A SINGULAR MISSION

Nurse practitioner Naomi Cazeau, of the Adult Bone Marrow Transplant Service.
The clinicians and scientists of Memorial Sloan-Kettering share a vision and a singular mission — to conquer cancer.

They are experts united against a complex disease. Each type of cancer is different, each tumor is unique. Set free in surroundings that invite the sharing of ideas and resources, they attack the complexity of cancer from every angle and every discipline.

TO CONQUER CANCER

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A complete version of this report — which includes lists of our donors, doctors, and scientists — is available on our website at www.mskcc.org/annualreport.
In 2012 the leadership of Memorial Sloan-Kettering endorsed a $2.2 billion investment in a clinical expansion that will set the stage for a changing care paradigm into the next decade and beyond.

This decision occurred against a backdrop of strong financial results, new clinical leadership, and new collaborative relationships. These actions position Memorial Sloan-Kettering for a future in which we have every reason to believe we will witness historic progress against the complex set of diseases we know as cancer.

There are several tangible components of the clinical strategic plan, among them an outpatient cancer care building that we plan to develop along the FDR Drive at East 74th Street in conjunction with Hunter College of the City University of New York. The project, currently under review by city planning authorities, would enable us to provide cancer care in a facility designed to adapt to the ways in which cancer will be diagnosed and treated in the coming decades. We will provide leading-edge treatment for patients with hematologic cancers such as leukemia and lymphoma, head and neck cancers, and thoracic cancers. In addition, the facility will support our efforts to provide bone marrow transplants in the outpatient setting and provide a focused environment for early-stage clinical trials. The MSKCC building would be located next to the future home of Hunter College’s new Science and Health Professions building and its School of Nursing. The acquisition contract is contingent upon the necessary land use and zoning actions required for construction of the two proposed buildings.

Other elements of the capital program include the Josie Robertson Surgical Center on York Avenue, which will feature 12 operating rooms equipped to provide technologically sophisticated surgical care on an outpatient basis. The certificate of need was approved in 2012, the structure that was on the site has been demolished, and construction has begun. Also close to the main campus, MSKCC is planning a new facility that will house a clinical laboratory and research building and will accommodate all specialized testing, along with a cell bank, a cell therapy facility, and tumor procurement services. Regional investments include a 114,000-square-foot ambulatory care facility in Harrison, New York, that will offer advanced cancer care to area residents, closer to home. A “topping-off” ceremony took place on December 20 as the final steel beam of the structure was hoisted into place.

These programmatic investments require leadership and vision. Our new Physician-in-Chief, José Baselga, joined us on January 1, 2013. An internationally recognized physician-scientist, he comes to us from Massachusetts General Hospital (MGH) where he was Chief of the Division of Hematology/Oncology and Associate Director of the MGH Cancer Center. Dr. Baselga is no stranger to MSKCC. He completed a medical oncology fellowship at Memorial Hospital and remained as a faculty member on the Breast/Gynecology Service from 1994 through 1996, after which he returned to his native Spain. His responsibilities include the management of patient care delivery in Memorial Hospital as well as at MSKCC clinics and regional sites. Dr. Baselga will also focus on clinical strategic planning and will oversee clinical and translational research. He succeeds Physician-in-Chief Robert E. Wittes, who stepped down after a decade of exemplary service.

Thomas J. Kelly, Director of the Sloan-Kettering Institute for the past 11 years, announced that he would be stepping down as of March 1, 2013. He is returning to full-time research and will remain an SKI faculty member. Dr. Kelly joined MSKCC in early 2002 and led the largest expansion in SKI’s history to broaden the scope of its basic and translational research activities and focus on the most promising areas of biomedical research as they relate to cancer. A search committee is actively recruiting his successor.

We are pleased with our financial results for 2012, particularly because our operating results for 2011 included non-recurring prior year gains. Our clinical volume continues particularly because our operating results for 2011 included non-recurring prior year gains. Our clinical volume continues...
In a striking example of the integration of clinical studies and fundamental laboratory discoveries at MSKCC, the US Food and Drug Administration announced in August that the drug enzalutamide had been approved for the treatment of men with metastatic prostate cancer. The results of a large, multicenter phase III study, led by an MSKCC physician-scientist, showed that enzalutamide significantly increased survival in men with advanced disease. Investigators led by Howard I. Scher, Chief of MSKCC’s Genitourinary Oncology Service, first presented these findings at the American Society of Clinical Oncology’s Genitourinary Cancers Symposium in February 2012. Laboratory work conducted by Charles L. Sawyers, Chair of our Human Oncology and Pathogenesis Program, and colleagues was instrumental in the development of this novel therapy. The basic research that resulted in its development, and the collaborative efforts to bring that discovery into a clinical setting, will serve as a model for future drug development at academic institutions.

The excellence of our researchers and research community received a ringing endorsement from The Starr Foundation, which provided renewed support for The Starr Cancer Consortium and Tri-Institutional Stem Cell Initiative totaling $105 million. The Starr Cancer Consortium is a collaboration among MSKCC, the Broad Institute of Harvard and MIT, The Rockefeller University, Weill Cornell Medical College, and Cold Spring Harbor Laboratory. The Tri-Institutional Stem Cell Initiative is a collaboration among MSKCC, The Rockefeller University, and Weill Cornell Medical College.

Our clinicians and scientists received numerous awards and honors in 2012, far more than we could ever mention here, so we will single out just a few of the most significant. Structural Biology Program Chair Nikola Pavletich and Immunology Program Chair Alexander Rudensky were named members of the National Academy of Sciences (NAS). The NAS is one of our country’s most prestigious scientific societies and election to its membership is one of the highest honors given to scientists working in the United States. Drs. Pavletich and Rudensky join the ten MSKCC researchers who were already NAS members.

In addition, neuro-oncologist Lisa M. DeAngelis was elected to the Institute of Medicine (IOM). Dr. DeAngelis is Chair of MSKCC’s Department of Neurology and Co-Executive Director of the Brain Tumor Center. Election to the IOM is considered one of the highest honors in the fields of health and medicine. She joins MSKCC’s 17 other IOM members.

In December, President Obama appointed Charles Sawyers as one of six new members of the National Cancer Advisory Board (NCAB). Dr. Sawyers will serve on a body that advises on some of our nation’s most pressing issues in cancer research and treatment. In yet another honor, Dr. Sawyers was elected President of the American Association for Cancer Research, the world’s oldest and largest professional organization dedicated to advancing cancer research.

And Clifford A. Hudis, Chief of the Breast Cancer Medicine Service, was elected President of the American Society of Clinical Oncology, the world’s leading professional organization representing more than 30,000 physicians who care for people with cancer.

The many achievements and successes over the past year would have been impossible without the support of our staff, who performed exceptionally in every important area. The dedication, energy, compassion, and creative spirits of the men and women of Memorial Sloan-Kettering are what power this extraordinary institution and make us thrive.

In the pages that follow, you will meet ten MSKCC researchers — basic scientists and clinicians — all of whom are working to answer critical questions about cancer. Each one is making seminal contributions to the development of new and innovative therapies that will improve and prolong the lives of the patients we see today and those we will care for tomorrow.
Ping Chi, MD, PhD

MEDICAL ONCOLOGIST, MELANOMA AND SARCOMA SERVICE
CANCER BIOLOGIST, HUMAN ONCOLOGY AND PATHOGENESIS PROGRAM
GEOFFREY BEENE JUNIOR FACULTY CHAIR

When I began looking for a place to start my career, I was drawn to Memorial Sloan-Kettering’s Human Oncology and Pathogenesis Program [HOPP] because I believed it would give me sufficient time to focus on my lab research without having to give up caring for patients.

HOPP was founded to bring together a diverse group of physician-scientists who could advance scientific knowledge while moving laboratory discoveries more rapidly into the clinic.

HOPP researchers tend to ask questions about the molecular causes of cancer. And although we work primarily in the lab, we meet regularly with our physician colleagues to discuss new cases, so we never lose sight of the practical and clinical considerations that go in to making decisions about patient care.

As a clinician, I see patients with melanoma and many different types of sarcomas. [Sarcomas are a diverse group of tumors that arise in soft tissues such as muscle, fat, and cartilage, and in bones.]

I am looking at genetic changes such as mutations, which affect the sequence of DNA [our genetic code], and also epigenetic changes, which do not involve alterations in our genetic code but can control whether a gene is turned on or off.

I also study the cellular context in which genetic and epigenetic changes occur. This is important in my research because sometimes—depending on the signals that are activated by nearby cells—such changes can lead to cancer and may represent a specific Achilles’ heel for a certain cancer type that we may be able to target with drugs.

My lab is using many different approaches to look for novel diagnostic and prognostic biomarkers and targets in sarcomas. These include mapping epigenetic changes, studying the cells of different types of sarcomas and the formation of disease in mouse models, and using high-throughput screens, which can evaluate gene expression signatures in thousands of samples at the same time. Even though we have come very far with targeted therapy, we still have a poor understanding of the biology of many types of sarcoma, and we don’t have a lot of treatment options.

A new focus of my lab is studying something called malignant peripheral nerve sheath tumors. The only treatment that can cure these tumors is surgery, and the surgery is very difficult because the tumors often surround crucial nerves. So I’m trying to identify ways to develop targeted therapy for advanced tumors. Understanding their genetic alterations could lead to the development of clinical trials in which drugs or combinations of drugs are selected to work against a particular target.

One of my goals is to get more involved with early-stage clinical trials that exploit the same molecular mechanisms I’m studying in the lab. In particular, for patients with a rare gastrointestinal tumor called GIST, there is a new treatment strategy that targets a gene called ETV1 and potentially could be used as an alternative to and a more effective therapy than the current therapy, a drug called imatinib (Gleevec®), which targets a different gene. My colleagues and I hope to start a clinical trial for this new treatment strategy sometime in 2013.
Interventional radiologists are physicians who use image guidance—including CT and PET scans, ultrasound, x-rays, and MRI scans—to perform minimally invasive procedures that diagnose and treat disease and provide relief from symptoms.

Thermal ablation of a tumor is among the procedures we do routinely. The technique involves placing a needle into a tumor and applying extreme heat or cold to kill the cancer while preserving surrounding healthy tissue. This has traditionally been done under CT guidance, which allows us to visualize the tumor and know where to place the needle. But the CT image can’t tell us whether we have fully destroyed a tumor.

A method that could provide real-time, image-based monitoring of tumor ablation would be a valuable tool. MSKCC is one of the only hospitals in the world with an interventional PET-CT, which is housed in our Center for Image-Guided Interventions. We’re investigating a novel, MSKCC-developed way to use PET scans to assess—while a patient is still on the table—whether we have completely ablated a tumor.

In addition to destroying tumors, thermal ablation may also help activate tumor-specific T cells [a type of immune cell]. When we ablate a tumor, we create an inflammatory reaction in which these cells are recruited to clean up the dead cancer cells. In the process, the T cells have the opportunity to learn that the cancer has certain markers that they might be able to recognize in order to fight cancer that has spread to other parts of the body. Generally, though, after ablation the immune response in humans is weak and not sufficient to eradicate tumors elsewhere in the body or to prevent disease progression. However, what we’ve discovered in our research in mice...
is that when we give a drug called ipilimumab (Yervoy™) after tumor ablation we can boost the immune system and see a response at other sites in the animal’s body. We’re now investigating this approach with a variety of cancers and with several different drugs; and, in collaboration with colleagues on the Breast Cancer Medicine Service, we’ve begun a small clinical study in women with localized breast cancer. We are looking at applying this technique to other cancers as well.

Another area of investigation in our group involves a new ablation tool called irreversible electroporation (IRE), which does not use heat or cold to kill cancer cells. This technique uses an electrical current to punch holes in cell walls, irreversibly destroying the cells. Some organs, such as blood vessels, are not made up of just cells—there’s also a protein structure. Because IRE targets only cells, we may be able to target a tumor abutting a major blood vessel, killing the cancer cells while the vessel wall—because it’s made up of these structural proteins—remains intact.

MSKCC interventional radiologists also can treat certain liver tumors by blocking branches of the hepatic artery in a procedure called embolization. With their blood supply cut off, the tumors die. We have expanded our use of the technique to include treatments for liver cancer using beads that deliver chemotherapy or radiation directly to tumors. The beads combine the therapeutic effects of radiation or chemotherapy with those of embolization.

In light of recent advances in individualized therapy for many types of cancer, molecular profiling of tumors is vital for guiding treatment decisions. To perform the necessary studies, larger amounts of tissue are required than in the past. One of the things we do as interventional radiologists is to obtain tumor samples by doing needle biopsies. We currently have a major initiative to develop innovative ways to determine—during a needle biopsy—when we have collected enough tissue. One technology we’re in the preliminary stages of exploring is spectral analysis, which involves shining a special light on a specimen as soon as it’s removed from a patient to allow us to see whether we have enough cells in the sample.

With so much progress in the field of interventional radiology we’ve also established an Oncologic Interventional Radiology Fellowship in which fellows are exposed to the full range of interventions and which also includes time to pursue research. These are just a few examples of the ways in which our IR group is working to enhance and improve the care we provide to patients. 

(Below, from left) Drs. Sofocleous and Solomon collaborate on an IR procedure; (at right) Dr. Solomon meets with a patient before beginning an IR procedure.
Our immune system has evolved highly sophisticated ways of fighting infection that hinge on its ability to distinguish between healthy cells and cells harboring intruders such as viruses.

This system provides powerful protection against pathogens, but it comes at a price: Unless the different cell types of the immune system are controlled precisely, they may inflict severe damage on the body’s own cells and tissues.

My laboratory studies T lymphocytes, white blood cells that are vital in the immune system’s response to infection.

One of the areas we focus on is a subset of T lymphocytes called regulatory T cells. We’ve found that regulatory T cells are critical for keeping other white blood cells in check, thereby playing an important role in controlling immune system reactions. In the absence of regulatory T cells, instead of attacking foreign cells, the immune system attacks normal cells and tissues, which may lead to sometimes fatal inflammatory responses.

Understanding how these cells function—what genes and biological pathways underlie their ability to regulate the immune system—has many potential clinical applications. For example, numerous studies have shown that most tumors are infiltrated by regulatory T cells, which are believed to suppress immune responses to cancer. Drugs that act by inhibiting regulatory T cell function might offer new and effective ways to control the disease.

Other potential therapies that act by boosting or targeting these cells are being explored to treat conditions characterized by an overactive immune system—including autoimmune disorders such as rheumatoid arthritis, psoriasis, and diabetes. Previously, we and others found that a gene called Foxp3 plays a critical role in the generation of regulatory T cells both in and outside of the thymus, a small organ located underneath the breastbone. In a series of recent studies, we reported that a distinct DNA element within the Foxp3 gene is essential for the process by which extrathyMIC regulatory T cells—those regulatory T cells that are generated outside of the thymus—mature and acquire specialized traits and functions.

In one of our studies, published in the journal Cell in July 2012, we discovered that one unique function of these cells is to control the immune system during pregnancy. Our findings suggest that extrathyMIC development of regulatory T cells emerged during evolution to prevent a mother’s immune system from attacking the fetus. In mice that lack the aforementioned Foxp3 DNA element, extrathyMIC regulatory T cells do not develop normally, causing pregnant females to lose their embryos more frequently and develop complications reminiscent of those seen in human pregnancies.

In another study, published in Nature in February 2012, we found that extrathyMIC regulatory T cells control allergic and asthma-like inflammation in the gut and lungs. My colleagues and I are now exploring whether subsets of regulatory T cells generated in and outside of the thymus play a role in different types of cancer.

We biologists tend to study an organism by breaking it down into parts—essentially attempting to reduce it to a mechanism. And it used to be that immunologists focused largely on specific parts of the immune system, overlooking its entirety and the interactions that take place between the immune, endocrine, and nervous systems. Working with MSKCC clinicians, we are now starting to apply our knowledge about the ways regulatory T cells control multiple body functions to practical use. Clinicians are trained to treat the entire organism. Their perspective is vital in bringing the scattered parts together.

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I knew this would be a field that would explode in my lifetime, and that has already turned out to be true in so many ways.

To a great extent, brain tumor surgery has become a form of personalized medicine, as we are now better able to devise strategies for removing a particular tumor both before and during the procedure.

At Memorial Sloan-Kettering, we have the ability to perform complex surgery with the patient awake and responsive, using advanced navigation systems. We choose an awake approach when the tumor is located in a deep area of the brain or near a region involved in speech expression or speech comprehension.

Before going into the operating room, we first perform a traditional MRI, which I liken to a low-resolution Google map. It tells us what the brain looks like and where the tumor is located. Then, we perform a functional MRI, which shows where the brain activates when the patient speaks or moves.

I was initially drawn to surgery because it couples intellect with manual elegance. When I was training in the 1990s, it was obvious that opportunities to improve brain surgery were on the horizon — technologies were becoming available that would allow us to operate more safely and effectively.

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The highest-resolution, three-dimensional map is obtained in the operating room. We stimulate particular areas of the brain while talking to the patient until we determine — to the exact millimeter — where nerve cells that control speech and motion reside. We are also fortunate to have access to an MRI scanner in the operating room for both brain and pituitary tumor surgeries, which allows us to see, as the surgery proceeds, how much of the tumor has been removed and how much remains.

With all of these tools — managed by our team of surgeons, anesthesiologists, neurophysiologists, and neurologists — we are able to design a brain operation in real time that takes into account the preservation of a patient’s quality of life. We are also able to remove tumors that would have been previously considered inoperable.

Outside of the operating room, I have been translating my research involving stem cells to brain tumors.

One of the most striking aspects of stem cells is their flexibility and versatility. They can adapt and respond to both intrinsic signaling and their immediate environment to become a specific type of cell.

Glioblastoma multiforme — one of the most common and aggressive types of brain tumor — is recognized for its heterogeneity in appearance and genetics. Scientists have suspected that a small portion of this type of tumor may consist of cancer stem-like cells, which could repopulate the tumor after treatment. This is what drove us to apply some of my previous stem cell research to glioblastoma multiforme and discover that a subset of these tumors can generate their own blood vessels from tumor stem-like cells. Instead of simply recruiting blood vessels, which many other tumors do, glioblastoma multiforme tumors can actually contribute to making their own blood vessels.

As a physician-scientist, when I’m operating on a patient and taking out a tumor, I am also thinking about the tumor’s cellular components. I think physician-scientists have a unique role in that they are so close to the subject of study that they are positioned to ask the right questions in the laboratory.

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Proteins are amazing machines. Inside the cells, they act like members of an orchestra, carrying out specific functions at specific times and in regulated ways. And what we do in my laboratory is try to understand how proteins work—primarily by determining what they look like in three dimensions. If you can see something, you can get an idea of how it functions.

My colleagues and I focus primarily on ion channels, proteins that reside within the plasma membrane surrounding cells. The plasma membrane can be likened to a balloon that separates the inside of the cell from the outside, and these channels span the thickness of the balloon.

The plasma membrane is a very thin layer, but it’s incredibly important because it’s what contains the cell, and cells also communicate through it. Membrane proteins—proteins such as ion channels that reside within the membrane—are gatekeepers and relay stations that allow each individual cell to communicate with its neighbors and interact with its environment. In comparison to proteins found within cells, membrane proteins are typically more difficult to study and consequently relatively little is known about their inner workings.

Ion channels act like molecular sieves, only letting their particular ion go in or out of a cell. So a calcium channel is selective for calcium, allowing calcium to pass in and out of a cell but not, for example, sodium or potassium.

Recently, my lab revealed for the first time the three-dimensional structure of two proteins found in humans, one of which—K2P1—regulates the flow of potassium ions in and out of cells, and the other—called Orai—which regulates the flow of calcium. In both cases we used a technique called x-ray crystallography, which is a method of determining the shape or structure of proteins.
a protein at an atomic level of detail. In order to do this, we first produced large amounts of the proteins, then purified and coaxed them to form crystals—solid structures in which all the proteins are symmetrically lined up. A typical crystal would contain approximately a trillion protein molecules. Next, we illuminated the crystals with an intense beam of x-ray light generated by a massive instrument called a synchrotron located at Brookhaven National Laboratories on Long Island. As the x-rays interact with each individual protein molecule within the crystal, the rays are diffracted at different angles. Captured on a screen, the diffracted rays form a unique pattern. From these patterns we were able to derive detailed information about the unique shapes of K2P1 and Orai. Finally, using a computer, we built an atomic model—an architectural mockup that shows every atom of both proteins. From this work, we obtained the first visualizations of any human potassium channel and of any calcium channel.

By drawing the detailed three-dimensional model of K2P1 and studying this model atom by atom, we and other scientists will be able to come up with ideas about how this channel works and test our ideas in the lab. It’s too early to say with certainty, but I think there are good reasons to be hopeful that our study will guide the development of certain types of drugs in the future.

Orai has been detected in almost every tissue of the body, which underscores the importance of calcium signaling in all sorts of biological processes. We still have much to learn about calcium signaling, but one setting in which we understand it relatively well is in T cells—a type of immune cell. Calcium is an important signaling molecule in these cells, and Orai is essential for the activation of the immune response. As with K2P1, this is very early work, but I hope that what we’ve revealed about the structure of Orai may set the stage for the development of drugs that target this channel and can be used to control the activation of the immune system in the context of autoimmune diseases, allergy, cancer, and other disorders.

In a small way, what we do is like going to the moon for the first time. We were the first people to see what K2P1 and Orai look like, and now we’re telling others. There are very few places besides MSKCC where I could have done what I’ve done with these projects. The institutional support of basic science has been crucial, the mindset that says, “We want you to try difficult things. We want you to take risks.” Researchers here can commit to trying to answer basic science questions, limited only by their own imaginations. As we have seen time and again, research that seeks to address fundamental biological questions relating to the inner workings of cells can lead, in the long run, to novel therapies for human diseases that wouldn’t have been possible without the commitment to basic science research like that ongoing at the Sloan-Kettering Institute.
respond to drugs, there are genetic factors that influence the way they respond to radiation. So both genetic and tumor-specific factors may contribute to the way a tumor responds to a fixed dose of radiation. Ultimately, we want to understand in real patients, exposed to real doses of radiation, why some respond well and others poorly.

A project in my laboratory is aimed at learning more about this variation in radiation sensitivity. We’ve taken lymphoid cell lines from individuals whose entire genomes have been sequenced and we’ve irradiated them. Then we’ve looked at the way the cells handle the radiation. What we see is a spectrum of response that exactly mirrors what we observe in people. Then, because these genomes are fully known, we can do association analyses. These are genome-wide association studies to attempt to relate what it is about the radiation-sensitive or resistant people that makes them sensitive or resistant. We’re doing these studies in collaboration with colleagues in Memorial Hospital’s Clinical Genetics Service and the Sloan-Kettering Institute’s Computational Biology Program and are looking to discover whether there are common parts of the genome that associate with radiation sensitivity or resistance. Understanding these variations will help radiation oncologists make the best treatment decisions for each patient. For example, if we know a tumor has built-in radiation resistance mechanisms we might not use radiation at all, perhaps use a higher dose, or use a drug to make the tumor more radiosensitive.

We’re entering a new and exciting era in radiation oncology, and these are just several of the many initiatives and research efforts under way at MSKCC.
In 2004, I collaborated on an evidence-based practice project that resulted in the development of a patient-education book for families of MSKCC patients called *A Guide for Caregivers*. In 2009, I collaborated on another evidence-based practice project within the Department of Radiation Oncology in which we produced guidelines to manage skin reactions in patients receiving radiation.

It was through my work with women who received pelvic radiation for rectal and anal cancer that I decided to undertake a research study focused on improving patient quality of life. I pursued the research under the auspices of the Nursing Fellowship Program.

Radiation to the pelvic area for the treatment of colorectal or gynecologic cancers can create a number of side effects in female patients, most notably vaginal stenosis, a narrowing and shortening of the vagina. As a result, women may experience pain with intercourse and have difficulty tolerating a pelvic exam, limiting the effectiveness of this critical examination.

To minimize the effects of stenosis, women are often advised to use a vaginal dilator. Historically, adherence to this therapy is low and there has been little follow-up to determine its effectiveness.

I joined MSKCC in 1991 directly from nursing school, and began my career caring for neuro-oncology and neurosurgery inpatients. The care of these patients attracted me because it provided me with the experience to build a strong foundation of nursing skills. But more importantly, I felt I could offer a great deal to patients with neurologic problems.

Many such patients have motor or sensory deficits, and helping them overcome these challenges allowed me to begin fulfilling the aspiration that drew me to nursing—to help patients have the best quality of life during and after treatment.

In 1999, I moved to ambulatory care [treatment delivered to outpatients], working first on the Endocrinology Service and later with the Department of Radiation Oncology, where I again worked with neuro-oncology patients who received radiation as part of their treatment. For the next ten years I cared for gastrointestinal cancer patients receiving radiation therapy. Today, I work in the Gastric and Mixed Tumor Service, a surgical oncology service.

Early in my career I knew I wanted to pursue nursing research. I became an active member of MSKCC’s Ambulatory Nursing Research Council and was also the ambulatory care representative on the institution’s first Evidence-Based Practice Nursing Council.

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I joined MSKCC in 1991 directly from nursing school, and began my career caring for neuro-oncology and neurosurgery inpatients. The care of these patients attracted me because it provided me with the experience to build a strong foundation of nursing skills. But more importantly, I felt I could offer a great deal to patients with neurologic problems.

Many such patients have motor or sensory deficits, and helping them overcome these challenges allowed me to begin fulfilling the aspiration that drew me to nursing—to help patients have the best quality of life during and after treatment.

In 1999, I moved to ambulatory care [treatment delivered to outpatients], working first on the Endocrinology Service and later with the Department of Radiation Oncology, where I again worked with neuro-oncology patients who received radiation as part of their treatment. For the next ten years I cared for gastrointestinal cancer patients receiving radiation therapy. Today, I work in the Gastric and Mixed Tumor Service, a surgical oncology service.

Early in my career I knew I wanted to pursue nursing research. I became an active member of MSKCC’s Ambulatory Nursing Research Council and was also the ambulatory care representative on the institution’s first Evidence-Based Practice Nursing Council.
My study was focused on evaluating adherence to and the effectiveness of using vaginal dilators to minimize stenosis. We accrued 115 women who had been treated for gynecologic and colorectal cancers at either MSKCC’s main campus or the regional network sites. This broad sample gave our study a statistically relevant representation of the population. And another wonderful aspect of the study was that it was conducted with a multidisciplinary team approach that included both radiation oncologists and nurses. It was professionally gratifying to bring together this mix of specialists to support the protocol. It made the study even more meaningful, relevant, and thorough.

The study opened in 2009 and we are now performing data analyses. I’m hopeful that the study’s work will shed light on the long-term benefits of vaginal dilator use and will lead to the improvement of patients’ quality of life.

I am committed to continuing to conduct research as I carry on my clinical work. I love caring for and interacting with patients, which has remained my goal since my first day of nursing. And it’s only from this hands-on experience that I can learn about and understand the clinical needs of patients, which will serve as an inspiration for my next research project.

Ms. Law wishes to thank many people for their support and contributions to her study, including Clinical Nurse Specialist Joanne Frankel Kelvin, radiation oncologist Karen Goodman, Senior Research Specialist Bridget Thom, Senior Research Biostatistician Elyn Riedel, Research Study Assistant Ashlyn Tom, Department of Nursing leadership, the Nursing Fellowship Program, and the Geri and ME Fund for nursing at MSKCC.
For example, we have discovered that a mutation that affects cells’ epigenetic programs—in this case by changing the structure of chromosomes—occurs in a number of different types of leukemia. Changing that chromosomal structure causes a gene expression program to get turned on when it shouldn’t. And for these types of leukemia, we now know exactly what the protein is that allows that to happen. The protein is called a histone methyltransferase (HMT), and we’re working to develop small-molecule inhibitors of the protein that are now in clinical trials at MSKCC in several adult leukemias. The research is particularly exciting because this is the first small-molecule inhibitor of an HMT to be evaluated in humans.

It’s becoming increasingly clear that many types of cancer have abnormalities in histone methyltransferases and that these abnormalities are likely part of the tumor development process in many cancers. So what we are learning with these new approaches in leukemia is likely to have a much broader impact as we understand more about how abnormalities in HMTs are working to drive cancer development.

Many would argue that the next big development in cancer research will be in the field of epigenetics—the study of changes in gene expression that are controlled by factors other than changes in an individual’s DNA sequence. These proteins and mechanisms contribute to cancer development in ways that we didn’t really understand ten years ago.
in the clinic. It will provide the infrastructure—the people, research, and technologies—to help move exciting new discoveries from the lab into clinical trials as quickly as possible. We will use next-generation genomic sequencing technology to characterize leukemia samples from patients in order to understand, in as much depth as possible, which mutations are present in leukemias and how they work together; develop mouse model systems of those leukemias; and then test newly developed therapies to support the rationale for ultimately doing clinical trials in patients.

Our goal is to streamline the process and encourage multiple MSKCC labs to work together using a common approach. The synergy that will develop will make it easier for the institution to support these efforts and for pharmaceutical companies and others to work with us.

We’ve gotten to the point where basic research discoveries are making a difference for patients. One of the things that makes me most proud as a pediatric oncologist who specializes in treating children with leukemia is when I talk to the parents of a child who has leukemia and I’m able to tell them that we’re making discoveries that are likely to influence the course of the disease for the better. It’s incredibly motivating to think of the comfort it gives parents to know that there are people focused on studying leukemia as deeply and intensely as we are, and if our efforts don’t make a difference for their child, we expect they will for patients in the future.

I don’t know of a better goal than being able to look back in ten or 15 years and say that I had a tangible influence on the way that people are treated for cancer. That is definitely what drives me and the people I work with here.
In biology, our focus instead is on deriving meaningful insights into biological processes. So while we still like to design clever algorithms to solve problems, the most important thing is asking the right question in the first place.

For most of the history of modern biology, scientists have studied one gene at a time. In the late 1990s, it became possible to simultaneously measure the expression of thousands of genes using microarrays (also known as DNA chips), giving a kind of genome-wide “snapshot” of gene activity in each sample of cells.

More recently, there has been another technological revolution, called next-generation sequencing, and now biologists are rapidly amassing even more comprehensive data. For example, for each tumor sample that we study, RNA sequencing produces millions of short sequences (“reads”) that we match to the genome and analyze statistically. [Like DNA, RNA is a large biological molecule consisting of strings of nucleotides; RNAs are transcribed from the genome using regions of DNA as a template.]

This statistical analysis allows us to measure changes in expression for thousands of protein-coding genes as well as RNAs that don’t code for proteins, to detect alterations in the way these genes were processed and to map their mutations.

Computational biology is a broad field, but a short explanation for what most computational biologists do is that we use computational methods to help make sense of very large amounts of biological data. I trained as a mathematician, and mathematicians are often attached to finding elegant solutions to problems.

My lab develops computational and statistical methods to exploit this data in order to study how genes are expressed in regular cells and to learn what goes wrong when gene expression gets dysregulated in cancer cells.

The methods we use often come from machine learning. These are algorithms that “learn” from data to build a model that can be used to make accurate predictions. Many
people have interacted with machine learning applications, perhaps without realizing it. For example, when you use a digital camera to take a picture, face detection software locates the faces in the field of view to allow the camera to focus better. The face detector was “trained” on a big data set of photos where the locations of faces were known, so that it could learn to discriminate between face and non-face patches of photos. It can then be applied to new data—in this case, new faces in the photo you want to take—and accurately predict the faces.

In our work, rather than faces, the data sets are made up of large quantities of data derived from different kinds of sequencing or microarray technologies. Often our models are designed to predict how genes are regulated. In one project, we are trying to train a model that will teach us more about the mechanism of gene silencing—a process that is dysregulated in many diseases, including cancer—using small pieces of RNA called microRNAs. In another project, we are trying to decode the information in regulatory regions of the genome that govern differentiation of stem cells into fully specialized cell types.

As a computational scientist, I came to Memorial Sloan-Kettering because I wanted to be immersed in an exciting biomedical science environment and work on important problems in cancer biology. This is an amazing place because scientists are encouraged to be ambitious and tackle big questions—which sometimes involves generating huge and complex data sets. Through close collaboration with experimental labs here, we have started to leverage the power of computational and machine learning methods to advance cancer research.
There are at least 20 types of breast cancer with distinctive features in their morphology [structure and form] that allow them to be identified by microscopy. These tumors collectively are known as special types of breast cancer. Each of them is relatively rare, but together they account for about 25 percent of all breast cancers.

The most common type of breast cancer, called invasive ductal carcinoma of no special type, makes up the other 75 percent of breast cancers. As the name implies, the category comprises all breast cancers that cannot be classified as one of the special types. This group of tumors is diverse, with varying morphologic characteristics, clinical behaviors, molecular features, and responses to therapies. Although special types of breast cancer have been known for a long time, patients with the rare types are currently treated in the same way as patients with ductal cancers.

Recent large-scale studies investigating the genetic basis of breast cancer have focused on the common type of the disease, and have confirmed that these tumors are diverse in their repertoire of gene mutations. Little is known, however, about the genetic characteristics of rare types of breast cancer.

The main goal of my lab is to develop a classification system for all breast cancers that is reflective not only of features seen under the microscope, but also of their molecular characteristics and response to therapies. To achieve this goal, we are studying the special types of breast cancer that are not categorized as ductal carcinoma.
In recent years, we and others have found that tumors from each of these rare types are less diverse than ductal carcinomas at the molecular level, and that some of these special types of breast cancer are underpinned by very specific genetic changes.

By using the molecular tools that we have access to here at MSKCC, we can now determine, at an unprecedented level, the genetic alterations that drive the malignant behavior of the cells of each special type of breast cancer. Once we know which mutations cause these cells to be malignant or are required for cancer cell survival, we can start figuring out how to develop therapies tailored for patients with these rare types of breast cancer. Furthermore, when we find the molecular drivers for these cancers, we will be able to go back and look for the same drivers in ductal carcinoma and then further classify subsets of these common tumors into groups that have molecular features and behave like the rare types.

Our laboratory is also developing approaches to assess the molecular alterations in breast cancer cells by analyzing blood samples from breast cancer patients. We know that cells and genetic material from breast cancers can be found in the blood. We are currently applying cutting-edge genetic tools to compare cancerous materials obtained from blood samples with those from the tumors themselves. Our aim is to determine how the information obtained from these "liquid biopsies" can be employed to guide treatment decision making for breast cancer patients.

I have only been at MSKCC a short time, but I already appreciate what a fantastic institution this is. The critical mass of basic, translational, and clinical scientists is unparalleled, and the clinicians are world-class. There is no other place that brings together people of this caliber who are working on issues that affect cancer patients. I have already had the chance to set up collaborations with other scientists and clinicians at MSKCC, and am looking forward to developing my research program with the aim of matching breast cancer patients with the therapy that will be most beneficial for them.
## Statistical Profile

### PATIENT CARE

<table>
<thead>
<tr>
<th></th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient Admissions: Adults</td>
<td>21,039</td>
<td>21,932</td>
<td>22,652</td>
<td>22,983</td>
<td>23,139</td>
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<tr>
<td>Patient Admissions: Children</td>
<td>1,650</td>
<td>1,537</td>
<td>1,494</td>
<td>1,503</td>
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<tr>
<td>Total Admissions</td>
<td>22,689</td>
<td>23,469</td>
<td>24,346</td>
<td>24,486</td>
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<tr>
<td>Total Patient Days</td>
<td>139,847</td>
<td>140,224</td>
<td>143,532</td>
<td>140,990</td>
<td>149,368</td>
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<tr>
<td>Average Patient Stay (days)</td>
<td>6.2</td>
<td>6.0</td>
<td>5.9</td>
<td>6.1</td>
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<td>Bed Occupancy Rate</td>
<td>88.0%</td>
<td>88.5%</td>
<td>83.7%</td>
<td>82.2%</td>
<td>87.0%</td>
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<td>Outpatient MD Visits: Manhattan</td>
<td>384,889</td>
<td>406,024</td>
<td>418,415</td>
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<td>Outpatient MD Visits: Regional Network</td>
<td>81,995</td>
<td>94,293</td>
<td>97,658</td>
<td>103,098</td>
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<td>Total Outpatient Visits</td>
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<td>500,317</td>
<td>516,073</td>
<td>535,900</td>
<td>541,474</td>
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<td>Screening Visits</td>
<td>28,888</td>
<td>27,369</td>
<td>23,373</td>
<td>20,518</td>
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<td>Surgical Cases</td>
<td>18,035</td>
<td>19,233</td>
<td>19,362</td>
<td>19,374</td>
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<td>Radiation Treatments and Implants: Manhattan</td>
<td>58,494</td>
<td>57,856</td>
<td>59,223</td>
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<td>Radiation Treatments and Implants: Network</td>
<td>43,550</td>
<td>47,987</td>
<td>47,926</td>
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<td>Total Radiation Treatments and Implants</td>
<td>102,044</td>
<td>105,843</td>
<td>107,149</td>
<td>112,008</td>
<td>110,765</td>
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<td>X-ray Examinations and Special Procedures</td>
<td>346,157</td>
<td>358,052</td>
<td>392,809</td>
<td>377,360</td>
<td>391,187</td>
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<tr>
<td>Clinical Investigation Protocols (open to accrual)</td>
<td>522</td>
<td>507</td>
<td>552</td>
<td>552</td>
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### STAFF

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<thead>
<tr>
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<th>2008</th>
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<td>Sloan-Kettering Institute Members</td>
<td>133</td>
<td>140</td>
<td>142</td>
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<td>Hospital Attending Staff</td>
<td>727</td>
<td>768</td>
<td>804</td>
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<td>Registered Nurses</td>
<td>1,734</td>
<td>1,845</td>
<td>1,945</td>
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<td>Support Staff</td>
<td>7,945</td>
<td>8,321</td>
<td>8,613</td>
<td>8,989</td>
<td>9,244</td>
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<td>Total Staff*</td>
<td>10,505</td>
<td>11,039</td>
<td>11,469</td>
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<td>Volunteers</td>
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<td>917</td>
<td>942</td>
<td>1,058</td>
<td>1,018</td>
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*In 2012, 34 staff members held appointments in both the Institute and the Hospital.

### EDUCATION

<table>
<thead>
<tr>
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<th>2010</th>
<th>2011</th>
<th>2012</th>
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<tbody>
<tr>
<td>Residents and Clinical Fellows — Positions</td>
<td>418</td>
<td>436</td>
<td>447</td>
<td>440</td>
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<tr>
<td>Residents and Clinical Fellows — Annual Total</td>
<td>1,609</td>
<td>1,651</td>
<td>1,625</td>
<td>1,676</td>
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<td>Research Fellows</td>
<td>254</td>
<td>303</td>
<td>295</td>
<td>321</td>
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<td>Research Scholars</td>
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<td>121</td>
<td>132</td>
<td>131</td>
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<tr>
<td>Research Associates</td>
<td>87</td>
<td>90</td>
<td>94</td>
<td>82</td>
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<tr>
<td>Graduate Research Assistants</td>
<td>—</td>
<td>—</td>
<td>23</td>
<td>29</td>
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<tr>
<td>PhD Candidates</td>
<td>232</td>
<td>227</td>
<td>231</td>
<td>225</td>
<td>222</td>
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<tr>
<td>MD/PhD Candidates</td>
<td>29</td>
<td>28</td>
<td>26</td>
<td>21</td>
<td>21</td>
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<tr>
<td>Registrants in CME Programs</td>
<td>2,115</td>
<td>2,395</td>
<td>2,554</td>
<td>2,533</td>
<td>3,968</td>
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<tr>
<td>Medical Observers</td>
<td>561</td>
<td>572</td>
<td>541</td>
<td>526</td>
<td>566</td>
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<tr>
<td>Medical Students</td>
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<td>399</td>
<td>391</td>
<td>429</td>
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<tr>
<td>Nursing Students</td>
<td>72</td>
<td>109</td>
<td>105</td>
<td>142</td>
<td>178</td>
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<td>Social Work Students</td>
<td>6</td>
<td>6</td>
<td>6</td>
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<td>6</td>
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<tr>
<td>Radiation Oncology Technology Students</td>
<td>15</td>
<td>15</td>
<td>14</td>
<td>14</td>
<td>13</td>
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<tr>
<td>Cytotechnology Students</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>Physical Therapy Students</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>7</td>
<td></td>
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<tr>
<td>Occupational Therapy Students</td>
<td>5</td>
<td>4</td>
<td>3</td>
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</tbody>
</table>
### Financial Summary (in thousands)

#### Operating Revenues

<table>
<thead>
<tr>
<th>Year</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient Care Revenue</td>
<td>$1,606,989</td>
<td>1,723,313</td>
<td>1,854,776</td>
<td>2,141,421</td>
<td>2,201,941</td>
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<tr>
<td>Grants and Contracts</td>
<td>163,352</td>
<td>167,495</td>
<td>166,327</td>
<td>190,948</td>
<td>185,160</td>
</tr>
<tr>
<td>Contributions Allocated to Operations</td>
<td>108,844</td>
<td>126,250</td>
<td>117,323</td>
<td>130,791</td>
<td>144,497</td>
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<tr>
<td>Royalty Income</td>
<td>94,131</td>
<td>62,232</td>
<td>68,663</td>
<td>77,510</td>
<td>78,350</td>
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<tr>
<td>Other Income</td>
<td>41,963</td>
<td>43,144</td>
<td>44,874</td>
<td>48,351</td>
<td>51,167</td>
</tr>
<tr>
<td>Investment Return Allocated to Operations</td>
<td>116,546</td>
<td>103,998</td>
<td>100,389</td>
<td>104,699</td>
<td>75,877</td>
</tr>
<tr>
<td>Transfer of Board-Designated Annual Royalty Annuitization</td>
<td>33,122</td>
<td>37,156</td>
<td>41,578</td>
<td>46,417</td>
<td>51,709</td>
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</table>

**Total Operating Revenues:** $2,164,947

#### Operating Expenses

<table>
<thead>
<tr>
<th>Year</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compensation and Fringe Benefits</td>
<td>$1,164,155</td>
<td>1,286,536</td>
<td>1,361,032</td>
<td>1,466,667</td>
<td>1,582,212</td>
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<tr>
<td>Purchased Supplies and Services</td>
<td>684,872</td>
<td>757,863</td>
<td>772,968</td>
<td>835,621</td>
<td>879,219</td>
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<tr>
<td>Provision for Bad Debts and Assessments</td>
<td>6,823</td>
<td>10,881</td>
<td>11,046</td>
<td>18,285</td>
<td>17,541</td>
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<tr>
<td>Depreciation and Amortization</td>
<td>175,870</td>
<td>171,806</td>
<td>175,494</td>
<td>195,461</td>
<td>210,810</td>
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<td>Interest Expense</td>
<td>59,023</td>
<td>64,997</td>
<td>47,931</td>
<td>57,098</td>
<td>54,894</td>
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<tr>
<td>Less Fund-Raising Expenses Transferred to Non-Operating Income (Expenses)</td>
<td>(36,048)</td>
<td>(40,320)</td>
<td>(43,926)</td>
<td>(44,665)</td>
<td>(47,305)</td>
</tr>
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</table>

**Total Operating Expenses:** $2,054,695

**Income from Operations:** $110,252

#### Philanthropy (in thousands)

<table>
<thead>
<tr>
<th>Year</th>
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<th>2010</th>
<th>2011</th>
<th>2012</th>
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<tr>
<td>Philanthropy</td>
<td>$279,103</td>
<td>166,247</td>
<td>237,666</td>
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#### Capital Spending (in thousands)

<table>
<thead>
<tr>
<th>Year</th>
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<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Capital Spending</td>
<td>$345,135</td>
<td>226,049</td>
<td>262,371</td>
<td>223,251</td>
<td>258,613</td>
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#### Balance Sheet Summary (in thousands)

<table>
<thead>
<tr>
<th>Year</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assets</td>
<td>$6,578,522</td>
<td>6,068,707</td>
<td>6,448,415</td>
<td>6,790,005</td>
<td>7,795,608</td>
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<tr>
<td>Liabilities</td>
<td>2,354,618</td>
<td>2,467,135</td>
<td>2,550,889</td>
<td>2,848,843</td>
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<tr>
<td>Net Assets</td>
<td>$3,223,904</td>
<td>3,601,572</td>
<td>3,897,526</td>
<td>3,941,162</td>
<td>4,233,052</td>
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</tbody>
</table>

### Notes

- **2012 Total Operating Revenue:** $2,788,701
- **2012 Total Operating Expenses:** $2,697,371
- **2012 Compensation and Fringe Benefits:** $1,582,212
- **2012 Purchased Supplies and Services:** $879,219
- **2012 Depreciation and Amortization:** $210,810
- **2012 Interest Expense:** $54,894
- **2012 Provision for Bad Debts and Assessments:** $17,541
- **2012 Less Fund-Raising Expenses Transferred to Non-Operating Income:** $(47,305)
Boards of Overseers and Managers

as of December 31, 2012

Douglas A. Warner III
Chairman
Richard I. Beattie
Vice Chair of Boards
Chair, Board of Managers, Memorial Hospital
Clifton S. Robbins
Treasurer
James D. Robinson III
Honorary Chairman
Marie-Josée Kravis
Vice Chair of Boards
Chair, Board of Managers, Sloan-Kettering Institute
Norman C. Selby
Secretary
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The Campaign for Memorial Sloan-Kettering maintained strong momentum in 2012, with gifts and pledges as of December 31 totaling more than $2.752 billion. Generous supporters from around the world contributed cash gifts totaling $235.6 million over the course of the year, including a record-setting $61.3 million in December. This marks the largest December total in Center history, and the 130,000 gifts processed during the month helped bring 2012 to an especially strong close.

As the Campaign continues ahead of pace toward its $3.5 billion goal under the leadership of co-chairs Douglas A. Warner III and Louis V. Gerstner, Jr., and President Craig B. Thompson, its ongoing achievement is sustained by the generosity of donors at every level. Benefactors of Memorial Sloan-Kettering generated a 30 percent increase in new pledges in 2012 from the previous year, and the commitment and support for the Center’s lifesaving mission demonstrated by its donors is exemplified by the following:

- David M. Rubenstein committed $10 million to establish the David M. Rubenstein Center for Pancreatic Cancer Research, which enables the launch of an intensive, fully integrated effort designed to achieve meaningful progress against one of the deadliest forms of cancer.
- The Tow Foundation pledged $8 million to underwrite the work of the Michael G. Harris Cell Therapy & Cell Engineering Facility (formerly the Center for Cell Engineering), which develops advanced cell-based cancer therapies through cutting-edge molecular and cellular techniques. This brings the Tow Foundation’s total giving to the Campaign to more than $38 million.
• The Estate of Samuel Mitchell, a long-standing donor to Memorial Sloan-Kettering, made a bequest of $3.6 million in unrestricted funds to the Center, part of the more than $37 million in bequests received by the Center in 2012.

• The Thompson Family Foundation made a $3.8 million grant in support of prostate cancer research at Memorial Sloan-Kettering as part of a groundbreaking international collaboration with Israel’s Weizmann Institute of Science.

The driving commitment to making a difference is also displayed by the thousands of volunteers and participants in athletic fundraisers who support Memorial Sloan-Kettering’s work with their efforts each year. The newest of these fundraisers, The RBC Decathlon, challenges professionals in financial services to compete against one another in ten separate athletic events. In 2012, participants in The RBC Decathlon raised nearly $1.3 million for pediatric cancer research. In ten separate athletic events. In 2012, participants in The RBC Decathlon raised nearly $1.3 million for pediatric cancer research.

And despite the cancellation of the 2012 ING New York City Marathon in the aftermath of Hurricane Sandy, members of Fred’s Team registered to participate on behalf of Memorial Sloan-Kettering showed extraordinary commitment, raising a total for the year of more than $46 million to support high-priority research at the Center.

Memorial Sloan-Kettering benefits enormously from the generosity of its committed benefactors, whose support at every level helps the Center’s physicians and scientists pursue the most-promising avenues of research and advance the standard of care for the benefit of patients around the world.
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