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According to Stanford Cancer Center director Beverly S. Mitchell, MD, “Scientists have begun to decode cancer’s molecular secrets by studying the signaling pathways that regulate cellular activities such as proliferation and survival. Many of these pathways are radically altered in cancer cells as a result of genetic mutations.”

Groundbreaking technologies developed at Stanford and elsewhere enable researchers — many from once-disparate fields of study — to understand the genetic basis of cancer and the molecular machinery of the cancer cell. Cancer experts are even capable of exploiting the molecular mechanisms by which cancer develops to unearth potential, highly specific drug targets with reduced side effects.

The Promise of Molecular Therapeutics in Cancer

Personalized molecular therapeutics is the future of cancer treatment, and the future is here at Stanford.

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Dr. Sunwoo will collaborate with noted researcher Michael Clarke, MD, professor of medicine-oncology, whose laboratory team discovered breast cancer stem cells. It is anticipated that a synergy developed between the two labs will result in more targeted therapies for cancers of the head and neck and improved quality of care for patients after treatment.

Gifts from the Harold Simmons Foundation, Jill and John Freidenrich and a number of other generous friends of Stanford will help fund the work of John Sunwoo, MD, a cancer surgeon and scientist who specializes in the molecular biology and immunology of head and neck cancers.

Dr. Sunwoo’s laboratory, supported in part by the Stanford Cancer Center, will focus on head and neck cancer stem cell research, building on Stanford’s pre-eminent program in cancer stem cell science. Cancer stem cells are the “mother cells” from which cancerous tissue are formed.

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DEAR FRIENDS

We are tremendously excited about our newest program in the Stanford Cancer Center, the development of new approaches to cancer treatment. Stanford has always been in the forefront of technology development, but the application of new technologies – such as the use of carbon nanotubes to deliver drugs and new diagnostics like the Firefly instrument to detect whether a drug is hitting its target – is where we can have the most impact on cancer patients.

This issue of our newsletter is devoted to telling you about some of our current projects that we hope will lead to significant advances in the treatment of some cancers in the future. From the dawning of new ideas through their testing in the laboratory to the application in cancer clinical trials is a long road. It is our purpose to identify some of the most promising of these ideas and support them with “seed funds” that will allow them to be tested more rapidly.

As always, the most difficult part of the process is actually getting new treatments to patients in the clinic. We have a wonderful group of clinicians who meet weekly to discuss what we refer to as “early-phase” clinical trials that are primarily directed at cancers that have failed initial treatment. These trials are specifically designed to determine the best doses and frequency at which new treatments can be given.

One of the most exciting aspects of a molecularly based therapeutics program is the prospect of determining which patients are likely to respond to a drug in advance of its being given, so-called personalized medicine. As a result of basic scientific discovery, it is possible to identify the way in which some drugs activate signals that cause cancer cells to die. The ability to monitor the effects of drugs at a molecular level within the cancer itself will markedly reduce the numbers of patients that need to be treated to determine whether a treatment is effective.

We hope that you will share in our excitement about “molecular therapeutics” at Stanford.

Sincerely,

Beverly S. Mitchell, MD
Director

The Promise of Molecular Therapeutics in Cancer

CONTINUED FROM PAGE 1

groundbreaking molecular therapeutic research. The identification of promising molecular targets has led to the development of many exciting new drugs for which an antitumor mechanism of action has been clearly delineated,” Dr. Mitchell said.

Potential therapies currently under development at Stanford include drugs that cut off a tumor’s blood supply (angiogenesis inhibitors) or trick a cancer cell into “committing suicide.” These and other compounds are designed to interfere with signaling pathways that control growth and spread of tumors.

Amato Giaccia, PhD, professor of radiation oncology and director of radiation biology at the Stanford University School of Medicine, discovered that connective tissue growth factor (CTGF), a “signaling chemical” that regulates cell proliferation, migration and adhesion, is overproduced in pancreatic cancer. Subsequent studies have detected elevated CTGF levels in late-stage breast cancer, glioblastoma and sarcomas.

Giaccia’s laboratory group has found strong evidence linking CTGF to tumor progression, including tumor cell survival and metastasis (cancer spread).

Based on the results of these studies, Giaccia developed a monoclonal antibody named FG-3019 that has been shown to block tumor growth and spread in animal models of pancreatic cancer. Cancer Center researchers recently began a clinical trial to assess the use of FG-3019 in patients with advanced local and metastatic pancreatic cancer.

LEADERSHIP IN HEDGEHOG PROTEIN SIGNALING RESEARCH

Anthony Oro, MD, PhD, is one of a growing phalanx of Stanford researchers investigating the role of the Sonic hedgehog (Shh) signaling pathway in cancer development – in his case, hair stem cells and basal cell carcinoma, a cancer originating in the hair follicle stem cell. Sonic hedgehog is one of three known types of Hedgehog proteins that play a critical role in embryonic development.

The Hedgehog gene was named for the pointy projections that occur on furry embryos missing the gene. Work on this developmental pathway led to a 1995 Nobel Prize.

Oro, an associate professor of dermatology who works in both the clinic and the laboratory, has found that the skin and scalp provide an accessible means to study the role of stem cells in the regeneration of an organ, because many organs need the same growth signals that hair follicles use to grow.

“Achieving new advances in understanding how hair follicle stem cells are regulated, studies of hair cycling will likely contribute greatly to studies of how normal and cancer stem cells are regulated,” Oro said.

Oro’s laboratory team also has found that a number of the same pathways involved in basal cell carcinomas also play a role in prostate, pancreatic and lung cell development as well as in the cancers these organs develop.

“Because basal cell carcinomas are dependent on Hedgehog signaling, they are a great model to study so we can develop therapeutics for other, more lethal Hedgehog-dependent cancers, such as lymphoma and prostate and pancreatic cancer,” Oro said.

Oro’s interest in the uniquely named gene began when he was a post-doctoral scholar in the lab of Matthew Scott, PhD, professor of developmental biology and genetics.

In the 1990s, Scott found that a mutation in the human Patched gene is linked with Gorlin’s syndrome, a rare, inherited condition manifesting in skin cancers and birth defects. He then showed that Patched mutations also occur in basal cell carcinoma, the most common skin cancer, and in medulloblastoma, the most common malignant childhood brain cancer.

Cover story Continued
Scott’s laboratory group then found that Shh signaling, which controls production of the Patched protein, stimulates growth of the cerebellum. Thus, Scott demonstrated, the cerebellum cancer medulloblastoma could be understood as resulting from excessive Shh signaling.

In subsequent experiments, Oro showed that overexpression of the Shh protein in epithelium (tissue that lines both skin and internal cavities in the body) can give similar tumors to basal cell carcinomas. Many researchers have subsequently found the overexpressed Shh gene in a number of other human cancers.

Phillip Beachy, PhD, professor of development biology – along with Scott – is one of the foremost experts in understanding developmental biology – along with Scott – is one of the foremost experts in understanding developmental biology. Beachy’s earlier research found that the Hedgehog protein reduced growth of prostate cancer cells. Several pharmaceutical companies are now exploring the use of such drugs to treat human cancers.

Beachy also has discovered that, in some circumstances, the Hedgehog protein causes a number of cancer cells, such as those in prostate tumors, to become highly aggressive. In landmark experiments, Beachy showed that inhibiting the Hedgehog protein reduced growth of prostate cancer cells, as well as cancers of the lung and gastrointestinal tract.

Another of Stanford’s leaders in the study of the Hedgehog protein is James Chen, PhD, assistant professor of chemical and systems biology. Chen recently received a prestigious Pioneer Award from the National Institutes of Health for his research. The award will provide $2.5 million in research funding over the next five years.

Some of Beachy’s colleagues, including Scott, had studied mutations in genes coding for proteins that interact with Hedgehog; the researchers found that people with those mutations were at greater risk for several cancers.

Beachy showed that a drug known to inhibit the Hedgehog signaling pathway reduced the size of brain tumor cells in a lab dish and in mice. In later experiments, that same drug effectively blocked the growth of cells from cancers of the lung, gastrointestinal tract and prostate. Several pharmaceutical companies are now exploring the use of such drugs to treat human cancers.

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Among Chen’s research interests is how the Hedgehog pathway regulates tissue patterning and cancer development in vertebrates. He uses both genetic and chemical techniques to study Hedgehog signaling in cell migration processes and tumor formation in zebrafish, a model for human cancer. Chen’s lab group is also developing new genetic and chemical technologies to evaluate the role of individual Hedgehog pathway components in embryonic development.

Chen, with David Solow-Cordero, PhD, also directs the Cancer Center’s High-throughput Bioscience Center, which provides the means to identify new therapeutic targets.

The Promise of Molecular Therapeutics in Cancer

CONTINUED FROM PAGE 3

Catalyzing Interdisciplinary Research

Dr. Mitchell emphasizes that “translational research is not only about therapeutics.”

“Scientists in the Cancer Center’s Molecular Therapeutics Program are also developing new methods of identifying cancer biomarkers, new methods of diagnosis, new methods of patient follow-up and new methods of prevention,” she added.

“The role of the Cancer Center is to facilitate interdisciplinary research, and the Molecular Therapeutics Program is one of the most exciting outcomes of that. We have been able to catalyze these interactions through the leadership of Dr. Dean Felsher and Dr. Brandy Sikic,” Dr. Mitchell said.

Dean Felsher, MD, PhD, associate professor of medicine-oncology, and other researchers have studied together how many oncogenes like myc can cause cancer in normal human and mouse cells in tissue culture or in specific tissues.

To date, his group has developed transgenic mouse models of lymphoma, leukemia, bone cancer, lung cancer and liver cancer, and many other pre-clinical models of human diseases.

“Our mouse models of cancer enable researchers to study the molecular and cellular basis of cancer development in its full complexity, through multiple stages, in a normal tissue environment and in an organism,” Felsher explained.

“They provide the possibility to examine chemoprevention and therapeutic strategies in such contexts, and can define which correct dosages and avoiding toxic side effects arising from faulty signaling pathways.”

Enter the Era of Personalized Cancer Treatment

Ultimately, the goal of the Molecular Therapeutics Program is to “personalize” medicine. A possible scenario: After a patient is diagnosed, a tumor sample will be analyzed for genetic mutations known to be critical in the disease. Patients will then be matched with targeted treatment based on the genetic makeup of their tumor.

This process – called pharmacogenetics – will also assist cancer specialists in determining the best treatments for each patient.

Cover Story Continued

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YW NAMED STANFORD CANCER CENTER’S FIRST NADIA VAN CAMP FELLOW

Furong Yu, PhD, has joined the laboratory of Lawrence Recht, MD, as the first Nadia Van Camp Fellow. Yu will spearhead a project designed to repair injuries to the central nervous system produced by both cancers and their treatments.

Yu received her PhD from the Chinese National Academy of Science, after which she spent five years at the University of Massachusetts Medical School studying the molecular mechanisms of Parkinson’s disease.

She brings an extensive experience in molecular biology to this new endeavor, where she will focus primarily on characterizing the mechanisms involved in the differentiation of embryonic stem cells into cortical neurons after transplantation.

The eventual goal of this project is to functionally repair brain and spinal cord injuries resulting from tumor or treatment by transplanting cells that will actually replace those that have been injured or are dead. This important work will be made possible by the generous support of Nadia and Peter Van Camp, to whom the Cancer Center is most grateful.
Stanford’s Firefly 3000 is one of only three in the world.

The nanoscale automated instrument is capable of performing almost 100 protein assays of extremely small samples of stem cells, rare biological materials and clinical specimens in hours rather than days.

The Promise of Molecular Therapeutics in Cancer
CONTINUED FROM PAGE 5
genesis have oncogenic or tumor suppressor activity,” he said.

Dr. Felsher is also principal investigator on a National Institutes of Health-sponsored Major Equipment Grant to assess the use of a Firefly 3000, a nanoscale automated instrument that is capable of performing almost 100 protein assays of extremely small (as little as 4 nanoliters of material or as few as 25 cells) samples of stem cells, rare biological materials and clinical specimens in hours rather than days. Stanford’s Firefly 3000 is one of only three in the world.

“I am excited about the opportunity to bring state-of-the-art science to patients in the clinic. I believe this technology will be very useful in helping us develop new targeted therapeutics,” Felsher said.

Dr. Sikic and his lab group study the mechanism of multi-drug resistance coded by the MDR1 gene and its product, P-glycoprotein (Pgp), a trans-membrane “pump” that is responsible for drug efflux (flow outward) and resistance to many cancer chemotherapeutics. MDR1 is believed to be significant in the clinical response to anticancer therapies, for example, paclitaxel (Taxol), which is widely used to treat breast and ovarian cancers and lymphoma.

The Sikic group also uses microarray technology to profile gene expression of ovarian cancer, acute leukemias, germ cell cancers and brain tumors. With Alejandro Sweet-Cordero, MD, assistant professor of pediatrics, the Sikic lab screens cancer specimens from patients at diagnosis and after chemotherapy to identify gene expression patterns that may further understanding of how cancer develops as well as to determine response to treatment.

In the clinic, Dr. Sikic is investigating the prognostic significance of resistance gene expression in cancers, and the pharmacokinetic consequences of MDR modulation, development of new modulators of drug resistance and clinical trials using monoclonal antibodies, small molecule inhibitors and various cancer chemotherapeutic regimens.

He also leads a group of clinical investigators, nurses and research coordinators who are carrying out early – Phase I and II – clinical studies of new anticancer therapies. These studies are a natural extension of the discovery research within the Molecular Therapeutics Program, which Dr. Sikic co-leads with Dr. Felsher. Whenever possible, new molecular markers that reflect whether a drug is hitting its target are incorporated into these trials. This approach makes it possible to finish the studies before any side effects from the drug become evident.

Through the program, patients and referring physicians have access to the newest anticancer treatments either developed at Stanford or supported by the National Cancer Institute and pharmaceutical companies. The team meets weekly to discuss current and pending protocols and to review all patients in study trials.

Leukemia & Lymphoma Society grant will fund studies of myelodysplastic syndromes.

The Leukemia & Lymphoma Society (LLS) has awarded a new $6.25 million, five-year Marshall A. Lichtman Specialized Center of Research (SCOR) grant to a group of noted Stanford Cancer Center researchers. The interdisciplinary study consists of five projects focusing on “Molecular and Cellular Characterization of Myelodysplastic (MDS) Syndromes,” and is headed by Beverly S. Mitchell, MD, George E. Becker Professor of Medicine at Stanford University and director of the Stanford Cancer Center.

The proposal integrates a strong clinical program in MDS with laboratory expertise in stem cell biology and the use of mouse models with Stanford’s extraordinary technical capabilities.

Myelodysplastic syndromes are a collection of related disorders in which damaged bone marrow cells can’t mature into functional blood cells. The defect occurs at the level of blood-forming stem cells. The disorders frequently progress to acute myelogenous leukemia (AML).

There are few data on the molecular characterization of the aberrant stem cell population and on how MDS develops, Dr. Mitchell explained.

“New treatments for MDS based on a more extensive understanding of the cellular and molecular anomalies underlying defective hematopoietic stem and progenitor cells function are urgently needed,” she said.

The grant proposal was based on the extensive clinical database and accompanying bone marrow samples that have been collected by Peter Greenberg, MD, emeritus professor of medicine-hematology and director of the Stanford MDS Center, and depends strongly on the capabilities of other Stanford investigators in hematopoietic stem cell biology. His research group includes Jason Gotlib, MD, MPH, assistant professor of medicine-hematology, a clinical investigator who specializes in the treatment MDS and myeloproliferative syndromes.

Other SCOR researchers include Irving Weissman, MD, professor of pathology and of developmental biology and director of the Stanford Institute for Stem Cell Biology and Regenerative Medicine, a pioneer in the field of normal and leukemic hematopoietic stem cells.

Dr. Weissman’s group recently identified new molecular markers that can help identify the MDS stem cells that have progressed to AML, and are working to develop therapies to target these abnormal MDS stem cells.

Also in this group is Steven Arlandi, MD, PhD, associate professor of medicine-hematology, an expert on telomerase, a protein that is expressed in stem cells and progenitor cells and is overproduced in the vast majority of human cancers.

Dr. Mitchell’s group is focusing on the role of an important protein that shuttles around within the cell to control production of RNA. Michael Cleary, MD, professor of pathology and of pediatrics, heads another of the projects. Cleary’s research focuses on developmental pathways that regulate hematopoietic cell growth and differentiation and are disrupted as normal cells become cancerous, particularly in leukemias and lymphomas.

The Sikic group also uses microarray technology to profile gene expression of ovarian cancer, acute leukemias, germ cell cancers and brain tumors. With Alejandro Sweet-Cordero, MD, assistant professor of pediatrics, the Sikic lab screens cancer specimens from patients at diagnosis and after chemotherapy to identify gene expression patterns that may further understanding of how cancer develops as well as to determine response to treatment.

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An Expansive, Inclusive Approach to Cancer Research

Groundbreaking technologies developed at Stanford and elsewhere enable researchers — many from once-disparate fields of study — to understand the genetic basis of cancer and the molecular machinery of the cancer cell.

The Cancer Center’s Molecular Therapeutics Program provides a cross-disciplinary forum for investigators in clinical and basic sciences to interact, foster resources to develop and analyze new therapies and validate these therapies in early clinical studies. The program stimulates researchers in chemistry, molecular imaging, bioengineering and systems biology to interact with scientists working in the areas of cancer stem cells, cancer biology, cancer imaging and lymphoma.

The program encourages investigator-initiated studies. For example, oncologist Alice Fan, MD, is principal investigator on a Phase II study “interrogating” the use of statins to treat certain non-Hodgkin’s lymphomas. Her collaborators on this project include Dean Felsher, MD, PhD, associate professor of medicine-oncology, pathology and molecular imaging; Steven Coutre, MD, associate professor of medicine-hematology; Yasodha Nath纳斯am, MD, PhD, associate professor of pathology; and Christina Kong, MD, associate professor of pathology.

Fan is also interested in using molecular methods of “delivering” standard cancer chemotherapy (such as doxorubicin) to focus the drug’s effects on the tumor and not normal surrounding tissues. Doxorubicin is a mainstay of lymphoma therapies and breast and other solid tumors. But it is also highly toxic to the heart.

Fan and Felsher are collaborating with John Goldsbie, MD, MPH, assistant professor of medicine-hematology, to develop the novel Firefly 3000 nanoscale protein detection technology for measurement of changes in cells from patients treated for leukemia.

Fan is also teaming with Felsher and nanotechnology expert Hongjie Dai, PhD, professor of chemistry, to assess the effectiveness of applying doxorubicin to microscopic carbon nanotubes and injecting the drug into the bloodstream of mice with tumors (see p. 10).

Beverly Mitchell, MD, and her lab group are developing new therapies for hematologic malignancies (cancers of the blood and lymph nodes). She is working with hematologist Bruno Medeiros, MD, on a Phase II study of a novel molecule called AS1411, both alone or in combination with cyclophosphamide, a standard therapy for primary refractory or relapsed acute myelogenous leukemia.

Dr. Mitchell is also principal investigator on a highly collaborative multimillion dollar SCOR grant from the Leukemia & Lymphoma Society to study stem cells in myelodysplastic syndromes (see p. 7).

Chemists Paul Wender, PhD, and Dai are working with Cancer Center scientists and clinicians to create new, natural cancer drugs and generate nanotechnology therapeutics and diagnostics.

Wender’s research combines chemistry, biology and medicine. Specifically, his lab designs novel drugs and molecularly manipulates existing drugs to boost their activity and reduce unwanted side effects. The latter is accomplished by developing systems that enable or enhance passage of drugs and diagnostic probe molecules across biological barriers (for example, skin and cell membranes), thereby making the drugs and probes more effective.

One of Wender’s projects focuses on synthesizing a form of bryostatin, a marine natural product that appears to have a wide range of therapeutic uses, but is difficult and expensive to produce.

Wender’s lab has found a way to construct synthetic bryostatin molecules that mimic the natural drug’s actions, including its ability to trigger apoptosis (“programmed cell suicide”) in cancer cells without destroying normal cells.

His group also has developed a new molecular strategy for overcoming cancer cell resistance to paclitaxel (Taxol), a cancer drug used to treat breast, ovarian and several other solid tumors. The strategy enables the medication to enter drug-resistant cancer cells without being detected by the membrane pumps that drive these cancer cells to expel drugs.

In her lab in the department of bioengineering, Jennifer Cochran, PhD, melds approaches in chemistry, engineering and biophysics to study complex biological systems.

Cochran uses rational design and combinatorial chemistry to develop tumor-targeting proteins as therapeutics as well as imaging agents that will allow researchers to diagnose tumors and also follow disease progression. Her lab creates molecules called peptides that, for example, block the supply of blood that a tumor needs to grow and metastasize.

The peptides are engineered through computational modeling and a process called “directed evolution.” With this method, Cochran combs a natural process of producing and replicating proteins while introducing small errors that result in a diversity of traits. Then her lab group generates millions of different mutant versions of these molecules and sorts through them to find peptides that have the properties they are targeting.

With researcher Sanju Sari Gambhir, MD, PhD, who directs the Molecular Imaging Program at Stanford, Cochran also has developed a way to “tag” tumors with probes that show up very brightly in diagnostic imaging systems. By precisely linking tumors to “contrast agents,” she hopes to develop a non-invasive system that will detect tumors early, before they’ve spread.

Cochran recently was awarded a V Foundation Scholar Award of $100,000 over two years to support research to pioneer a new class of peptides as alternatives to monoclonal antibodies for targeted cancer therapy.

Specifically, the Cochran lab is developing cystine knot peptides as a new class of tumor-targeting agents for uses in cancer diagnosis and therapy. Cystine knot peptides possess several desirable qualities for use as drugs or diagnostic agents, including the fact they are non-toxic; however, they do not naturally bind to tumor-specific receptors. Cochran and her colleagues are using molecular design and engineering to custom-tailor the peptides’ binding specificities to target receptors that are overproduced in human cancer.

“This new platform has the potential to be applied to virtually any target of interest across a spectrum of cancers. It is our hope that these agents will enable physicians to identify patients who are appropriate candidates for molecular therapies while also using them to follow tumor responses,” said Cancer Center director Beverly Mitchell, MD.

Pictured (left page): Alice Fan, MD (Alan Yatagai Photography). (Above) Jennifer Cochran, PhD, assistant professor of bioengineering.
From Chemistry to Cancer Therapy

Stanford researchers are getting down to nanometer scale to speed the diagnosis and treatment of cancer – a point where they may eventually be able to tailor therapies to a patient’s individual cancer.

A nanometer is one billionth of a meter – about 1/80,000 the width of a human hair. Because of their size, nanoscale devices are readily able to interact with molecules on the surface and inside cells. And since cancer occurs at the nanoscale level and inside cells, nanotechnology offers a new range of subatomic tools to provide cancer researchers innovative ways to diagnose and treat cancer.

Stanford scientists are developing nanodevices to detect cancer at its earliest stages, pinpoint its location within the body, administer anticancer drugs specifically to cancerous stages, pinpoint its location within the body, and treat cancer.

Researchers innovative ways to diagnose and treat cancer:

1. Because of their size, nanoscale devices are readily able to interact with molecules on the surface and inside cells.
2. And since cancer occurs at the nanoscale level and inside cells, nanotechnology offers a new range of subatomic tools to provide cancer researchers innovative ways to diagnose and treat cancer.
3. Stanford scientists are developing nanodevices to detect cancer at its earliest stages, pinpoint its location within the body, administer anticancer drugs specifically to cancerous stages, pinpoint its location within the body, and treat cancer.
4. In recent experiments, Dai and graduate student Zhuang Liu delivered carbon nanotubes loaded with an anticancer drug that targeted breast cancer cells while leaving normal tissues unharmed.
5. First they coated the nanotubes with a molecule named polyethylene glycol (PEG), which has three branches on one end, then attached molecules of a form of the anti-breast-cancer drug paclitaxel to each branch. Each of the 100 nanometer-long nanotubes carried about 150 drug molecules (see illustration, page 11).
6. Thanks to their size, the nanotubes were able to pass through the porous blood vessels of a tumor but could not penetrate healthy blood vessels.
7. Dai explained that the branched PEG is stable in the bloodstream for an extended period, which enables the nanotubes to home in on and treat a tumor before being excreted from the body.
8. The researchers found that breast tumors treated with the paclitaxel-laced nanotubes were less than half the size of the tumors treated by the second-most effective treatment, the standard form of paclitaxel known as Taxol. The tumors treated with the carbon nanotube-delivered therapy had a higher percentage of cell death and a smaller percentage of proliferating cells.
9. Dai and Liu estimated that 10 times more of the nanotube-delivered paclitaxel was absorbed by breast cancer cells than the amount of Taxol.
10. “This uptake means that when linked to new delivery methods, smaller doses of drugs could be used to achieve the same effects as other treatments, with fewer side effects,” Dai said.
11. Dai and his colleagues have been exploiting the physical properties of carbon nanotubes for several years. For example, they have used carbon nanotubes exposed to a laser beam to essentially “fry” cancer cells without harming surrounding, healthy tissues.
12. “An interesting property of carbon nanotubes is that they absorb near-infrared light waves, which are slightly longer than visible rays of light and pass harmlessly through our cells,” Dai explained.
13. But if a carbon nanotube is zapped with near-infrared light, electrons in the nanotube get excited and start to release excess energy – in this case, heat.
14. Dai’s group is also collaborating with oncologists Dean Felsher, MD, PhD, and Alice Fan, MD, on a clinical study to assess the efficacy of carbon nanotubes loaded with the anticancer drug doxorubicin for the treatment of lymphoma.

Polyethylene glycol (PEG) (Illustration courtesy of Dai laboratory.)

Pictured above: Hongjie Dai, PhD, and graduate student Zhuang Liu (Alan Yatagai Photography).

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The sixth researcher is Dean Felsher, MD, PhD, associate professor of medicine-oncology, pathology and molecular imaging, who is well known for his studies on the role of the myc protein, which is essential to the development and maintenance of hematopoietic tumors. Dr. Felsher will supervise the genetic studies of the MDS stem cells.

The SCOR program is the Leukemia & Lymphoma Society’s largest research grant, with total funding of $178.25 million since its inception in 2000.

The program brings together teams of researchers representing different disciplines in collaborative efforts to discover new approaches to treat patients with leukemia, lymphoma and myeloma. Awards go to groups that best demonstrate outstanding scientific promise facilitated by the synergy that will occur from their combined efforts.

“The sixth researcher is Dean Felsher, MD, PhD, associate professor of medicine oncology, pathology and molecular imaging, who is well known for his studies on the role of the myc protein, which is essential to the development and maintenance of hematopoietic tumors. Dr. Felsher will supervise the genetic studies of the MDS stem cells.”

The SCoR program is the Leukemia & Lymphoma Society’s largest research grant, with total funding of $178.25 million since its inception in 2000.

The program brings together teams of researchers representing different disciplines in collaborative efforts to discover new approaches to treat patients with leukemia, lymphoma and myeloma. Awards go to groups that best demonstrate outstanding scientific promise facilitated by the synergy that will occur from their combined efforts.

“Dr. Mitchell and the team of Stanford investigators are undertaking cutting-edge research to reach a better understanding of the root causes of MDS,” said Louis DeGennaro, PhD, Chief Scientific Officer of the Leukemia & Lymphoma Society.

Pictured above: Peter Greenberg, MD.