticles Zero In on Tumors

A new class of nanoparticles that home in on tumors and then attract additional nanoparticles to the site could play an important role in diagnosing and treating cancer, according to researchers at La Jolla, CA. Because the nanoparticles increase their own accumulation inside tumors, the nanoparticles could be used to deliver larger quantities of MRI image-enhancing agents or cancer drugs into tumors. Ruoslahti and colleagues demonstrated that when tagged with fluorescent molecules, the nanoparticles made images of breast-cancer tumors in mice three times brighter than they would be if the particles did not self-amplify. Imaging is crucial in diagnosing cancer, and a threefold brightness is a substantial improvement for imaging, Ruoslahti says. The particles also induce blood clotting in 20 % of the blood vessels inside tumors, a property that could be used to destroy tumors by killing their oxygen flow.

Moreover, the new results, published in the Proceedings of the National Academy of Sciences, show that the self-amplifying property could be added to drug-carrying nanoparticles. "If you can get threefold more of drug into tumor, that's a big difference," Ruoslahti says.

Interest in using nanotechnology to detect and cure cancer has surged in recent years. By specifically targeting tumors, nanoparticles show promise in minimizing harm to surrounding tissue and reducing the side effects of invasive cancer treatments such as surgery and chemotherapy. One approach to targeting tumors is to coat the surface of nanoparticles with biological molecules that bind to receptor molecules found only in tumor tissue.

Ruoslahti's team takes this approach by coating iron-oxide nanoparticles with a special peptide that is attracted to blood protein clots found in the walls of tumor blood vessels. When injected into mice with breast cancer, the nanoparticles seek out tumors and bind to the blood-vessel walls. But then the nanoparticles go a step further. For reasons the researchers do not yet understand, the particles induce more clotting, which attracts even more nanoparticles so that their numbers build up in the tumors. The researchers found that the amplification also worked when they used liposomes--tiny liquid-filled spheres made of fat molecules--instead of iron-oxide nanoparticles. That means the self-amplifying process depends on the peptide, and the researchers could use different nanoparticles for various functions. For instance, the magnetic iron-oxide nanoparticles that they use could be employed for diagnosing cancer in humans, because they are popular MRI image-enhancing agents. Liposomes, on the other hand, could be used to carry cancer drugs. "The novelty here is the self-amplification. The technology would be applicable to just about any other nanoplatform that we use for tumor targeting, whether for imaging or therapeutic purposes."

Ruoslahti also plans to test other similar peptides that could cause clotting in much more than 20 % of the blood vessels to choke off the tumor's oxygen supply.

Making the technique safe and effective will take a lot more work. When injected into mice, the peptide-coated nanoparticles trigger an immune response, and the liver tries to get rid of them. The researchers currently avoid this by injecting a decoy particle to take the immune system's attention away from the peptide particles. But to be efficient, "we should be able to engineer a nanoparticle that does what we need it to do without the help of other nanoparticles". It would be crucial to localize the nanoparticles inside tumors so that they do not cause clots in the liver, lungs, and other organs, and so that drug-carrying nanoparticles do not accumulate in the organs. It would also be important to control the clotting that the peptides induce inside tumor blood vessels, because the clots could dislodge from the vessels and enter into the brain, heart, or other areas.

Cheap and Easy Screening for Breast Cancer



Z-Tech's 12-petaled breast-screening device

A new device that screens for breast cancer by measuring electrical resistance in tissue could soon become a painless, radiation-free, and less costly alternative to mammography for women at high risk for the disease. The company developing the technology, Z-Tech, based in Westford, US, recently completed international trials of screening methods on 3,500 subjects at 28 different sites. A paper detailing the outcome of the two-year trial will be submitted later this year for peer review, the company says. But preliminary results indicate that the device catches more cancers and has fewer false positives than film mammography, most notably in patients younger than 50 years of age. Steven Nakashige, CEO of Z-Tech, says the company's test works best on women with dense breast tissue, an area where mammography is generally at its weakest. The test also takes only a few minutes and doesn't require a specially trained technician. Its simplicity, as well as the fact that it doesn't emit the potentially harmful ionizing radiation associated with mammography x-rays, could make it an effective tool in battling this deadly and most frequently diagnosed cancer in women. "We believe this would significantly increase [screening] compliance rates, which would help detect cancers earlier," says Nakashige. "And if you detect cancers earlier, you can reduce mortality rates." Some observers say that Z-Tech's technology, while an improvement over mammography, needs to perform dramatically better if the aim is to encourage regular screening of the population at an earlier age. The worry is that false positives, even when there are fewer compared with mammography, would increase on an absolute basis.

So-called electrical-impedance scanning also faces competition from a variety of other emerging screening technologies that use everything from low-level microwaves to infrared light to locate breast tumors. The challenge with using impedance measurements is to make it sophisticated enough so that it can distinguish between cancers and less serious abnormalities--a complex computational task that engineers at Dartmouth College, in Hanover, US, are tackling.

The Z-Tech system works on two principles: that malignant tumors permit electricity to pass through more easily than noncancerous cells do, and that the left and right breasts of a healthy woman typically exhibit the same electrical characteristics. A disposable flower-petal-shaped disc is attached to each breast. Each of the 12 petals on the disc is an electrode, which bends over the contour of the breast and sticks to the skin with a light adhesive. A mild current is then applied between the electrodes in more than 300 combinations, and the data is sent to a bedside computer for immediate analysis. "Our device compares one breast to the other breast and looks for the breast that has the higher [electrical] impedance," explains Nakashige. "It uses an algorithm and determines if the impedance exceeds a certain threshold. If it does, there's a high probability it's cancerous." He emphasizes, however, that the test doesn't produce an image of the breast and can't detect tumor types. "Our device is really a screening device. It's more of a yes-no answer that you get. You just want to determine whether someone should go on for diagnostic [mammography, ultrasound, or MRI] testing or go home."

Genetic search closes in on 'Alzheimer's mutation'

The long-suspected link between Alzheimer's disease and abnormalities in the way amyloid protein is processed in the brain has been confirmed at last – a significant step on the path to an effective drug treatment for the condition.

Usually harmless, the amyloid precursor protein is thought to trigger neurological damage when it is broken down and transformed into toxic fragments of beta-amyloid. Previous studies have shown that people with Alzheimer's have reduced levels of several proteins involved in processing amyloid. To find out whether low levels of any of these proteins could cause the production of toxic beta-amyloid, Peter St George-Hyslop at the University of Toronto in Canada and colleagues studied the DNA of 6861 people, 46% of whom had had Alzheimer's.

Those with the disease proved significantly more likely to have mutations in the gene *SORL1*, which usually produces a protein that binds amyloid and transports it to an area of the cell where it can be harmlessly recycled. To demonstrate that mutate variations in *SORL1* could trigger the disease, the researchers treated cells in the lab to deactivate the gene. This, they found, led to a substantial increase in the production of toxic beta-amyloid. "Where *SORL1* is absent or defective, it allows the amyloid to float off into other areas where it is degraded," says St George-Hyslop. The team have identified two regions of *SORL1* which they believe harbour the disease-causing mutations, but have not yet found the mutations themselves. When they do, they hope this will lead them to a drug that increases *SORL1* activity in people with variant forms of the gene.

Journal reference: Nature Genetics

'Quiet Revolution' May Herald New RNA Therapeutics

Scientists at the University of Oxford have identified a surprising way of switching off a gene involved in cell division. The mechanism involves a form of RNA, a chemical found in cell nuclei, whose role was previously unknown, and could have implications for preventing the growth of tumour cells.

RNA plays an important and direct role in the synthesis of proteins, the building blocks of our bodies. However, scientists have known for some time that not all types of RNA are directly involved in protein synthesis. Now a team of scientists has shown that one particular type of RNA plays a key role in regulating the gene implicated in control of tumour growth. The research is published on the current issue of Nature.

The Human Genome Project identified about 34,000 genes responsible for producing proteins. The remaining part -- in fact, most of the genome -- constituted what was considered to be "junk" DNA with no function. However, latest estimates show that this "junk" DNA produces around half a million varieties of RNA of unknown functions. "There's been a quiet revolution taking place in biology during the past few years over the role of RNA," says lead author Alexandre Akoulitchev. "Scientists have begun to see 'junk' DNA as having a very important function. The variety of RNA types produced from this "junk" is staggering and the functional implications are huge."

The particular form of RNA that has been of interest to Dr Akoulitchev's team is involved in regulation of the dihydrofolate reductase gene (DHFR), determining whether the gene is "on" or "off". The DHFR gene produces an enzyme that controls thymine production, necessary in rapidly dividing cells. "Inhibiting the DHFR gene could help prevent the growth of neoplastic cancerous cells, ordinary cells which develop into tumour cells, such as in prostate cancer cells," explains Dr Akoulitchev. "In fact, the first anti-cancer drug, Methotrexate, acts by binding and inhibiting the enzyme produced by this gene."

Dr Akoulitchev believes that understanding how we can use the RNA to switch off or inhibit DHFR and other genes may have important therapeutic implications for developing new anti-cancer treatments.

Scientists Map Key Landmarks In Human Genome: New Method Reveals Positions Of Gene-regulating Nucleosomes

Dana-Farber Cancer Institute researchers have developed a powerful method for charting the positions of key gene-regulating molecules called nucleosomes throughout the human genome. The mapping tool could help uncover important clues for understanding and diagnosing cancer and other diseases, the scientists say. Moreover, it may shed light on the role of nucleosomes in the process of "reprogramming" an adult cell to its original embryonic state, which is a critical operation in cloning. David E. Fisher and colleagues describe their findings in *Nature Biotechnology*,

"This study presents the first global view of human nucleosome positioning," said Fisher. Although analyses of this type had been pioneered in simpler organisms such as yeast, those approaches were not feasible when applied to the massively larger and more complex human genome. The new technique "provides major new clues to the locations of many hallmark features of the human genome, such as where transcription factors bind, where transcription begins and possibly ends, and where there are other biologically important structural features," said Fisher. Transcription factors are proteins that bind to particular DNA sites in "promoter" regions of genes and turn the genes on or off.

The novel method employed gene microarray studies followed by sophisticated computational data analysis to pin down the nucleosome locations. The paper describes how the scientists used the technique to locate nucleosomes in 3,692 promoters (regions of DNA that interact with regulatory factors to control gene activity) within seven human cell lines, including malignant melanoma. Nucleosomes are spherical packing units for DNA. They consist of a length of DNA wrapped around a core, like ribbon around a spool that is made up of proteins called histones, and the nucleosomes are located along the chromosomes like beads on a string. Nucleosomes have multiple functions, including allowing several feet of DNA to be packed tightly into a cell's nucleus. They also regulate gene expression, or activity, by determining whether DNA sequences can be accessed by transcription factors, allowing the factors to regulate expression of a nearby gene.

It has long been assumed that these factors can't bind to a stretch of DNA that is bound by a nucleosome; they can, however, attach to DNA "linkers" between two nucleosomes. With their new method, Fisher's group found that transcription factor binding indeed typically occurs in the "linker" regions in between nucleosomes, rather than in the DNA regions that are tied up by the nucleosome complexes. These results suggest that nucleosome positioning controls the turning on or off of genes, and the nucleosomes can be relocated if cellular needs change.

Fisher said the work that led to the current paper began when his lab was studying the protein, MITF, made by one of these genes, which influences the expression of other genes. "We wanted to understand how MITF regulates target genes, and specifically where in promoter regions of those genes does MITF bind," Fisher said. "In the process of this work, we asked whether MITF is binding between nucleosomes or on top of nucleosomes, and that led us to devise a method to ask where the nucleosomes are located."

The researchers used gene microarrays to which DNA associated with single nucleosomes was added. The nucleosomal DNA was derived from several cancer cell lines including melanoma and breast cancer, as well as several normal human cell types. On a graph, the data displayed as a series of peaks and troughs corresponding to positioned nucleosomes and nucleosome-free regions, respectively. Analysis revealed that promoters of genes that had similar expression status (they were all "off" or "on") had related nucleosome locations. The technique successfully pinpointed the location of some nucleosomes previously found through other means, but the new method can be applied to the entire genomes of human and other cells, said the scientists. In the future, the techniques might be useful as a diagnostic tool, Fisher said.

Molecular Discovery Could Help Drugs Target Unhealthy Cells

University of Central Florida and University of California Riverside professors are a step closer to being able to deliver life-saving drugs through tiny molecules that would travel through the bloodstream and destroy only cancer-ridden cells.

In a paper published on Science Express, the scientists describe how they got an adsorbate molecule (anthraquinone) to pick up two carbon dioxide atoms and carry them in a specific direction on a flat copper surface. Their discovery helps scientists understand how they someday may be able to attach therapeutic drugs to molecules.

"It's significant because we wouldn't expect atoms to move that way," said UCF Talat Rahman, who co-authored the study with Sergey Stolbov. "Atoms tend to move randomly, like dust particles, and getting them to move in a specific direction will help in our understanding and manipulating of the region around atoms."

It's what sci-fi fans have seen on television -- nanotechnology that sends microscopic creatures to space or the body to make repairs. That sci-fi reality is years away, but this research is a step in that direction. "Right now the only way we can transport atoms is with instruments," Rahman said.

"Being able to control that at a molecular level would help us create a natural transportation system that could aid us in many ways in the future."

Next, the researchers want to see if they can make the molecule carrier go around corners, rotate its cargo or send out photons to let scientists know where it is located