



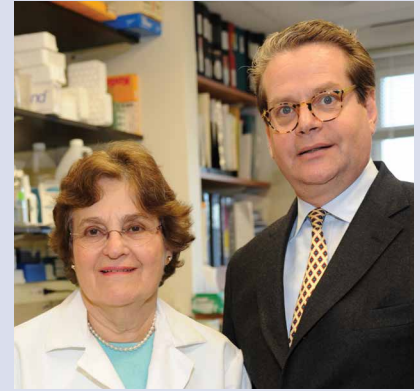
RESEARCH for
A **cure**
National Foundation
for Cancer Research
2013 Progress Report

NFCR MISSION STATEMENT

The National Foundation for Cancer Research (NFCR) was founded in 1973 to support cancer research and public education relating to prevention, early diagnosis, better treatments and ultimately, a cure for cancer. NFCR promotes and facilitates collaboration among scientists to accelerate the pace of discovery from bench to bedside. NFCR is about Research for a Cure — cures for *all* types of cancer.

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RESEARCH CURES CANCER

Dear Friends and Donors,

NFCR is about research to cure cancer.

NFCR's first forty years have been marked by significant scientific discoveries, medical advancements, and changes in technology that have revolutionized the way doctors treat all types of cancer today. But the paths toward discovery — the development of new anti-cancer treatments — are long and complex. Discoveries alone aren't enough, and NFCR is committed to fostering scientific creativity so we can translate promising cancer discoveries from laboratory bench to patient bedside.

NFCR funds research to cure cancer, and while our focus has always been to provide leading scientists the “adventure” funding to discover, we recognize that NFCR must also help advance these discoveries to the next level in order to develop new approaches to preventing, diagnosing, and treating cancer.

In that spirit, NFCR has marshalled leading scientists from around the world to build new, innovative research platforms for global collaboration that will accelerate the path to curing cancer. Only by sustained funding and by engaging partners and other institutions to work toward a strategy of better preventing, diagnosing, and treating cancer — only by working together — will research cure cancer.

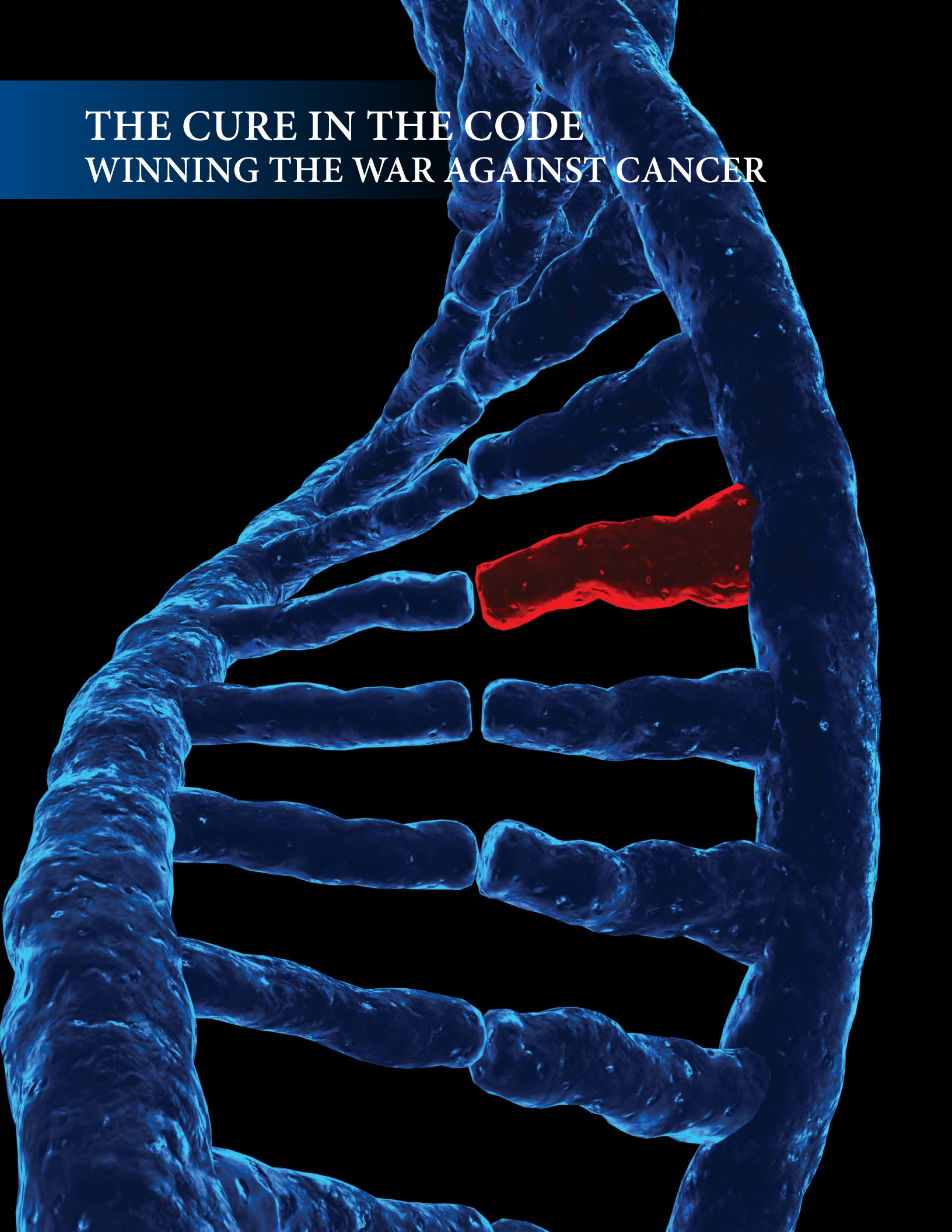
And that is what NFCR means by **Research** for a **Cure**.

Thank you and sincerely,

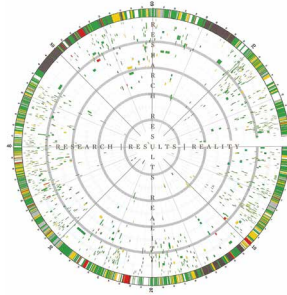
A handwritten signature in blue ink that reads "Franklin Salisbury, Jr.".

Franklin C. Salisbury, Jr.
President

THE CURE IN THE CODE
WINNING THE WAR AGAINST CANCER



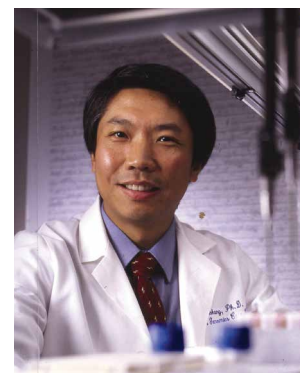
Catalyst for Discovery: NCFR Accelerates Pace of Cancer Research



A new era is dawning in the diagnosis and treatment of cancer. The black box that was the cancer cell has been opened. With grassroots support of millions of Americans, NCFR researchers have pioneered the redefinition of cancer as a genomic disease, transforming medicine and bringing hope to patients worldwide.

Cancer is often caused by errors in genes, usually multiple errors in those that control some aspect of cell growth and division. Though some of these errors may be inherited, most are acquired. Sunlight, cigarette smoke, toxins and aging itself help these errors accumulate. NCFR scientists worldwide are hard at work on cancer therapies that target the products of these broken genes — the very genes that make a cell cancerous. Unlike traditional chemotherapies and radiation, these new treatments aim to halt the processes that make a normal cell turn into a cancer cell in the first place — an approach with less collateral damage on healthy cells.

We are at a turning point in medicine. For decades the disease model was confined to what doctors could observe in tissues and organs. Now, being able to determine which genes and proteins are driving the cancer process in an individual patient, we can define more precise targets for cancer treatments. NCFR research is helping build a future where cancer is detected early so we can intervene before the cancer is visible under the microscope. This is molecular medicine — these new approaches to treating cancer are less miss and a lot more hit.



Real Teams Working Together Against Cancer

MOLECULAR SAWDUST HOLDS THE KEY

The Power of microRNA



Over the last two decades, our understanding of cancer at the cellular and molecular levels has increased exponentially. This new era of genomics is the most promising time ever in cancer research, but it also presents great challenges. We have more data and deeper insights than ever before, but to make sense of it requires not only talent and dedication, but also collaboration. No one person working alone can hope to unravel the mysteries of cancer, but dedicated scientists working together and building on each other's discoveries can make remarkable contributions to the improvement of patient care and **Research** for a **Cure**.

That is the story and the mission of NFCR, fostering scientific research from the lab bench to the bedside. It is also the story of NFCR Fellow Curt Civin, a pediatric oncologist specializing in leukemia research at the University of Maryland School of Medicine in Baltimore.

Dr. Civin revolutionized the field of leukemia research with his breakthrough discovery of CD34, the first — and still the best — marker of hematopoietic stem-progenitor cells ever found. His

subsequent isolation of the hematopoietic (blood-forming) stem cell opened entirely new directions in bone marrow transplant research, and led directly to cures for patients. The CD34+ transplantation technology created by the team of scientists in Dr. Civin's laboratory has been widely applied, and thousands of patients owe their lives to this new approach to treating cancer.

Since the discovery of CD34 — and, in part, because of it — the relative 5-year survival rates for all types of leukemia have increased dramatically. Despite these advances, there is still so much that needs to be done. Although patients with acute myeloid leukemia (AML) have shared the improvement in outcomes, AML remains the deadliest form of leukemia.

One of the most important molecules in the body for protection against cancer is a gene called p53. Research into the molecular basis of cancer shows that AML patients with the worst response to current therapies are those whose cancers have mutations in p53, causing it to no longer function. When p53 no longer functions, what can be done to help these patients?

The answer may lie in a recently discovered class of cellular molecules called microRNAs. These tiny bits of RNA — previously thought to be the “molecular sawdust” left over from the breakdown of much larger RNA molecules — have been discovered to have vital functions of their own. MicroRNAs profoundly influence which of each cell’s genes are made into proteins. A single microRNA may control the amounts of hundreds of different proteins in our cells.

If expressing individual genes can be likened to turning on light switches one at a time, microRNAs can be thought of as flipping circuit breakers, switching on entire buildings at once. In the cancer cell, entire pathways or sets of pathways — involved in cell growth or division, for example — can be activated by a single microRNA. Conversely, a single microRNA may be able to shut down a cancer cell.

Dr. Civin’s NCFR-funded research is focused on miR-34, a quintessential tumor-suppressor microRNA. When a mutant cell contains enough miR-34, a molecular self-destruct sequence is initiated that destroys the cell in a process called apoptosis.

It has been discovered that miR-34 is absent, or present at only extremely low levels, in most leukemia cells. It is as if leukemia cells have managed to cut the power to the apoptosis circuit.

Dr. Civin’s exciting new research strategy is to restore miR-34 to the leukemia cells and reset the circuit breaker for tumor suppression. This strategy aims to activate the leukemia cells’ own natural machinery to induce their self destruction through apoptosis. But miR-34 is a cancer molecule that has never been targeted previously in humans. How can we target miR-34 in leukemia?

REPURPOSING MALARIA TREATMENT FOR CANCER PATIENTS

Scouring the libraries and databases of existing clinical drugs, Dr. Civin’s team identified a set of drugs that were able to increase the amount of miR-34 in target cells. The most promising of these drugs came from an unexpected source.

Nearly two thousand years ago, Chinese herbalists described the use of the *Artemisia annua* plant as a remedy for malaria. The active ingredients isolated from this plant, known as Artemisinins, are now a standard treatment for patients with severe malaria, and they are routinely administered with no major toxicity. Dr. Civin’s research revealed that, in addition to their anti-malarial effects, these Artemisinins increased the levels of miR-34 in cells.

Would they be able to do this in leukemia cells as well?

Back in the laboratory, research conducted by Dr. Civin discovered that Artemisinins can indeed increase the levels of miR-34 in leukemia cells and inhibit their growth. Even more promising is the discovery that they can also achieve this result in leukemia cells with mutant p53 — giving hope to AML patients with the worst prognosis.

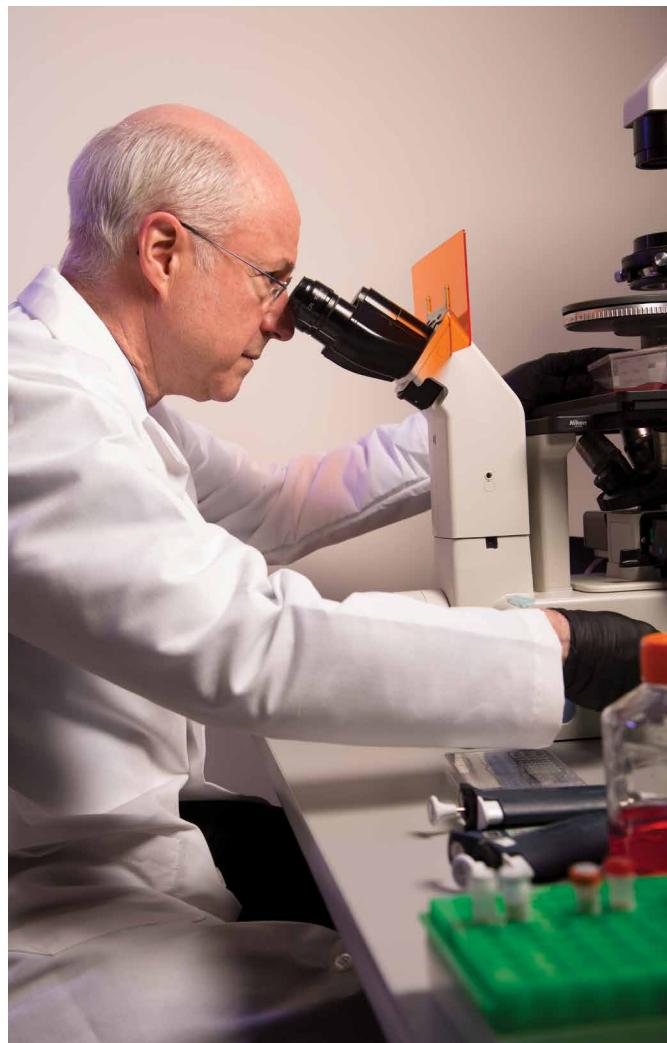
This could be a breakthrough discovery. Artemisinins are the first class of drugs that up-regulate miR-34 in a way that is both

independent of p53 and safe for clinical use. Clinical trials testing the efficacy of Artemisinins in AML patients will be underway in the very near future, bringing this new AML treatment into the clinic.

This is an entirely new approach to treating AML. Researchers focused on malaria are not thinking about leukemia; researchers focused on leukemia are not thinking about malaria — but with a willingness to share information and think critically across disciplines, scientists can recognize the potential of different treatments. In this case, they will repurpose a malaria drug as a novel treatment for cancer — a new treatment that provides new hope and might lead to cures. Dr. Civin’s innovative research offers promise that this can be accomplished.

What future might Dr. Civin’s research hold? Could this drug or this approach be applicable to other types of cancer? Are there other microRNAs that are critical for cancer? Might there be other cancer drugs — safe, effective, and readily available — that are waiting for scientists like Dr. Civin to repurpose them?

With your support and by working together, we will answer these questions. From discovery research to new treatments, Dr. Civin’s work has both exemplified and advanced the mission of NCFR: **Research for a Cure.**



TARGETING CANCER

Research Highlights

When it comes to finding a cure for cancer, the National Foundation for Cancer Research is hard at work. For 40 years, since 1973, NFCR has committed over \$320 million to funding discovery-oriented research and public education.

The path from a promising discovery to an effective cancer treatment often takes a decade or more. But from that process — of fits and starts, progress and setbacks, and finally more progress — grow the insights and advances that change the course of medicine.

The 21st century is seeing new approaches to treating cancer, driven by breakthroughs in basic science and emerging “omics” technologies; and NFCR-funded scientists are pioneering the redefinition of cancer as a genomic disease, driven by abnormal genes and proteins.

With the grassroots support of millions of Americans, the black box that was the cancer cell has been opened and NFCR-funded scientists are at work on new approaches to preventing, diagnosing, and targeting the very genes and genetic pathways that make a cell cancerous. NFCR is transforming medicine so that real hope for a cure is now within sight.

Every day NFCR-funded scientists are reporting breakthroughs and setting the stage for new approaches to treating cancer that will eliminate the suffering and death from cancer. Much more needs to be done, and until there is a cure, we will not be satisfied—too many lives are at stake.

Join us in our **Research** for a **Cure**.

Research for a Cure



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EARLY CANCER DETECTION AND MONITORING

Early detection and monitoring is critical for effective cancer treatments. The molecular differences that make cancer cells lethal also provide the clues for their detection, identification and visualization. Our scientists are developing new methods in molecular imaging technologies which are highly sensitive cancer detectors. A revolutionary technology recently developed by NFCR-funded researchers makes it possible to detect early-stage cancer through a simple blood test. We envision that the development of these new tools for early cancer detection and continuous monitoring during and after cancer treatment will significantly improve clinical management of various cancers and ultimately, improve patient survival.



CTC CHIP DETECTS CANCER IN BLOOD

Daniel A. Haber, M.D., Ph.D., Massachusetts General Hospital, has developed a revolutionary way — so sensitive that it can detect and capture one circulating tumor cell (CTC) out of a billion normal cells in the blood. CTCs may constitute seeds for metastases and may indicate the spread of a tumor. This technology may provide doctors with a new tool to rapidly detect invasive cancers at early stages by using an easily administered blood test. Licensed by *Johnson & Johnson*, the CTC Chip also allows characterization of genetic features of tumor cells, so doctors can identify and prescribe targeted anticancer treatments early on, before the cancer metastasizes. The CTC Chip could also enable doctors to monitor the effectiveness of their patient's treatment and make any necessary changes, increasing the positive effect of all cancer therapies. Dr. Haber's most recent work involves developing novel tumor models for various cancers, which would allow them to perform detailed measurements of gene patterns and compare gene profile in tumor cells that have entered the blood (CTCs) with that in primary tumor cells. This new approach will help scientists identify the genes that regulate cancer invasion and find new ways to suppress the ability of cancer cells to leave the primary tumor and spread to distant organs.



MOLECULAR IMAGING

James Basilion, Ph.D., NFCR Center for Molecular Imaging, Case Western Reserve University, is building a new technology platform that uses molecular imaging for early detection and improved treatment of cancer. Scientists in this center are utilizing an entirely new technique that permits the simultaneous imaging of multiple molecular markers. This makes it possible to identify cancer at a very early and more treatable stage, significantly improving patients' chances of survival.

Technologies developed at the Center can also help surgeons determine tumor margins during surgery. This could be particularly helpful for the treatment of glioblastoma multiforme (GBM), the most aggressive brain tumor in adults that often infiltrates into surrounding brain tissues. Dr. Basilion's new technique promises to facilitate molecular imaging of gliomas, may allow a more complete tumor resection and may even serve as a molecular targeting agent to deliver therapeutics.

Additionally, this novel approach may soon advance to clinical trials on breast cancer, where it will be further evaluated for its clinical use in helping surgeons to assess during surgery if all margins of lumpectomy specimens from the breast are free of cancer during surgery. Success with this technique could dramatically reduce the current re-excision rates of 20–60%, and more importantly, reduce or eliminate local recurrence due to “surgically missed” cancerous tissues.



DETECTING OVARIAN CANCER

Robert C. Bast, Jr., M.D., MD Anderson Cancer Center. Ovarian cancer remains the most lethal of the gynecological cancers, due largely to late diagnosis. Only 25% of ovarian cancers are detected at an early stage — when the disease is highly curable. Renowned ovarian cancer scientist, Dr. Robert Bast, has found that more early cancers can be detected with a “two-step” strategy that first analyzes blood biomarkers and secondly, performs transvaginal sonography (TvS), an imaging technique to visualize the ovary. This new approach for early detection of ovarian cancer has shown promising results in clinical evaluation over the past 12 years. With additional positive results from a large clinical trial that is underway in the United Kingdom involving 200,000 women, Dr. Bast's two-step strategy may obtain FDA approval in a few years. Currently, Dr. Bast is leading his team to develop a more sensitive imaging technology called Superconducting Quantum Interfering Device (SQUID), which is several orders of magnitude more sensitive for detecting small, early-stage tumors than other technologies, such as PET, CT, and MRI. Dr. Bast's mission to develop this new, ultra-sensitive two-step detection strategy could greatly increase early detection and diagnosis of ovarian tumors, at a stage that would offer the best opportunity for a cure for many patients.

ADVANCING PERSONALIZED MEDICINE THROUGH ANALYSIS OF CANCER PATHWAYS AND DRUG RESISTANCE

What makes cancer cells different and dangerous? Among the myriad genetic alterations observed in tumors, only some propel cancer cells to proliferate abnormally, survive inappropriately and resist the drugs administered to destroy them. Furthermore, every cancer is different as multiple pathways can lead to the lethal growth of cancer. To know which alterations represent important therapeutic targets, we need to understand their place in the vast molecular network that underpins cellular function. We are using multiple genomic, proteomic, computational, and *in vivo* approaches to build a comprehensive “wiring diagram” for cancer cells and their molecular environment. This blueprint will lead us to better, more sophisticated strategies to control individual cancers and combat drug resistance.

TARGETING GLIOBLASTOMA



Webster K. Cavenee, Ph.D., Ludwig Institute for Cancer Research, is a renowned leader in identifying the genetic underpinnings of glioblastoma multiforme (GBM), the most common and lethal form of brain tumor, and creating innovative therapeutic approaches against this disease. Dr. Cavenee and his team demonstrated in glioblastomas of two patient cohorts that a modification of tumor suppressor protein, PTEN, is associated with and may cause the upfront and acquired tumor resistance to the targeted therapy, EGFR inhibitors. In addition, their research demonstrated that PTEN modification is an independent indicator of poor prognosis. Current work is identifying the molecular players of PTEN modification and determining the regulators of this important event. This ongoing research is highly significant as it will lead to a novel form of therapy whereby patients are treated for their own personalized tumor mutation spectra as well as with agents targeting the mechanisms their tumor uses to elicit

resistance. Dr. Cavenee’s approach holds the promise to improve patient’s therapeutic responsiveness and extend length of survival from the deadly brain tumor.

STOPPING METASTASIS



Danny Welch, Ph.D., NFCR Center for Metastasis Research, The University of Kansas Cancer Center, is addressing metastasis, the most lethal aspect of cancer, which is related to more than 90% of all cancer deaths. Center researchers and collaborators have discovered six metastasis suppressor genes, including *KISS1*, which when expressed in metastatic cells, renders the cells incapable of growing into a secondary tumor. Researchers are focused on identifying how these genes and their proteins function to suppress metastasis. The data generated will be crucial for translating their laboratory discoveries into new anti-metastasis therapies for cancer, including: breast, prostate, colon, ovarian, and pancreatic cancers, melanoma, and others.

In addition, scientists at the Center are unraveling another mystery of metastasis: Why are some patients more susceptible to metastasis than others? The answer seems to lie in the genetic variations of DNA in the tiny cellular structures called mitochondria. With continued investigation in this intriguing field, Dr. Welch envisions that one day, patients could have their mitochondrial DNA in a blood sample sequenced within an hour. The test results could then guide physicians to prescribe more aggressive treatment for patients at risk for metastasis, while sparing others from toxic therapies.

BIOMARKERS AND NEW THERAPEUTIC TARGETS

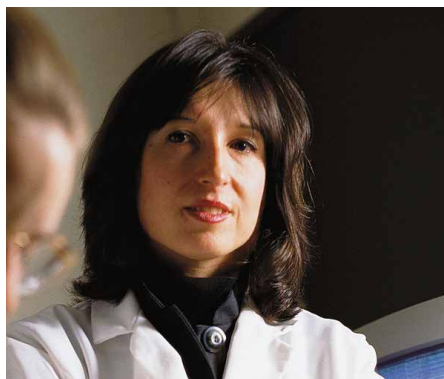


Wei Zhang, Ph.D., NFCR Center for Cancer Systems Informatics, MD Anderson Cancer Center, is identifying microRNAs (miRNAs) — tiny cellular molecules that are closely associated with cancer development and progression — that may serve as diagnostic

markers and clinical-stage markers for colorectal cancer. Dr. Zhang is investigating candidate miRNA biomarkers in a patient's plasma for early detection, staging, predicting treatment response, and evaluating prognosis. In 2013, the team further validated that miR-148a and miR-141 are two effective biomarkers for late stage tumor detection. These plasma miRNA markers hold important clinical relevance and could give oncologists new diagnostic tools for colorectal cancer, improving disease management and patient survival.

Recently, Dr. Wei Zhang led his research team in developing streamlined pipelines for efficient processing and systematic analysis of genomic datasets generated from tumor tissue samples. Their efforts could significantly accelerate pace of research in identifying novel genetic mutations as potential biomarkers for early diagnosis and staging of colorectal, gastric and other cancers.

ANTI-CANCER DRUG DESIGN AND DISCOVERY

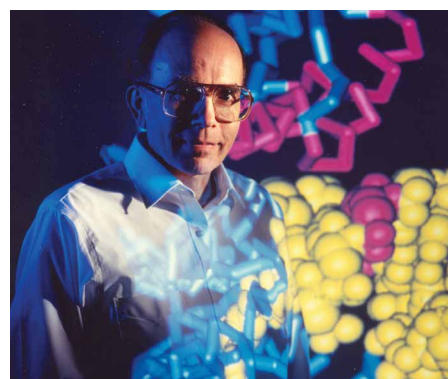


Alanna Schepartz, Ph.D., Yale University, has developed anti-cancer beta-peptide inhibitors to address one of the biggest challenges in drug discovery — how to block “protein-protein interactions” within cells that are closely related to diseases such as Alzheimer’s and cancer. Beta-peptide inhibitors represent a new generation of anti-cancer drugs that are highly effective and specific in targeting almost any cancer-related protein-protein interaction. This new class of drugs may positively impact the treatment of almost half of all cancers. In the past year, Schepartz Laboratory of Chemical Biology synthesized a series of peptide molecules that may provide a new therapeutic approach to inhibit EGFR — an important protein that is

often mutated in non-small cell lung cancer. The molecules developed by Dr. Schepartz’ team can be used to inhibit even those forms of EGFR that are resistant to current therapies. Continued development of these new therapeutic agents could lead to new and more effective ways of treating patients whose lung cancer has already developed resistance to current therapies.



Alan C. Sartorelli, Ph.D., Yale University School of Medicine, is leading his team of researchers to design, synthesize and characterize new anticancer drugs that could deliver better therapeutic benefits to patients. One of the new drugs designed and synthesized in his lab is lomustine (onrigin®), which causes damages in DNA in tumor cells. This drug has shown remarkable clinical response in some patients with acute myeloid leukemia (AMT) in phase II clinical trials. Dr. Sartorelli is now developing new approaches that would further improve treatment efficacy of lomustine. Another novel agent, Triapine, was originally designed and synthesized in the Sartorelli lab, and has now been tested in over twenty phase I and phase II clinical trials by his and other groups. In a recent trial, Triapine has demonstrated pronounced anticancer effects on cervical and vaginal cancers. This novel agent is now advancing to large scale Phase III clinical trials, moving closer to becoming a new FDA-approved anticancer drug for women battling cervical or vaginal cancer. Dr. Sartorelli is currently evaluating Triapine for treatment of ovarian cancer, further expanding the potential clinical application of this promising new drug.



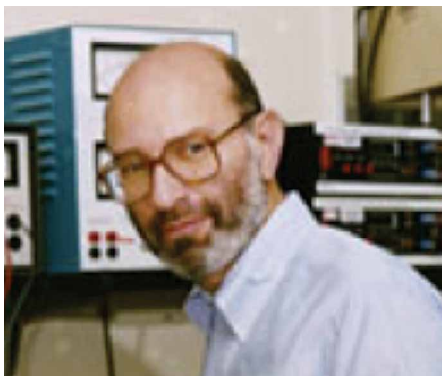
Paul Schimmel, Ph.D., Scripps Research Institute, is seeking to understand why human aminoacyl tRNA synthetases (AARSs), which are among the essential enzymes involved in the protein synthesis machinery found in all organisms, have distinct additional vital activities that are involved in pathways relevant to treating cancer and other diseases. In 2013, Dr. Schimmel’s team discovered that one type of AARS, tyrosyl-transfer RNA synthetase (TyrRS), also plays an important role in platelet production and maintenance. Platelets, the tiny blood cells responsible for forming blood clots, are often damaged during chemotherapy, which can leave patients with dangerous bleeding disorders. Dr. Schimmel’s team is working to develop a treatment based on TyrRS that can correct this damaging side-effect, improving the quality of life for all post-chemo cancer patients suffering from platelet deficiency.



Daniel Von Hoff, M.D. & Laurence Hurley, Ph.D., NCI Center for Targeted Cancer Therapies, TGen, are developing new targeted cancer therapies and improving the treatment efficacy of existing therapies for pancreatic cancer. During 2013, their team continued to identify, design, and synthesize new agents that specifically target the mutant

K-Ras gene which is found in almost all of the most aggressive types of pancreatic cancer. Importantly, since normal cells do not have mutated K-Ras, new compounds developed at the Center will have minimally harmful effects on normal cells. Additionally, Drs. Von Hoff and Hurley are exploring a new approach that involves targeting this gene at the DNA level. They identified a unique DNA structure on the K-Ras gene that could be an ideal drug target. To date, researchers at the Center have screened a chemical library and identified 12 different “hit molecules” that could target this DNA structure. Further optimization of these hit molecules can potentially lead to a new class of K-Ras targeted agents. This previously unexplored approach will allow Drs. Von Hoff and Hurley to identify and develop new molecules that can be readily translated into the clinic, helping patients to fight one of the deadliest cancers in humans.

Janos Ladik, Ph.D., University Erlangen-Nürnberg, Erlangen, Germany, is conducting research on DNA intercalating agents — anti-cancer drugs that wedge themselves into the DNA double helix to interfere with cell division and the making of RNA and proteins. Rapidly dividing cells, such as cancer cells, are inhibited by certain intercalating agents. Dr. Ladik uses theoretical physics and super computers to investigate the use of DNA intercalating agents as cancer preventive agents, inhibiting the formation of cancer-causing mutations and, thereby, preventing cancer initiation.



Stanley Cohen, M.D., Stanford University School of Medicine, is working to understand the molecular players that regulate the growth of cancer cells vs. normal cells. One such player that has been a particular area of focus is TSG101, a multifunctional tumor

cell growth regulator that was previously discovered in Dr. Cohen’s lab. His latest research demonstrated that the TSG101 protein interacts with the important tumor suppressor gene p21; deficiency of the TSG101 protein leads to increased expression of p21 and slows down cell proliferation in human osteosarcoma cell lines. Continued research in this area will provide scientists with a deeper understanding of the complex molecular underpinnings of tumor growth and proliferation, and may lead to novel therapeutic approaches to treating cancer.

PERSONALIZED APPROACH FOR MORE EFFECTIVE TREATMENT



Kathryn B. Horwitz, Ph.D., University of Colorado Denver, is working to understand why certain breast cancer tumors are resistant to hormone therapy, even if the growth of those tumors is also fueled by estrogens. New research in Dr. Horwitz’s lab has discovered that luminal breast tumors, which account for 75% of all breast tumors, actually contain four different types of cancer cells, some of which are resistant to hormone therapy. This is as if a woman had four different kinds of breast cancer. Her team is working to isolate each of these four cell types, and discover drugs that attack each one. Then they will reassemble tumors using multiple cell types and test combination therapies that attack not just one, but all the malignant cells in a tumor. This research looks to develop a better long-term approach to treatment, converting breast cancer into a chronic and survivable disease.



Waun Ki Hong, M.D., MD Anderson Cancer Center, is leading the development of personalized molecular targeted therapies for lung cancer. His Biomarker-Integrated Approaches of Targeted Therapy for Lung Cancer Elimination (BATTLE) program, which is in part funded by NCFR, employs an adaptive clinical trial design in which patients are assigned to the treatment drug to which they are most likely to respond, based on their personal biomarker profile identified through tumor biopsies. Clinical application of these findings is underway in developing and testing personalized therapeutic strategies for non-small cell lung cancer (NSCLC).

Dr. Hong’s ongoing research in this area holds extraordinary promise to improve patient survival in lung cancer — the leading cancer killer in America. The BATTLE program is now serving as a model for personalized medicine throughout the world, for lung cancer and other cancer types as well. Dr. Hong’s team is forging ahead with several newly initiated clinical trials built on the success of the original BATTLE program. These new BATTLE programs have the potential to make major contributions to the field of lung cancer in three ways: providing a better understanding of how this disease progresses and which genes and pathways are responsible; testing novel clinical agents and drug combinations to develop individualized and targeted treatment approaches; and ultimately, finding ways to prevent this deadly disease.

ANGIOGENESIS: SHUTTING DOWN CANCER



Harold F. Dvorak, M.D., Beth Israel Deaconess Medical Center, is an NFCR Fellow and Albert Szent-Györgyi Prize winner for his discovery of vascular permeability factor/vascular endothelial growth factor (VPF/VEGF). The growth factor VEGF plays a central role in angiogenesis or the formation of blood vessels in and around malignant tumors. Dr. Dvorak's NFCR-funded research has led to the development of the anti-angiogenic therapies, a new generation of anti-cancer drugs that target tumor blood vessels. His recent discoveries have identified and characterized at least six different kinds of blood vessels in tumors, but current anti-angiogenic therapies primarily act against only one of them. His group has already discovered new therapeutic targets on the other five vessel types, with the goal of improving the effectiveness of anti-

angiogenic therapy by attacking the entire tumor environment.



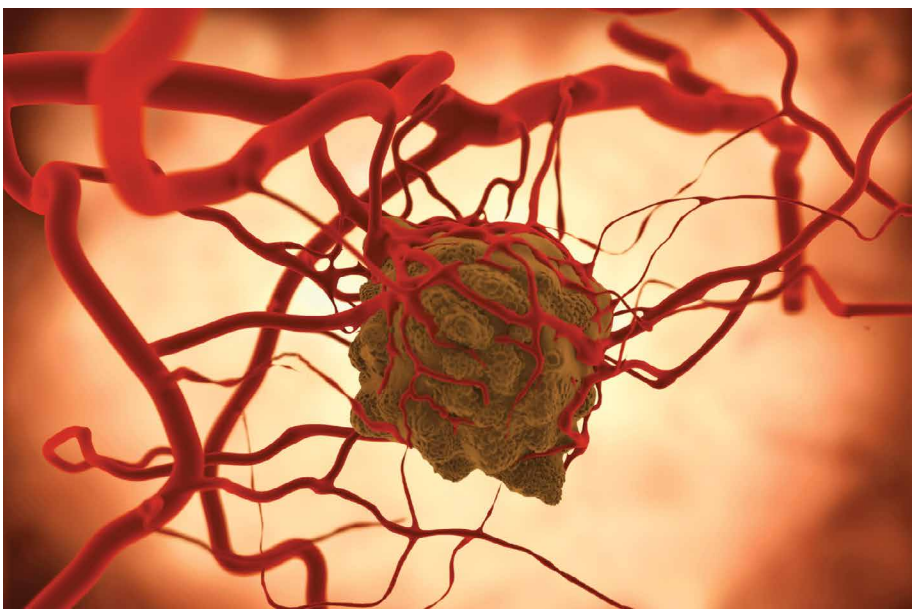
Rakesh K. Jain, Ph.D., Massachusetts General Hospital, has discovered new ways of preventing resistance to anti-angiogenic therapy in glioblastoma patients. Dr. Jain's earlier research has demonstrated that anti-angiogenic therapy works through normalizing the abnormal, leaky vessels that usually surround and penetrate tumors, improving delivery of both chemotherapy drugs and the oxygen that is required for effective radiation therapy. Although some patients initially respond positively to this therapy, in all cases the tumors eventually regrow and invade healthy areas of the brain. Identification of biomarkers that indicate tumor progression during therapy is urgently needed to guide the development of new treatments that will stop cancer growth. Recently, Dr. Jain's team identified that inflammatory cells called macrophages are

increased in tumor areas after antiangiogenic therapy. The increase of macrophages directly correlates with a shorter survival time for patients. These results suggest that tumor-associated macrophages may participate in escape from antiangiogenic therapy and that targeting macrophages may be a new strategy to prevent tumors from invading healthy tissue as well as new blood vessel formation in brain tumors after antiangiogenic therapy. The team is continuing to examine the molecular characteristics of drug resistance after antiangiogenic therapy, with the goal of developing new molecularly targeted agents to circumvent resistance to current antiangiogenic agents, and bringing improved treatment benefits to the patients.

OVERCOMING DRUG RESISTANCE



Susan Band Horwitz, Ph.D., Albert Einstein College of Medicine, is a leading pharmacologist who deciphers how tumors develop resistance to Taxol, and develops new strategies to overcome its resistance in tumors. It is currently difficult to predict which patients will respond well to Taxol and which will be resistant. In 2013, her team developed a new tool to help oncologists make this prediction. By comparing cell lines that are resistant to Taxol with those that are sensitive to the drug, they were able to determine that a specific protein, galectin-1, was associated with resistance to Taxol. When they blocked expression of galectin-1, the resistant cells became sensitive to the treatment. Tumor cells also secrete galectin-1 into the blood stream, which could allow a simple blood test to determine whether a patient is likely to respond to Taxol treatment. Dr. Horwitz's work will help doctors provide the right therapy to patients, saving precious time by avoiding treatments that are unlikely to work.

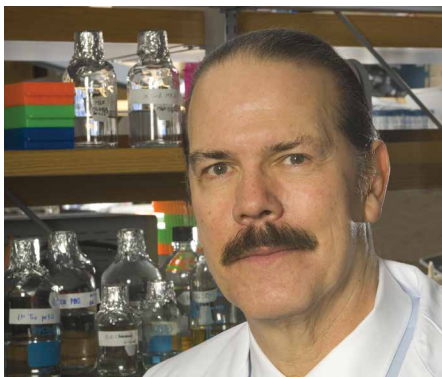


Artist's rendering of angiogenesis: Blood vessels grow towards and infiltrate a tumor mass, supplying it with the oxygen and nutrients it needs to grow and survive.

INNOVATIVE THERAPIES



Wayne Marasco, M.D., Ph.D., NFCR Center for Therapeutic Antibody Engineering, Dana-Farber Cancer Institute, Harvard Medical School, is discovering and engineering therapeutic antibodies for clinical applications in cancer. The Center has established a library containing 1.6 billion different human sFv antibody-displaying phages, a tremendous resource for developing monoclonal antibody-based targeted therapies. Equipped with extensive experience in the field of human monoclonal antibody engineering, Center researchers have recently isolated two high-affinity human monoclonal antibodies that specifically attack a tumor antigen named carbonic anhydrase IX (CAIX) on renal cell carcinoma (RCC) — the most common form of kidney cancer. These reagents are being developed as new immunotherapies and diagnostic tools for kidney cancer patients who currently have no effective treatment options. Dr. Marasco expects that once proven effective in their pre-clinical experiments, these anti-CAIX antibodies can quickly advance to clinical testing for treatment of RCC patients. In addition, these antibodies may also have broader use in other CAIX-associated cancers, such as oral cancer and lung squamous cell carcinoma.



Paul B. Fisher, M.Ph., Ph.D., Virginia Commonwealth University School of Medicine, has developed a novel gene therapy to treat both early stage and metastatic

prostate cancer, the second leading cause of cancer-related deaths in men in the U.S. This new therapeutic, which he calls a *Cancer Terminator Virus (CTV)*, is a virus genetically reprogrammed to infect and destroy only tumor cells, leaving normal cells unharmed. And the more aggressive the cancer cells, the more potent the virus' effects are. One of the main hurdles in clinical application of viral therapies is that therapeutic viruses are often trapped in the liver and destroyed by the immune system before they reach the targeted cancer cells. To shield the viruses from the immune system, Dr. Fisher's team encloses them in microbubbles, which are "decorated" with molecules on their surface that specifically bind to cancer cells. The therapeutic cargo is then released at the site of the tumor using ultrasound. Dr. Fisher is now working to combine the Cancer Terminator Virus with another drug that blocks MCL-1 — a protective protein made by cancer cells — which will make CTV even more effective in combination. This powerful new approach is making steady progress and may translate into the clinic to treat patients with prostate cancer.

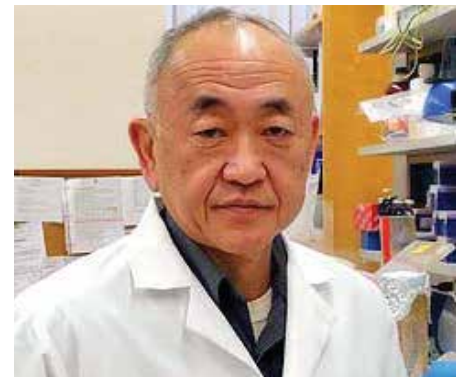


Laurence J.N. Cooper, M.D., Ph.D., MD Anderson Cancer Center, is pioneering the development of a new forward-thinking technology which genetically engineers human immune cells for the treatment of certain types of leukemia and lymphoma. Over the past year, convincing clinical data has emerged to demonstrate that this novel immunotherapy holds great promise to offer a new, safe and effective way to treat patients with leukemia and lymphoma which has stopped responding to all other therapies. This promising new treatment is moving into the next round of clinical trials in 2014.

With NFCR's funding, Dr. Cooper continues to move forward towards his goal — to develop an "off-the-shelf" therapy in which

the engineered immune cells can be pre-assembled and frozen in large quantities and simply thawed and infused back to the patients "on demand." More intriguingly, Dr. Cooper's innovative genetic engineering approach reduces the need to "match" donors with patients, allowing the immune cells prepared from a single donor to be used in multiple recipients. This novel approach is a paradigm shift in immunotherapy, which allows prompt delivery of a safe and effective new immunotherapy to patients in need without delay.

CHINESE HERBAL MEDICINE: ADJUVANT TO CHEMO AND RADIATION THERAPY



Yung-Chi Cheng, Ph.D., Yale University School of Medicine. While the therapeutic effects of traditional Chinese medicine (TCM) have been documented anecdotally for centuries, they have been too often discounted by modern medicine as "alternative therapy" because there was little rigorous scientific proof of their effectiveness. For the last 12 years, with NFCR support, Dr. Cheng has explored the therapeutic properties of PHY906, an ancient Chinese herbal medicine formula. In preclinical research, Dr. Cheng discovered that PHY906 effectively decreased intestinal injury from anticancer drugs including irinotecan (Camptosar®) and etoposide (Toposar®). PHY906 is currently being evaluated in Phase II clinical trials as an adjunct to chemotherapy in the treatment of colorectal cancer patients. Dr. Cheng's latest laboratory research demonstrated that PHY906 also decreases the toxicity of abdominal irradiation, while increasing the response of tumors to radiation therapy. With further investigation, PHY906 has the potential to be used as an adjuvant to radiation therapy for colorectal cancer.

PHY906 could also improve the effectiveness of Sorafenib (Nexavar®), the only approved drug for treatment of patients with liver cancer. A phase I/II clinical trial with liver cancer patients has been initiated for evaluating PHY906 as an adjuvant to Sorafenib. The outcomes of these clinical trials could have a major impact for the treatment of colorectal and liver cancer patients in the future, and PHY906 could become one of the first FDA-approved oral herbal medicines for anticancer treatment.

DEVELOPING NANOTECHNOLOGY-BASED CANCER THERAPIES

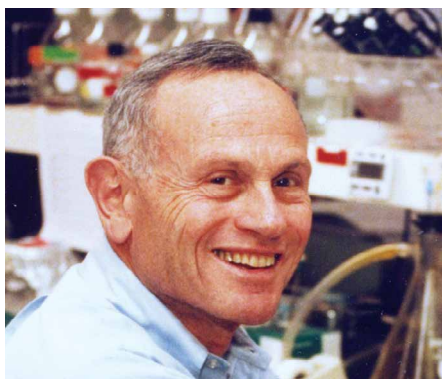
Most cancer drugs are blunt instruments. NFCR scientists are working at the molecular level to engineer new therapeutic agents that can home in on cancer cells and selectively destroy them with little or no side effects. Designing these nanoscale “smart bombs” requires multiple rapidly-advancing technologies and the expertise to combine them. Critical components of therapeutic nanoparticles include: (1) a targeting mechanism that identifies cancer cells by the molecules they express; (2) a destructive mechanism such as a toxin, antibody or tumor suppressor gene that disables cancer cells; and (3) molecular packaging such as a liposome or other material that allows the therapeutic agent to traverse the body efficiently.



Esther H. Chang, Ph.D., Georgetown University, has developed a nanoscale drug delivery system that carries anticancer agents directly to both primary and metastatic tumor cells, significantly enhancing a tumor’s sensitivity to chemo- and radiation therapy. The nanocomplex, based on liposomes, has been used successfully in a phase I clinical trial to deliver the tumor suppressor gene

p53 to patients’ metastatic cancer cells in a safe, non-toxic way. Using ovarian cancer models in their recent experiments, Dr. Chang’s team successfully delivered the p53 gene to the tumor, sensitizing the cancer to chemotherapy. These results are highly significant since ovarian cancer in two-thirds of patients becomes resistant to cisplatin, the first line treatment of chemotherapy, and Dr. Chang’s p53 nanocomplex may offer an effective alternative for these patients. In addition, because the anticancer effects of the combination of p53 nanocomplex with cisplatin are far greater than using cisplatin alone, less of the chemotherapy drug is needed to treat the tumor, which therefore decreases harmful side effects of the treatment.

CANCER PREVENTION



Michael B. Sporn, M.D., Dartmouth Medical School, is establishing new approaches for prevention and treatment of various cancers. His highly fruitful research has resulted in the development of several triterpenoid compounds which have potent preventative effects against liver cancer, melanoma, and highly aggressive lung cancer. Dr. Sporn’s team also demonstrated that triterpenoids combined with other anti-inflammatory agents such as histone deacetylase inhibitors were effective in preventing pancreatic, breast and lung cancer in tumor models. Moreover, they found that the triterpenoid, CDDO-Me, increased the therapeutic activity of two chemotherapy agents, carboplatin and paclitaxel, in treatment of lung cancer in tumor models.

Dr. Sporn’s latest research on the anticancer drug olaparib is particularly exciting. Olaparib is currently tested in Phase III clinical trials for treatment of BRCA-mutated ovarian cancers. Results from his lab show

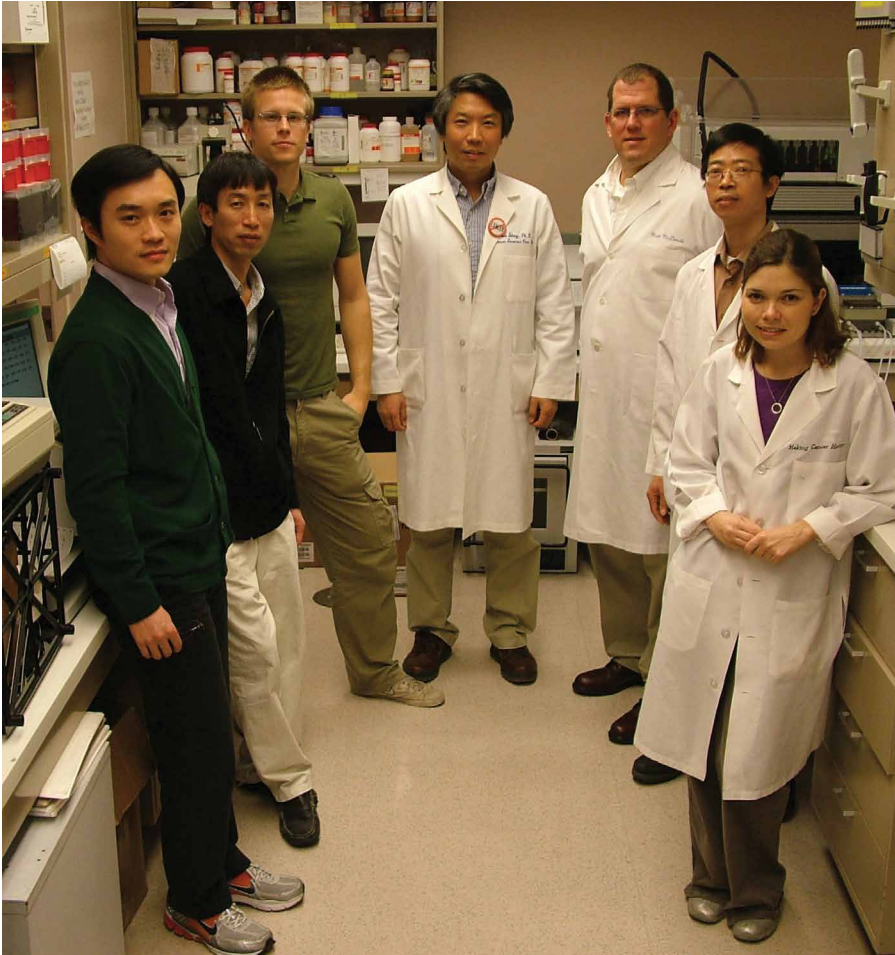
that this agent markedly inhibited the progression of carcinogenesis in breast cancer tumor models that harbor aberrant BRCA and p53 genes. Dr. Sporn’s finding is highly relevant since a BRCA mutation sharply increases a woman’s risk for breast cancer and many women with such a mutation would elect to undergo bilateral prophylactic mastectomy if practical chemoprevention is not available as an alternative. Olaparib is thus an excellent potential candidate for preventative use for these women, so that they may be able to effectively prevent breast cancer without painful and disfiguring surgery.



Helmut Sies, M.D., Heinrich-Heine-Universität, Düsseldorf, Germany, is well recognized for his discovery of the skin cancer prevention effects of the micronutrient, lycopene, the antioxidant found in tomatoes and carrots. Dr. Sies’ research is recently focused on selenium (Se), a trace metal essential for good health. Dietary or supplemental Se is incorporated into selenoproteins — critical cell proteins that have anti-oxidation functions. Expression of a selenoprotein, Sepp1, has been shown to be suppressed in cancer tissue of colorectal cancer patients. Dr. Sies’ team showed that various dietary Se compounds stimulate the secretion of selenoproteins and this may protect the intestine from oxidative damage. Latest research data from his lab also suggest that the Sepp1 protein secreted from intestinal cells may provide immune cells in the gut with selenium, which is required for a proper immune defense. Research will continue to delineate how intestinal selenoproteins are involved in selenium-mediated colorectal cancer prevention, as well as to determine the genetic and metabolic factors that may affect the individual outcome of dietary supplementation.

A NEW PLATFORM FOR INTERNATIONAL COLLABORATION

NFCR Launches Center for Cancer Systems Informatics at MD Anderson



In the fight against cancer, collaboration is critical. Why collaborate? Because working together we will be far more effective than working alone. Cancer is a very complex disease, and no single organization can hope to decipher the complex web of genes, pathways, molecules, and receptors that allow cancer to function – much less design drugs that attack these targets effectively. Most of today’s drug-development programs fail, often in the expensive clinical-trial phases where a new drug is tested in patients. Frequently, the failure happens because the researchers had an imperfect understanding of the genetic networks regulating the disease and the molecular pathways the new drugs were designed to target. Collaboration will allow researchers to identify errors early in

the development process, reducing the risk of failure.

In the age of cancer genetics, promising new treatments rely on identifying the molecular profile of each patient’s cancer, then exploiting vulnerabilities that stem from the underlying genetics. This “pharmacogenomic” approach allows doctors to choose the optimal therapy that will be most effective and least toxic for each individual patient. Such targeted cancer therapies may prove to be more successful and less costly than conventional treatments. In order to successfully develop new anti-cancer drugs using pharmacogenomics, well designed and organized collaborations between scientists and physicians are essential.

Recognizing the critical importance of collaboration in the fight against cancer, NFCR established the **Center for Cancer Systems Informatics** in June 2013. Under the directorship of Professor Wei Zhang, Ph.D. at MD Anderson Cancer Center, the Center will be the hub for international collaboration on many projects led by NFCR. In partnership with leading cancer research institutes and organizations around the world, the Center will provide the resources and technical platforms necessary to streamline the processing and systematic analysis of genomic and other cancer-related datasets. The collaborative efforts of the Center could significantly accelerate the pace of developing advanced tools for early diagnosis and more effective therapies for the treatment of various cancers.

Since its establishment, this new NFCR Center has played a central role in two international collaborative projects. The inaugural project — **Geographic Mapping of Gastric Cancer Genomes** — is already demonstrating the importance of the Center’s collaborative approach for the development of new cancer treatments.

Gastric cancer is the fifth most common cancer and the third leading cause of cancer death worldwide, accounting for 6.8% of all newly-diagnosed cancer cases and 8.8% of cancer deaths globally. It is also especially susceptible to genetic mutation, even compared with other cancers. This is because the stomach sits on the front lines of our interaction with the environment. Contaminated foods that contain mutagens, spoiled foods that release high energy oxidant products and free radicals, foods and drinks that are too acidic and too alkaline — all the bad things we ingest may cause mutations in important genes at several locations in our stomach.

For these reasons, gastric cancer is a highly heterogeneous disease. The molecular profile of genetic mutations varies substantially, not only from one patient to the next, but also between two tumors within the same patient,

and even from one region to the next within the same tumor. Researchers do not yet have a complete picture of just which genes are commonly mutated in gastric cancer, nor have they determined whether these mutations can be successfully targeted by new therapies in patients.

The success of this project depends upon international collaborations — including both private-public and industrial-academic partnerships. The quality of the analysis performed at the Center is directly affected by the quality of human gastric cancer tissue samples available to the researchers. In many ways, cancer tissue can be considered the center of the molecular-medicine universe. In partnership with the Tissue Bank Consortium in Asia (TBCA) — another collaborative program established and supported by NFCR since 2008 — in Tianjin, China, the new Center will be able to work directly with scientists at the Tianjin Cancer Institute and Hospital, performing all of their experiments on gastric cancer specimens of the highest quality. This partnership will both improve and accelerate the results of this high-impact research project.

In their efforts to map the molecular paths that gastric cancers follow — and to identify key points along those paths that can be targets for treatment — researchers at the NFCR Center for Cancer Systems Informatics have formed a strong multi-disciplinary team.

Joining NFCR in this global gastric cancer research initiative is a diverse group of cancer researchers, representing 10 partners from academia and industry in both the U.S. and China. Center Director Dr. Wei Zhang, who is also an Adjunct Professor at the Tianjin Medical University Cancer Institute and Hospital, will work closely with the scientists at the Tianjin Cancer Institute to conduct genomic data analysis on the entire set of gastric cancer samples used in this project. Based on preliminary data, the researchers anticipate that this project will discover important and exciting results about gastric cancer. Dr. Zhang is already preparing a research paper detailing these findings, which he plans to submit to a first-class, peer-reviewed scientific journal once the project finishes in early 2014.

As its second project, this new Center is also a pivotal part of **The Global Brain Cancer**

Alliance, an international collaborative network that is committed to accelerating laboratory and clinical research on glioblastoma multiforme (GBM), a deadly form of brain cancer, with the goal of significantly improving survival of patients with this disease. The Center will play a critical role in genomic data analysis and sequencing of patients' tumor tissue samples, providing crucial genetic information to help

guide novel clinical trials that have never been conducted with GBM patients before.

NFCR has initially committed \$600,000 of research funding to the Center over five years, and we are confident that the Center will continue to play a critical role in high impact cancer research for years to come. The collaborative projects represent the leadership role of NFCR and our partner institutions in battling cancer on a global scale.



Haixin Li, M.D., Ph.D, TMUCIH-NFCR Joint Tissue Banking Facility, Tianjin Medical University Cancer Institute and Hospital (TMUCIH). Well-characterized tumor specimens, carefully gathered and preserved in a well-managed biorepository, constitute one of the most valuable resources for cancer researchers. Genetic data from tumor specimens, coupled with the development of technologies

to assay the molecules and pathways in tumor cells, allow researchers to gain deeper understanding of the roles cancer-related genes, proteins and pathways are playing in different types of cancer. This approach is revolutionizing modern cancer therapy.

Scientists at the Joint Tissue Bank collect and maintain biospecimens (tumor tissues and matching blood samples) from cancer patients fighting all types of cancer; this rapidly growing biorepository includes over 42,000 fresh frozen tissue samples and nearly 46,000 blood samples.

The TMUCIH-NFCR Joint Tissue Bank is part of the NFCR Tissue Bank Consortium in Asia (TBCA), a source of biospecimens essential to cutting-edge cancer research. NFCR provides consortium members access to a web-based biospecimen locator, enabling cancer researchers to determine the availability of suitable biospecimens. By providing cancer researchers access to many different types of high quality tumor specimens, the TBCA plays an increasingly important role in cancer research.

The TBCA operates in total compliance with the highest international standards, and is governed by a TBCA Steering Committee made up of leading scientists from universities, research hospitals, and biopharmaceutical companies in the United States and China.

In recent years, TBCA initiated a gastric cancer research project which performs the “next generation” genomic profiling on gastric cancer samples collected from cancer patients, to identify genes that are responsible for gastric cancer and its metastasis. The project will also identify biomarkers that could be useful both for gastric cancer diagnosis and the development of new targeted therapies. The scale of this project and the quality of the tumor samples provided by TBCA are unprecedented, and the preliminary results are very encouraging. The success of this leading-edge research project will positively impact many areas of gastric cancer diagnosis and treatment, saving more lives from this deadly disease.

2013 SZENT-GYÖRGYI PRIZE

TARGETED CANCER THERAPY: A NEW PARADIGM FOR TREATING CANCER

Dr. Alex Matter was awarded the 2013 Szent-Györgyi Prize for Progress in Cancer Research for his contributions to the development of Gleevec®, the first drug specifically targeting a molecular lesion in cancer. The Prize was presented to Dr. Matter at an awards ceremony in Washington, D.C., on April 5, 2013 at The National Press Club. John Castellini, President and CEO of PhRMA, delivered the keynote address.

Dr. Alex Matter's pioneering research in probing the molecular anatomy of tumor cells in search of cancer-causing proteins — represents the start of a new era in cancer treatment. Gleevec was the first drug that translated the insights of molecular cancer biology into a highly effective anti-cancer therapy, which offered proof that molecular targeting works in treating cancer.

This first targeted cancer therapy, imatinib mesylate, or Gleevec, contributed to a major breakthrough in the treatment of chronic myelogenous leukemia (CML), followed by its successful application to other malignant cancers by turning off the signal of the protein causing these cancers. With Gleevec, the outcome of treating CML went from the dismal and often deadly to a nearly 100% survival with few or no side-effects.

The successful development of Gleevec led to a paradigm shift in new cancer treatments. These research discoveries demonstrated that it is possible to counteract cancer by specifically inhibiting the activity of key oncogenic molecules was nothing short of phenomenal. Dr. Matter's research made it possible to turn deadly cancers into treatable diseases.

"Alex Matter is the father of targeted cancer therapies," said former Chinese Minister of Health Zhu Chen, M.D., Ph.D., co-winner of the 2012 Szent-Györgyi Prize and the Chair of the 2013 Prize Selection Committee. "Dr. Matter's leadership in building the synergy between pharmaceutical companies, universities, and research hospitals changed both the way we treat CML and our approach to developing new anticancer drugs."

"I am deeply honored to receive the 2013 Szent-Györgyi Prize," said Dr. Matter, CEO of the Experimental Therapeutic Centre of Agency for Science, Technology and Research (A*STAR) in Singapore.



Dr. Zhu Chen, Co-Chair of 2013 Prize Selection Committee; Dr. Alex Matter, 2013 Prize Recipient; Dr. Sujuan Ba, Co-Chair of 2013 Prize Selection Committee.

"Dr. Matter is a pioneer. Overcoming many barriers, he was able to translate the insights of molecular cancer genetics and tumor biology into a new approach for treating cancer. This was Albert Szent-Györgyi's vision," said Sujuan Ba, Ph.D., co-chair of the Szent-Györgyi Prize Selection Committee and Chief Operating Officer of NFCR.

The Szent-Györgyi Prize honors a scientist for a seminal discovery that has resulted in a breakthrough in cancer research. The prize is awarded annually to a scientist, nominated by colleagues or peers, who has contributed outstanding, significant research to the fight against cancer, and whose

accomplishments have helped improve treatment options for cancer patients. The prize is named in honor of NFCR co-founder Albert Szent-Györgyi, M.D., Ph.D., who won the Nobel Prize for Science and Medicine in 1937 for his discovery of Vitamin C.

ABOUT ALEX MATTER, M.D.

Dr. Alex Matter received his medical degrees from the Universities of Basel and Geneva, and completed his doctoral thesis at the Institute of Pathology at the University of Basel. He held fellowships at the Swiss National Science Foundation and the Swiss Academy for Medical Sciences. Dr. Matter is currently CEO of the Experimental Therapeutics Centre, A*STAR, Singapore, having spent five and a half years as Director of the Novartis Institute for Tropical Diseases (NITD), from October 2003 to February 2009. Prior to this role, Dr. Matter was Global Head of Oncology Research for Novartis Pharmaceuticals Corporation, Head of Novartis Institutes for BioMedical Research in Basel and Global Head of Translational Research.

Dr. Alex Matter previously held teaching positions at the University of Basel and the European University Confederation of the Rhine. He has published more than 100 scientific articles, several book chapters in the area of oncology and hematology, and is emeritus Professor of the Medical Faculty of the University of Basel and an Honorary Adjunct Professor of the Department of Pharmacology, YLL School of Medicine, National University of Singapore.

Dr. Matter is a member of the American Association for Cancer Research, the National Medical Research Council in Singapore, and the Board of Curiox, a Singapore-based start up company. He is also an elected member of the Swiss Academy of Medical Sciences. Dr. Matter is the recipient of the 13th Warren-Alpert prize and the AACR-Bruce F. Cain Memorial Award.

THE LUCY FUND

Spreading the Word, Stopping the Spread — of Metastatic Cancer

ABOUT LUCY

Lucy Stanovick was a daughter, sister, wife, mother of two, and an Associate Professor at East Stroudsburg University in Pennsylvania. In 2008, Lucy was diagnosed with stage IV metastatic breast cancer at the age of 42. Upon her diagnosis Lucy researched her specific condition. She came across a troubling statistical discrepancy, which stated that less than 5% of current funding supports research into the mechanisms of metastasis, even though over 90% of cancer mortalities are due to metastatic cancer — cancer that has spread from its original location to vital organs within the body.

Treatment options for metastatic cancers are limited and there is no cure. Determined to change that, Lucy sought out Danny Welch, a leading metastasis researcher and Director of the NCCR Center for Metastasis Research. She then founded *The Lucy Fund*, which cultivated grassroots support for metastatic cancer research, with the ultimate goal of being able to treat late stage cancers as chronic, rather than deadly, diseases.

Lucy always knew *The Lucy Fund's* efforts would come too late to save her life. While she passed away on August 16, 2012, her legacy lives on with her family, friends, supporters, and *The Lucy Fund* itself. To date, the Fund has raised over \$230,000 for metastatic cancer research.



Lucy Stanovick, founder of The Lucy Fund, and her sister, Beth Elicker, at the 4th Annual Party4Life.

THE LUCY FUND 5K

In the Spring of 2013, Beth Elicker, Lucy's sister, searched for a way to honor her sister and continue the mission of *The Lucy Fund*. After giving it some thought, Beth decided to organize the 1st Annual *Lucy Fund 5k* on June 30, 2013 in Cape Elizabeth, Maine. The event was a success with over 150 runners and walkers raising over \$10,000 to support metastatic cancer research.

THE LUCY FUND'S 6TH ANNUAL WEST END PARTY4LIFE

By the Next Generation, For the Next Generation

On July 27, 2013, the 6th Annual West End *Party4Life* was held at the Jaycee Fields in Kunkletown, PA.

"This year's *Party4Life* was particularly difficult as my mom passed away in August of 2012," said Nick Stanovick, Lucy's son and co-organizer of the event. "We are honored to carry the torch in her memory and continue the fight against metastatic cancer. My mom was an inspiration to so many, and this event carries on her legacy."

This year's event was themed "By the Next Generation, For the Next Generation", highlighting the desire of Lucy's children, Nick and Katie, and others who knew and loved her, to continue her mission for the next generation.



Sisters Jody and Kelly Hoffman share a smile before the balloon release at the 6th Annual West End Party4Life.

This year's festivities included music, raffles, karaoke, lawn games, a balloon release, dinner, dancing, and more! The balloon release was particularly moving as participants watched their balloons disappear into the sky as Lucy's favorite song, "Seasons of Love," played in the background. The activity left everyone at the party deeply grateful for the loved ones in their lives and filled everyone with the motivation to continue Lucy's mission. The community raised over \$8,000 dollars at this year's *Party4Life*, and attendees left with the confidence that *Party4Life* will continue to grow for generations to come.

PARTY4LIFE GOES TO COLLEGE

Susquehanna University hosted the first *Party4Life* on a college campus on September 28, 2013 and raised over \$1,300 to benefit *The Lucy Fund*. The day of the event, Alpha Phi Omega and Zeta Tau Alpha helped to run carnival games, musical chairs, raffles, a buffet, and a balloon release. "We were excited and honored to come together as a community, continue Lucy's legacy and do our part to raise support and awareness for metastatic cancer research," said Samantha Phillips, organizer of the event.

Play4theCure

TAKING ACTION AGAINST CANCER

In 2013, 600 sports teams joined *Play4theCure*, and together contributed \$285,000 to life-saving cancer research. Fierce rivals put aside their differences and played for a greater purpose. Softball, lacrosse, soccer and field hockey teams banded together, engaging their communities to make a difference.

The field hockey community whole heartily embraced *Play4theCure* as over 450 teams hosted events in 2013. “It is extremely important that children recognize from a young age what it means to work for something bigger than themselves. Being a part of our junior high field hockey team is more than just playing field hockey,” said fundraising coordinator Samantha Moses of Carlisle Junior High Field Hockey. “Cancer affects so many families in our communities, and it’s important that we all work together to cure cancer once and for all. *Play4theCure* is one small way that our team, families and community can donate toward eliminating cancer.



2013 Wheeler High School



Thousands of young NCFR supporters are helping us solve the cancer puzzle.



We are proud to have Longstreth Sporting Goods, a company specializing in women’s field hockey, lacrosse and softball equipment as a sponsor of NFCR *Play4theCure* initiative. Thanks to Longstreth, all *Play4theCure* field hockey teams played with the iconic *Play4theCure* game ball, and wore *Play4theCure* wristbands during their events.

Yoga, Pilates and other stretch-based exercise studios joined in on the campaign, putting a slight twist on the name by calling it *Stretch to the Cure*. More than 60 studios and wellness facilities donated proceeds from one or more classes to NFCR. Some even held special classes for the cause to raise awareness and additional funds for cancer research.



Century High School, Sykesville, MD, forms pink ribbon to raise awareness,

**PLAY
4THE
CURE**
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GOLF FOR A CURE

NFCR held its 10th Annual Memorial Golf Classic and Dinner Party on September 23, 2013 at the Kenwood Golf and Country Club in Bethesda, MD, bringing together hundreds of donors, volunteers, sponsors and participants to join in a friendly round of golf and support cancer research. The event featured a scramble-style tournament, a barbecue lunch and dinner party and award ceremony after the game. The day scored a hole-in-one and raised more than \$57,000 for NFCR



The Winning Foursome at the 10th Annual Memorial Golf Classic — From Left to Right: Jay Reilly, Kevin Lee, Mike Morrel, Ron Holsey



Event Chair Melissa Siegel with sponsor representatives from Whole Foods, Bethesda, MD



DAFFODILS & DIAMONDS

The 32nd Annual Daffodils & Diamonds Ladies Luncheon took place on March 14, 2013 at the Columbia Country Club in Bethesda, Md. Nearly 300 cancer research advocates from the community came together for a champagne reception, runway fashion show, lunch and silent and live auctions. Their generosity garnered more than \$70,000 for life-saving breast and ovarian cancer research, making for another very successful event in the long history of Daffodils & Diamonds fundraisers.



Attendees enjoying the event



Committee Members Jennifer Davidson, Gail Peterson and Polly Sturm

EXTRAORDINARY SUPPORT

2013 was distinguished by the extraordinary breadth and depth of support for NCFR. An unprecedented number of donors, corporations, foundations and institutions made gifts totaling \$13,650,000. We are deeply grateful to all of our donors for their generosity and confidence in our vision of **Research** for a **Cure**. Every gift, large and small, is an investment in new and better ways to prevent, diagnose and treat cancer. NCFR is about cancer research, for research will cure cancer.

On these pages, we are pleased to recognize those donors, corporations, foundations and institutions who made significant gifts to the National Foundation for Cancer Research in 2013.

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