Meet Our
Research Grant Recipients

The Chemotherapy Foundation®
Our Chemotherapy Laboratory

Built in 1970, the Foundation Laboratory in the Division of Hematology/Oncology at the Icahn School of Medicine at Mount Sinai has remained at the core of our expanded research grant program. Rebuilt in 1989 and relocated to larger facilities in Mount Sinai in 2004, the Rochelle Belfer Chemotherapy Foundation Laboratory, under the direction of Samuel Waxman, M.D. (seated center), is an acknowledged research center for the development of differentiation therapy, which is the process of reversing the behavior of cancer cells into mature, functioning cells and programming their death.

Today, this novel concept of cancer treatment—which began in this Laboratory—is a clinical reality. For example, retinoic acid (a derivative of Vitamin A) has become a standard differentiation treatment for acute promyelocytic leukemia.

The combination of the Vitamin A derivative with arsenic trioxide followed by chemotherapy results in 95% cure of patients with acute promyelocytic leukemia and is used worldwide. This is the first demonstration of an almost total cure of a form of acute leukemia using differentiation therapy, whereby cytotoxic chemotherapy is not required for most patients. Preclinical trials include identification of gene defects in retinoic acid function associated with breast cancer, development of a new generation of differentiation agents in collaboration with the National Cancer Institute, and identification of targets to block the development of metastases in breast cancer and melanoma. Research in the Lab has resulted in a promising clinical trial combining Bortezomib with Fulvestrant as treatment for breast cancer. The Laboratory also continues its basic studies of the mechanisms of differentiation, metastasis, tumor dormancy, and selective cancer cell apoptosis (programmed cell death) in leukemia and breast cancer.

In addition to The Chemotherapy Foundation’s ongoing support, all the principal investigators in the Laboratory are funded by grants from the National Institutes of Health and by the Samuel Waxman Cancer Research Foundation, a long-time partner of The Chemotherapy Foundation.

As supporters of The Chemotherapy Foundation, you are active partners in providing the vital opportunities to develop and advance promising new research avenues.

We acknowledge with sincere gratitude the significant support of the Alpern Family Foundation and the Halbert family in establishing the original laboratory.
A majority of tumors are caused by mutations or disruption of the chemical instructions (i.e. “software instructions”) that control the genome, which collectively reprogram normal cells into cancer tissue. Under the direction of Dr. Ari Melnick, Gebroe Family Professor of Hematology/Oncology and Chair of the Hematologic Malignancies program at Weill Cornell Medical College, researchers are developing ways to identify, understand and therapeutically reprogram molecular software (known as the “epigenome”) that encodes the chemical instructions that determine the behavior of tumor cells. In recent research, Dr. Melnick and colleagues discovered that malignant lymphomas, including Hodgkin’s disease, are able to evade the immune system by aberrantly erasing a set of specific epigenomic programs. These programs normally are responsible for producing proteins that enable the immune system to “see” lymphoma cells. Dr. Melnick’s group discovered the specific enzyme that erases these key instructions and developed a therapy that can block this enzyme and render lymphoma cells vulnerable to attack by the patient’s own immune system. This innovative therapeutic approach merges the concepts of immunotherapy and epigenetic therapy modifying the tumor cell's software programs in such a way as to drive the patient’s T-cells to attack and potentially eradicate the disease. Based on these results the investigators initiated a multicenter collaborative group to rapidly translate this new therapy into clinical trials. Notably this collaborative effort is showing that this mechanism may be relevant to solid tumors such as lung cancer as well. In addition to support of The Chemotherapy Foundation, Dr. Melnick receives a special annual award from the Sol and Dina Sloan Memorial Research Fund.

Dr. Iannis Aifantis is a Professor and Chair of the Department of Pathology at the New York University School of Medicine, and a member of the Laura and Isaac Perlmutter Cancer Center. His laboratory focuses on the study of normal and malignant hematopoiesis. More specifically, the Aifantis Lab has been a pioneer in the study of acute leukemia including lymphocytic (ALL) and myeloid (AML) leukemia. The lab has added during the last decade a large number of novel tumor suppressors and oncogenes (NOTCH1, FBXW7, EZH2, TET2, ASXL1 to mention a few), generated related animal models of leukemia and offered a detailed analysis of their function. At the same time, the laboratory focuses on novel compounds and biological agents that can affect such oncogenic triggers and tests them in pre-clinical disease models, including targeted animals and human xenograft models. The goal of such studies is to use this knowledge to rationally design clinical trials targeting human acute leukemia. Moreover, the Lab has recently identified a number of novel oncogenic drivers, including novel tumor suppressors that control epigenetically disease initiation and progression (including PRC2 complex, Cohesin complex, TET gene family). Such studies led them to an effort to map the epigenome of human leukemia and use such knowledge to design drugs targeting epigenetic regulators that control gene transcription at multiple levels, including DNA and histone modification. Finally, the Aifantis Laboratory is developing computational methods and platforms analyzing the leukemia transcriptome, including the expression of novel, unannotated long non-coding RNAs (lncRNAs) in human acute leukemia. Of special note is the additional Postdoctoral Oncology Fellowship awarded by the Feldman family to Dr. Aifantis in 2015 to focus further on a project he and his team have been developing, which has been one of the most important stories in the field of human leukemia. In this project, research focuses on an enzyme called TET2 that is able to modify (methylate) DNA. TET2 is mutated in a large fraction of leukemia patients. TET2 mutations can predict failure to respond to current therapy protocols (chemotherapy), making the understanding of TET2 function very important both biologically and clinically. The Aifantis team has generated animals that carry the exact human mutations found in leukemia patients. These animals develop myeloid cancers with age, exactly like the human patients. Using these animals, the team is trying to understand how TET2 mutations lead to leukemia, why are such mutations resistant to current chemotherapy protocols, and whether one can use experimental drugs to target their activity and lead to leukemia remission. Support by this Fellowship Award was key for the generation of these precise models of human disease and is essential for a better knowledge of acute leukemia progression and for the introduction of future targeted therapies.
Human T-cell lymphoblastic leukemias are highly aggressive tumors. Over the last decade, Adolfo Ferrando, M.D., Ph.D., Professor in the Departments of Pediatrics and Pathology at Columbia University Medical Center, has been studying the genetic programs and molecular alterations responsible for uncontrolled growth, proliferation and survival in human leukemia cells. Using a combination of genomic tools for mutation detection and analysis of gene expression, Dr. Ferrando initially defined different molecular groups of T-cell leukemia defined by the activation of key oncogenic factors and characterized by a unique signature of gene expression and different prognosis. Following on this work, Dr. Ferrando contributed to the identification of mutations resulting in the aberrant activation of the NOTCH1 gene in over 50% of patients with T-cell leukemia. The fundamental importance of this finding resides in their capacity to block the activity of NOTCH1 with drugs known as gamma-secretase inhibitors. Over the last years, Dr. Ferrando’s lab has analyzed the oncogenic function of NOTCH1 and the antileukemic properties of blocking NOTCH1 signaling. These studies have uncovered the role of NOTCH1 as a master regulator of cell growth and metabolism upstream in leukemic cells and identified the interaction of NOTCH1 with major oncogenic factors such as MYC and the PI3K-AKT pathway. In addition, Dr. Ferrando has demonstrated that inhibition of NOTCH1 signaling can effectively reverse resistance to glucocorticoid therapy in T-cell leukemias. Current work in the Ferrando lab supported by The Chemistry Foundation uses high throughput assays and Systems Biology approaches to identify therapeutic targets aimed to reverse chemotherapy resistance in ALL. These studies will ultimately lead to new therapies for high-risk leukemia patients.

Steven Itzkowitz, M.D., Professor of Medicine and Oncological Sciences, Director of the Gastroenterology Fellowship Program (seated) and research associates at the Icahn School of Medicine at Mount Sinai are investigating the role of inflammation in the development of colon cancer. Studies are elucidating clinico-pathological risk factors for developing colorectal cancer in patients with inflammatory bowel disease (IBD). Other work has elucidated that patients with IBD who have a history of cancer and receive immunosuppressive medicines for their IBD are at no greater risk of developing new or recurrent cancer than those who did not receive these medicines. This work is now being confirmed in a regional prospective study which will be expanded to a national network. Dr. Itzkowitz helped demonstrate the efficacy of a new non-invasive stool DNA test for early detection of colorectal cancer in the general population and spearheaded a study to develop a similar test in patients with IBD. He is Co-Chair of the Guidelines Committee of the New York Citywide Colorectal Cancer Control Coalition in New York City.
The most aggressive forms of human cancer are invariably associated with loss of the differentiated features of the original tissue. The laboratory directed by Antonio Iavarone, M.D., Professor in the Departments of Neurology and Pathology and the Institute of Cancer Genetics at Columbia University Medical Center, discovered that the molecular pathways normally operating to maintain self-renewal and the undifferentiated state during embryonic development are aberrantly recruited by aggressive tumors. Such pathways ultimately transfer the phenotypic traits of embryonic stem cells to cancer cells. Dr. Iavarone’s lab identified the inhibitor of differentiation (Id) proteins as the master transcriptional regulators that direct those functions. They produced genetic and biochemical evidences that the Id2 protein is a physiologic target of the Retinoblastoma tumor suppressor protein Rb during development. In the absence of Rb, unchecked Id2 activity prevents differentiation and the post-mitotic state in the nervous system. Concurrently, the genetic and epigenetic oncogenic events that mark tumor initiation and progression in mouse and humans converge on direct or indirect activation of Id protein function. The result is that unrestricted Id activity becomes essential to maintain most of the phenotypic hallmarks that distinguish the aggressive neoplasm (relentless proliferation, invasiveness and metastasis, neo-angiogenesis). Their most recent work identified the degradation machinery for Id proteins and determined that Id proteins may escape from it to accumulate in cancer cells. From this, they moved towards the notion that unrestricted Id signaling in quiescent neurons engages a well-defined transcriptional network that leads to rejuvenation, a process associated with resumption of the neurons’ intrinsic ability to elongate the axonal processes. This work provided new and unexpected links between stem cells, cancer and neurobiology. Among a constellation of potential tumor targets for intervention, few other molecules offer the attractive targeting mechanisms against multiple essential traits of cancer that might be associated with the ablation of Id function in malignant pediatric brain tumors.

The most recent research interest of Dr. Iavarone’s lab is to tackle the problem of the link between stem/progenitor cells with cancer stem cells in the nervous system. Their findings indicate that transcription factors establish interconnected networks to initiate and maintain a defined tumor phenotype. They have discovered that reconstruction of those networks in normal cells is sufficient to confer the malignant properties associated with that particular phenotype. Their ultimate goal is to understand the signaling systems involved in initiating and maintaining pediatric brain tumors and use this understanding to identify new targets for pediatric brain tumor therapy.
Dr. Sandra Demaria is Professor of Radiation Oncology and Pathology in the Department of Radiation Oncology at Weill Cornell Medical College. The goal of her work is employing conventional therapeutic modalities, such as chemotherapy and radiation, as tools to promote antitumor immunity and vaccinate the patient against his/her own cancer. Her approach is based on the hypothesis that the combination of an agent that kills tumor cells with a timely stimulation of the cellular immune response results in a synergistic effect whereby reduction in tumor burden is combined with increased antigen presentation and development of an effective antitumor response capable of eliminating systemic metastases.

Dr. Demaria and co-workers were the first to demonstrate that apoptosis induced by chemotherapy is not immunologically silent and can be associated with the generation of maturation signals for dendritic cells (DC) (Journal of Leukocyte Biology, 2005). This work has important implications for the effects of chemotherapy on antitumor immunity.

In collaboration with colleagues from radiation oncology, Dr. Demaria’s group has pioneered the use of ionizing radiation therapy (RT) as a tool to generate an in situ vaccine in mouse breast cancer models. They have shown that RT can elicit antitumor immunity when used in combination with a growth factor that enhances the number of DC (International Journal of Radiation Oncology, Biology and Physics, 2004). Second, they demonstrated for the first time that local RT to an established breast cancer that has already spread systemically can induce an antitumor immune response capable of inhibiting systemic metastases, if combined with immune checkpoint inhibitors (Clinical Cancer Research, 2005, Seminars in Radiation Oncology, 2015). In addition, they showed that RT induces chemokines and NKG2D ligands in tumors that attract antitumor T cells (Journal of Immunology, 2008 and Radiation Research, 2010) and promote the interaction of cytolytic T cells with tumor cells (Journal of Clinical Investigation, 2012). Third, the importance of dose and fractionation of radiotherapy in determining its ability to synergize with immunotherapy was demonstrated (Clinical Cancer Research, 2009), and the presence of immune regulatory circuits that influence the response to treatment with radiotherapy and immunotherapy was shown in experimental models (Clinical Cancer Research, 2009 and 2012, Journal for ImmunoTherapy Cancer 2014, Cancer Research 2015) and breast cancer patients (Cancer Investigation, 2011, Clinical Cancer Research 2012). Results of these studies support a new paradigm for the use of radiotherapy in treatment of patients with metastatic cancer, and initial clinical translation of this approach is promising (Lancet Oncology, 2009, Cancer Immunology Research 2013, Journal of the National Cancer Institute 2013, JAMA Oncology 2015). Recently, her lab has demonstrated that the DNA damage response triggered by radiation is coupled with activation of viral defense pathways that lead to tumor rejection (Nature Communications, in press).

In the next year(s) Dr. Demaria’s work will continue to be focused on identifying actionable molecular targets to enhance responses to treatment with immunotherapy in pre-clinical models and in cancer patients.

Dr. Demaria has received both a special Breast Cancer Research Award from the Joyce and Irving Goldman Family Foundation and a research grant from The Chemotherapy Foundation since 2003 in partial support of her studies. Additional funding has been provided by grants from the NIH/NCI, the American Cancer Society, the Department of Defense Breast Cancer Research Program, the Breast Cancer Alliance, and Breast Cancer Research Foundation.

Samir Parekh, M.D., Associate Professor of Medicine, Division of Hematology/Oncology at the Icahn School of Medicine at Mount Sinai, and his associates have identified aberrantly methylated and silenced genes in mantle cell lymphoma and developed a novel combination of hypomethylating agents and histone deacetylase inhibitors to overcome resistance to Bortezomib in this difficult-to-treat lymphoma.

Mantle cell lymphoma is characterized by a relapsing course with median survival of around 2-4 years for most patients. Using an integrative genomic approach with a high resolution custom-designed microarray, Dr. Parekh’s lab has analyzed a large number of mantle cell lymphoma samples and demonstrated striking hypermethylation throughout the genome in this disease. Moreover, they have identified a panel of hypermethylated genes that have tumor suppressor functions in other cancers, like breast cancer, glioma, and leukemia, and are excellent targets for future research in mantle cell lymphoma. The team also developed a combination of DNA hypomethylating agents and histone deacetylase inhibitors that can reactivate several of these silenced tumor suppressors to therapeutic benefit. Interestingly, the combination of hypomethylation and histone deacetylase inhibition could even overcome resistance to Bortezomib, a promising new therapeutic agent for this disease. The lab is now working on identifying specific signatures of drug resistance and response to individualize therapy in MCL, in collaboration with a large ongoing trial at the NIH. These studies will lay the foundation for epigenetic analysis and therapy in MCL.

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Doris Germain, Ph.D., Professor, Division of Hematology/Oncology at the Icahn School of Medicine at Mount Sinai, and her associates most recent discovery is first pregnancy-dependent oncogene. Breast cancer affects 140,000 women each year and the number of young women affected is rising. While pregnancy at a young age is associated with a reduction in the overall lifetime risk of developing breast cancer compared to that in women who never had children, pregnancy is also associated with an immediate but transient increased risk of breast cancer in all women, with the risk peaking within 5 years after pregnancy. Moreover, since women tend to have their second child within 5 years of having their first, there is a concern that a second pregnancy at a time where the breast is at higher risk of breast cancer may amplify the risk. Conversely, a large analysis of the effect of breast-feeding using combined results from 47 studies, involving a total of 50,302 women, revealed that extended lactation is protective against breast cancer (Collaborative Group on Hormonal Factors in Breast, 2002). This study suggested that the cumulative risk of breast cancer could be reduced by half should the period of breast-feeding be increased (Collaborative Group on Hormonal Factors in Breast, 2002). In addition, breast-feeding was found to be protective against more aggressive tumors, but the mediators of the protective effect of lactation have not been identified. The Germain laboratory has found that the protease Pappalysin-1 is an oncogene, but its oncogenic function can only be unleashed during post-partum involution. Further, they found that the length of the lactation prior to the involution is critical in limiting the effect of Pappalysin-1.

1. More specifically they discovered that the protective effect of lactation is linked to the presence of inhibitors of Pappalysin-1 in breast milk. Since Pappalysin-1 is also expressed in breast cancer in older women, they are currently testing the possibility that conditions found in the mammary gland during involution can be recapitulated in an old mammary gland. The Germain group also investigates the role of the diabetes drug metformin against the Pappalysin tumors and, therefore, they are working toward a targeted therapy of pregnancy-associated breast cancer. In the past, Dr. Germain’s discoveries have led to a phase II clinical trial, which was completed last year. Her hope is that the current research on pregnancy-associated breast cancer will also lead to a clinical trial in the near future. In addition to funding from The Chemotherapy Foundation, Dr. Germain has also received the Robert D. Feldman Memorial Research Grant for breast cancer since 2008 in partial support of her studies.

Eduardo F. Farias, Ph.D., Assistant Professor in the Division of Hematology/Oncology at the Icahn School of Medicine at Mount Sinai (ISMMS), has focused his studies on Vitamin A signaling and epigenetic reprogramming in breast cancer. Dr. Farias’s research is focused on the role of nuclear receptors on the normal mammary gland development, tumor biology, cell signaling in tumor and metastasis and epigenetic reprogramming in breast cancer. He found that CRBPI plays a crucial role in tumor cell differentiation by retinoids and also showed that isotypic-specific RAR activation has a strong anticancer activity in three transgenic mouse models MMTV-neu, wnt1 and -myc, and discovered a new and unexpected pro-proliferative function of RARy in both normal and transformed mammary epithelium stem cells. In collaboration with Dr. Aguirre-Ghiso at ISMMS, he is studying the role of p38 and NR2F1, important co-receptors that participate in the activation of RARs, in the control of tumor dormancy and early dissemination in breast and head and neck cancers. In collaboration with Dr. Samuel Waxman at ISMMS, he showed that a Sin3 chromatin modulator decay peptide to a specific target induces epigenetic reprogramming resulting in anti-estrogen and retinoid responsiveness in human triple-negative breast cancer TNBC cells and animal models. His lab developed and tested small molecule inhibitors (SMIs) to target the PAH2 domain of Sin3 that show reversion of epithelial to mesenchymal transition (EMT) in TNBC models in vitro and in vivo and inhibition of tumor growth and metastatic dissemination with inhibition of the expansion of the stem cell compartment. His most recent published data shows that the inhibition of Sin3 function using peptides or SMIs selectively inhibits the expansion of the cancer stem cells and impair wnt and TGFβ signaling, two pathways associated with tumor progression and metastasis dissemination. Moreover, by combining the Sin3 SMIs with FDA approved RARx agonists AM80 (tambutotecin) in adjuvant settings in pre-clinical models of TNBC, Dr. Farias significantly reduced the lung metastasis, blocked residual disease in the bone marrow and impressively extended the overall survival of the mice treated with the SMI/AM80 combination. This new therapeutic approach opens a window of opportunity for the treatment of relapsed TNBC patients that until now do not have any other therapeutic alternative.
Other Program Accomplishments

Progress in the fight against cancer has also been accomplished by the Foundation’s innovative support and leadership:

**Medical Research**

In 1970, two years after our founding, we built, equipped and staffed the Chemotherapy Foundation Laboratory in the Division of Medical Oncology at the Mount Sinai School of Medicine. In 1974 we added a tumor cell lab. With support from the Foundation and other major sources, the Rochelle Belfer Chemotherapy Foundation Laboratory was rebuilt in 1989 and relocated to larger premises in Mount Sinai in 2004. It has the latest facilities for utilizing molecular biology in cancer treatment.

We have also provided funds to start and support an ever-growing number of innovative cancer research projects in other Divisions at the now Icahn School of Medicine at Mount Sinai and at three other major New York City medical centers: New York University School of Medicine, NYU Langone Medical Center; Weill Cornell Medical College; and Columbia University Medical Center. These include grants to leading investigators who have conducted research studies in breast cancer, brain tumors, gastrointestinal cancer, hematologic malignancies and malignant melanoma, among others.

Foundation support with initial funds also helped to establish two Bone Marrow Transplantation Units (Hackensack University Medical Center and Mount Sinai Medical Center) and three Oncology Units (NYU Medical Center, New York Medical College, and Long Island Jewish Medical Center).

**Professional Education**

Since the early 1970s we have sponsored professional symposia to inform physicians of practical advances to improve patient-care management and survival, and to foster the exchange of concepts that will lead to tomorrow’s new treatments.

We were at the forefront in promoting another new approach in 1983, when we organized and solely supported the first breast cancer chemoprevention conference to discuss the feasibility of a controlled chemotherapy study for women at high risk of developing breast cancer. In 1987 we continued that innovative tradition by sponsoring the first international workshop on breast cancer chemoprevention. Researchers concluded that a major study of breast cancer prevention with Tamoxifen was justified by available reported data. Five years later (summer 1992) the NCI launched a five-year clinical trial using Tamoxifen for the chemoprevention of breast cancer that involved 13,000 patients. Spearheaded by Dr. Bernard Fisher, scientific director of the National Surgical Adjuvant Breast and Bowel Project (NSABP), he and his NSABP colleagues, working with the National Cancer Institute, designed and implemented this first breast cancer prevention trial in the United States. The results were published by Dr. Fisher in 1997 and showed the ability of Tamoxifen to reduce recurrence of either invasive or pre-invasive events by 40%.

Our annual three-day Chemotherapy Foundation Symposium, now managed by Physicians’ Education Resource, LLC (PER), is an outstanding event on the professional calendar. We are confident that our ongoing efforts to stimulate the scientific imagination will continue to reap rewards in our progress against cancer.
The Challenge Continues — Why Now Is The Time To Help

We are in the midst of a period of unprecedented rapid expansion of biomedical knowledge. The BRCA breast and ovarian cancer susceptibility genes were discovered in the 1990s and greatly enhanced our understanding of these cancers. In June 2000 the sequence of genes on human chromosomes was completely deciphered. These discoveries are spearheading the development of new molecules for diagnosis and treatment.

Going forward we need to continue to learn the answer to our deepest questions, and participate in the translation of dramatic new scientific information into breakthrough improvements in cancer management. The challenge is to meet this potential with adequate research support, so as not to let this opportunity slip away, for the failure to follow-up these discoveries will delay key cancer breakthroughs. Clinical trials based on these new discoveries are an essential component of such advances.

While chemotherapy made significant inroads into improving outcomes, science has moved beyond targeting cell division and its DNA. Modern drug regimens are now partnering with drugs arising from new concepts seeking targets beyond the tumor cells into the tumor microenvironment, including blood vessels and other cells present in tissues. The early intense focus on the tumor cell diverted us from key aspects of this environment that is being increasingly studied in the era of molecular targeted therapy. The best example of a successful story in this regard is the greatly improved eradication of breast tumor cells that have high expression of the growth factor receptor HER2 or erbB2 when combined with chemotherapy. In addition to these conceptual advances, there are technical advances in drug delivery. These weapons include monoclonal antibodies, and anti-body drug complexes that are used as "smart bombs" zeroing in on the cancer cell and delivering cancer-killing drugs. Because we have identified key molecules on the surface of cancer cells, which mark them as distinct from normal cells, we have been able to make anticancer vaccines that instruct the body to kill the cancer by natural methods. This, in turn, has led to better understanding of the immune system and new molecules that release the natural brakes that our body activates to limit normal tissue damage as bystanders in the body’s reaction to infection (and to tumors). This new ability to unleash the immune system against cancer cells has revolutionized the treatment of melanoma, and is revitalizing immunotherapy for lung cancer and is gradually extending to treat a great variety of advanced cancers heretofore resistant malignancies. These developments are being worked through trials in a vast array of human malignancies and represent the most exciting possibilities for prolonged control of advanced malignancies that have become realities only in the last few years.

It is clear that your support at this critical moment can do more than it ever could, because the scientific tools are more refined and the goals more sharply defined. The Chemotherapy Foundation has long led the fight against cancer by supporting the best scientists, physicians, and educators, those best positioned to use modern science to defeat an ancient enemy. Now is the time for all of us to work together even harder to grasp the victory against many additional cancers within our reach. The strength of our Foundation is our capacity to seek out talent among institutions to improve emerging multidisciplinary treatments across the cancer spectrum.

Established in 1968, The Chemotherapy Foundation is a public foundation dedicated to the control, cure, and prevention of cancer through innovative medical therapies. The Foundation funds selected laboratory and clinical research at four major New York City medical centers. Our annual three-day Chemotherapy Foundation Symposium each November, now managed by Physicians' Education Resource, LLC (PER), is an outstanding event on the professional calendar. The Foundation also publishes free public education literature. Continuing contributions from the public, the business community, and the profession are needed to increase the range and reach of these essential programs. Donations to The Chemotherapy Foundation, 183 Madison Avenue–Suite 403, New York, NY 10016 are tax deductible.