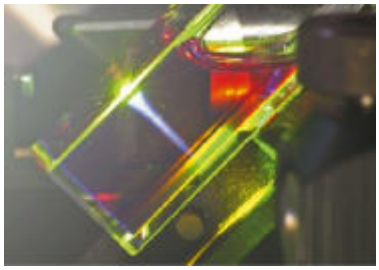
Energy

FONDAZIONE SILVIO TRONCHETTI PROVERA

FTP

A Boost For Solar Cells With Photon Fusion

An innovative process that converts low-energy longwave photons (light particles) into higher-energy shortwave photons has been developed by a team of researchers at the Max Planck Institute for Polymer Research in Mainz and at the Sony Materials Science Laboratory in Stuttgart. With the skillful combination of two light-active substances, the scientists have, for the first time, manipulated normal light, such as sunlight, to combine the energy in photons with particular wavelengths (Physical Review Letters, October 4, 2006). This has previously only been achieved with a similar process using high-energy density laser light. The successful outcome of this process could lay the foundation for a new generation of more efficient solar cells.
The efficiency of solar cells today is limited, among other reasons, by the fact that the longwave, low-energy part of the sunlight cannot be used. A process that increases the low level of energy in the light particles (photons) in the longwave range, shortening their wave length, would make it possible for the solar cells to use those parts of light energy that, up to now, have been lost, resulting in a drastic increase in their efficiency. The equivalent has only been achieved previously with high-energy density laser light which, under certain conditions, combines two low-energy photons into one high-energy photon - a kind of photonic fusion. This is a significant step forward for the scientists at the Max Planck Institute for Polymer Research and at the Sony Materials Science Laboratory. In developing this process, they have succeeded, for the first time, in pairing up photons from normal light, thus altering the wavelength. They used two substances in solution, platinum octaethyl porphyrin and diphenylan-thracene, which converted the longwave green light from a normal light source into shortwave blue light.



Similar to the process in laser light, this also pairs up photons, but in a different way. When a molecule is manipulated by laser light to take up two photons, which is only probable if it is literally bombarded with a laser beam of photons, the molecules in this case only receive one photon. Two photon partners are brought together between the molecules via a different mechanism called triplet-triplet annihilation. By selecting different, corresponding "matchmaker" molecules, it is possible to combine the energy from photons from the entire sunlight spectrum. The two substances developed by the researchers as "photon matchmakers" have quite different properties. Whereas one serves as an "antenna" for green light (antenna molecule), the other pairs the photons, connecting the two low-energy green photons into one high-energy blue photon, which it transmits as an emitter (emitter molecule). This is what happens in detail: first the antenna molecule absorbs a green low-energy photon and passes it to the emitter molecule as a package of energy. Both molecules store the energy one after the other in "excited" states. Then, two of the energy-loaded emitter molecules react with each other - one molecule passes its energy package to the other. This returns one molecule to its low-energy state. The other, conversely, achieves a very high-energy state that stores the double energy package. This state rapidly collapses when the large energy package is sent out in the form of a blue photon. Although this light particle is of a shorter wave length and higher in energy than the green light emitted initially, the end effect is that no energy is generated, but the energy from two photons is combined into one.
The process is very interesting in chemical terms as the molecules must be carefully matched to allow the energy to be transmitted efficiently, and neither the antenna nor the emitter molecules are

allowed to lose their energy through shortcuts. The researchers therefore had to synthesize an antenna molecule that absorbed longwave light and store it for so long that the energy could be transferred to an emitter. Only a complex metal organic compound with a platinum atom in a ring-shaped molecule was suitable for this purpose. The emitter molecule, on the other hand, must be able to take the energy package from the antenna and hold on to it until another excited emitter molecule is found for the subsequent photon fusion.

As this procedure allows previously unused parts of sunlight to be used in solar cells, the scientists are hoping that it offers the ideal starting point for more efficient solar cells. To optimize the process and to bring it closer to an application, they are testing new pairs of substances for other colors in the light spectrum and are experimenting with integrating them in a polymer matrix.

Printing Fuel Cells

A new printing process could cheaply make complex fuel-cell reformers, and other microscale devices.

A technique based on an inexpensive process used to print electronic circuit boards has been developed for constructing complex three-dimensional devices, such as a micro-reformer for fuel cells. The new method could be a versatile way to more cheaply and easily create microscale devices, making it practical to fabricate fuel cells for recharging two-way radios. It could also help make some types of chemical manufacturing safer and more efficient, and produce wireless-tire air-pressure sensors inexpensive enough to be standard issue in new cars.

The process works by building up hundreds of layers of specially formulated inks containing various materials, such as polymers, metals, and ceramics, to form a three-dimensional structure, complete with hollow passages and chambers sealed inside, says Arthur Chait, CEO of EoPlex Technologies, in Redwood City, CA, the startup company that developed the new technique. For each layer, the technology prints both the materials that will make up components of the final device and space-holder materials that will help support the next printed layer.

Each layer is cured by a flash of ultraviolet light before the next layer is printed, and once all of the layers have been printed, the whole assembly is fired at high temperatures, about 850 degrees Celsius , depending on the materials used. These materials have to be carefully selected so that they shrink at the same rate during the firing, and so that the space-holding materials can diffuse through the other materials, leaving behind empty spaces.

One of the company's first devices, a fuel-cell "reformer" for stripping hydrogen from methanol, will supply enough hydrogen for micro fuel cells that recharge 20-watt two-way radios used in emergency areas, where grid power isn't reliably available. The 300-layer device shows the complexity possible with the printing technique, Chait says. The layers form a total of 33 discrete components, such as heating coils, catalyst beds, "chambers, passageways, a diffuser section, a reformer section, and a combustion section," he says. Methanol is fed into the device, and the combination of steam and catalysts free the hydrogen. The entire reformer is the size of two dominoes.

Small reformers have been built before by researchers at MIT, the Pacific Northwest National Laboratories, and the University of Illinois. But so far, with the exception of a shoebox-size device, they have been inefficient, transforming only a "very small percentage" of the energy in the fuel into electricity, says Klaus Jensen, professor at the MIT Microsystems Laboratory. If EoPlex has indeed succeeded in making a small device that works well in a fuel cell, he says, "that would be a very important advance."

A Practical Fuel-Cell Power Plant

GE's advance allows for a solid-oxide fuel cell to use coal-based fuels at costs approaching that of conventional power plants

GE’s new solid-oxide fuel-cell prototype

One of the most efficient ways to produce power at future coal-gasification power plants is with solid-oxide fuel cells, which use the hydrogen from the gas stream to generate electricity through chemical reactions. This is more efficient than simply combusting the gas stream from coal gasification. And unlike other types of fuel cells, the solid-oxide variety can operate at very high temperatures and efficiencies, and be scaled up to provide cities with power.
But among the various challenges to developing the technology, manufacturing cost has been a potential deal breaker. Now, researchers at GE have demonstrated a manufacturing method that assembles layers of ceramic and electrolyte materials cheaply so that the final product can be built for about $800 a kilowatt, which starts to approach the $500-to-$550-per-kilowatt cost of building a conventional gas-fired power plant.

GE's six-kilowatt prototype achieves 49%efficiency in converting fuel into electricity, which compares favorably with the 35% efficiency of conventional coal-burning power plants. "I do believe GE has established a new state of the art," says Wayne Surdoval, of the National Energy Technology Laboratory, part of the U.S. Department of Energy, which is funding this project and others aimed at producing better solid-oxide fuel cells. "The bottom line," he adds, is that the GE prototype "is a particularly inexpensive fuel cell to make. Basically, you are using simple manufacturing techniques using fairly inexpensive materials in the cell." Surdoval likens the process to making pizza dough. Three sets of materials--representing the two electrodes and one electrolyte that make up each layer of a fuel cell--are mixed and put through two rollers that squeeze them. "You have three different doughs, you flatten each one, then layer them, then flatten them," he explains. "Then basically, you bake it."

The process paves the way for mass manufacture, according to Kelley Fletcher, the advanced-technology leader for sustainable-energy programs at GE Global Research "People have made fuel cells that make more power, and people have also made ones that have done this efficiency level," he says. "But to do so in one package, and at the cost estimate that we have done, is the real achievement here." Previous prototypes have cost thousands of dollars per kilowatt to manufacture, he says.

Life Sciences

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Experts crack cancer 'gene codes'

US scientists have cracked the entire genetic code of breast and colon cancers, offering new treatment hopes. The genetic map shows that nearly 200 mutated genes, most previously unknown, help tumours emerge, grow and spread. The discovery could also lead to better ways to diagnose cancer in its early, most treatable stages, and personalised treatments, Science magazine reports.

The Johns Hopkins Kimmel Cancer Center says the findings suggest cancer is more complex than experts had believed. The mutated genes in breast and colon cancers were almost completely distinct, suggesting very different pathways for the development of each of these cancer types. Each individual tumour appeared to have a different genetic blueprint, which could explain why cancers can behave very differently from person to person, the scientists said. "No two patients are identical," co-author Victor Velculescu explained.
Now researchers will study how these mutations occur in breast and colon cancers. Previous cancer gene discoveries have already led to successful detection and treatment strategies. For example, the breast cancer drug Herceptin targets a breast cancer cell receptor made by the Her2-neu gene. Blood tests for hereditary bowel cancer are based on the APC gene.

Dr Anna Barker, of the National Cancer Institute, said: "Maximising the numbers of targets available for drug development in a specific cancer means that patients will ultimately receive more personalised, less toxic therapies.In the future, scientists hope to be able to tailor plans for preventing or treating cancer to each person's individual genetic profile. Studies like this can help us to accomplish this goal."

Waves for Non-Invasive Cancer Treatment

Magnetic resonance guided focused ultrasound (MRgFUS) ablation procedures have provided relief for thousands of women with uncomfortable uterine fibroids. Now researchers are hoping to apply this non-invasive outpatient procedure to more malignant problems, such as liver cancer, in the near future.ExAblate 2000, from GE Healthcare and InSightec, integrates Magnetic Resonance Imaging (MRI) with focused ultrasound energy.
The procedure has already proved successful in the removal of benign uterine tumours around the world and at St Mary's Hospital and Imperial College London, where it is being tested. However, the procedure has yet to be approved for coverage under the National Health Service, although it has been approved by the Food and Drug Administration in the US.ExAblate 2000 is also undergoing a trial to treat liver cancer.

The technology works by directing high intensity focused ultrasound waves into the body at a specific tissue via a transducer. At the focal point, the ultrasonic 'beam' brings the temperature of the targeted tissue to 65-85°C, which destroys the damaged tissue.The thermal imaging capabilities of the MRI scanner provide three dimensional, real-time images and a temperature map of the targeted tissue, so that a physician has a high degree of control over the outcome of the therapy.
While capturing an MR image of uterine fibroids has been successful, Lynn Golumbic, director of marketing at InSightec, said the liver's constant movement makes imaging difficult.
'You take a picture of a liver in one position and then it might move,' she said. 'We can't exactly stop it, but what we can do is stop the breathing for short periods of time so we can treat this.'
Another problem with MRgFUS technology being applied to the liver is that bones absorb ultrasonic energy and much of the liver is located between the rib cages.Golumbic said the liver is just one example of the design challenges that researchers at InSightec's headquarters in Israel are facing when trying to apply this technology to various body organs.
'We have clinical trials going on right now for brain tumours. We also have liver and breast, and we are also developing some technology in order to treat prostate cancer,' she said. 'The list is really unlimited but the question is how to develop the combination of the hardware and the software to reach in to all these things.'Golumbic said that getting the technology to its current stage has been challenging enough.

InSightec was founded in 1999 when GE Healthcare (then GE Medical Systems) and Elbit Medical Imaging transferred their proprietary technology to the company to enable it to concentrate on developing the MRgFUS surgery both companies had investigated.
Golumbic said more than 2,000 patients have been treated with ExAblate 2000 at 35 installations around the world.'It's slowly picking up and the reasons are obvious — because if you can go and have a non-invasive treatment that enables you to go back to work and back to normal life immediately, why wouldn't you?' she said. 'With traditional surgery, most of your recovery time is spent recovering from your incision. With this, we are treating the problem while letting you get on with your life.'

Listening To The Sound Of Skin Cancer: New Technique Detects Signature Of Metastasizing Melanoma

Researchers at the University of Missouri-Columbia can now detect the spread of skin cancer cells through the blood by literally listening to their sound. The unprecedented, minimally invasive technique causes melanoma cells to emit noise, and could let oncologists spot early signs of metastases -- as few as 10 cancer cells in a blood sample -- before they even settle in other organs. The results of the successful experimental tests appear in the Oct. issue of the journal Optics Letters.

The team's method, called photoacoustic detection, combines laser techniques from optics and ultrasound techniques from acoustics, using a laser to make cells vibrate and then picking up the characteristic sound of melanoma cells. In a clinical test, doctors would take a patient's blood sample and separate the red blood cells and the plasma. In a healthy person, the remaining cells would be white blood cells, but in a melanoma patient the sample may contain cancer cells. To find out, doctors would put the sample in saline solution and expose it to rapid-fire sequences of brief but intense blue-laser pulses, each lasting just five billionths of a second.

In lab tests, the Missouri-Columbia team was able to detect melanoma cells obtained from actual patients, showing that the method can spot as few as 10 cells in saline solution. The dark, microscopic granules of melanin contained in the cancer cells absorb the energy bursts from the blue-laser light, going through rapid cycles of expanding as they heat up and shrinking as they cool down. These sudden changes generate loud cracks -- relative to the granules' size -- which propagate in the solution like tiny tsunamis. The sound waves produced by melanin are high-frequency ultrasounds, meaning that they cannot be heard by the human ear, even if amplified. However, researchers can pick them up with special microphones and analyze them with a computer. Other human cells do not contain pigments with the same color as melanin, so the melanin signature is easy to tell apart from other noises, said John Viator coauthor of the paper. And the presence of melanin granules in the blood is an unmistakable sign. "The only reason there could be melanin in the human blood is that there would be melanoma cells," he said.

This new blood test would allow for a much more timely diagnosis of metastasis and with early diagnosis comes early treatment and increased likelihood for survival. As one of the most aggressive forms of cancer, if a melanoma is not removed at its earliest stages, it will penetrate into the deep layers of the skin. From there its cells can break off and pass into the circulatory and lymphatic systems, spreading to other organs and creating metastases even after the original melanoma has been surgically removed.

An earlier metastasis warning, as this blood test provides, could alert oncologists to the cancer when it's at its earliest stages in other parts of the body and help them to begin a quicker counterattack. "Our method can help doctors plan treatment to battle the spread of the disease," he said. Current techniques to monitor the disease spread and recurrence have proven to be inaccurate, time-consuming and painful. This new blood test would enable physicians to have a more accurate method of monitoring for metastasis since its results would be almost immediate. "It could take just 30 minutes to find out if there are any circulating cancer cells," . The team is now planning a pilot study on actual blood samples from patients, and larger clinical studies will need to be done, but the test shows great promise for early detection of the spread of this disease, according to Viator.

The team is also working to extend the reach of its technique to other types of cancer. Because of melanin, melanoma is the only type of cancer whose cells will strongly absorb all wavelengths of light, emitting ultrasounds that stand out from those of other cells. But artificial materials could also be introduced, to act as light absorbers -- and as noise makers. "We're looking for methods to attach other kinds of absorbers to cancer cells. For example, gold nanoparticles could be attached to the cells using proteins that bind to special receptors on the cells' membranes. With their own photoacoustic signature, the gold particles would then signal the presence of cancer cells£”

'Quality Control Check' Protein May Help Target Anti-Tumor Drugs

Mayo Clinic researchers have found that a protein that initiates a "quality control check" during cell division also directs cell death for those cells damaged during duplication. This knowledge represents a potential "bulls eye" for targeting anti-tumor drugs. The findings appear in the current issue of Science.

The researchers examined a protein called cyclin-dependent kinase 2 (CDK2), which works as a "quality control inspector." As normal cells divide, they pause in the replication process when they find inaccurate genetic code embedded in their DNA. The health and well-being of offspring cells depends on accurate genetic code transfer from one generation of cells to the next. The Mayo researchers showed that when errors in genes are irreparable, CDK2 modifies another cellular protein -- FOXO1 -- to send a signal that results in the death of the cell. This protein-to-protein relationship invites targeted drug intervention to control unregulated growth of cancer cells.

"Quality control within dividing cells is essential because mistakes during duplication of the genetic code can lead to cancer," says Donald Tindall, co-leader of the Mayo Clinic Cancer Center prostate cancer research program. "CDK2 is a key protein component in the cellular mechanism that leads to repair of damaged DNA."If cells pass this quality control checkpoint, they can resume the process of dividing into two daughter cells. If, however, major irreparable discrepancies occur in the genetic code, cells are shunted toward a molecular sequence that leads to death, or apoptosis. Cells have the genetic knowledge to sacrifice themselves for the greater good of the organism rather than to pass on errant genetic codes that can lead to disease. Genetic errors that sneak past the cell's quality control check-points can make the cell prone to develop into cancer.

The Mayo researchers documented that CDK2 infuses high energy into another cellular protein, FOXO1, switching it on as the initial link in a signal that tells the cell to set itself up for apoptosis. CDK2 adds phosphorylation to a specific serine residue on the chain of amino acids that make up FOXO1. In case of robust errors found in the genetic code, CDK2 signals FOXO1 to explicitly call for the cell to produce a set of proteins leading to apoptosis.

"If the cell has minor alterations in the DNA code that can be repaired, those repairs are made," says author Haojie Huang. "If the genetic message cannot be repaired, our studies show that CDK2 can initiate the steps necessary for cells to order the production of genes involved with cell death, and the errant cell dies without propagating its damaging genetic message to progeny cells of its own."

"As patients and their physicians seek to control or cure tumors, research is providing new approaches to limiting cancer from growing and spreading," Dr. Tindall said. "With this new understanding of the biology driven by critical dual functions of CDK2, the cancer community can focus on ways to regulate a mechanism that the cell contains to prevent damaged genetic messages from being inherited and spread in proliferating tumor cells."

A new Alzheimer's vaccine

New approaches to immunizing patients against the harmful protein buildup characteristic of Alzheimer's disease offer hope for safer treatments.

Vaccination against Alzheimer's disease is one of the most promising treatment strategies. But safety concerns arising after initial human trials have slowed clinical development of such vaccines. Now new research that aims to bring the benefits of vaccines without the harmful side effects are raising hopes once again for this largely untreatable disease.

Alzheimer's vaccines work by preventing or clearing the buildup of a protein, known as beta-amyloid, which clogs the brains of Alzheimer's patients. A patient can be injected with either an active or passive vaccine. Active vaccines contain the protein itself, triggering the body's immune response to produce protein-specific antibodies that tag the protein for clearance. Passive vaccines, on the other hand, contain antibodies to the protein and therefore may not require an active immune response. Animal tests of both approaches have been promising: animals given the vaccines showed less buildup of the toxic protein and better performance on cognitive tests. But an early clinical trial of an active vaccine, sponsored by the Ireland-based Elan Corporation, was stopped in 2002 after four patients developed encephalitis, an inflammation of the brain. Later, autopsies of these patients' brains showed that despite the inflammation, the vaccine did clear the toxic protein from the brain. The challenge now is, are there other ways to use the immunotherapy approach to get the benefits without the adverse effects.

The NIA (National Institute on Aging) in Bethesda is sponsoring a new trial, announced at the Society for Neurosciences conference, of a different type of antibody therapy: intravenous immunoglobulin (IVIg), a blood product used to treat immune disorders. IVIg contains a mix of different antibodies, including one against amyloid. Because the product has already been used to treat thousands of people with immune disease, scientists say it is unlikely to cause the inflammatory problems seen in the first Elan trial. "We have a good understanding of the side effects and how to avoid them," Elan is also testing a passive vaccine, currently in clinical trials. Scientists still aren't sure exactly what part of the complex immune response is necessary to successfully clear amyloid protein or what part triggers excessive inflammation, as seen in the first Elan trial. But second-generation vaccines that create more-targeted responses might soon answer that question.

William Bowers and Howard Federoff of the University of Rochester Medical Center, in New York, are working on a gene-therapy vaccine delivered via a stripped-down herpes-simplex virus. Their vaccine carries both the code for the toxic protein and the code for genes involved in different aspects of the immune response. "We can shape the response and evoke different kinds of antibodies and different immune responses," says Bowers, who presented his findings at the neuroscience meeting. In addition, the researchers can use different genetic "promoters"--genetic sequences that control where and when a gene is expressed--to target the vaccine to specific cell types. By testing different varieties of the vaccines, they hope to tease apart each component's effects.

Penn researchers provide insights into how the immune system avoids attacking itself

Discovery may have applications in cancer biology and autoimmune disease

A finding by University of Pennsylvania School of Medicine researchers about how immune cells "decide" to become active or inactive may have applications in fighting cancerous tumors, autoimmune diseases, and organ transplant rejection.

Gary A. Koretzky describes, in the current issue of Nature Immunology, one way in which T cells may develop tolerance to host cells and proteins. Koretzky and colleagues found that small fatty acids called diacylglycerols (DAGs), and the enzymes that metabolize them, are critical players in the molecular pathway that leads to activity versus inactivity.

Immune cells called T lymphocytes recognize invaders in the body, such as viruses, bacteria, tumor cells, or allergens. Normally, T cells are activated by a complex series of signals that end with the destruction of the foreign substance. However, some T cells are not activated, in fact they are inactivated by a process called anergy or tolerance. This process helps prevent immune cells from attacking themselves and other normal cells and proteins.

"How T lymphocytes become activated or inactivated has been one of the major questions in the field of immunology," says senior author Koretzky. "Our discovery shows that DAGs are critical for T-cell activation so these cells can respond to foreign invaders. However, when DAGs are chemically modified by enzymes called diacylglycerol kinases, T cells become tolerant or unresponsive to foreign substances and to self."

The discovery was made by studying mice that had been engineered to lack diacylglycerol kinases (DGKs). Although T cells from these knock-out mice were normal in most respects the induction of tolerance was impaired. When DAGs could not be chemically altered because the DGKs were absent, the T cells were hyperreactive to foreign antigens and could not be made tolerant to host cells. Hyperreactivity was shown when purified T cells from DGK knockout mice were stimulated by antigen in a culture dish. The failure of the T cells to become tolerant was demonstrated in experiments where mice were treated with a toxin from staphylococcal bacteria that should have induced unresponsiveness. Instead, the T cells produced about five times more of an immunity factor than did cells from normal mice.

The hyperreactive state, if controlled, might be beneficial to the body under some circumstances; for example, some T cells might be made more effective at eliminating tumors. The research team is continuing to study DGK knock-out mice to see if they are more resistant to tumors. If the hyper-reactive T cells in these mice recognize the tumor cell as a foreign invader, then the tumor might be eliminated or reduced. Conversely, if the tolerant state could be induced in a controlled manner, it might benefit individuals with autoimmune disease or help prevent rejection of transplants.

Database for Disease

A genetic "roadmap" will help to find treatments for diseases, by looking at the signatures that drugs leave behind.

A newly developed genetic "roadmap" promises to streamline the drug discovery process. Called the Connectivity Map, this public database matches drug compounds with diseased cells and the processes occurring within them. "The reason it's so difficult to find those disease and drug connections is that the languages in which they are conventionally described are very different," says Justin Lamb, at the Broad Institute in Cambridge, MA. "A physician would describe a disease in terms of its physical symptoms, whereas a chemist would describe drug actions in terms of binding that chemical to a particular protein." The researchers want to bridge that gap using a common language: gene-expression signatures.

At any point in time, some genes in a cell are expressed, or "on", while others are not. And a cell's particular profile of activity is known as its gene-expression signature. When cells are exposed to a drug, that signature changes: some genes that were expressed are turned off and vice-versa. And different drugs leave different signatures. It is these signatures that the researchers used to build the Connectivity Map.Lamb and his colleagues conducted a pilot study on a select number of compounds and cell types to create the first installment of the map, reported recently in the journal Science. They chose 164 molecules that were biologically active, including drugs approved by the FDA and compounds commonly used as tools in the lab. They tested the molecules on four types of cancer cells--breast cancer, prostate cancer, leukemia, and melanoma--looking at how the compounds affected gene expression in those cells.

The researchers did the analysis using DNA microarrays. These tiny glass chips are coated with thousands of short sequences of DNA that refer to parts of the human genome that often differ between individuals. For a given drug or cell type, the chips produce a unique pattern corresponding to the particular genes expressed. For example, the hormone estrogen might cause breast cancer cells to express certain genes, but have no effect in a prostate cancer cell, and that difference would be visible on the DNA chip.The researchers then developed a computer program to compare the signatures to each other and rate the strength of the connections. The data from even this relatively small number of cell types and compounds, Lamb says, has yielded two new findings, described in papers in the journal Cancer Cell.

One compound, a plant-derivative called gedunin, was identified through a conventional screening method as interfering with the hormone androgen in prostate cancer cells, which is an important strategy in treating the disease. But the exact mechanism of how gedunin blocked the androgen signaling pathway wasn't clear. When the scientists searched the Connectivity Map for compounds that had similar activity to gedunin, though, they found matches to compounds that inhibit heat shock proteins and thus suppress androgen receptor activity. The other finding involved a specific type of leukemia that was resistant to traditional chemotherapy. A team led by Scott Armstrong, at Harvard Medical School and Children's Hospital, determined the signature of the drug-resistant cells, queried the Connectivity Map, and found a match to sirolimus, a drug currently used to prevent rejection after organ transplantation. When they tested the drug in the lab, the scientists found that it re-sensitized the leukemia cells to chemotherapy, reversing the drug resistance.

The team plans to expand the map to cover all 1,400 or so drugs approved by the FDA, an effort that should take between one and two years. "We wanted to make data that was broadly useful, so that requires a systematic approach to data generation," Lamb says. "And then if you can make that database accessible to the world in a way which is easy for the world to interact with it, that would solve a lot of problems for a lot of people."