



**Terence J. Irmen**

## From The Chairman's Desk

The Forbeck Foundation is now in our 25th year of operation. Many of the original volunteers assembled by *George and Jennifer Forbeck* in 1984 remain active with the Foundation, serving on the Board of Trustees and the Scientific Advisory Board. The magic of the Forbeck Foundation is the close working relationship between these two groups.

The Board of Trustees has no personnel changes to report, however we continue to focus on succession planning by passing many key responsibilities onto the younger generation of trustees. It is critical that we ensure the long term viability of the Foundation. The most significant succession change is *Jamie Forbeck Collins* is transitioning into the role of Foundation Administrator, the role formerly held by *Jennifer Forbeck* since the Foundation began. We wish Jamie success in her new role, and are filled with gratitude for Jennifer's tremendous leadership and selfless contribution to the Foundation.

The original mission developed during our first year continues as our beacon for growth, promoting advances in the field of oncology, particularly pediatric oncology. This past year's forum addressed the very important and current topic "*Immunotherapy and Breaking Tolerance*" co-chaired by *James Allison, PhD* from Memorial Sloan Kettering, and *Stan Riddell, MD* from Fred Hutchinson. The 2009 topic on "*The Biology and Treatment of Primary Brain Tumors*" will be co-chaired by *Tracy Batchelor* from Harvard Medical School and *Luis Parada* from the University Of Texas Southwestern Medical Center. We plan to continue these forums as long as there is work to be done.

Our Scholar Board organized the fourth Forbeck Scholar Retreat held in Lake Geneva, Wisconsin last September. The retreat was chaired by *David Fisher, MD,*

*PhD*, a member of our Scientific Advisory Board. Participants in the retreat have indicated that this year's event was another overwhelming success. The Scholar Board held another great fundraising event following the retreat to help raise funds for next year's retreat. The Scholar Board will now be led by *Chandler Dimberg, Galen Eckland* and *Molly Ring* as *Jamie* takes on her significant role with the Foundation.

We believe that we can make a difference and that a cure for cancer is immanent, however it is important to remain grounded enough to be prepared for a long battle. The continuation and improved quality of life for millions lies in the balance. We thank the many dedicated volunteers who have contributed so much to make the Forbeck Foundation the force that it is becoming in the fight against cancer. We also thank the many donors who have continued to be generous, despite the very pessimistic financial news. On behalf of the Forbeck Foundation, let me say thank you all for your continuous support.

**Terence J. Irmen**  
Chairman, Board of Trustees

### In This Issue...

From the Chairman's Desk .....	1
Scientific Advisory Board .....	1
Forum 2008: <i>Immunotherapy &amp; Breaking Tolerance</i> .....	2
Forums 2009 and 2010 .....	6
INRG update .....	6
Awards: 2008 Scholars .....	7
Scholar Board .....	8
Scholar Board Donors .....	9
Scholar Retreat .....	9
Benefactor List .....	10
Financial Report .....	11
Trustees & Scientific Advisory Board	12
Mission & Strategies .....	12

## SCIENTIFIC ADVISORY BOARD REPORT

Funding for basic scientific research seems to have reached an all time low on both sides of the Atlantic. Major funding bodies do not appear to have the capital to finance



**John T. Kemshead**

grant proposals from investigators with excellent track records in research. This is despite the fact that it is clearly an exciting time to be in research as we learn more and more about ourselves and this disease's impact upon us.

Over the last 25 years so much progress has been made in understanding how cells control division, and how they communicate with each other. This knowledge, along with activities such as the sequencing of the human genome, is key to developing new strategies for the treatment of cancer. This is a long-term process that will truly take decades to solve. Breakthroughs that are commonly reported in the press are normally years away from coming to fruition if they happen at all. Unfortunately, such a message is not deemed to be

*Continued on page 11*

### Visit our new and improved web site.

- View the video
- Donation Information
- Upcoming meetings
- Blue Jean Ball
- Past Forum Summaries

# 2008 Forbeck Forum: XXIV<sup>th</sup> Annual Forum

November 6–9, 2008 Hilton Head Island, South Carolina

## Subject: Immunotherapy and Breaking Tolerance

- I: Local and Systemic Host Response to Cancer
- II: Immunologic Checkpoints and Cancer Vaccines
- III: Adoptive Cellular Therapy
- IV: Genetic Modifications of Immune Cells for Cancer Therapy

### Chairmen

James Allison, PhD	Memorial Sloan Kettering	New York, NY
Stanley Riddell, MD	Fred Hutchinson Research Center	Seattle, WA

### Participants

Prof. Thomas Blankenstein	Charite	Berlin, Germany
Philip D. Greenberg, MD	University of Washington	Seattle, WA
Michael Jensen, MD	City of Hope Medical Center	Duarte, CA
Carl June, MD	University of Pennsylvania	Philadelphia, PA
Cornelis J. M. Melief, MD, PhD	Leiden University Med Ctr	Leiden, Netherlands
Jeffrey Mouldrem, MD	Univ of Texas MD Anderson	Houston, TX
David Munn, MD	Medical College of Georgia	Augusta, GA
Drew Pardoll, MD, PhD	Johns Hopkins	Baltimore, MD
Nick Restifo, MD	National Cancer Institute	Bethesda, MD
Steven A. Rosenberg, MD, PhD	National Cancer Institute	Bethesda, MD
Michael Sadelain, MD, PhD	Memorial Sloan Kettering	New York, NY



James Allison, PhD



Stanley Riddell, MD

## 2008 Conference Report on “Immunotherapy and Breaking Tolerance”

by Stanley Riddell

The 2008 Forbeck Symposium focused on new developments in cancer immunotherapy. Recent findings in tumor immunology have provided a clearer understanding of the complex interaction between the host immune system and cancer. Significant progress has been made in elucidating the function and regulation of immune cells, in identifying molecules that are expressed by tumor cells and can be targeted for immune-based therapy, and in understanding the mechanisms by which tolerance to self-antigens is maintained. These insights have resulted in a significant transformation in the field of tumor immunology over the past several years, and led to the development of novel therapies that are now yielding encouraging results in a subset of human cancers.

The goals of this meeting were to review the progress made in understanding the host immune response to a developing cancer and the mechanisms that growing tumors employ to impede the host response, and discuss strategies that can be used to overcome regulatory checkpoints and promote effective immunity to cancer, either by adoptive T cell transfer or by vaccination.



The first session of the meeting was chaired by **Jim Allison** and dealt with the local and systemic host response to cancer. **Drew Pardoll** led off the session with a presentation provocatively entitled “Immunotherapy: saving our field to save cancer patients”. He reviewed data demonstrating how oncogenic signaling pathways involving the transcription factor Stat 3 can shift the immune response from an adversary of tumor progression to one that supports tumor growth. He illustrated how studies in animal tumor models have revealed the importance of combining therapeutics that disrupt pathways that inhibit effective antitumor immunity and with those that enhance antitumor immunity. He argued that combination therapies to simultaneously disrupt inhibition of immunity and to augment host responses to the tumor are likely to be most effective and expressed concern that it will be difficult for academic scientists to assemble the reagents to perform such trials due to the proprietary interests of pharmaceutical companies. He expressed concern that even though the tools available for clinical translation are unprecedented, well

conceived approaches such as vaccines that have proven effective in preclinical models, may fail in the clinic if the trial design and patient populations are not appropriately selected.

**David Munn** focused his presentation on the role of indoleamine 2,3-dioxygenase (IDO) in tumor induced tolerance. IDO has been shown to play a key role in tolerance to the fetus in pregnancy and in mucosal tolerance to foreign antigens. David presented data showing that IDO is expressed by some tumors, and is upregulated in dendritic cells in tumor bearing animals. These IDO expressing dendritic cells suppress

*“The 2008 forum was a very productive meeting from my perspective. I was able to hear data and ideas from leaders in the field, in areas that I don’t personally work in, but that are relevant and helpful in my research. Several collaborations have resulted, which I’m sure is due in part to the informal, personalized style of the meeting. Thanks for all your efforts in organizing it.”*

**David Munn, MD,**  
MCG Cancer Center

effector T cell responses to the tumor and drive the expansion of regulatory T cells in the local tumor environment that inhibit the immune response to the tumor. Based on these observations, IDO is a potential target for pharmacologic inhibition and David described a small molecule IDO-inhibitor drug that is now in Phase I clinical trials. This drug (1-methyl-D-tryptophan) has synergism in animal tumor models when combined with chemotherapy or tumor vaccines.

**Russell Jones**, one of the Forbeck Scholars, presented his work on the metabolic adaptations to cellular stress. The ability of a tumor cell to grow depends on the cell utilizing available energy resources, and the increased proliferation of cancer cells requires changes in energy metabolism to meet metabolic demands. The mammalian AMP-activated protein kinase (AMPK) is an evolutionarily conserved energy rheostat that alters the transcription of many genes that are key to mediating the response to cellular energy stress, and cancer cells utilize this pathway to enable their rapid proliferation. He explained how AMPK may function as a transcriptional activator by the modification of chromatin and this pathway could provide a target for novel therapies.

**Stefanie Sarantopoulos** was the second Forbeck Scholar to present at the meeting. Her work has focused on alterations in B lymphocyte homeostasis after allogeneic hematopoietic stem cell transplantation, and how B cell responses may contribute to the pathogenesis of chronic graft versus host disease and a graft versus leukemia effect. The premise of this work is that the severe depletion of B cells resulting from intensive chemoradiotherapy results in elevated levels of B cell activating factor (BAFF) in an effort to restore B cell homeostasis. High BAFF levels may contribute to the defective censoring of autoreactive B cells. Dr. Sarantopoulos explored a potential relationship between BAFF levels and B cell numbers in transplant recipients with and without chronic GVHD and found that an elevated BAFF/B cell ratio was associated with chronic GVHD. This suggests that high BAFF levels in the setting of allogeneic stem cell transplant may contribute to the development of alloantibodies and autoantibodies that are characteristic of chronic GVHD and identifies BAFF as a potential target for therapy.

**Thomas Blankenstein** returned to the topic of T cell recognition of tumors and described a very elegant model of

cancer in transgenic mice that develop sporadic tumors as a consequence of rare spontaneous activation of a dormant oncogene. These tumors express a strong antigen to which the mice are not initially tolerant. If immunized prior to tumor development, an immune response is elicited and the mice are protected from cancer development. In non-immunized mice, potentially tumor-reactive T cells are rendered tolerant by the growing tumor. These results reveal a failure of immunosurveillance of an immunogenic tumor, and illustrate some of the obstacles to developing immune based therapies for sporadic tumors in humans. This generated considerable discussion about the relevance of murine tumor models for recapitulating the issues relevant to human cancer. There was general agreement that the models that are currently available, while superior to previous models for deriving basic insights into mechanisms that may be responsible for the failure of the immune system to recognize tumors, still have limitations.



*When asked if he planned any new research projects, or had modified existing research due to the meeting, **Thomas Blankenstein**, said, "Any good discussion somehow changes the way of thinking a bit and influences future research, be it direct or indirect."*

The afternoon session on day one was chaired by **Drew Pardoll** and reviewed what has been learned about immunologic checkpoints and how therapeutics are being developed based on inhibiting regulation of the immune system and the progress in cancer vaccines. **Jim Allison** led off this session and described the role of CTLA-4, which is a transmembrane protein expressed on T cells that serves as a key negative regulator of adaptive immune responses. CTLA-4 acts as a brake on T cell responses and plays a central role in maintenance of peripheral tolerance and shaping a developing T cell response. He presented data on treatment of patients with metastatic melanoma with an anti-CTLA-4 antibody to allow activation of anti-tumor immune responses. With this agent alone, there is a 16% response rate in advanced melanoma with one third of the responses consisting of complete and durable tumor regression. A high proportion of responding patients develop autoimmune toxicity that can be

treated with systemic corticosteroids. Jim made the point that anti-CTLA-4 is an agent that may be substantially more effective if used in combination with vaccines or cell therapy. This prompted a vigorous debate about the difficulty developing combination therapies prior to FDA approval of the individual components of an experimental regimen.

**Kies Melief** presented data on the use of a synthetic long peptide vaccine for human papilloma virus (HPV)-associated premalignant lesions. HPV causes cervical cancer and can also chronically infect the vulva where it induces a lesion termed vulval intraepithelial neoplasia (VIN) that eventually progresses to cancer. Many vaccines have been tried unsuccessfully, and Melief proposed that one reason for failure is due to inadequate stimulation of the CD4 helper T cell arm of the immune system. Studies in mice showed that immunization with a vaccine comprised of long overlapping peptides from the HPV E6 and E7 antigens that can elicit CD4<sup>+</sup> and CD8<sup>+</sup> T cells mediated the eradication of established HPV positive tumors. This approach was then tested for efficacy in women with high-grade VIN. Each patient received three immunizations of E6/E7 peptides formulated in an adjuvant. Vaccinated patients did not have side effects, E6 and E7-specific T cell responses increased after the vaccine, and objective clinical responses were observed in a high proportion of patients, providing encouraging evidence that premalignant lesions might be eradicated by immunotherapy.

**Joshua Brody** was the third Forbeck Scholar to present and discussed the development of vaccination for B cell lymphoma in preclinical models and in patients. Dr. Brody employed direct intratumoral injection of CpG oligonucleotides that activates Toll like receptor 9 (TLR9) and enhances the ability of antigen presenting cells to induce an immune response to tumor-associated antigens. In a murine model of lymphoma this approach elicited tumor-specific CD8<sup>+</sup> cytotoxic T cells that mediated tumor regression but also activated CD4<sup>+</sup> regulatory T cells. The effect of regulatory T cells could be mitigated by transferring T cells from vaccinated animals into lymphodepleted mice, which resulted in expansion of the transferred effector T cells and improved the antitumor effect. The CpG intratumoral vaccine was administered to patients without toxicity. Several objective clinical responses were observed. Responses occurred at non-injected tumor sites and

*Continued on page 4*

correlated with induction of a tumor-specific CD8<sup>+</sup> T cell response. The investigators are planning a future trial incorporating storage of post vaccine lymphocytes for cell transfer after lymphodepletion.



**Jeff Moldrem** presented his studies targeting PR1, an antigenic epitope derived from the proteinase 3 gene that is overexpressed in acute and chronic myeloid leukemias. Dr. Moldrem discovered the PR1 epitope several years ago and has performed clinical trials of PR1 peptide vaccination in patients with myeloid leukemia that demonstrated the induction of immune responses in 53% of patients and objective clinical responses in 18% of patients. Patients with antitumor responses had PR-1 specific T cells with a higher functional avidity than non-responders. Dr. Moldrem generated a monoclonal antibody to the HLA A2/PR-1 peptide complex, one of very few monoclonal antibodies to defined MHC/peptide determinant. He presented data showing the HLA A2/PR1 monoclonal antibody specifically stained acute myeloid leukemia cells that express proteinase 3 but not promyelocytes suggesting that leukemia cells process the PR3 protein differently than their normal bone marrow counterparts. This monoclonal antibody also mediated complement dependent cytotoxicity of leukemic cells both in vitro and in mice engrafted with human leukemia. These results support efforts to target PR1 by vaccination or adoptive T cell therapy and suggest a potential therapeutic role for the HLA A2/PR1 monoclonal antibody.



The second day of the symposium focused on cell based immunotherapy for cancer. **Steven Rosenberg** opened the session on adoptive T cell therapy by discussing work at the Surgery Branch of the National Cancer Institute on of T cell therapy for metastatic melanoma. This approach relies on the isolating T cells from surgically resected melanoma specimens, expanding these T cells in interleukin-2 and administering large numbers of cells selected for tumor reactivity back into patients. The partial and complete response rate after adoptive immunotherapy was dramatically improved when lymphodepleting chemotherapy was administered prior to adoptive T cell

transfer. Lymphodepletion results in an increase in serum levels of IL-15 and IL-7 that promote T cell survival and proliferation and depletes regulatory T cells and suppressor cells. Several lymphodepletion regimens have been examined including the use of myeloablative total body irradiation requiring hematopoietic stem cell support. The toxicity with this approach is tolerable and response rates of 50-72% in patients with advanced and otherwise incurable melanoma are now being achieved. A key determinant of the antitumor response is the persistence of transferred T cells and the reason T cells fail to persist in some patients is unclear. There are obstacles to making this therapy available to more patients including the inability to obtain tumor-infiltrating lymphocytes (TIL) from many patients. A solution to this problem is to use gene transfer approaches to express the T cell receptor genes from highly avid tumor reactive T cells in peripheral blood T cells from the patient. Data demonstrating that this can be effective in humans was presented, although response rates in patients that received gene-modified T cells were significantly lower than those from whom TIL could be obtained. The remarkable success of T cell therapy for advanced melanoma validates the potential to employ the immune system to treat even advanced solid tumors.

**Stanley Riddell** continued on the issue of T cell persistence and presented experiments in a nonhuman primate model that addressed the intrinsic qualities of antigen-specific T cells that are required for their persistence in vivo after adoptive transfer. T cells can be divided into antigen inexperienced naïve T cells (T<sub>N</sub>) and into broad classes of memory cells termed central memory (T<sub>CM</sub>) and effector memory (T<sub>EM</sub>). These cells have a distinct phenotype, homing properties, and transcription profile, and serve distinct functions in the immune response. T cells that are generated for cancer immunotherapy differentiate into effector cells while in culture and their origin (from T<sub>N</sub>, T<sub>CM</sub>, or T<sub>EM</sub>) has not been known for certain in any clinical trial. Cell sorting was used to purify T<sub>CM</sub> and T<sub>EM</sub>, and antigen-specific effector T cell clones were derived from each subset and used for adoptive transfer. T cell clones from both memory subsets had equivalent cytolytic and proliferative capacity. However, CD8<sup>+</sup> T<sub>E</sub> clones derived from T<sub>EM</sub> survived in the blood for only a short duration after adoptive transfer, and failed to persist in lymph nodes, bone marrow, or peripheral tissues. By contrast, T<sub>E</sub> clones derived from T<sub>CM</sub> persisted in the blood long-term after

adoptive transfer, migrated to memory T cell niches, and responded to antigen challenge. These results have implications for the types of T cells that should be selected for adoptive transfer, and for strategies to derive tumor-reactive T cells for immunotherapy of cancer by gene insertion.

**Nicholas Restifo** continued on the theme of qualitative properties of T cells that enable their survival in vivo and antitumor activity. He presented data using T cell receptor transgenic mice as a source of naïve tumor-specific T cells and a murine model of melanoma. In this model, transfer of naïve T cells is effective in eradicating the tumor providing the tumor bearing mice are vaccinated to activate the T cells in vivo and interleukin 2 is administered. This model was used to determine the importance of lymphodepletion for the efficacy of transferred T cells and suggested that higher doses of total body irradiation resulted in more effective T cell mediated antitumor activity. The model also enables the analysis of culture conditions for activating the T cells ex vivo and analyzing their antitumor activity in vivo. T cells cultured with antigen in IL-2 exhibited greater cytotoxicity and cytokine production in vitro than T cells cultured in IL-15 but were less effective in mediating tumor regression on a per cell basis after adoptive transfer. Remarkably, the most effective T cells were those derived by culture in antigen and IL-21, which promoted proliferation of the cells but not differentiation to effector cells. These results suggest that the typical measures used to assess tumor reactivity of T cell products may not provide information on the quality of the cells or their potential for antitumor activity in vivo.

*"I absolutely loved the Forbeck Forum. It was the best scientific meeting that I have been to in several years, and I go to at least a dozen each year. What I liked was the extended discussions between scientists. The format shattered the (somewhat) artificial veneer that scientists show at meetings. Given the size of the meeting, it was easy to interrupt other presentations."*

**Nicholas P Restifo, MD,**  
National Cancer Institute,  
Bethesda, MD



**Carl June** presented the first talk on the use of genetically modified T cells for adoptive therapy of human malignancies and viral diseases. He discussed the construction of chimeric

antigen receptors based on the fusion of a single chain antibody that targets a tumor cell surface molecule to the signaling components of the T cell receptor. He presented data showing that these artificial receptors can be readily expressed in human T cells using lentiviral vectors and confer recognition and lysis of tumor cells in vitro. Dr. June showed that the surface membrane glycoprotein mesothelin is overexpressed on mesothelioma, ovarian, and pancreatic tumors and described the engineering T cells with a lentiviral vector encoding a chimeric antigen receptor that targeted mesothelin. This receptor also incorporates the signaling domains of CD28 and CD137, which provide T cell proliferative and survival signals upon recognition of mesothelin positive tumor cells. These engineered T cells were transferred into immunodeficient mice that were engrafted with established mesothelin bearing tumors and could mediate complete tumor eradication at low effector to target ratios. The incorporation of the CD137 domain was important for T cell persistence in this model. This work illustrates the potential to target solid tumors by engineering immune effector cells for tumor recognition.



The final session of the symposium continued the theme of genetic modification of immune cells for cancer therapy. **Philip Greenberg** reviewed the impediments to effectively treating patients with established malignancies, and discussed how genetic modification of T cells could be used to circumvent these impediments. He initially focused on the problem of isolating T cells with high functional avidity for the tumor from tumor bearing patients, and described how this might be overcome by assembling a library of T cell receptor genes that could be introduced into patient T cells. He described the use of yeast expression display as a method for rapidly screening for mutations in low avidity T cell receptors that enhance avidity. Expression of a tumor-specific T cell receptor in patient T

cells can result in cross pairing with endogenous receptors and this both reduces the levels of the tumor-specific receptor and could result in an deleterious specificity. He described ways in which pairing, expression, and function of the introduced receptor could be optimized by modifications to the introduced receptor chains. Finally, he described an elegant mouse model in which T cells expressing dual receptors for a tumor antigen and a viral antigen respectively, were used to define how tolerance to tumor antigens develops, is maintained, and can be broken to promote tumor eradication.



**Michael Jensen** described a variety of genetic engineering strategies for T cells including a novel approach to treating human glioblastoma, a malignant brain tumor that is notoriously resistant to radiation and chemotherapy. Glioblastoma expresses a form of the IL13 receptor and Jensen developed a tumor targeting receptor that consisted of a mutated IL-13 molecule that binds to the tumor form of IL-13R, and is linked to the T cell receptor zeta chain. T cells that express this “zetakine” after gene transfer recognize and lyse glioblastoma cells in vitro and in mice. He is now investigating this approach in an ongoing clinical trial in which patients with recurrent glioblastoma receive direct intratumoral injection of engineered T cells. This treatment has had remarkably little toxicity in the initial patients, and he has observed encouraging evidence of antitumor activity. He then described efficient methods for expressing multiple genes in primary T cells to enhance their antitumor activities and described a novel approach for editing regulatory genes using zinc finger nucleases to permanently disrupt the coding sequence at a targeted site.

**Lili Yang** was the last Forbeck Scholars to present at the meeting. Her work focused on the use of lentiviral vectors to target tumor antigen expression in dendritic cells. For this purpose, the lentivirus envelope consisted of a viral

glycoprotein from the Sindbis virus that was engineered to be specific for a dendritic cell surface protein, termed DC-SIGN. This resulted in efficient introduction of tumor antigen and maturation of dendritic cells. Injection of this lentiviral vector into mice with established tumors promoted tumor regression and induced a CD8<sup>+</sup> T cell response to tumor antigen. Thus, lentiviral targeting of tumor antigen to dendritic cells could provide a novel alternative to current vaccine regimens for inducing immune responses to tumors.



The formal meeting concluded with a presentation from **Michele Sadelain** on redirecting T cells to be specific for leukemia by expressing a chimeric antigen receptor that targets the CD19 molecule on leukemia cells. He described novel constructs of the receptor that incorporate costimulatory ligands for T cells and provide for auto- and trans-costimulation of tumor-reactive T cells. These receptors provide improved antitumor activity in murine models, and may provide for more efficient activation of T cells in human tumor therapy. Michele is conducting a clinical trial of engineered T cells specific for CD19 to treat patients with chronic lymphocytic leukemia and presented the results in the initial patients suggesting that the engineered T cells can infiltrate very large tumors become activated into effector cells. Many B cell malignancies express CD19 and this appears to be a very promising approach for such patients.

The format of the Forbeck meeting provided for vigorous discussion and debate amongst the participants, and there was unanimous enthusiasm amongst the participants for this type of small focused meeting on a topic in cancer biology. The level of scientific discussion was outstanding and identified many opportunities for future collaborations that will hopefully result in the development and rapid translation of improved approaches to treat cancer through immune manipulation.



# Forum Planning

## 2009: The Biology & Treatment of Primary Brain Tumors



Chairmen: Dr. Luis Parada, Dr. Tracy Batchelor

Primary tumors of the central nervous system are the leading cause of cancer death in children, and a tumor of growing incidence in adults, in whom it is equally difficult to treat. Only recently have researchers begun to understand the basic genetic derangements that play a central role in the growth of these tumors.

In children, brain tumors are found in a variety of unusual types, some with unique and characteristic appearance on pathologic examination and in their clinical behavior. In adults, primary brain tumors usually arise in astrocytes, supporting cells of the nervous system, and, in their most common, and poorly differentiated form, these tumors, called gliomas, are exceedingly difficult to treat with any of the common approaches (surgery, chemotherapy, or radiation therapy).

Major efforts are now underway to elucidate the genetic changes in these tumors, the pathways and receptors activated by these changes, and the changes found in the tumor micro-environment. Animal models for some of these kinds of brain tumors have now been developed, and are providing information on tumor behavior and response to experimental treatment. New clinical trials are finding that these tumors are highly dependent on new blood vessels, and respond to treatments that destroy these vessels. Abnormal receptors and activated growth pathways are found on the cell surface or inside these tumors and these may be the subject of new treatments as well. New information indicates that multiple aberrancies may exist in the signalling portfolio of these tumors, thus requiring multiple sites of attack.

In this rapidly evolving state of knowledge, the meeting, to be led by Dr. Tracy Batchelor of the Massachusetts General Hospital's Cancer Center, and Dr. Luis Parada of Univ Texas Southwestern Medical Center, should provide an exciting opportunity for bringing together leaders in the genetics, biology, and treatment of this important group of tumors.

## 2010 Forum: Cancer Genomics

Cancer is a genetic disease. Increased cancer susceptibility can result from inherited mutations in tumor suppressor genes and oncogenes as well other genes. In addition, in both inherited and the much more common sporadic cancers, mutations arise that drive the development and progression of the cancer. Decades of research have led to an understanding of the types of pathways that become genetically and epigenetically compromised in cancer; however, a comprehensive understanding of the genetics of all tumor types is not yet available.

In recent years it has been possible to bring therapies to the clinic that are targeted to genetic defects found in cancer. The classic example is the treatment of Chronic Myelogenous Leukemia (CML). This disease is characterized by a specific chromosomal translocation resulting in the breakage and fusion of two genes to create the novel, mutant BCR-ABL fusion gene. The expression of this fusion gene drives tumorigenesis in this disease. The development of a class of agents that inhibit the BCR-ABL oncoprotein, which is only expressed in CML cells has dramatically improved the prognosis of patients diagnosed with CML. Many other such anti-cancer drugs have been developed or are under development for treating other types of cancer. However, the ability to more broadly implement these types of therapies will require a much more comprehensive understanding of the genetic and epigenetic changes in different cancers.

Since the successful sequencing of the human genome, there have been rapid advances in the development of genomic technologies. These new technologies, which are advancing and evolving at a rapid rate, have made it possible to gain a much more detailed understanding of the types of genetic changes present in cancers. For example, it is now possible to detect genetic changes that occur at low frequencies in different cancer types and begin to explore whether such mutations are bystander mutations or whether they influence the development and progression of cancer, and therefore might guide the development of targeted therapeutics. In addition, it is also possible to map the types of genome rearrangements that occur in cancers in much greater detail. In parallel with these developments has been the development of sophisticated bioinformatics needed for analyzing the much larger data sets being generated. Over the next few years, it is likely that our ability to explore the genetics of individual cancers will expand by a previously unanticipated extent.

In this rapidly evolving field, the meeting, to be led by Dr. Richard Kolodner of the Ludwig Institute for Cancer Research and Dr. Michael Stratton of the Sanger Center, will provide an important opportunity to explore our current state of knowledge of Cancer Genomics and the directions in which the field is moving.

## International Neuroblastoma Risk Groups (INRG)

Since 1986, the Foundation has sponsored and funded a series of conferences to standardize the international neuroblastoma criteria for diagnosis and response to treatment. Acceptance of these standards allows the principal neuroblastoma research groups around the world to compare results and compile large data bases used for improving diagnosis and treatments for this childhood cancer.

The rapid explosion of medical knowledge and technology has necessitated frequent updates to these standards.

Fall of 2005, the Foundation funded the fourth such meeting chaired by Drs. Susan Cohn and Andy Pearson. Fifty-two delegates attended, representing the six major pediatric study groups around the world.

*"We have submitted 4 papers to the Journal of Clinical Oncology discussing the new International Neuroblastoma Risk Group Classification Schema, the new INRG staging system, recommendations for evaluating of minimal residual disease, and standard operating procedures for examining tumor biology. In addition, Kate Matthay is in the process of writing INRG recommendations for evaluating disease using MIBG.*

*When I met with you a few years ago, I told you that I would make sure that we completed this project. While this project has been VERY challenging, it has also been VERY rewarding. At the neuroblastoma meeting in Japan, there are several papers that are going to be presented using the data we collected on over 8,000 children with neuroblastoma diagnosed around the world.*

*Andy and I are delighted that the papers have finally been published, but we realize this is just a beginning. We have plans to dramatically expand the database and develop a better infrastructure for additional research studies like the one I have attached in which the database was mined to examine the outcome of patients with low stage, MYCN amplifying tumors. We also envision modifying the INRG classification system as tools to analyze the entire genome become more accessible."*

Susan I. Cohn, MD  
University of Chicago  
Children's Hospital



In December, 2008, three articles were published in the Journal of Clinical Oncology. Links to the full articles are posted on our web site at [www.wgfrf.org](http://www.wgfrf.org)

## Awards: “FOCUS on the FUTURE” Program

### The FORBECK SCHOLAR AWARD

The William Guy Forbeck Research Foundation is pleased to sponsor this program to further the advance of cancer research. The “Scholar Award” recognizes promising young scientists working in this field.

The Foundation looks for outstanding clinician or post-doctoral fellows with an interest in cancer research. Award recipients are invited to attend the Foundation Forum held in November in Hilton Head Island, South Carolina. After receiving this award, scholars are invited to participate in the Scholar Retreat held in Lake Geneva, Wisconsin.

Nominations are made by letter of recommendation from the applicant's director of studies, including a short synopsis of the applicant's research interest and a brief explanation of why this individual is recommended. Nominations are due in the spring of each year.

The Foundation received a number of very qualified applications for the 2008 Forbeck Scholar Award. The Scientific Advisory Board selected four outstanding young scientists to attend the 2008 Forum in Hilton Head and receive this award. The Foundation was pleased to present this year's Scholar Award to these talented young researchers.

### 2008 SCHOLARS

**Joshua Brody, MD** is presently a Clinical Fellow in Medical Oncology at Stanford University School of Medicine. Joshua was nominated by *Dr. Ronald Levy, MD* and is currently focusing his research efforts on developing a new strategy of immunotherapy for mantle cell lymphoma. He plans to take advantage of the early post-transplant period, when the immune system is depleted, and during which T cells can expand rapidly, and proposes to administer educated T cells together with an idiotype vaccine during this window of immunologic opportunity. According to Dr. Levy, “Josh does it all. He...sees patients, does preclinical modeling, designs and executes the trial and develops and performs the immune monitoring assays.” Joshua received a B.S. in Molecular and Cellular Biology from Harvard University and his MD from Stony Brook School of Medicine.

**Russell (Rusty) Jones, PhD** is an Assistant Professor at McGill University. Rusty's past research has provided critical insight into understanding how energy stress controls cell growth, proliferation, and survival. In addition, he has developed a research program aimed at identifying signal transduction pathways that influence cell proliferation in the immune system. Going forward, Rusty plans to focus his research efforts on determining how cancer cells drive proliferation and mediate adaptation strategies to survive stress, and understanding the role of metabolism in the regulation of immunity. Rusty received his PhD and BS from the University of Toronto, and was nominated to the Scholar program by *Dr. Craig B. Thompson*.

### **Stefanie Sarantopoulos, MD, PhD**

was nominated to the Scholar program by *Dr. Jerome Ritz* of the Dana-Farber Cancer Institute. According to Dr. Ritz, “Stefanie is a very talented new investigator...focusing her efforts in areas of translational research in the hope that her research will have a direct impact on patients undergoing allogeneic stem cell transplantation.” At the Dana-Farber Cancer Institute, Stefanie is currently focused on studying the immune pathology of chronic graft versus host disease (GVHD) after allogeneic hematopoietic stem cell transplantation (HSCT). She received her MD and PhD degrees from Boston University School of Medicine and subsequently trained in Internal Medicine at Boston Medical Center. While at Boston Medical Center she served as Chief Medical Resident.

*“A hearty thank you for giving me the opportunity to meet and collaborate with physician-scientists at the recent Symposium. The experience was terrific (and invaluable really). I can't imagine having had the chance to meet with those guys otherwise. ...your efforts are high impact because you get at the root of the issue—face the major unknowns in biology and disease in order to develop treatments. I'm really looking forward to the Scholar Retreat this fall!”*

**Stefanie Sarantopoulos, MD, PhD**

**Lili Yang, PhD** is currently a Postdoctoral Fellow at California Institute of Technology, and was nominated to the Scholar program by *Dr. David Baltimore*. The theme behind Lili's research is the use of gene transfer technology to reprogram the immune system. She is currently leading two programs - the first, in HIV therapy, involves antibody gene transfer into CD34+ cells in HIS mice. The second uses gene transfer approach to target antigen genes to dendritic cells. Dr. Baltimore states that “[Lili's] prodigious energy, imaginative science and remarkable knowledge of the immunologic literature have allowed her to make progress on many fronts.” Lili received her PhD in Biology from Caltech.

*“...the most impressive meeting that I have ever attended. It is amazing that you gathered almost all the best intelligence of the immunotherapy field in the same room, and created a unique environment for people to share information, and discuss/debate freely. What I learned from this single meeting is probably more than the sum of all the related meetings that I have attended in the past years.”*

**Lili Yang, PhD**



*Lily Yang, Stefanie Sarantopoulos, Joshua Brody, Russel Jones*

# Foundation Scholar Board



**Jamie Collins**

As the 4th year of Scholar meetings in Lake Geneva, WI ended we said goodbye to our first graduating class. *Edward Attiyeh, Nabeel Bardeesy, Anthony G. Letai and Kim Rathmell* have moved on but they assured us that the time spent at the Forbeck meetings has changed their research forever. *Kim Rathmell* wrote, "Thanks so much for all you do! You have made a difference for science and cancer research for more people than you can imagine!"

While it is great to get such positive responses back from participants, we still battle the question of how to quantify the accomplishments of the Foundation. This year at the Blue Jean Ball we heard a 3rd year Scholar, *Kim Kelly*, list 4 collaborative research projects that she is part of thanks to the Scholar Retreat. As difficult as it may be to quantify the success of these meetings it is obvious that the many years of Retreats and Forums are inspiring innovative paths for cancer research.

We have an incredible list of Mentors and Scholars lined up for the 5th Scholar Retreat, September 17th to 20th. The range of science that will be represented will be Stem Cells, Cellular Imaging, Immunotherapy and Micro RNA. This meeting continues to be a melting pot for innovative science.

Thank you to the Scholar Board and *Gretchen Oettinger*, of Culture 22, for all their work on the Blue Jean Ball. Aside from the rain it was a successful and fun event. We need everybody's help, especially in this tough economy, to ensure the future of these meetings. The next Blue Jean Ball will be September 19th. Consider becoming a Sponsor!

My thanks to all,

*Jamie*

**Jamie Forbeck Collins**

## SPONSOR A SCHOLAR

The Foundation greatly appreciates all donations, which go directly towards funding Forbeck Foundation scientific think tanks. One such Forum is the Scholar Retreat, held annually in Lake Geneva, WI. Scholar and Mentor attendance at the Retreat involves travel costs, honorariums, meals, housing and general meeting costs, all of which are funded by your generosity.

The Forbeck Foundation has been noted as one of the most tightly financed cancer research organizations. Traditionally, about 85% of the total annual expenses directly support the annual forum, specific Foundation projects, and the annual meeting of the Scientific Advisory Board. The Scholar Board follows the same financial principals.

We are very grateful to everyone who participated at the Blue Jean Ball and helped us fund all of the 2008 Scholars. If you would be interested in directly funding a part of these meetings please contact us for details by visit our web site, [www.wgfrf.org](http://www.wgfrf.org) or e-mail us at [jrboard@wgfrf.org](mailto:jrboard@wgfrf.org).

### How you can help...

- Mark your calendars for the Blue Jean Ball on September 19th
- Become a Sponsor!
- Contact us at [jrboard@wgfrf.org](mailto:jrboard@wgfrf.org) to volunteer

## SPONSORSHIP LEVELS

Retreat Sponsor	\$10,000
Sponsor a Mentor	\$2,500
Research Sponsor	\$1,000
Blue Denim Donor	\$500

or

## SPONSOR A SCHOLAR

4-year pledge	\$1,000/yr
---------------	------------

## MEMBERS OF THE SCHOLAR BOARD

Seth Beers	Denver, CO
Brendan Cashman	Winnetka, IL
Chandler Dimberg	Chicago, IL
Benjamin Collins	Lake Geneva, WI
Jamie Collins	Lake Geneva, WI
Galen Eckland	Oregon, WI
Brian Fanning	Lake Geneva, WI
Liz Fanning	Lake Geneva, WI
Jeannie Gallucci	Chicago, IL
Michael Goetsch	Chicago, IL
Lisa Hoffman Garrison	Chicago, IL
Aaron H. Jesser	Chicago, IL
Bridgid Kyle	Chicago, IL
John E. Lehman, III	Chicago, IL
Cindy Maher	Lake Geneva, WI
Nicole Mazzei Goetsch	Chicago, IL
Christoph Oettinger	Schaumburg, IL
Glenn D. Pankau	Chicago, IL
Dick Payne	Lake Geneva, WI
Mollie Ring	Chicago, IL
Bryant Rowan	Chicago, IL
Aaron Taylor	Chicago, IL
Chrissie Taylor	Chicago, IL
Nicole Vaughan Rowan	Chicago, IL



**Scholar Board Friday night dinner**

## 4TH SCHOLAR RETREAT

September 11-14, 2008 - Lake Geneva, WI

The 2008 Scholars' Retreat was held in September 2008 at Lake Geneva, representing the 4th year since the birth of this program. The event was a smashing success. The Retreat is an annual meeting which is attended by the most current 4 years of Forbeck Scholars in addition to a panel of faculty mentors. The meeting follows the Forbeck Forum model of up to 5 slides maximum, with extensive scientific exchange and discussion. However there are several important distinctions of this meeting, relative to the Forum, distinctions which significantly impact the experience for the scholars.

Scholars Retreats are attended by young cancer researchers who inherently focus on a diverse cross section of topics within the field of oncology or related disciplines within biomedical research. Since Scholars are selected on the basis of their achievements as well as the focus of the Forum which they attend, the mixing of Scholars from different years brings together unique combinations of expertise.

In addition to the diversity of scientific subject matter, the Scholars are also diverse in their stages of professional development. While some are still working within a mentor's lab, others (on the older end of the spectrum) are likely to have started their own independent laboratories. This mix of "seniority" provides a novel opportunity for the attendees to learn the inside scoop from friends and colleagues who have recently "been there" for many of the challenges inherent in a biomedical research career. For

example extensive discussions involve faculty job searches, negotiations, compensation (start-up packages), grant writing strategies, "how to get invited to a meeting," or "how to get invited to write a review." Other notable topics include "how to respond to journal editors" and "is it better to publish two small stories or one large story" and "how to manage scientific collaborations without losing credit for your work."

This year's attendees were a particularly animated group. The quality of the science was outstanding. The very best technologies were routinely employed and every scholar had a truly interesting story to tell. Although their discoveries are too extensive to review here, it is abundantly clear that the Retreat was housing future leaders in multiple important research areas.

Five faculty mentors also attended. Although they tried their best to keep up with their younger colleagues at the bar, not all of them succeeded (the younger generations seem to be breaking records). But mentors' scientific presentations proceeded as among a collection of peers. I know I can speak for the other mentors in saying that the "learning" was happening with equal flux in both directions. Fortunately there were at least a few morsels of career advice which the mentors could offer, to exploit their greater experience, if not age. The keynote speaker was Chuck Sherr from St. Jude's Hospital, who participated together with the other mentors Anindya Dutta, Martine Roussel, Norman Sharpless, and myself.

David E. Fisher MD, PhD  
Massachusetts General Hospital

## 2008 ATTENDEES

### SENIOR INVESTIGATORS

Anindya Dutta, MD, PhD	University of Virginia	Charlottesville, VA
David E. Fisher, MD, PhD	Massachusetts Gen Hosp	Boston, MA
Martine Roussel, PhD	St. Jude Children's Research Hosp.	Memphis, TN
Norman Sharpless, MD,	University of North Carolina	Chapel Hill, NC
Charles Sherr, MD, PhD	St. Jude Children's Research Hosp.	Memphis, TN

### 2004 SCHOLARS

Edward Attiyeh, MD	Children's Hospital of Philadelphia	Philadelphia, PA
Nabeel Bardeesy, PhD	Dana Farber Cancer Institute	Boston, MA
Anthony G. Letai, MD, PhD	Dana Farber Cancer Institute	Boston, MA
W. Kimryn Rathmell, MD, PhD	Univ. of North Carolina	Chapel Hill, NC

### 2005 SCHOLARS

Kimberly Kelly, PhD	Massachusetts General Hospital	Charlestown, MA
Ingo K. Mellinshoff, MD	University of California	Los Angeles, CA
Michal Safran, PhD	Dana Farber Cancer Institute	Boston, MA
Benjamin B. Williams, PhD	Dartmouth Medical School	Hanover, NH

### 2006 SCHOLARS

Benjamin L. Ebert, MD, PhD	Broad Institute of Harvard & MIT	Boston, MA
Carla F. Bender Kim, PhD	MIT & Children's Hospital	Boston, MA

### 2007 SCHOLARS

Kristina Cole, MD, PhD	Children's Hosp of Philadelphia	Swarthmore, PA
Jun Lu, PhD	MIT	Cambridge, MA
Carl Novina, MD, PhD	Dana Farber	Boston, MA
Andrea Ventura, MD, PhD	Sloan Kettering	New York, NY

## SPONSOR A SCHOLAR

*These donations directly fund a Scholar's attendance and participation in the meeting for 4 consecutive years.*

Dalco Metals  
Mary Jo & Dick Pfeil  
Tom and Pat Theys

## SPONSOR A MENTOR

*Mentor Sponsors fund a senior investigators attendance at a Scholar Retreat.*

William Blair & Company  
Geneva Investment Management

## RESEARCH SPONSOR

*Research Sponsors fund a scientist's attendance for 1 year to the Scholar Retreat.*

Lars & April Brunk  
Julie K. Ieronimo  
Cindy Maher Forbeck, RE/MAX Realty  
Christian & Michelle Nimmo  
Nancy Paullin

## BLUE DENIM DONORS:

*Each year, our sponsors pledge a generous contribution, allowing the Scholar Board to cover all up front expense.*

E Street Denim  
Interstate Insurance Group  
Thomas & Jacqueline Murray

*Many people help make the Scholar Retreat a success, through general contributions or through donation of event supplies and auction items.*

## COMMENTS FROM SCHOLAR RETREAT

*"The exposure to people of broad experience is invaluable...The ability to have long conversations in an unburied atmosphere with people whose expertise is different from my own is useful and fun."*

**Tony Letai, MD, PhD**

*"This is by far the best format for a meeting..."*

**Andrea Ventura, MD, PhD**

*As much as I love science on a daily basis whenever I am attending one of the Forbeck meetings I find science even more exciting and enjoyable. I feel that the Forbeck meetings contribute to my scientific development both with ideas and collaborations.*

**Michal Safran, PhD**

*Thank you for the invitation to the Scholar's retreat. I think you've set up a very unique meeting whose effects on the participants will be felt over many many years. Congratulations!*

**Anindya Dutta, MD, PhD**

# Founding Sponsors

Mr. Richard Barnes	Mr. and Mrs. Robert P. Evans	Mr. and Mrs. John T. Geldermann	Mr. and Mrs. William J. McGinley
Chicago Children's Charities	Mr. and Mrs. A. George Forbeck	Mr. and Mrs. Thomas A. Geldermann	Mr. and Mrs. Michael O'Brien
Mr. Thomas H. Dittmer	The Traders Foundation	Mr. and Mrs. Hayden Leason	Mr. and Mrs. Richard Pfeil

*In the early years of the Foundation, each of the Founding Sponsors made a commitment for \$5,000 per year for five years. These pledges provided a stable financial basis for the Foundation and allowed efforts to be concentrated on establishing the Foundation and organizing programs.*

***In grateful acknowledgement of our donors... (from January, 2008 thru March, 2009)***

## Benefactors

<p>Mrs. Orval Adam          Lisa and Bob Aiken          The Andersons          Margaret and Bill Anderson          Robert J. Aarix          The Arthurs          Marnie Atkinson, Jr          Rotchy and Julie Barker          Alben F and Clara G Bates Foundat'n          Sarah and Ralph Bogan          Mr. and Mrs. John A. Bollero, Jr.          Charles and Cecilia Bona          Jay and Kathy Bothwick          Thomas and Melva Breitenstein          Maud F. Brennan          Captain Dusty's, Inc          Charlie and Linda Carey          Juels and Sally Carlson          Brendan and Danielle Cashman          Thomas and Jacqueline Cashman          Bruce and Davi-Ellen Chabner          Edward B. Chez and Camille DeFrank          CME Group          David and Dorcas Collins          Drs. Robert and Cynthia Conlon          Joe and Joella Cramblit          Robin and Ed Culbertson          Mr. and Mrs. Hanley Dawson          Gale and Dave Dewar          Karen and Barry Diekelman          Diane and Chandler Dimberg          Mr. and Mrs. Charles H. Drawdy, Sr.          Sally Edwards          Margaret Eklof          Elgin Toyota          Linda P. Ellis          Rebecca Esch          Ira K. and Susan C. Evans          Scott and Wendy Farber          George Farnsworth          William Fleming and Jane Crosby          Jennifer and George Forbeck          Ed and Sally Frick          Rob and Ann Furst          Mrs. William B. Gage, Sr          Rachel and Bill Gage</p>	<p>Frank and Kathleen Gazzolo          Mr. and Mrs. J. Jeffrey Geldermann          Jane F. Geldermann          Robert and Dana Geldermann          Stephen and Carolyn Geldermann          Tom Geldermann          Geneva Investment Mgmt of Chicago          The Geraghty Group, Inc          Eric and Diane Geschke          Ray Geschke          Ginger and Hollis J. Griffin          Dean &amp; Lois Griffith Foundation          Babbie and Don Guscio          John and Dana Hagenah          Peter and Coby Hannon          Prudy and Charles Harker          Anna and Paul Harmon          Sandra and John Hatch          John and Helen Hawkinson          Healing Baskets, Inc          Don and Marilyn Hedberg          Dr. and Mrs. Martin D. Herman          Jay and MaryBeth Hicks          Jim and Ann Howard          Judith A. Howe          Mr. and Mrs. Daniel R. Huber          Mortimer G. and Susan V. Huber          J. P. Ieronimo          Jim Irmen          Terence J. Irmen          Bill and Jan Jackson          Jesse and Dawn Marie Jacobs          D. Elaine Jacobsen          Jerome and Kathleen Jacobson          Aaron and Darcy Jesser          Jesser and Farber, LLP          Kathy and Arvid Johnson          Bill and Helen Johnson          Gary and Sally Jouris          Robert H. Joyce          Mary O. Kasten          John and Margaret Kinzer          William M. Kinzer          Mr. and Mrs. Peter C. Kostantacos          Kay and John Kyle</p>	<p>John and Mimi Kyle          Mr. and Mrs. Montague T. Laffitte          Lake Geneva Country Club          Lake Geneva Oral Surgery          Tobey and Dennis Lannert          Curtis and Barbara Lansbery          Thomas and Juliann Larimer          Wendell and Shirley Larimer          Larry and Sue Larkin          Mr. and Mrs. Hayden Leason          Michael and Stephanie Leason          Bill Lehman and Diane Bjorkman          John W. LoCicero          Mrs. Sandra Loebmann          George and Karen Ludington          Mr. and Mrs. James P. MacMillan          Martha and James Maddock          Mr. and Mrs. Robert A. Marks          Judy and Michael Mason          Edgar and Lisa Massini          Nan and John Mattson          Mr. and Mrs. Mark C. McCloy          McCullough's          Mrs. Maggie McGinley Field          Dr. and Mrs. Thomas W. McNeill          Judith and Stephen Miller          Paul and Joan Millichap          Stephen and Kim Minette          Gail Modetz          Lawrence and Colleen Moelmann          Robert &amp; Patricia Moore Foundation          Alice J. Morava          J. P. Morgan Chase Foundation          Gregory and Terri Mueller          Courtney and Sean Murphy          Robert and Mary Ellen Muskat          Betsy and Hardy Nalley          Leslie Nolan          Chuck and Barbara Obligato          Karl G &amp; Lucy T. Otzen          Irene and Eric Pagh          Palmetto State Bank          Annette M. Peterson          Luanne and Douglas Peterson          Mary Jo and Dick Pfeil</p>	<p>Phibro Animal Health Corp          Caroline and William Philipbar          Barbara J. Phillips          Anthony and Charlene Picone          Brian and Dana Porter          John and Annie Porter          Posen Family Foundation          Lois L. Pratt          Hank and Sally Proesel          QBE          Laurie Quindel          Fred and Mary Reed          Glen Reed          Jane and Bill Reilly          Mary Kay Ring          William and Susan Rolander, Jr.          Martine F. Roussel, PhD          Stefanie Sarantopoulos, MD, PhD          Patricia L. Schmidt          Robert &amp; Rebecca Schutz          Shodeen Family Foundation          Paul and Cecile Smith          Judith Fox Smyth          Neoma B. Sorensen          Brent and Holly Starck          Tobias and Shari Steivang          E. C. Styberg Foundation, Inc          Rebecca Swangstu          Colleen and Bob Sweeney          Jane P. Sweetsir          Luis E. and Beth Taveras          The Terlato Family          Thompson Foster Street Foundation          Bill and Barbara Turner          John P. Vaile          Nan B. Vaile          Mr. and Mrs. John M. Volkhardt          Jake and Mary Lynn Vrabel          William M. Walsh          Murray and Pat Wheeler          Mr. and Mrs. Boyd Whitney Jr.          David and Nancy Williams          Jeannette M. Windon          Susan and David Wright          Norah and Rick Zeroka</p>
---	---	--	---

## Honoraria

Jacki Barron	Benjamin Collins	Mary Kay Ring
Bruce and Davi-Ellen Chabner	Montague and Ann Laffitte	William Terlato and Terlato Wine Group

## In Memorium

<p>Bonnie Abwarish          Mary G. Altier          Joan Barry          Alberte Bartlett          Jerome R. Bona          Maude Brennan          Lindsay Noble Buchanan          Weechie Carroll          Helen Chitrowski          Harriet Drawdy          Harry Strouse Eklof          William Guy Forbeck          Jane McMillan Frackelton          William H. Frackelton, MD</p>	<p>Frank Gallucci, Sr.          Frank Gallucci, Jr.          Joan Geldermann          Pearl Geschke          Richard Goodemote          Elizabeth Graves          Mary Agnes Huber          Donna Irmen          Ruth Kane          Shirley Kaufman          Richard A. Kinzer          Gary K. Laatsch          Larry S. Lawrence</p>	<p>Corey Lazzeroni          John E. Lehman, Jr.          James Lehman          Marvin David Libman          Stuart Malina          William Marscher          William J. McGinley          Dorothy F. Michaud          Florence Milstein          Robert T. Morava, Sr.          Violet Neukirch          Richard H. Pfeil, Sr.          Walter "Wally" Phillips</p>	<p>Linda Porec          Robert J. Porter          Ronald E. Pratt          Claudia          Mark Reader          Frederick L. Reed          Betty L. Reed          Richard J. Ring          Sarah Schultz          Daniel J. Shelley          Lillian M. Shodeen          Robert W. "Bobby" Smyth          Florence Tiersky</p>
---	--	---	---

The accounting firm of *Cherry, Bekaert and Holland* audits the Foundation's financial records annually.

The Foundation has established a very sound financial position. Steady growth in income has allowed the Foundation to expand its program in additional funded projects and now through the efforts of the Scholar Board, the "Scholar Retreat." The Trustees continue to aim at a very high mark - that 85% of the total expense goes directly to support scientific programs. In 2008, 83.7% of all expenses funded scientific activities.

**BASIS OF SUPPORT**

The William Guy Forbeck Research Foundation desires and has a broad base of support. Of major significance to the Foundation are the contributions from many individuals and their families. Many people have chosen to use the Foundation as a fitting memorial gift. A number of corporations and other foundations have also supported the Foundation with contributions, some having very rigorous qualifications for grants.

In 2008, the Scholar Board raised funds through their Fallfest fund raising event and contributions. They met their goal in funding most of the costs of the Scholar Retreat.

**EXPENSES**

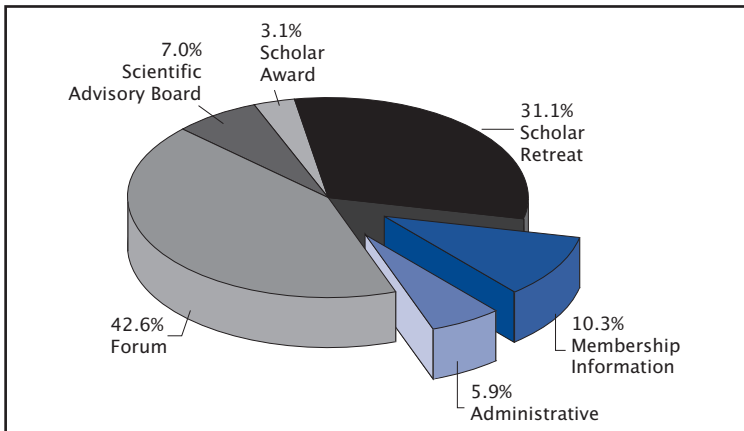
Historically, 85%-90% of the total expenses go directly to supporting the annual Forum and Foundation projects.

Membership information costs include the annual newsletter, member mailings, the video and the web page.

In 2008, the Foundation had unusual expenses for completing a significant upgrade to the Foundation video, and for a major revision of the Foundation web site.

The Foundation Trustees participate at their own expense. Administration expenses include auditing costs, as well as printing and postage expense.

Members of the Scientific Advisory Board attend the Forum meeting in Hilton Head and hold their annual meeting at that time. The SAB provides the technical direction for the Forum and the Foundation.



newsworthy and therefore the truth becomes distorted and the expectations of the public are let down.

In light of this reality as the Foundation comes to its 25th anniversary year it is pertinent to reflect on what we do, how we spend our funds and where we are going. Firstly, it is a major achievement to reach such an anniversary and a complement to *Jennifer, George and Jamie* along with the Board of Trustees who have taken so much care to ensure the future of the organization. Also, of particular note is the formation of a "Scholar Board" to focus on the scholar meetings and to potentially step up to the main board as and when required.

Even in times of low levels of funding for science a Foundation needs to consider how best to use its resources. It would be only too easy to give grants for research but in reality the levels of funding needed to be successful in today's complex scientific environment markedly outweigh the resources of the Foundation. In fact to make this strategy a reality our funding base would have to increase beyond anything envisaged by the Trustees today. Funding success also means funding failure as in reality more research projects fail than succeed; this being the nature of the development process. This is perhaps a bitter pill to swallow when everyone wants to see their finances put to the best use possible. This is why the Foundation sticks with its philosophy of not funding science directly but does this in an indirect fashion by promoting the interaction of scientists and clinicians to further their research.

The benefits of this approach to driving science forward may also not be immediately obvious, but it is sowing seeds for future collaborations and interactions. This is particularly pertinent for the scholar meetings where creating an environment for interaction of scholars with key investigators and repeating this over a four-year cycle can have significant benefits.

For the immediate future we are looking at consolidation, making sure that the scholar meeting runs to the same high standard as the main meeting in Hilton Head. After that, hopefully the economic tide will have turned and we can think of the next steps for the Foundation. In the interim the 25th anniversary for the Foundation should be a time to celebrate as well as reflect. It is a major achievement and all of those who have worked so diligently on behalf of the Foundation have something to be truly proud of.

John T. Kemshead  
Chairman, Scientific Advisory Board

## Board of Trustees

**David R. Barry, Jr.**  
Chicago, IL

**James A. Buchanan**  
Portland, ME

**Jamie Forbeck Collins**  
Lake Geneva, WI

**Charles H. Drawdy**  
Hampton, SC

**Jennifer K. Forbeck**  
Hilton Head Island, SC

**Edward R. Frick**  
Pinehurst, NC

**Jeffrey C. Hadden**  
Weston, MA

**Hon. William G. Herbkersman**  
Bluffton, SC

**Terence J. Irmen**  
Sylvania, OH

**T. L. Sam Irmen**  
Maumee, OH

**Charles H. Jesser, CPA**  
Chicago, IL

**Ann Huber Kasten**  
Winnetka, IL

**Montague T. Laffitte**  
Bluffton, SC

**Hayden Leason**  
Ft. Lauderdale, FL

**Michael H. Leason**  
Aquadilla, PR

**Luis E. Taveras, PhD**  
Ortley Beach, NJ

**William Terlato**  
Lake Forest, IL

**Thomas Theys**  
Fontana, WI

**Henry Wagner, Jr. MD**  
Hershey, PA

**Nancy Wellard**  
Hilton Head Island, SC

### EMERITUS TRUSTEES

**Joseph W. Black, MD**

**Hon. Sol Blatt, Jr.**

**Thomas A. Geldermann**

**Hon. Ernest F. Hollings**

**Joseph Laver, MD**

## Scientific Advisory Board

**Bruce A. Chabner, MD**  
Massachusetts General Hospital  
Boston, Massachusetts

**David E. Fisher, MD, PhD**  
Massachusetts General Hospital  
Boston, Massachusetts

**Michael Jensen, MD**  
City of Hope  
Duarte, CA

**Jan Karlseder, PhD**  
The Salk Institute  
La Jolla, CA

**John T. Kemshead, PhD**  
Baxter Healthcare, Chicago, IL and  
University of Manchester, England

**John D. Minna, MD**  
University of Texas  
Dallas, Texas

**Jean Y. J. Wang, PhD**  
University of California, San Diego  
La Jolla, CA

### EMERITUS MEMBERS:

**Garrett M. Brodeur, MD**  
Children's Hospital of Philadelphia

**Webster K. Cavenee, PhD**  
Ludwig Inst for Cancer Research

**Alan D'Andrea, MD**  
Dana Farber Cancer Institute

**Isaiah Fidler, DVM, PhD**  
MD Anderson Hospital

**Arnold I. Freeman, MD**  
Hadassah Hospital (Israel)

**Ed Harlow, PhD**  
Massachusetts General Hospital

**Michael B. Kasten, MD, PhD**  
St Jude Children's Hospital

**Philip A. Pizzo, MD**  
Stanford Univ School of Medicine

## In Appreciation



Our heartfelt thanks go to all the people who have worked to make the activities of the Foundation a success.

We are grateful to the Scientific Advisory Board and the Forum participants, the scientists and clinicians whose leadership and effort are the front line in the war against Cancer.

Our special appreciation goes to the Foundation trustees, the Junior Board and volunteers whose thoughtfulness, time and energy have done so much for the success of the Foundation and the Forums.

Most importantly, our thanks go to the hundreds of donors, individuals, businesses and foundations, whose financial support assures our continued work in Cancer research.

Sincere Thanks,

George and Jennifer Forbeck

## Mission

The mission of the William Guy Forbeck Research Foundation is to promote advances in the field of oncology, particularly pediatric oncology, by shortening the cancer research timetable.

## Strategies

While the Foundation may provide grants for pilot research studies and educational efforts, its centerpiece activity will be an annual Forum, a scientific roundtable held at Hilton Head Island, South Carolina.

Attending the Forum each year will be twelve to fifteen physicians and scientists who will meet in a completely private "think tank" environment, where they can exchange ideas freely in the hope of building on each other's ideas, knowledge, and experience.

The objective is not to discuss published research, but rather to provide a forum for the cross fertilization of ideas, concepts, and observations.

Participants will be invited to the Forum based on the recommendation of the Foundation's Scientific Advisory Board, a distinguished panel of physicians and scientists.

We fully support the activities of the William Guy Forbeck Research Foundation Junior Board, particularly the Scholar Retreat.

*It is through your generous support that continuing research in the field of childhood cancer can be ensured. Contributions are tax deductible for federal IRS purposes. The IRS file number is 580063499. For additional information please fax: (843) 837-3088, visit our web site [www.wgfrf.org](http://www.wgfrf.org) or write: William Guy Forbeck Research Foundation, 23 Peninsula Drive, Hilton Head Island, South Carolina 29926*