RESEARCH for A cure

National Foundation for Cancer Research
2012 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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ARCHITECTURE OF DISCOVERY

The National Foundation for Cancer Research is an innovative cancer charity with a deep scientific base and a truly collaborative approach to cancer research reaching global dimensions. A leading research charity in the fight against cancer, since 1973 NFCR has spent over $300 million taking risks and funding pioneering research in angiogenesis, metastasis, targeted cancer therapies, chemoprevention, nanotechnology and bioinformatics.

All this by providing the best scientists with both the "adventure" funding to discover, as well as sustained funding to actually translate those discoveries from bench to bedside. In remarks before the American Association for Cancer Research, former *Fortune* magazine editor, Clifton Leaf, described NFCR's approach as a model for supporting research that will cure cancer:

> *NFCR…has invested in building research tools and infrastructure in a small and scalable way … to see if it works first. Management … has not been about setting new rules, filling out more forms and micromanaging what researchers can and can't do. It is about creating an environment that works...to liberate science.*

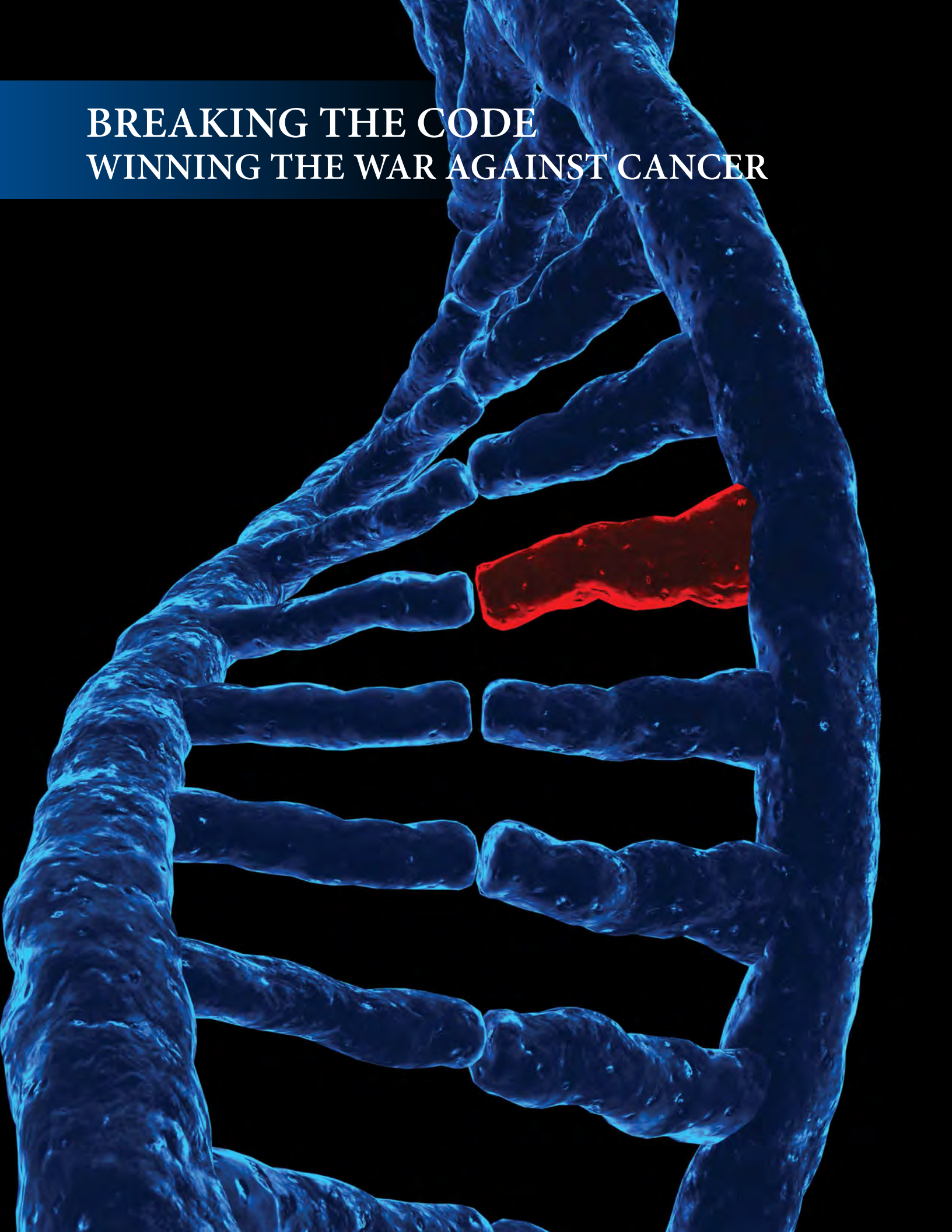
Research takes time and needs unwavering support. NFCR gives reason to hope in the progress being made against cancer — new treatments brought into the clinic, patients saved, and cures delivered. NFCR-funded research provides both the architecture of discovery and the roadmap to new approaches for treating cancer.

NFCR is about ***Research*** for a **Cure** — Cures for all types of cancer.

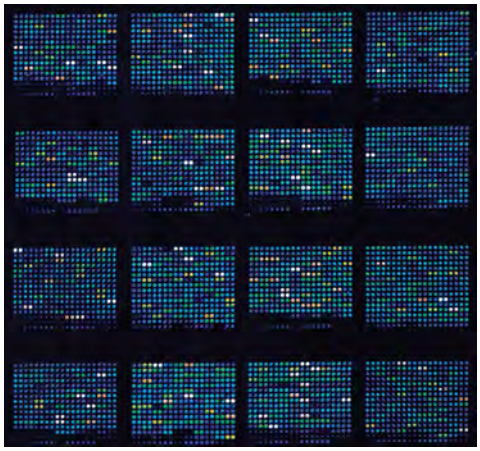
Thank you and sincerely,

Franklin C. Salisbury, Jr.
President

BREAKING THE CODE
WINNING THE WAR AGAINST CANCER

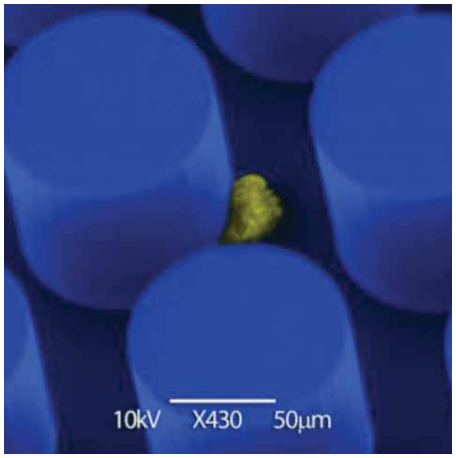


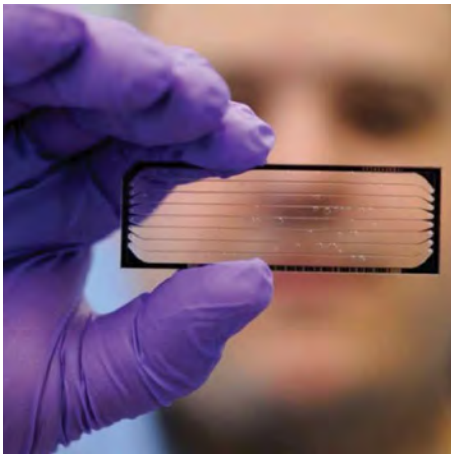
This is a time of great discovery in cancer research, but the path from a promising discovery to an effective cancer treatment often takes a decade or more. But from that process — of fits and starts, progress and setbacks and finally more progress — grow the insights and advances that change the course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



We are entering a revolutionary period in cancer therapy that is being driven by breakthroughs in cancer prevention, diagnostics and treatment. The convergence of scientific research and emerging technologies is building an unprecedented understanding of cancer as a genetic disease, driven by abnormal genes and proteins.

An important part of this revolution is a shift away from a one-size-fits-all approach to a more individualized, patient-centric approach to cancer treatment. For decades, oncologists were limited by — and treatments were confined to — what we could observe in tissues and organs. Advances in basic science research now make it possible for doctors to "see" biological processes in real time at the genetic, molecular and cellular levels.

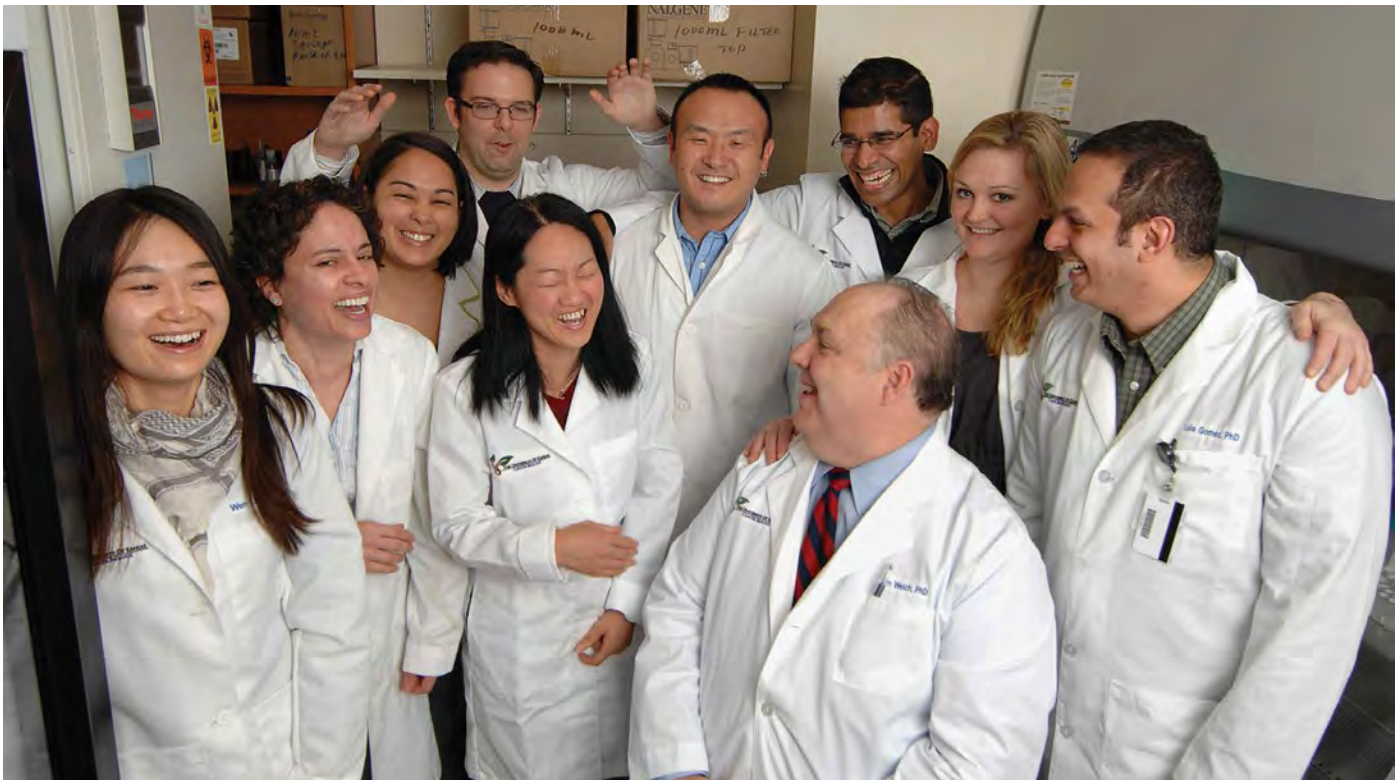




NFCR-funded scientists are investigating cancer cells in great depth and characterizing the many steps and complex mechanisms involved in the disease process called cancer. We are beginning to unravel cancer's mysteries, and are using these discoveries to develop ways to preempt cancer before it becomes life-threatening, to detect it early and to treat patients on a much more individual basis. NFCR research is setting the stage for exponential progress, and providing a foundation to eliminate suffering and death due to cancer in the next decade.

This is what we mean by ***Research*** for a **Cure**.

METASTASIS: CANCER'S MENACING BALLET



NFCR RESEARCH CENTER AT UNIVERSITY OF KANSAS CONFRONTS COMPLEXITIES OF METASTASIS

This is a time of great discovery in cancer research, and the convergence of science and advanced technologies is setting the stage to eliminate the suffering and death due to cancer. NFCR Scientists are making these breakthroughs, unraveling cancer's mysteries, and using these discoveries to treat each cancer patient as never before.

NFCR scientists are identifying previously unrecognized steps and complex mechanisms involved in the disease processes we call cancer. Not the least of which is the genetic heterogeneity of cancer cells. The cancer cells within each patient's tumor are different, and NFCR researchers are finding that metastasis, one of the ten "hallmarks" originally thought common to all cancer cells, uses a different playbook.

When cancer cells metastasize, the fallout is devastating. Metastasis is the ultimate cause of death in the vast majority of cancer patients: fewer than 10% of cancer deaths are caused by the primary tumor; 90% stem from metastases in vital sites like the lungs, liver, bones and brain. Yet fewer than 8% of the scientists applying for research dollars even mention the word "metastasis." This is a very frightening statistic and translates to less than 5% of today's research dollars going towards studying cancer metastasis.

The National Foundation for Cancer Research is one of the few cancer research charities that focuses on metastasis. And leading this research is Danny Welch, PhD, founding director of the NFCR Center for Metastasis Research at the University of Kansas Medical Center (KUMC) where the focus of Dr. Welch's research is on the science of tumor progression and the regulation of cancer metastasis.

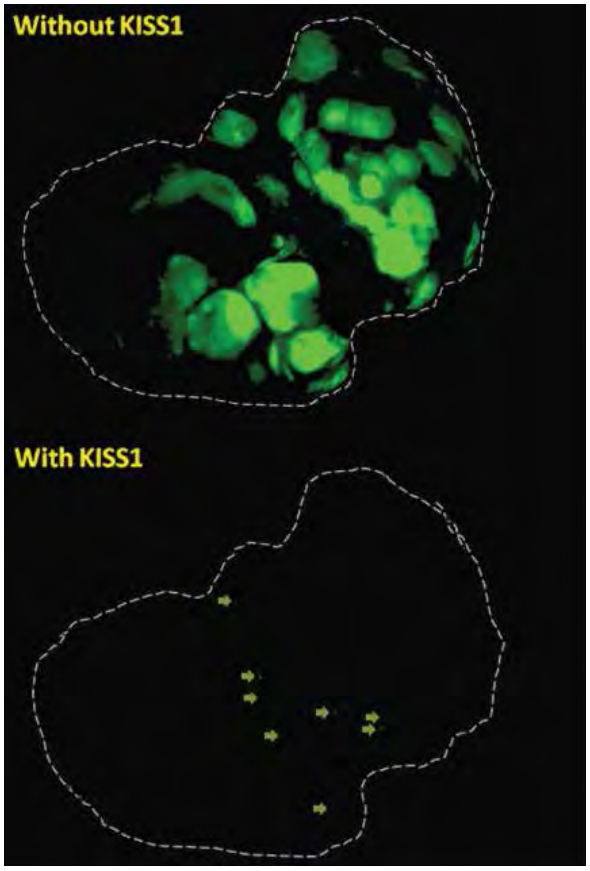
Researchers at the NFCR Center for Metastasis Research at KUMC are focused on research to suppress the metastatic growth of cancer. To that end — and with NFCR support — Welch has now discovered eight genes that suppress metastasis. Genes that specifically regulate the process of metastasis have already proven to be useful tools to elucidate molecular mechanisms and are showing great promise to become markers and/or targets for anti-metastatic therapy.

Research being directed by Welch is proving instrumental in finding better ways of pinpointing and reining in metastatic tumor cells.

In order to do this, scientists first need to understand the genetic programs and molecular mechanisms that drive cancer metastasis.

For starters, not every tumor invades or metastasizes. And then, metastasis involves more than the ability of some cancer cells to leave the original tumor and spread to other parts of the body. Metastasis is better explained by likening tumors to seeds that will grow only in the right type of soil and in the correct climate. Welch says this explains how metastases occur preferentially in certain organs and why some people have aggressive cancers and why others do not.

Before cancer cells leave the primary tumor, they begin communicating with cells in a patient's bone marrow. Those noncancerous bone marrow stem cells then go to different sites of the body and appear to pave the way for metastatic tumor cells to follow. The cancer cells follow the bone marrow cells, adhere and then form cancerous colonies in their new location. Colonization is a key component of metastasis, but cancer researchers only recently began to recognize the importance of this final colonization step, due in part to work published by the NFCR Center for Metastasis Research at KUMC.



THE STORY OF KISS1

Welch has been hard on the heels of metastasis since 1989. The first gene capable of quashing this deadly process, NM23, had been discovered and Welch was convinced there were others. Several scientific publications from the 1950s — including the first complete chromosome analysis of malignant melanoma cells — had also caught his eye.

Then, while on the faculty at the Pennsylvania State University College of Medicine in Hershey, Welch observed that as melanoma cells switched from benign to malignant, a particular piece of chromosome 6 vanished about 80 percent of the time. He hypothesized that replacing this missing chromosome would reduce the cells' malignancy — specifically their ability to spread — akin to repairing a runaway vehicle's faulty brakes.

His hunch proved correct. By comparing cells with and without chromosome 6 — searching for genes that slowed metastasis — Welch discovered a gene he called **KISS1.**[1] To figure out how this gene might muzzle metastasis, Welch's NFCR research team fluorescently labeled **KISS1**-expressing melanoma cells in mice, turning them green and tracking them to the lungs. After nine months, the melanoma cells which expressed **KISS1** gene did not colonize or form tumors in the lungs. When isolated and re-injected under the skin, those same melanoma cells traveled back to the lungs but failed to form tumors there.

Thus, **KISS1** allows every step of metastasis except growth at the secondary site. In other words, **KISS1** works to suppress metastasis by preventing the final colonization step of metastasis:. Scientists at the NFCR Center for Metastasis Research have now shown that beyond melanoma, the **KISS1** gene also blocks the spread of ovarian, pancreatic, prostate and breast cancer cells.

Critics have suggested that studying metastasis is as futile as closing the stable door after an escaped horse. But the research being done at the NFCR Center for Metastasis Research leads Welch to a very different conclusion — it may not be necessary to corral the horse back into the stable, if it can be kept from running amok in the pasture.

If doctors could somehow "turn on" the **KISS1** gene or replace the protein made by the **KISS1** gene, Welch believes that physicians could hold cancer cells that have already spread in a dormant state. As a result, metastatic cancer could conceivably become a chronic, controllable disease. By translating **KISS1** and other metastasis suppressor genes for patient use, scientists at the NFCR Center for Metastasis Research at KUMC are working to develop a new approach to treating cancer.

Imagine if Welch could invent a switch to turn on the **KISS1** gene in cancer patients. This could be an important new approach to treating multiple types of cancer. Still, there are plenty of unanswered questions. If **KISS1** is given after metastases have started to grow, will it still work? How long will **KISS1** hold cells in a dormant state? Will there be a carryover effect enabling them to stay dormant? Is **KISS1** working directly on the tumor cells or is an intermediary — think a hired assassin — involved? Would examining **KISS1** in tumors help doctors determine which patients wouldn't need to undergo nasty chemotherapy?

Such are the important questions the NFCR Center for Metastasis Research is tackling with your support.

[1] Welch originally called this gene "SS1" for "Suppressor Sequence 1", but colleagues told him "it was a boring name no one would remember." So he added the letters 'KI' in front, spelling "KISS1", to remind people this gene was discovered in Hershey, Pennsylvania. When Welch discovered BRMS1 (Breast Cancer Metastasis Suppressor 1), he wanted to create a matching set and name it "Hug1," but the Genome folks said "no". Scientists have now identified 30 metastasis suppressor genes, eight of them discovered by Dan Welch with NFCR-funding.

ACCELERATING DISCOVERY RESEARCH HIGHLIGHTS

The National Foundation for Cancer Research is an innovative cancer charity with a deep scientific base and a truly collaborative approach reaching global dimensions. NFCR accelerates the pace of cancer research by recognizing innovative research while it is still in its infancy, and by providing scientists with the "adventure" funding to discover what they are then able to seek government funding to prove. Like a "Laboratory Without Walls," NFCR promotes the sharing of ideas and information by engaging top researchers across a wide range of scientific disciplines, and from many research institutions.

With the grassroots support of millions of Americans, the black box that was the cancer cell has been opened, and NFCR scientists have pioneered the redefinition of cancer as a genetic disease, making possible new approaches to treating cancer and transforming medicine so that real hope for a cure is now within sight.

NFCR scientists are at work on new anti-cancer drugs that target the very genes and genetic pathways that make a cell cancerous. These new targeted cancer therapies are proving more effective, longer lasting and far less toxic than radiation and chemotherapy.

Today, more individuals diagnosed with cancer are surviving longer than ever before. Even those who ultimately succumb to cancer live longer and experience a much better quality of life than was possible just a few years ago. Every day at NFCR, our researchers report progress in the development of promising new treatments for cancer. But until there is a cure, we will not be satisfied — too many lives are at stake.

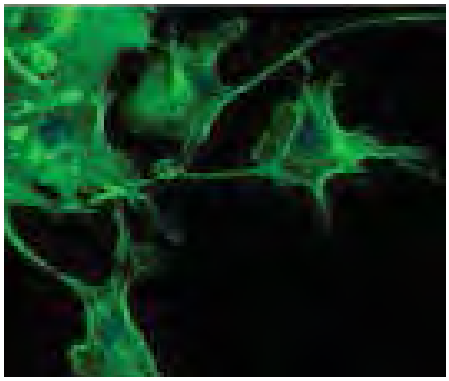
A leading research charity since 1973, NFCR has spent over $300 million to fund "high risk/high reward" cancer research at universities and research hospitals worldwide. The research funding we've provided is having a catalytic effect, and accelerating the pace of cancer research. In laboratories across the United States, England, Germany, and China, NFCR scientists are moving cancer research toward that ultimate goal — finding cures for all types of cancer.

EARLY CANCER DETECTION AND MONITORING

Early detection and monitoring is critical to mounting effective cancer treatments. The molecular differences that make cancer cells lethal when left unchecked also provide the clues for their detection, identification and visualization. We are developing new methods in molecular imaging technologies to develop highly sensitive cancer detectors. And, by combining implantable detectors with wireless data transmission technologies, we envision the development of tools for continuous monitoring during and after cancer treatment to signal remission and relapse, or even trigger micro-scale drug delivery systems for automatic therapeutic interventions.

CTC CHIP DETECTS CANCER IN BLOOD

Daniel A. Haber, M.D., Ph.D., Massachusetts General Hospital, has developed a revolutionary way to detect and capture circulating tumor cells (CTCs) in the blood. This technology may provide doctors with an unprecedented means of rapidly detecting invasive cancers by using an easily administered blood test. Licensed by *Johnson & Johnson,* the CTC Chip gives doctors the ability to detect the presence and genetic features of cancer cells circulating in a patient's blood — long before they become tumors. The CTC Chip revolutionizes early detection, so doctors can identify and prescribe targeted anti-cancer 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 treatment changes, increasing the positive effect of all cancer therapies.



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. Utilizing an entirely new technique that permits the simultaneous imaging of multiple molecular markers, scientists in this Center make 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 an operation and make it possible for more complete surgical removal of infiltrated tumor tissue such as brain cancer. One molecular imaging approach is currently being tested in a clinical trial to assess 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. In 2012, Dr. Bast's team began to identify new blood biomarkers to add to the existing CA125 biomarker test. They are developing a more sensitive imaging technology called Superconducting Quantum Interference Device (SQUID), improving the sensitivity to detect tiny, early-stage tumors by several orders of magnitude over current imaging technologies such as CT, MRI and PET-CT. 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 time that would offer the best opportunity for a cure for many patients.

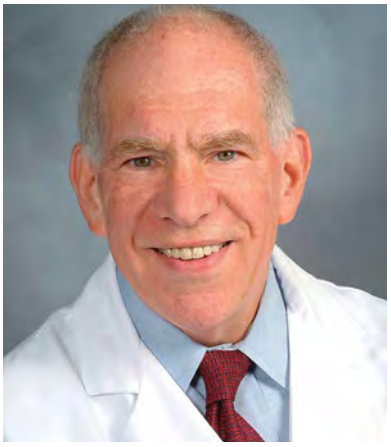
ADVANCING PERSONALIZED MEDICINE THROUGH ANALYSIS OF CANCER PATHWAYS AND DRUG RESISTANCE

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 brain cancer and creating innovative therapeutic approaches. In 2012, his team has made novel findings that extend our understanding of two of the cardinal features of brain tumors that make them so lethal: their ability to move around and invade normal brain tissue and their ability to take over normal blood vessels to provide the tumor with vital nutrients to grow. These results offer new insights into how brain cancer grows and thrives and suggest that a therapeutic approach that targets these cancer processes, in combination with mutation-directed therapies, might have increased effectiveness for patients.

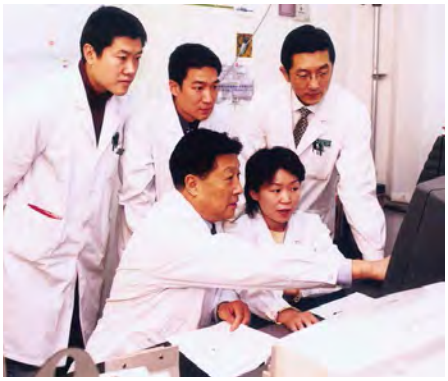
Ronald G. Crystal, M.D., Weill Medical College of Cornell University, is conducting research in collaboration with Dr. Viviane Tabar at Memorial Sloan Kettering Cancer Center on using the novel approach of recombinant proteins and antibodies to develop gene therapy to convert brain cells into antibody-producing cells. These antibodies will target the cancer cells in glioblastoma, the aggressive and currently uncurable brain cancer. The researchers at Dr. Crystal's lab have developed strategies and the technology to successfully deliver genes to the central nervous system, and this research will expand the technology and its application to cancer.



BIOMARKERS AND NEW THERAPEUTIC TARGETS

Curt I. Civin, M.D., University of Maryland. MicroRNAs (miRs) are potent regulators of normal and cancerous growth and may potentially be effective clinical agents or targets; the inefficient clinical delivery of miRs or miR-inhibitors is not a fully-solved problem. Dr. Curt Civin knows first-hand the involvement of miRs in leukemia, having previously discovered two miRs that regulate leukemia. In 2012, Dr. Civin also focused on miR-34, another miR which normally serves to suppress cancer growth. However, low levels of miR-34 in leukemia and other cancer types diminish its role on reducing these cancers. Dr. Civin's team identified, via high-throughput screening of a clinical compound library, a set of candidate drugs that upregulate levels of miR-34 in cells. One drug, Artemisinins, a new class of antimalarials, has activity against leukemia cell lines. Research is investigating how Artmisinins upregulate miR-34. This class of drugs may be repurposed for a new approach for leukemia therapy.

Wei Zhang, Ph.D., MD Anderson Cancer Center, is identifying microRNAs (miRs) — 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 miR biomarkers in patient's plasma for early detection, staging, predicting treatment response, and evaluating prognosis. Dr. Zhang's recent work has identified miR-141, 148a and 96 to be good biomarkers for late stage colorectal cancer in Caucasian patients. These promising biomarkers will be further validated using samples from the NFCR-TMUCIH Joint Tissue Bank at the Tianjin Medical University Cancer Institute and Hospital (TMUCIH) in China, to test if similar results are found in Asian colorectal cancer patients. Biomarkers will allow physicians to diagnose colorectal cancer earlier and tailor efficacious treatment for their patients — giving them the hope they deserve to fight their cancer successfully.



Xishan Hao, M.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, and are revolutionizing modern cancer therapy.

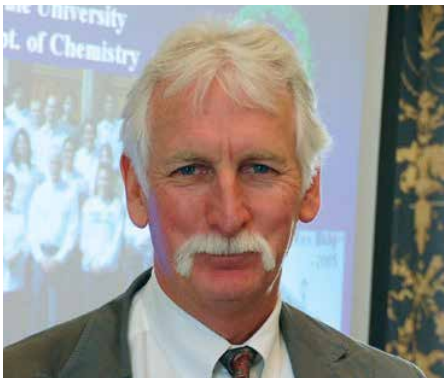
Scientists at the TMUCIH-NFCR Joint Tissue Bank collect and maintain biospecimens (tumor tissues and matching blood samples) from cancer patients fighting all types of cancer; this rapidly growing biorepository includes more than 31,000 fresh frozen tissue samples and over 30,000 blood samples.

The TMUCIH-NFCR Joint Tissue Bank is part of an 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.

ANTI-CANCER DRUG DESIGN AND DISCOVERY

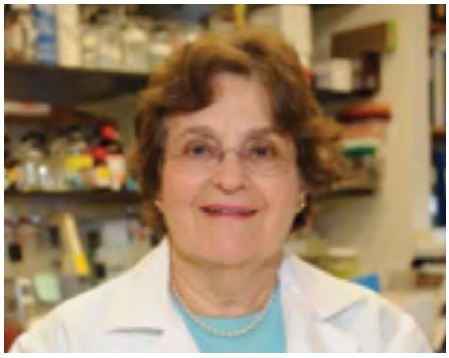
Alanna Schepartz, Ph.D., Yale University, has developed anti-cancer beta-peptide inhibitors to address one of the biggest challenges in drug discovery. Beta-peptide inhibitors represent a new generation of anti-cancer drugs that are highly effective and specific in targeting almost any cancer-related protein-protein interaction. Dr. Schepartz is designing beta-peptides against protein interactions involving PTHrP, a protein involved in breast cancer metastasis to bone. Beta-peptide inhibitors are a new platform technology that may positively impact the treatment of all major types of cancer.



William Jorgensen, Ph.D., Yale University, is a pioneer in the field of computational chemistry, developing methodology and computer software to rapidly and cost-effectively develop novel targeted drugs. Dr. Jorgensen has successfully applied his methodology to efficiently obtain a novel, potent anti-HIV drug and an anti-inflammatory agent for arthritis. In 2012, his team optimized and evaluated several potent lead compounds that inhibit FGFR1 (Fibroblast Growth Factor Receptor), which is implicated in pancreatic, breast, brain, salivary gland, ovarian, and prostate cancers, as well as Kaosi's sarcoma. With further testing and refinement, these potential drugs that target FGFR1 may be rapidly brought into the clinic as a new treatment approach.

Alan C. Sartorelli, Ph.D., Yale University School of Medicine, is a pharmacologist who designed laromustine, a drug demonstrating promising treatment benefits for patients with Acute Myeloid Leukemia (AML), other types of leukemia, brain tumors, lung and other types of cancer. Laromustine is a member of a class of chemotherapy agents called guanine O6-targeting drugs which modify cellular DNA. To make laromustine and similar agents target only cancer cells, Dr. Sartorelli has designed an inactive drug that converts to an active one only in low-oxygen or hypoxic cancer cells. With his innovative design, Dr. Sartorelli envisions these new targeted drugs will be effective in tumors that have been resistant to therapeutic intervention, providing hope to AML and other cancer patients that their cancer can be effectively treated.

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 2012, Dr. Schimmel's team discovered that one type of AARS, glycyl-transferase (GRS), is a potent anti-cancer agent for tumors which grow through tumor activator, ERK. Results show that GRS works through a cancer cell biomarker to shut down tumor activation by ERK. The biomarker and ERK are both associated with metastatic kidney cancer and small cell lung cancer. Patients are in dire need of new treatments as these cancers become resistant to standard therapies. GRS has the potential to become a therapeutic approach to help patients fight these cancers.



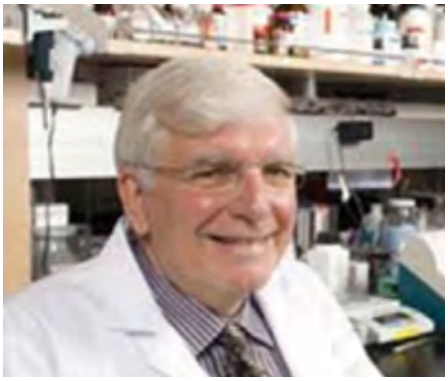
Susan Band Horwitz, Ph.D., Albert Einstein College of Medicine, is a leading pharmacologist who deciphers how tumors develop resistance to Taxol, and develops new strategies to overcome its resistance in tumors. In 2012, her team developed the first antibody to betaV-tubulin, a protein that may have a role in cancer-causation and in the resistance to Taxol. Antibody results indicate that betaV-tubulin may be a potential prognostic marker of breast, lung, ovarian, and prostate cancer. Also this past year, her team published their work on a promising combination therapy for ovarian cancer. Dr. Horwitz's team have shown that combining a MEK inhibitor with an estrogen receptor antagonist synergistically reduces tumor growth and the development of drug resistance compared to each single agent. Future plans to bring this drug combination into the clinic as a phase 1 trial are underway. The availability of such a combination approach could bring an effective treatment for the two-thirds of ovarian cancer patients who lose their lives due to drug resistance.

Stanley Cohen, M.D., Stanford University School of Medicine, is working to identify genes and understand how they may enable cancers to develop resistance to initially effective therapeutic agents. New target therapies against these genes can be developed to reduce or eliminate the resistance to the main therapy. His team recently discovered the gene, Txr1, whose expression alters the susceptibility of cancer cells to Taxol, the widely-used anti-cancer drug for treating lung, breast, ovarian, and prostate cancers. In 2012, his research showed that inhibiting another protein that modifies Txr1 proteins can stop the resistance to Taxol. Research continues by Dr. Cohen's team to further validate these findings with Txr1 as a way to provide a new therapeutic approach to reducing the resistance to Taxol.

Kathryn B. Horwitz, Ph.D., University of Colorado at Denver and Health Sciences Center, is working with breast cancer patients enrolled in a clinical trial to identify genes that can predict which tumors will respond to hormone therapies, and which will be resistant, even if the growth of those tumors is also fueled by estrogens. New biomarkers discovered through Dr. Horwitz's research will help doctors to provide a right therapy to the patients, saving precious time by avoiding treatments that won't work.

Waun Ki Hong, M.D., MD Anderson Cancer Center, is leading the development of personalized molecular targeted therapies for lung cancer. His NFCR-funded Biomarker-Based Approaches of Targeted Therapy for Lung Cancer Elimination project (BATTLE) 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) and mesothelioma.

Dr. Hong's ongoing research in this area holds extraordinary promise to prolong progression-free survival of these deadly cancers. This ground-breaking program is now serving as a model for personalized medicine throughout the world for other cancer types as well.
Dr. Hong's team is forging ahead with several newly initiated BATTLE clinical trials that will follow a similar strategy: BATTLE-2; BATTLE Prevention; and BATTLE Neoadjuvant. These programs have the potential to make major contributions to the field of lung cancer by providing a better understanding of how this disease progresses, identifying the genes and pathways responsible for NSCLC tumors, and developing individualized approaches to more effectively target them therapeutically and ultimately, to work towards prevention of this deadly disease.



Daniel Von Hoff, M.D., Laurence Hurley, Ph.D., Co-Directors, NFCR Center for Targeted Cancer Therapies, TGen, are developing new targeted cancer therapies and improving the treatment efficacy of existing therapies for pancreatic cancer. During 2012, their team identified and developed agents that specifically target the mutant K-Ras gene which is found in almost all of the most aggressive types of pancreatic cancer. Initial results are promising. Additionally, using a new approach that involves targeting this gene at the DNA level, Drs. Hurley and Von Hoff have successfully prepared a group of novel compounds that are very potent against pancreatic cancer cells and significantly reduce the level of K-Ras protein in the cells. Additive effects on reducing K-Ras protein were demonstrated when these new compounds are combined with other agents currently used to reduce K-Ras levels. This approach of targeting the K-Ras gene at the DNA level offers a new opportunity to treat pancreatic cancer not previously used.

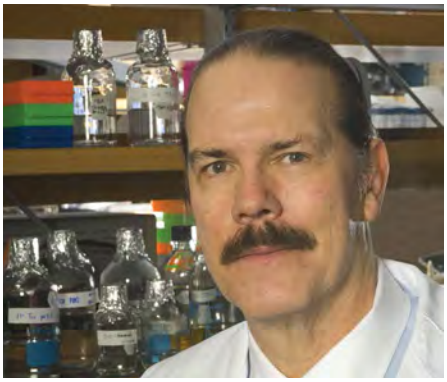
ANGIOGENESIS: SHUTTING DOWN CANCER

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 cell 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 demonstrate that the therapeutic effects of individual anti-angiogenic drugs distinguish among different types of tumor blood vessels. This research has significant clinical impact as it identifies the strengths and weaknesses of a new anti-angiogenic drug for treating cancer.

Rakesh K. Jain, Ph.D., Massachusetts General Hospital, has discovered new ways of preventing resistance to anti-angiogenic therapy in glioblastoma patients. 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. In 2012, Dr. Jain's team identified that inflammatory cells called macrophages are increased in tumor areas after anti-angiogenic therapy. The number of macrophages directly correlates with shorter survival time for patients. These exciting results suggest 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 anti-angiogenic therapy. If this therapeutic strategy can be developed, it will give glioblastoma patients renewed hope of winning the battle against this aggressive brain cancer.

INNOVATIVE IMMUNOTHERAPY



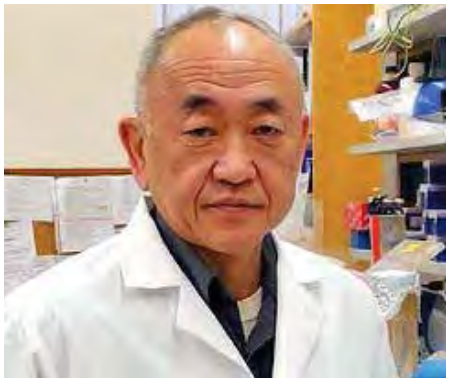
Paul B. Fisher, M.Ph., Ph.D.,Virginia Commonwealth University School of Medicine, has developed a novel gene therapy to treat early stage and metastatic prostate cancer, the second leading cause of cancer-related deaths in men in the U.S. This new therapeutic is a genetically reprogrammed virus, designed to specifically infect and destroy only tumor cells, leaving normal cells unharmed. The virus also delivers a natural product of our immune system, interferon gamma, which will seek out and destroy cancer cells that have metastasized. Dr. Fisher's team has now employed an ultrasound contrast agent, microbubbles, to deliver the effective therapeutics to the primary tumor site and to metastatic cancers. In addition, the microbubbles protect the virus from elimination by the immune system. This powerful new gene therapy approach is making steady progress towards treating patients with advanced prostate cancer in a clinical trial.

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. In 2012, Center researchers developed two high-affinity human sFv antibodies against a unique domain of a selected cancer target for renal cell carcinoma. These reagents are being developed as new immunotherapies and diagnostic tools for kidney cancer patients who currently have no effective cancer treatment. Recent research indicates increasing types of cancer cells also have the selected target suggesting these new antibodies may have a broader use in cancer therapy.

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 leukemia and lymphoma. The safety and feasibility of this novel immunotherapy continues to be demonstrated in his team's phase I clinical trials in patients with CD19+ lymphoma.

Currently, it can take up to 6 weeks to customize the engineered T cells for infusion into patients, during which time the patient's cancer may progress, rendering them too ill to receive the therapy. In 2012, Dr. Cooper's team forged ahead and began developing a new approach in which the modified tumor-specific T cells can be prepared from a single donor and infused into multiple recipients with CD19+ malignancies as an "off-the-shelf" therapy. The engineered cells can be pre-assembled and frozen so they can be simply thawed and infused "on demand."

CHINESE HERBAL MEDICINE: ADJUNCT TO CHEMOTHERAPY



Yung-Chi Cheng, Ph.D., Yale University School of Medicine. While the therapeutic effects of traditional Chinese medicines have been documented for centuries, they have been too often discounted by modern medicine as "alternative therapy" because there was little scientific proof that they could work. For the last 11 years, with NFCR support, Dr. "Tommy" Cheng has explored the therapeutic properties of PHY906, one of the ancient Chinese herbal medicine formulas. PHY906 could become one of the first FDA-approved oral herbal medicines for anti-cancer treatment. PHY906 will soon enter phase II trials where it is combined with chemotherapy in the treatment of colorectal cancer patients. Earlier trials have shown it alleviates unpleasant gastrointestinal side effects of chemotherapy. Research results have shown PHY906 also has anti-cancer properties and the trial will also investigate for this clinical benefit as well. PHY906 could also potentiate Sorafenib, the only approved drug

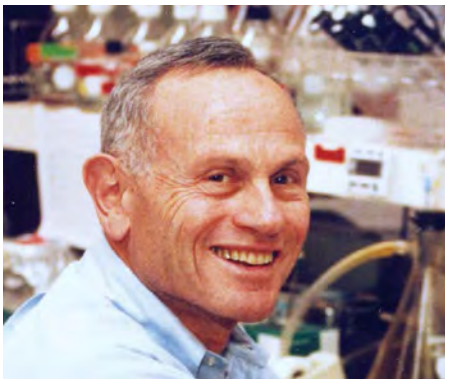
for treatment of patients with liver cancer, and a new clinical trial will begin soon to test this combination of drugs. The outcomes of these clinical trials could have a major impact for the treatment of colorectal and liver cancer patients in the future.

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 RNA interference (RNAi) molecule 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.

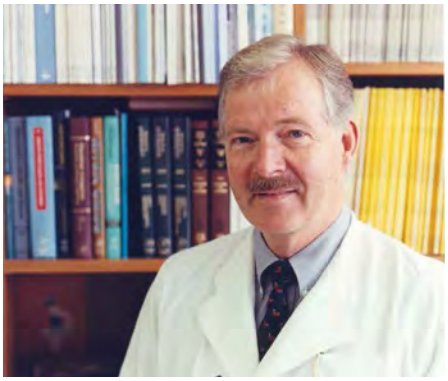
Esther H. Chang, Ph.D., Georgetown University, has developed a nanoscale, liposome-based tumor targeting drug delivery system that carries anti-cancer agents directly to both primary and metastatic tumor cells, significantly enhancing a tumor's sensitivity to chemo- and radiation therapy. A nanocomplex carrying tumor suppressor gene, p53, has already proved to be a safe, non-toxic potential targeted therapy and is now in a phase IB clinical trial treating patients with certain types of solid tumors. In 2012, using ovarian cancer models, 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 the first line treatment of chemotherapy, leaving patients with no effective treatment. These promising results give hope to women with ovarian cancer that the nanocomplex of p53, combined with chemotherapy, may be an effective treatment approach to save their lives.

CANCER PREVENTION



Michael B. Sporn, M.D., Dartmouth Medical School, is developing new triterpenoid compounds for the prevention and treatment of cancer. His highly fruitful research has resulted in several triterpenoid compounds which have potent preventative effects against liver cancer, melanoma, and highly aggressive lung cancer. Two of these agents have been evaluated in clinical trials for cancer treatment. In 2012, Dr. Sporn's team demonstrated that triterpenoids combined with other anti-inflammatory agents such as histone decarboxylase inhibitors were effective in preventing pancreatic, breast and lung cancer in tumor models. Moreover, the combination of agents was significantly more effective than the single agents alone. These new results demonstrate that a promising approach for chemoprevention of cancer is through drug combinations.

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. Cells that are rapidly dividing 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.



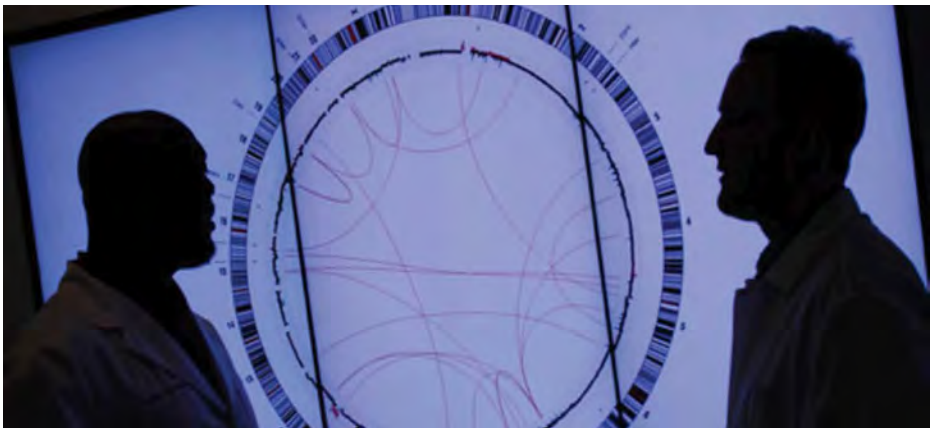
Helmut Sies, M.D., Heinrich-Heine-Universität, Düsseldorf, Germany, is well recognized for his discovery of the skin cancer prevention effects of the micronutrient, lycopene, the antioxidant found in tomatoes and carrots. Another focus of Dr. Sies' team is on selenium (Se), a trace metal essential for good health. Dietary or supplemental Se is incorporated into selenoproteins — critical cell proteins that have anti-oxidation functions. Expression of a selenoprotein has been shown to be suppressed in cancer tissue of colorectal cancer patients. In the last year, Dr. Sies' team showed that various dietary Se compounds stimulate the secretion of selenoproteins and this may protect the intestine from oxidative damage. Research will continue to delineate how dietary Se compounds are metabolized and how intestinal selenoproteins are involved in selenium-mediated colorectal cancer prevention. Dr. Sies' invaluable research results will help guide future cancer prevention trials to test more specifically if dietary Se can prevent colorectal cancer.

FIRST WHOLE GENOME SEQUENCING OF MULTIPLE PANCREATIC CANCER PATIENTS AT NFCR CENTER FOR GENOMIC MEDICINE

Research at the NFCR Center for Genomic Medicine at TGen in Scottsdale has two goals. Goal one is to get the best possible targeted new anti-cancer agents for cancer patients now. Goal two is to make a major impact against pancreatic cancer.

Pancreatic cancer has the worst survival rate of any cancer and is the fourth leading cause of cancer deaths in the United States. NFCR scientists are developing targeted therapies for patients with pancreatic cancer based on distinguishing characteristics of pancreatic cancer cells versus normal cells.

This approach relies on being able to identify new targets in pancreatic cancer cells, and to develop agents that hit these targets and preferentially kill pancreatic tumor cells.



In ground-breaking research[2] by scientists at the NFCR Center for Genomic Medicine, researchers demonstrated that the use of whole genome sequencing represents a compelling solution to obtaining detailed molecular information on tumor biopsies in order to provide guidance for therapeutic selection.

Whole genome sequencing — spelling out all 3 billion letters in the human genome — is a powerful method for making an impact against pancreatic cancer each patient at a time.

Using next-generation, or whole genome sequencing, researchers were able to generate an average of 132 billion mappable bases, or data points, for each patient, resulting in the identification of 142 cellular genetic coding events, including mutations, insertions and deletions, and chromosomal copy number variants.

This research was the first to report whole genome sequencing findings in paired tumor/normal samples collected from separate pancreatic cancer patients. By sequencing the cancers from each patient, researchers found multiple potential therapeutic targets, highlighting the need to examine the full spectrum of the genome and re-emphasizing the need to develop multiple avenues of therapeutics to match the specific medical challenges of each patient. Cellular pathway analysis of sequencing data from each patient was performed to identify processes that may be the most heavily impacted by cellular and gene expression alterations.

Cancer, and specifically here, pancreatic cancer, is a highly complex disease that ultimately will require a variety of treatment methods to control, and ultimately cure the disease. As researchers at the NFCR Center for Genomic Medicine continue to generate more information by sequencing the whole genomes of each patient, they will continue to discover the specific mechanisms that cause this cancer.

By continuing to sequence the whole genomes of cancer patients, clinical researchers will acquire a better understanding of the compendium of events that have a role in the disease, and strengthen our knowledge base for identifying and developing improved therapeutics. Research being done at the NFCR Center for Genomic Medicine has demonstrated the feasibility of applying whole genome sequencing approaches toward eventual personalization and precision therapy for patients with pancreatic cancer, and the application of this approach to cancer patients in the clinic.

Whole genome sequencing provides oncologists with the genetic blueprint and knowledge that is needed to crack the complex mysteries surrounding pancreatic cancer. This NFCR research directed by Dr. Von Hoff is a major step forward in the quest to apply whole genome sequencing to treating cancer in a real time fashion. Whole genome sequencing is a new approach toward finding better treatments and to making these treatments available to cancer patients who need them now. This is ***Research*** for a **Cure.**

[2]Daniel Von Hoff, MD, et. al., Genome-Wide Characterization of Pancreatic Adenocarcinoma Patients Using Next Generation Sequencing, PLoS ONE 7(10): e43192. doi:10.1371/journal.pone.0043192 (2012)

7TH SZENT-GYÖRGYI PRIZE

ACUTE PROMYELOCYTIC LEUKEMIA FROM HIGHLY FATAL TO HIGHLY CURABLE

Dr. Zhen-Yi Wang, and Dr. Zhu Chen were awarded the 7th Szent-Györgyi Prize for Progress in Cancer Research. The Prize was presented to Dr. Wang and Dr. Chen at an awards ceremony March 6, 2012 at the Westin Times Square in New York City. Former Congressman and healthcare leader — a cancer survivor himself — W.J. "Billy" Tauzin hosted the awards ceremony; Wei-Wu He, Ph.D., CEO, OriGene Technologies, Inc., delivered the keynote address.

Their's was truly innovative research; by combining arsenic trioxide of traditional Chinese medicine with western medicine, Dr. Zhen-Yi Wang and Dr. Zhu Chen successfully developed a new therapeutic approach to acute promyelocytic leukemia (APL). Drs. Wang and Chen provided dramatic improvement in the five-year disease-free survival rate of APL patients from approximately 25% to 95%, making this combination therapy a standard of care for APL treatment throughout the world — turning one of the most fatal diseases into a highly curable one.

A hematologist and clinical researcher at the Shanghai Second Medical University and Ruijin Hospital, Dr. Zhen-Yi Wang performed the first successful induction therapy on APL patients using all-trans retinoic acid (ATRA) which significantly increased the survival rate of APL patients. Dr. Zhu Chen — who spent years as a "barefoot doctor" in the countryside during the Cultural Revolution where he used many traditional Chinese remedies — pursued a medical degree at Shangrao Health School in Jiangxi, and started his career as a medical scientist working under Dr. Zhen-Yi Wang at the Shanghai Second Medical University. After his graduate studies with Dr. Wang, Zhu Chen continued his research in Paris at the Central Hematology Laboratory at Saint-Louis Hospital where he realized the transition from hematologist to molecular biologist, returning to the Shanghai Institute of Hematology at Rui Jin Hospital where he pieced together the complex picture of how chemistry might fight APL.

Previous research by Dr. Wang had demonstrated that ATRA is successful in treating most APL patients by inducing differentiation in affected cells. By further analyzing the genetics and phenotype of APL, Dr. Chen identified key mechanisms in ATRA as well as in arsenic trioxide — a compound used in traditional Chinese medicine for over 2,400 years — which led to remission in those APL patients for whom retinoic acid and chemotherapy had failed.

Dr. Sujuan Ba, Co-Chair of 2012 Prize Selection Committee; Dr. Zhen-Yi Wang, 2012 Prize Co-Recipient; Dr. Carlo M. Croce, 3rd Szent-Györgyi Prize Winner; and Dr. Zhu Chen, 2012 Prize Co-Recipient.

Dr. Chen hypothesized that the arsenic-containing remedies of traditional Chinese medicines might improve retinoic acid therapy, and that the two combined would be even more effective in treating APL.

His hypothesis proved correct and, working together, Drs. Zhu Chen and Zhen-Yi Wang combined ATRA and arsenic trioxide in clinical trials where they successfully demonstrated that the two drugs work synergistically to not only kill malignant cells, but also promote apoptosis, curing the disease in the majority of APL patients.

Through research such as this, Dr. Zhu Chen and Zhen-Yi Wang have transformed leukemia therapy, changing the face of medicine for those patients suffering from APL. "Their combined work has saved and will save millions of lives around the world both today and for future generations," said Dr. Beatrice Mintz, winner of the 6th Szent-Györgyi Prize and Co-Chair of the 2012 Prize Selection Committee. "I cannot imagine a better testament to the outcomes of investing in cancer research than what these two distinguished scientists have achieved."

In keeping with the spirit of nonconformity that NFCR co-founder, Albert Szent-Györgyi is known for, the selection of Drs. Wang and Chen has a significant meaning to those who work in the trenches of cancer research each day," said Sujuan Ba, Ph.D., Co-Chair of the Szent-Györgyi Prize Selection Committee and Chief Operating Officer at NFCR. "True scientific discovery comes from innovative ideas and dedicated research. These two scientists are inspirational as they both have devoted their lives to this work that will impact the world for generations to come."

"I am so glad to see that the efforts we have devoted to research on Leukemia these last several decades have led to solid clinical benefits to APL patients around world. We will continue our efforts in finding more effective therapies to treat cancers," said Dr. Wang. "It is quite humbling to know that our colleagues across the various scientific disciplines selected us. Scientists across the globe are working every day to cure cancer. I hope our work may continue to inspire others," said Dr. Chen, who in 2007 was appointed Chinese Minister of Health. "This is a great honor for Dr. Wang and me."

The Prize is named in memory of NFCR Co-Founder, Albert Szent-Györgyi, M.D., Ph.D., who won the Nobel Prize for Physiology and Medicine in 1937 for his discovery of vitamin C. The Szent-Györgyi Prize is awarded annually to a scientist, nominated by colleagues or peers, who has contributed outstanding, substantial research to the fight against cancer and whose accomplishments have helped improve treatment options for cancer patients.

CHINA-U.S. SYMPOSIUM

FUTURE DIRECTION OF GLOBAL COLLABORATION

The 2012 China-U.S. Symposium was a transformative opportunity for global collaboration and could well accelerate the development of new cancer therapies by building successful clinical trial networks for global oncology drug development programs

While in New York to receive the 7th Szent-Györgyi Prize for Progress in Cancer Research, Chinese Minister of Health Dr. Zhu Chen proposed that NFCR organize a special meeting of U.S. and Chinese cancer research scientists to develop a partnership between NFCR, the Chinese Ministry of Health, and Chinese Academy of Medical Sciences (CAMS). Minister Chen explicitly proposed that NFCR work to create the architecture for high-level U.S.-Chinese scientific collaboration to advance cancer prevention and treatments for several major types of cancer.

The China-U.S. Symposium on the State-of-the-Art of Cancer Research and Future Directions for Global Collaboration was an important first step towards promoting the advancement of translational and molecular medicine through more effective U.S.-China collaboration in cancer research and cancer therapy development.

The Symposium brought together leading cancer researchers from the United States and China to launch an actionable roadmap for international research collaboration that will accelerate innovative cancer research and discovery.

Held in Beijing on November 4–6, 2012, the China-U.S. Symposium brought together academicians, leading scientists from universities, research hospitals and U.S. and Chinese pharmaceutical and biotech companies for two days of mind-opening presentations and dynamic panel discussions.

The China-U.S. Symposium was an historic event and could well launch important collaborative efforts — including government-private-public partnerships — that could both shape and accelerate global drug discovery and clinical trials to help defeat cancer worldwide.

The Symposium opened with a Plenary Session featuring Minister Zhu Chen and Dr. Anna Barker, Director, Arizona State University Transformative Healthcare Networks and former Deputy Director of the U.S. National Cancer Institute (NCI). Minister Chen warmly greeted fellow Szent-Györgyi Prize winners Webster Cavenee, Peter Vogt, and Carlo Croce, Academicians of the U.S. National Academy of Sciences.

Peter Vogt, Professor in the Department of Molecular and Experimental Medicine at The Scripps Research Institute, spoke about the power of mutation while Carlo Croce, Professor and Director of the Ohio State University Medical Center's Institute of Genetics, shared his insights as to the causes and consequences of microRNA dysregulation in cancer. The presentations by Dr. Vogt and Dr. Croce were truly enlightening and generated heated discussions among the audience on the challenges, progress and new directions of global cancer research.

The Chinese Ministry of Health has launched the Chinese National Cancer Center Network (CNCCN) headquartered at the Cancer Institute & Hospital of the Chinese Academy of Medical Sciences in Beijing. Minister Chen asked NFCR to help CNCCN build international networks for multi-center global trials. The blueprint for building a Global Clinical Trials Network of Chinese Clinicians as Co-PIs was discussed at the China-U.S. Symposium. Clinicians from both the U.S. and China shared their insights as to the efficacy of U.S. cooperative clinical trial networks, and the current status of clinical research in China.

The proposal to build "Global Clinical Trial Networks" attracted positive feedback from many researchers participating in the China-U.S. Symposium. Scientists from biotech and pharmaceutical companies expressed great interest to explore conducting clinical trials in China. NFCR was assigned the role of conducting follow-up discussions with the CAMS as to how to best implement the Global Clinical Trial Networks initiative.

Intensive dialogue is critical to building bridges between and among cancer researchers regardless of scientific fields or national boundaries. CAMS President Dr. Xuetao Cao proposed that NFCR and CAMS jointly host this type of high-level U.S.-China Symposium on a biennial basis, and has instructed the International Affairs Office of CAMS to form an action plan with NFCR to organize an even larger 2014 China-U.S. Symposium on the State-of-the-Art of Cancer Research and Future Directions for Global Collaboration.

THE LUCY FUND RESEARCH FOR A CURE

The Lucy Fund took on new meaning in 2012 when founder, Lucy Stanovick, lost her four-year-long battle with metastatic breast cancer in August. Though Lucy may no longer be with us to lead the charge against this deadly disease, her legacy lives on through the Lucy Fund and the multitude of loved ones and supporters who continue the fight.

A 46-year-old mother of two and English professor, Lucy Stanovick was diagnosed with Stage IV — or metastatic — breast cancer in 2008. Shocked by how little medical help or information she could find about her cancer and little research was being done in metastatic cancer, Lucy took matters into her own hands and started a grassroots campaign to support metastatic breast cancer research. After learning about the KISS1 Gene, and the research being done by Danny Welch at the NFCR Center for Metastasis Research, Lucy teamed up with NFCR to form the Lucy Fund to raise money specifically for metastatic breast cancer research in an effort to make the disease a chronic, rather than deadly, condition.

The Lucy Fund is making a real difference in research to find a cure for metastatic cancer. Lucy showed how anyone, anywhere could make a difference in research for a cure. In the face of adversity, Lucy summoned the will to cure metastatic breast cancer. Her passion energized all of us, inspiring us to redouble our efforts to support research that focuses on cancer metastasis. This is her legacy.

A member of the NFCR Board of Advisors, Lucy has worked passionately and actively with NFCR to make a difference. Despite her worsening condition, in August Lucy and her supporters held a very successful **5th Party 4 Life** in Shawnee on the Delaware to raise support for the Lucy Fund.

Several Pennsylvania high school basketball teams also came together for a "Shoot for the Cure" tournament benefitting the Lucy Fund in January of 2012. Together these eight participating teams have now raised more than $1600 for metastatic breast cancer research.

Since 2008, Lucy has successfully mobilized support and raised more than $160,000 in support of the research being done at the NFCR Center for Metastasis Research. Lucy raised funds for research, awareness about metastatic cancer research, and reason to hope for a cure. The research supported by the Lucy Fund has provided new insights into the causes and mechanisms of cancer cell metastasis. The discoveries made by scientists at the NFCR Center for Metastasis Research have already stimulated new directions in prognostic and therapeutic research in other laboratories.

"Knowing from day one that she was on the clock, Lucy turned her efforts toward the impact she could make for generations to come. Her desire to bring about cures was remarkable. I will not give up," said Dr. Danny Welch, Director of the NFCR Center for Metastasis Research at the University of Kansas.

Lucy has touched countless lives, including those that will be saved. Thanks to Lucy's efforts and leadership, everyone she encountered was changed for the better, including the entire team at the National Foundation for Cancer Research.

"We are so saddened that we have lost Lucy — she is already missed. Lucy's passion, her spirit, her caring, and her fight live on. Lucy's courage in her battle against cancer and her ability to take action to make a difference for others touches all of us profoundly. We vow to keep her spirit and continue her fight against metastatic breast cancer," said Sujuan Ba, Ph.D., Chief Operating Officer at the National Foundation for Cancer Research.

Lucy was an inspiration. Her determination is a testament to what someone can do in support of research for a cure. The Lucy Fund is about the pledge Lucy Stanovick made to us, to future generations, and we will continue to push forward to make metastatic breast cancer a chronic, not deadly, disease. The Lucy Fund is about ***Research*** for a **Cure.**

Taking Action

NFCR ALL-STARS SPORTS PROGRAM

One of NFCR's most engaging and educational fundraising campaigns is the "NFCR's All-Stars" sports program. The campaign calls on teams from several different sports to join NFCR by holding a fundraiser in conjunction with one or more of their games or tournaments. The program raised more than $110,000 for NFCR in 2012 and has been particularly successful in softball, field hockey and yoga and Pilates.

More than 70 softball teams have participated to-date in the "NFCR's All Stars" program, raising more than $60,000 for NFCR's life-saving cancer research endeavors. The teams have run the gamut from high school to professional and have embraced the cause wholeheartedly. Some teams wore special uniforms or shoelaces to advocate for cancer research, some held bake sales and raffles to raise funds, some even recognized cancer survivors or had a way for loved ones to honor those who have fought cancer.

Field hockey was also a community that was very involved with "NFCR's All Stars." Like the softball teams, these young women across the country gave their all to raise awareness and funding for cancer research, knowing that it was crucial in order to find a cure.

2012 Duke Field Hockey Team.

New Answers for Cancer: NFCR Donors are helping us solve the Cancer Puzzle – helping us solve the scientific mysteries of cancer to bring promising new therapies to patients.

Against Cancer

A notable example of the success of this program is the event held by Georgetown University's field hockey team Oct. 5, 2012. The team raised funds online leading up to their game versus Rutgers University, and players wore purple T-shirts during the game in support of the cause. Thanks to their generous community, Georgetown field hockey raised an exceptional amount of more than $20,000 for NFCR.

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." The studios and wellness facilities raised funds by donating 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. More than 30 studios participated across the country, and NFCR hopes to see even more participants as the program grows.

Winston Churchill High School Soccer team in Potomac, Maryland huddles during their Kick Cancer Fundraiser.

GOLF FOR A CURE

NFCR held its 9th Annual Memorial Golf Classic and Dinner Party Sept. 24, 2012 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. Governor Martin O'Malley served as the honorary chair for the fundraiser. The event featured a scramble-style tournament, a barbecue lunch as well as a dinner party and award ceremony after the game. The day went off without a hitch and raised around $75,000 for NFCR.

The Potomac Group at Folger Nolan Fleming Douglas (Kevin Sourk, George Hill, Stanley Turesky, and Brian Baquer).

Cabinet Discounters foursome Mike Bliss, John Mikk, Jr., Darrin Mikk and Jeff Lewis.

DAFFODILS & DIAMONDS

The 31st annual Daffodils & Diamonds Luncheon took place March 15, 2012 at the Congressional Country Club in Potomac, MD. Nearly 300 influential women and cancer research advocates from the community came together for a champagne reception, runway fashion show, lunch and silent and live auctions. Their generosity raised more than $80,000 for life-saving breast and ovarian cancer research, making for another very successful event in the long history of Daffodils & Diamonds fundraisers.

Loyal Committee Members Gail Peterson, Malinda Lindsay and Claudia Neal.

Committee Member Anne F. Rouse and guest Marie Colturi.

EXTRAORDINARY SUPPORT

2012 was distinguished by the extraordinary breadth and depth of support for NFCR. An unprecedented number of donors, corporations, foundations and institutions made gifts totaling $14.5 million. 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. NFCR 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 2012.

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Includes all donors, foundations, corporations and institutions giving $500 or more between January 1, 2012 and December 31, 2012.

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The Legacy Society recognizes donors who have chosen to create a substantial legacy in cancer research by leaving a gift to NFCR through their estate, or by utilizing other planned gift vehicles to support NFCR's cancer research. We are grateful to these donors for their dedication and foresight and are proud to recognize them through membership in the NFCR Legacy Society.

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INDEPENDENT AUDITORS' REPORT

Board of Directors
National Foundation for Cancer Research, Inc.
Bethesda, Maryland

We have audited the accompanying consolidated financial statements of the National Foundation for Cancer Research, Inc. and Affiliates (collectively, the Foundation) which comprises the consolidated statements of financial position as of December 31, 2012 and 2011, and the related consolidated statements of activities, functional expenses and cash flows for the years then ended, and the related notes to the consolidated financial statements.

Management's Responsibility for the Financial Statements

Management is responsible for the preparation and fair presentation of these consolidated financial statements in accordance with accounting principles generally accepted in the United States of America; this includes the design, implementation, and maintenance of internal control relevant to the preparation and fair presentation of consolidated financial statements that are free from material misstatement, whether due to fraud or error.

Auditor's Responsibility

Our responsibility is to express an opinion on these consolidated financial statements based on our audit. We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audits to obtain reasonable assurance about whether the consolidated financial statements are free of material misstatement.

An audit involves performing procedures to obtain audit evidence about the amounts and disclosures in the consolidated financial statements. The procedures selected depend on the auditor's judgment, including the assessment of the risks of material misstatement of the consolidated financial statements, whether due to fraud or error. In making those risk assessments, the auditor considers internal control relevant to the entity's preparation and fair presentation of the consolidated financial statements in order to design audit procedures that are appropriate in the circumstances, but not for the purpose of expressing an opinion on the effectiveness of the entity's internal control. Accordingly, we express no such opinion. An audit also includes evaluating the appropriateness of accounting policies used and the reasonableness of significant accounting estimates made by management, as well as evaluating the overall presentation of the consolidated financial statements.

We believe that the audit evidence we have obtained is sufficient and appropriate to provide a basis for our audit opinion.

Opinion

In our opinion, the consolidated financial statements referred to above present fairly, in all material respects, the financial position of the National Foundation for Cancer Research, Inc. and Affiliates as of December 31, 2012 and 2011, and the changes in their net assets and their cash flows for the years then ended in accordance with accounting principles generally accepted in the United States of America.

Squire, Lemkin + Company, LLP

April 17, 2013

NATIONAL FOUNDATION FOR CANCER RESEARCH, INC.

AND AFFILIATES

CONSOLIDATED STATEMENTS OF FINANCIAL POSITION

	DECEMBER 31,	
ASSETS	2012	2011 (Restated)
Cash and cash equivalents	$ 2,481,706	$ 830,144
Accounts receivable	154,996	169,900
Contribution receivable	1,300,000	189,532
Prepaid expenses and other assets	455,575	449,396
Furniture and equipment, net of accumulated depreciation	43,714	50,837
Investments	7,108,227	7,373,894
Amounts held in trust by others	1,923,156	1,728,899
TOTAL ASSETS	$ 13,467,374	$ 10,792,602
LIABILITIES AND NET ASSETS		
LIABILITIES:		
Accounts payable and other liabilities	$ 736,569	$ 580,636
Research grants and contracts payable	2,234,418	1,710,118
Accrued compensation and benefits	122,954	116,086
TOTAL LIABILITIES	$ 3,093,941	$ 2,406,840
NET ASSETS:		
Unrestricted:		
Designated for research	$ 4,454,840	$ 4,521,709
Undesignated	2,503,932	1,739,800
Total unrestricted	$ 6,958,772	$ 6,261,509
Temporarily restricted	1,842,766	718,616
Permanently restricted	1,571,895	1,405,637
TOTAL NET ASSETS	$ 10,373,433	$ 8,385,762
TOTAL LIABILITIES AND NET ASSETS	$ 13,467,374	$ 10,792,602

The accompanying notes are an integral part of these financial statements.

NATIONAL FOUNDATION FOR CANCER RESEARCH, INC.

AND AFFILIATES

CONSOLIDATED STATEMENTS OF ACTIVITIES

	FOR THE YEAR ENDED DECEMBER 31, 2012				FOR THE YEAR ENDED DECEMBER 31, 2011 (Restated)			
	Unrestricted	Temporarily Restricted	Permanently Restricted	Total	**Unrestricted**	Temporarily Restricted	Permanently Restricted	Total
REVENUE AND SUPPORT:								
Public support	**$ 9,955,535**	$ 1,728,146	$ -	$ 11,683,681	**$ 11,581,307**	$ 676,421	$ -	$ 12,257,728
Bequests	**2,352,946**	-	-	2,352,946	**800,169**	102,044	-	902,213
Noncash support	**549,981**	-	-	549,981	**649,361**	-	-	649,361
Mailing list rentals	**339,611**	-	-	339,611	**343,924**	-	-	343,924
Net investment income	**671,121**	-	-	671,121	**50,977**	-	-	50,977
Change in value of split-interest agreements	**(20,498)**	27,999	166,258	173,759	**(29,249)**	(13,004)	(86,332)	(128,585)
Other revenue	**284,749**	-	-	284,749	**296,789**	-	-	296,789
Net assets released from restrictions	**631,995**	(631,995)	-	-	**833,140**	(833,140)	-	-
TOTAL REVENUE AND SUPPORT	**$ 14,765,440**	$ 1,124,150	$ 166,258	$ 16,055,848	**$ 14,526,418**	$ (67,679)	$ (86,332)	$ 14,372,407
EXPENSES:								
Program services:								
Research	**$ 5,070,789**	$ -	$ -	$ 5,070,789	**$ 4,823,367**	$ -	$ -	$ 4,823,367
Public education and information	**5,081,937**	-	-	5,081,937	**5,720,799**	-	-	5,720,799
Total program services	**$ 10,152,726**	$ -	$ -	$ 10,152,726	**$ 10,544,166**	$ -	$ -	$ 10,544,166
Supporting services:								
Fundraising	**$ 2,891,461**	$ -	$ -	$ 2,891,461	**$ 3,923,938**	$ -	$ -	$ 3,923,938
Management and general	**1,023,990**	-	-	1,023,990	**883,654**	-	-	883,654
Total supporting services	**$ 3,915,451**	$ -	$ -	$ 3,915,451	**$ 4,807,592**	$ -	$ -	$ 4,807,592
TOTAL EXPENSES	**$ 14,068,177**	$ -	$ -	$ 14,068,177	**$ 15,351,758**	$ -	$ -	$ 15,351,758
CHANGE IN NET ASSETS	**$ 697,263**	$ 1,124,150	$ 166,258	$ 1,987,671	**$ (825,340)**	$ (67,679)	$ (86,332)	$ (979,351)
NET ASSETS, BEGINNING OF YEAR	**6,261,509**	718,616	1,405,637	8,385,762	**7,086,849**	786,295	1,491,969	9,365,113
NET ASSETS, END OF YEAR	**$ 6,958,772**	$ 1,842,766	$ 1,571,895	$ 10,373,433	**$ 6,261,509**	$ 718,616	$ 1,405,637	$ 8,385,762

The accompanying notes are an integral part of these financial statements.

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