



Terrence J. Irmen

From The Chairman's Desk

The Forbeck Foundation is now in our 23rd 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 been strengthened with the addition of two new members, *Louis Taveras, PhD*, and *Tom Theys*. We look forward to the resources and talents that they each bring 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 "Stem Cells" co-chaired by *John E. Dick*, University of Toronto and *David Scadden*, Massachusetts General Hospital. The 2007 Forum topic on "MicroRNA" will be co-chaired by *Carlo Croce, MD*, Ohio State University and *Gregory Hannon, PhD*, Cold Springs Harbor Laboratories. We plan to continue these forums as long as there is work to be done.

Our Junior Board, led by *Jamie Forbeck*, organized our second Forbeck Scholar Retreat held in Lake Geneva, Wisconsin last September.

The retreat was chaired by *John Kemshead, PhD*, Chairman of the Forbeck Scientific Advisory Board. Participants in the retreat have indicated that this year's event was another overwhelming success. The Junior Board held another great fundraising event following the retreat to raise funds for next year's retreat.

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. 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.

Terrence J. Irmen
Chairman, Board of Trustees

In This Issue...

From the Chairman's Desk	1
Scientific Advisory Board	1
Forum 2006: <i>Stem Cells</i>	2
Junior Board	6
Junior Board Donors	6
Scholar Retreat	7
INRG update	8
Awards: 2006 Scholars	8
Forums 2007 and 2008	9
Benefactor List	10
Financial Report	11
Trustees & Scientific Advisory Board ...	12
Mission & Strategies	12

SCIENTIFIC ADVISORY BOARD REPORT

Looking back, Looking into the future.



John T. Kemshead, PhD

For the first time in many years survival rates of many of the major cancers have increased slightly in Western society. This is obviously good news but a lot remains to be done. Our understanding of the cancer process in both adults and children continues to grow changing the way in which we think about developing new cancer treatments. Take for example last years Forum where the concept of cancer stem cells was discussed. Understanding that not all cancer cells in an individual are identical, opens up the opportunity of developing treatments that eliminate the cancer stem cell. If you can target therapy to this cell then the cancer can ultimately be eliminated and the patient cured. Conventional drug therapy is not this selective leading to research into new agents that are far more selective in their activity.

The Foundation has been playing a prominent position in focusing on the newer forms of cancer therapy. These not only supplement conventional chemotherapy and radiotherapy but also prevent damage to normal tissues and prevent drug resistance. Several of the Foundations main forums held in Hilton Head have focused on exploiting our new knowledge on the molecular biology of cancer.

Continued on page 11

2006 Forbeck Forum: XXIInd Annual Forum

November 2–4, 2006 Hilton Head Island, South Carolina

Subject: Stem Cells

- I: Stem Cell Properties
- II: Self Renewal
- III: Cancer Stem Cells & Environmental Control
- IV: Therapy & Metastasis

Chairmen

John E. Dick, PhD	University of Toronto	Toronto, Ont, Canada
David Scadden, MD	Massachusetts General Hospital	Boston, MA

Participants

Philip Beachy, PhD	Stanford University	Stanford, CA
Ann Chambers, PhD	London Health Sciences Centre	London, Ont, Canada
Michael F. Clarke, MD	Stanford University School of Medicine	Palo Alto, CA
Hans Clevers, MD, PhD	Uppsalalaan - Hubrecht Laboratory	Utrecht, the Netherlands
R. Keith Humphries	British Columbia Cancer Agency	Vancouver, BC, Canada
Craig T. Jordan, PhD	Univ of Rochester School of Medicine	Rochester, NY 14642
Haifan Lin	Yale University School of Medicine	New Haven, CT
Luis F. Parada, Ph.D.	UT Southwestern Medical Center	Dallas, TX
Professor Chris Potten	EpiStem, Ltd.	Manchester, UK
Derek van der Kooy, PhD	University of Toronto	Toronto, Ont, Canada
Amy Wagers, PhD	Joslin Diabetes Center	Boston, MA
Irv Weissman, MD	Stanford Univ School of Medicine	Stanford, CA



John E. Dick



David Scadden

2006 Conference Report on “Stem Cells”

by John E. Dick, Ph.D. & David Scadden, M.D.

The 2006 Forbeck Forum focused on a question that has captured the attention of cancer scientists and cancer research funding bodies around the world. Does every cell in a tumor, whether liquid (leukemia) or solid, have equal ability to sustain cancer growth or are some cells within the tumor more potent than others? The answer to this question has the potential to alter research approaches away from studying the cellular and molecular properties of entire tumor tissue towards focusing on the tumor-initiating cells or so called cancer stem cells (CSC). There was much discussion at the Forum on the best nomenclature for these cells. Evidence is emerging that for many tumors, they are organized as cellular hierarchies that are sustained by stem-like cells in much the same way as normal organs. To gain a clearer picture of CSC and their similarities and differences to normal stem cells, the 2006 Forum assembled 12 leading stem cell

scientists whose interests ranged from stem cell biology of lower organisms like *Drosophila* to blood and intestinal stem cells of the mouse, to normal human hematopoietic stem cells (HSC) and leukemic stem cells (LSC). The unifying theme of the Forum was that progress to identify and characterize CSC from different tumors and to understand their importance in cancer will only come when we understand how normal stem cells for each organ actually work. One field of study, normal or neoplastic, informs the other. Indeed by understanding the genetic and epigenetic programs that govern normal stem cells, we can begin to understand how the neoplastic process subverts normal stem and progenitor cells. This Forum showed once again the value in bringing together investigators from different stem cell areas that often do not talk to each other and having them focus their attention on one question. New research directions were formulated,

collaborative research projects were developed, and ultimately a strategy for progress in this promising field was stimulated in each of the participants.

As with many areas of biology our understanding of regulatory mechanisms that stem cells use comes from lower organisms. **Philip Beachy** described studies of a central pathway in pattern formation in *Drosophila*, the hedgehog (Hh) pathway and how this pathway regulates stem cells from several different tissues and how this pathway becomes subverted in cancer. For example, Hh activation in the skin leads to basal cell carcinoma. Yet pathway blockage in normal tissues does not result in abnormalities unless tissue injury ensues. He described how injury drives otherwise quiescent stem cells to be activated becoming migratory and proliferative. If pathway activity is sustained because of injury or inflammatory processes, additional epigenetic and genetic changes can arise resulting in

the generation of abnormal CSC. He demonstrated that even in non-epithelial tissues, such as multiple myeloma (MM), the MM-initiating cell (MM-IC) fractions had higher Hh pathway activation than the larger non-MM-IC fractions. This knowledge can be harnessed by developing therapies that block Hh activated CSC.

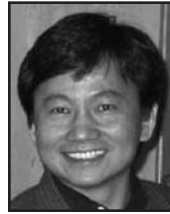


Hans Clevers

described how his long term studies of the genes that play a central role in the growth and differentiation of the cell types that make up the small and large intestine are revealing the genetic signature for each developmental stage from an intestinal stem cell, to the transit amplifying (TA) progenitors, to the mature lineages. Deregulation of many of these genes results in abnormal growth and neoplastic growth. He showed the role of the WNT pathway on the proliferation of TA progenitors, the Notch pathway in lineage determination, and the tissue specific pattern of function of effector genes such as Tcf4. Finally, he provided evidence of an intestinal stem cell in an unexpected location amongst the Paneth cells of the small intestine or the bottom of the crypt of the large intestine. These cells express a putative stem cell marker, GPR49. These studies provide the first glimpse of this new intestinal stem cell and how it may be involved in early steps of adenoma formation, the first step of the neoplastic process. Given the expression in normal stem cells and a small subset of neoplastic cells, this new marker may be pointing to a new marker for a colon CSC showing the power of combining normal stem cell and cancer stem cell studies.

The Forbeck Scholar **Joseph Wu** talked about his work aimed at controlling human embryonic stem cell (hES) development into mesodermal lineages and ultimately into cardiac mesoderm under controlled conditions. He developed a genetic reporter construct for cardiac differentiation that enabled multiparameter imaging by flow cytometry, bioluminescence, and functional imaging by PET. Collectively imaging techniques such as this will have important uses in all of stem cell biology.

Evidence is emerging that epigenetic factors play a large role in regulating stem cell function. Certain repressors expressed in stem cells prevent the activation of differentiation programs. However how these epigenetic factors function in stem cells is poorly understood. **Haifan Lin** again demonstrated



how discovery of fundamental control mechanisms of stem cells from lower organisms can lead to new insights of mammalian stem cell biology. He described the pathway of discovery from identification of the role that the gene PIWI plays in germ stem cells in *Drosophila* to its involvement in epigenetic silencing, in the process linking non-coding RNA such as RNAi and miRNA to epigenetic silencing. He showed that some proteins that interact with PIWI are canonical epigenetic factors that together to create a functional unit that regulates chromatin. PIWI and some PIWI interacting proteins such as HP1 regulate self renewal, a biological property that is central to the stem cell state. Moreover since PIWI genes are also involved in RNAi processing and function, this new work links non-coding RNA to chromatin regulation in addition to more well known functions of RNAi in post-translational modification. Interestingly the human version of PIWI, called HIWI is dysregulated in human germ cell tumors and GI cancer. Silencing of HIWI in human cancer cell lines results in reduced growth pointing to central importance in the neoplastic process.

Since stem cells are long lived and able to produce large numbers of progeny, mechanisms must be in place to guard against DNA damage within the stem cells and to prevent neoplastic transformation. One such mechanism was proposed some 40 years ago by Cairns that suggested since many stem cells must divide asymmetrically to yield one daughter stem cell and one committed daughter cell, that the original DNA template of the parental stem cell segregated to the daughter stem cell only. Since this template is not copied it is much less susceptible to mutation. **Derek van der Kooy** described a series of experiments to determine if this model held for neural stem cells (NSC). He utilized as an assay for NSC in vitro

sphere formation and marked primordial and newly replicated DNA strands with BrdU and 3H-TdR. He showed evidence of unequal strand segregation into NSC daughters. However, he also described the difficulties in showing that the resulting daughters were true NSC and that every NSC segregated DNA in this fashion. Therefore he turned to a more robust stem cell system of the germ stem cells of the fly ovary where cell position denotes stem cell or progenitor cell identity. With various genetic mutants he was able to alter the ratio of symmetric and asymmetric cell division and was able to provide some support for the asymmetric DNA strand segregation. However he also presented the uncertainties of the system to prove that all stem cells behave according to the principles of this hypothesis.

Self renewal and how this process is regulated by intrinsic and extrinsic factors is a central problem in stem cell biology. Self renewal is the key biological property that distinguishes a stem cell from any other cell type and it must be dysregulated in cancer. **Keith Humphries** described studies of the



role of Hox genes in regulating self renewal and how this process can be harnessed for stem cell-based therapeutics as well as how dysregulation occurs in leukemia. Forced expression of Hox B4 results in enormous expansion of HSC number in vitro and in vivo, however enforced expression in progenitor cells cannot convert them into stem cells. He also made a point of distinguishing between self renewal potential and the actual execution of the self renewal program. He also described how Hox genes altered because of chromosomal fusion to partner genes can co-operate to disrupt self renewal and block differentiation resulting in leukemia.

Self renewal was also the theme of a presentation by **Michael Clarke**. He described studies on the gene Bmi1 which is implicated in the self renewal of several stem cells including HSC because KO mice rapidly lose HSC after birth and succumb to bone marrow failure. Bmi1 represses a number of genes including p53, p16, and p19. When each of these KO are combined with

2006 Conference Report

Continued from Page 3

Bmi1 KO they are able to provide partial rescue of the HSC defect. Interestingly when all three downstream genes are mutated, the BM failure is rescued and HSC number restored. Careful analysis of these animals indicates that early progenitor cells gain self renewal potential, a property such cells have normally lost. If these animals are followed over time or are serially passaged, leukemia often arises. These studies begin to address the question of whether leukemia arises from the stem cell compartment or from the progenitor pool whereby these cells acquire self renewal potential.

The Forbeck Scholar **Ben Ebert** described an interesting series of studies to understand the basis for 5q- MDS. This disease is characterized by abnormal differentiation. He focused on the minimally deleted region and carried out an shRNA screen to determine if any of the genes within the deleted interval affects differentiation. He focused on erythroid differentiation and identified one gene RPS14 that blocks erythroid differentiation. He discussed other ribosomal genes that are also involved in other hematological diseases suggesting a link between this family of genes and erythroid maturation. He also described studies where he developed a genetic signature of erythroid gene expression that was predictive of MDS patients who respond to specific drug treatment. Ultimately such studies have enormous value in developing more effective therapeutics.



Irv Weissman

described a wide range of studies on HSC biology from early development, to the effects of aging on HSC and leukemogenic alterations in HSC. He carried out a lineage fate mapping study using genetically marked chimeras. He showed that the yolk sac blood island is not clonal. Interestingly he also showed that the endoderm derives from a limited number of precursors. For example the intestine seems to derive from 12-14 precursors in large clonal patches. He also described the intrinsic and extrinsic changes that occur in HSC as they age. Aged HSC produce fewer lymphoid

cells and more myeloid cells. As well the self renewal potential of the myeloid cells is increased. Collectively this insight into aged HSC shows that the age-related alterations in immune function can be traced back to HSC rather than age effects on the mature immune cells themselves. The alterations in HSC cycling, self renewal and the increased propensity for myeloid development including precursors that retain self renewal capacity provides mechanistic framework that explains the increased incidence of AML with age. Finally, he discussed the pathway of leukemogenesis from the normal cell of origin to the appearance of fully leukemic blasts. He argued that the initiating events must occur in HSC but that these are “pre-leukemic” in that they differentiate normally, the clone sustains additional genetic alterations. The mutations that occur leading to the generation of an LSC might occur in myeloid progenitors that now possess self renewal, thereby acting as self renewing leukemic stem cells. He proposed that examination of the hematopoietic system, especially the stem cell compartment by sequencing might uncover the number and sequence of pre-leukemic genetic alterations that occur before AML arises.



Amy Wagers presented a series of studies aimed at understanding the biology of murine HSC. She described a series of parabiosis experiments to examine

HSC migration in a non-transplant situation and showed that while many HSC cycle every day, it still takes 7 to 16 weeks to obtain full donor chimerism. She then reported on studies to identify HSC-specific genes involved in mobilization and discussed her work on EGF1 which is differentially expressed to high level in long term repopulating HSC compared to multipotential progenitors. Knockout of this gene results in enhanced mobilization and HSC cycling. Thus EGR1 acts to limit HSC cell cycle when mobilized linking HSC proliferation and mobilization. She argued that regulators of this type are needed to ensure that HSC that are mobilized from their niches do not cycle since HSC that cycle do not home back to their niches. She also described her studies on the role of specific classes of

osteoblasts in the niche (especially osteopontin expressing) and how they respond to HSC, that is the activity of HSC on regulating the osteoblasts that are involved in niche creation.

The Forbeck Scholar **Carla Bender-Kim** described her studies on the identification of a putative lung stem cell. These cells were identified on the basis of cell surface marker expression using an assay of naphthalene challenge. Most lung cells are damaged with this agent, but the stem cells are spared and able to contribute to lung regeneration. She termed these cells the broncho-alveolar stem cell (BASC). She then targeted expression of the k-ras oncogene to the BASC and showed that their number increased dramatically before tumor appearance. She showed that ras expression could only generate tumors if it was expressed in the BASC cell types. She showed that the tumor stem cells within the generated tumors could self renew as assayed by serial passage. Collectively her studies point the way to characterize in more detail the BASC and how they play a role in tumorigenesis. Finally they provide the basis to identify the human equivalent cell types in human lung cancers.

The majority of our understanding of cancer biology has come from studies in murine models of cancer. However, there is need to understand neoplastic processes in primary human cells because of subtle differences between the two species. **John Dick** discussed studies examining the hematopoietic stem cell hierarchy of primary human normal and leukemic cells using xenotransplantation systems. He showed that there were significant similarities between HSC and LSC, both contained stem cells with variable capacity for repopulation and self renewal organized as a hierarchy. The heterogeneity in LSC repopulation capacity poses a challenge to discover the molecular basis for this variable capacity and for the development of therapies that target the most quiescent LSC. He discussed one approach to eradicate LSC by targeting their ability to traffick in vivo and inducing them to differentiate. His group treated AML with specific monoclonal antibodies to CD44 and showed that they could interfere with LSC migration to the bone marrow and alter their

Continued on page 5

stem cell fate via induction of differentiation. He also described a series of experiments to create an experimental system of human leukemogenesis by transducing normal human stem/progenitor cells with viral vectors expressing oncogenes including MLL fusion proteins. Upon transplantation into the xenograft models, all mice developed human ALL and AML. Such models could be powerful tools to study the leukemogenic process in primary human cells. Finally he discussed recent studies to determine the identity of the CSC that underlies human colon cancer. He showed a robust xenograft assay for the colon cancer initiating cell (CC-IC), that could be used to quantify their number. In addition the CC-IC could be enriched by sorting on the basis of CD133 expression.



The normal cell within which brain cancer arises is not well understood. **Luis Parada** described a series of experiments in murine models where he targeted oncogenes to be expressed in specific neural cell types. He provided evidence that the hippocampus retains cells with neurogenic potential, these cells express the marker GFAP. By targeting a GFAP-cre marker to these cells, he showed that a stem cell defect occurred if cells expressing GFAP were ablated. He then went on to show that if the oncogene NF1 was expressed only in GFAP expressing cells in the context of a p53 deletion, that changes occurred in the sub ventricular zone before the appearance of tumor. He looked earlier in development at radial glial cells and showed that he only obtained brain tumors when the radial glia were targeted with NF1 expression as opposed to targeting other parenchymal cells. He then went on to examine the involvement of other regulatory genes to determine their role in oncogenesis as well as the sequence of activity. For example he showed that p53 mutations need to occur before the action of NF1 or no tumors developed, the addition of PTEN leads to alterations in neural stem cell function prior to frank tumor development.

Craig Jordan discussed his accumulating evidence for molecular distinctions between malignant stem cells in leukemia and normal hematopoietic stem cells. Prominent among these differences is activation of NF-kB and PI-3K, but other features such as increased oxidative state, immunoproteasome and gammaH2Ax suggest a cell under stress. One of the issues raised was whether the cancer stem cell has acquired the capacity to tolerate stress or has accumulated changes that themselves induce a state of cellular stress. Intervening to exploit that state is one hypothetical means of developing cancer stem cell therapies that may not similarly be toxic for normal stem cells. Craig showed his results with several agents that have a differential effect on leukemic stem cells compared with normal HSC. One of these, parthenolide, is moving forward toward clinical testing.

The sequence of events and cell types that are involved in leukemic progression are poorly understood. This knowledge would aid in the more effective design of new therapeutics. The Forbeck Scholar **Catriona Jamieson** presented data showing the sequence of events in the evolution of human CML from chronic phase to blast crisis. She showed that the phenotype of the LSC changes during disease progression so that in the blast crisis phase the LSC shows properties consistent with their evolution from a committed progenitor population. She then examined the molecular alterations between the CML LSC and the normal committed progenitors which they resembled and showed the inappropriate activation of the WNT pathway. This result opens the way for a new avenue of therapeutics that target the LSC.

The most lethal component of cancer is when the tumor becomes metastatic, spreading from the primary site. A major question is whether every cell that migrates from a tumour has the potential to initiate metastatic growth. **Ann**



Chambers discussed a series of studies where she examined the proportion of cells that are able to initiate a micrometastasis and how many of these actually grow into a tumor at the distal site. She described a classical fluctuation analysis to calculate

the rate of metastatic cell generation in a well studied murine model. She showed that only 2% of primary cells generated a micrometastasis and only 1% of those actually generated a tumor. She showed that majority of the micrometastatic cells remained dormant. Even in a highly metastatic cell line, a large proportion of the cells are not metastatic. She argued that the generation of metastatic cells is very rapid and not likely due to mutational events, rather due to epigenetic changes within the population of cells. Her presentation sparked significant discussion as to whether many of the micrometastases are inherently able to form tumors but cannot due to an inhospitable microenvironment, or whether many micrometastatic cells are not CSC and only the CSC within a tumor are able to initiate metastatic growth. The group felt that much effort should be made to identify and purify the metastatic cells within tumors.



"I just wanted to thank you again for inviting me to your superb symposium. It was without doubt one of the best meetings I've ever attended. You'll be pleased to know that at least one exciting collaboration has been born thanks to the Forbeck symposium. Catriona Jamieson and I have a project planned to test a new drug for leukemia. Hopefully, this is an effort she'll be able to describe at future meetings of the scholars. In addition, I had a great talk with Ben Ebert about a possible collaborative project as well.

Aside from the clear advantage of getting these people together, there is another benefit you should know. Most of the attendees were laboratory scientists, who rarely (if ever) come in contact with the reality of treating cancer patients. For these scientists, spending time with people who have directly experienced the tragedy of cancer and have committed themselves to improving cancer care is an important type of inspiration. The scientists are all dedicated people, but it doesn't hurt to help them remember the human side of what they do. In the end, I think that may be every bit as important as the exchange of scientific ideas."

Craig T. Jordan

Foundation Junior Board



Jamie Forbeck

The Junior Board has had another amazing year. Fall Fest 2006 and the Scholar Retreat are improving every year. Members of the Junior Board worked hard throughout the three days of events, and did a wonderful job as always. Several new members were crucial to the success of the event. *Brian and Liz Fanning* created beautiful theme baskets for the silent auction. *Dick Payne* provided constant help in the weeks approaching the event as well as throughout.

It is a treat having the doctors return for multiple years. We have seen them grow and make major accomplishments in the few years we have been hosting these meetings. One of the most exciting things for me was hearing about all the scientific papers that were published and recognitions received by our scholars. Participants were excited for their fellow Forbeck Scholars and were eager to hear about everyone's progress during the past year.

The enthusiasm in the Scientific community and the participant's progress made me realize that the unique format of this meeting should be treasured. A scholar mentioned the effect of one interaction resulting from the Retreat, "I don't think I would have gotten to meet him outside this forum. He has had a great impact on my thinking already."

The Fall Fest event was a fun, casual event this year. Blue grass music, raffles and our wonderful supporters made for another successful event. We once again covered the cost of the meeting and are looking forward to the 2007 Retreat and Event. *Galen Eckland* and *Chandler Dimberg* are chairing the fundraising event. This year the event will have a new theme. We will be hosting the "Blue Jean Ball" on September 16th. Make sure you put it on your calendar and come out to support this amazing group of scientists and this forum.



Chandler Dimberg

It is important to the Junior Board and the Foundation that our supporters recognize the difference that their donations and attendance at Fall Fest make

to these meetings and cancer research. With out the amazing support and generosity of the people of Lake Geneva, Chicago and everyone involved, the Junior Board would not be able to host these meetings. The Scholars suggested this meeting could rival other forums or awards and is evolving into not only an important meeting but an honor to attend.

Many Junior Board members came to dinner with the doctors the Friday of the event. They saw and heard the appreciation for their hard work from the doctors and also that their contributions are important in making a difference in the progress of cancer research.

My thanks to all,

Jamie Forbeck Collins

MEMBERS OF THE JUNIOR BOARD

Brendan Cashman	Chicago, IL
Benjamin Collins	Southern Pines, NC
Jamie Collins	Southern Pines, NC
Renee Dahlstrom	Chicago, IL
Chandler Dimberg	Chicago, IL
Galen Eckland	Oregon, WI
Brian Fanning	Lake Geneva, WI
Liz Fanning	Lake Geneva, WI
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Glenn D. Pankau	Chicago, IL
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Chrissie Taylor	Chicago, IL
Ryan Vaile	Chicago, IL

HOW TO CONTACT THE JUNIOR BOARD:

Junior Board page on the web site

www.wgfrf.org

Email: jamie@wgfrf.org

Contact any Junior Board member

HOW YOU CAN HELP

Mark your calendars! September 15th
 Donate auction items
 Help find sponsors.

CONTRIBUTIONS TO JUNIOR BOARD

SPONSORS: Each year, our sponsors pledge a generous contribution, allowing the Junior Board to cover the up front expenses of the Fallfest fund raising event. We are grateful for the support from

William Blair & Company
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Many people help make the Junior Board fund raising project a success, through general contributions or through donation of event supplies and auction items. Many thanks to the following:

Charles and Beth Boehland
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"I would like to thank you for allowing me to be a part of your amazing event. I was so moved by the people who contributed all their talents to a cause that touches so many people everyday. As I sit here today I am waiting news of one of my best friends daughters who had brain cancer and is in her two year scan. After sitting through way to many C.T.'s M.R.I.'s along with so many other things that come with the territory, I know how long the wait is for my beloved friend and her family. The strength you and your family have to put sorrow into a Foundation that can bring joy to others is beyond words to me. I would love to help with your cause in any way I can."

Annemarie Konicek

2nd Scholar Retreat

September 14-17, 2006 - Lake Geneva, WI

For the second year running the Forbeck Foundation's Junior Board organized a superb meeting for the Forbeck Scholar awardees. The venue for the meeting was, once again, George Williams College, Lake Geneva, Wisconsin. *Jamie Forbeck* and members of the Junior Board worked tirelessly to ensure that the participants had everything they needed for a successful meeting. The efforts of the Junior Board were matched by the enthusiasm of both the senior scientists and the scholars attending the Forum.

The concept behind these meetings is to provide a basis for scientific collaboration and the exchange of ideas to further the field of cancer research. To attend the scholars meeting the candidates have to be selected initially to attend the prestigious Foundation's meeting in Hilton Head. The caliber of the scholars that apply to attend this meeting is beyond reproach and each year it becomes more difficult for the Foundation's Scientific Advisory Board to select four scholars out of the applicants.

The two day meeting is split into four sessions, each mentored by one of the senior scientists presenting on their area of expertise. The remainder of each session is devoted to talks from the scholars. Each scholar attending the main Forbeck meeting has the opportunity to come to four of the scholar meetings, which are held on an annual basis.

In organizing the scholar meeting in Lake Geneva the Foundation is building a base for the best up and coming scientists to get to know each other and form relationships and collaborations that likely would never occur without their attending the forum. As in many areas of science, cancer research has become highly specialized and researchers tend to remain focused in their particular areas of interest. Whilst this is natural, if one looks at progress in science, this often occurs through the application of ideas in one field that are applied to another. A further benefit for the attendees is that, throughout their tenure the scholars get to interact with today's senior scientists who have built considerable reputations in the field of cancer research. This is again a wonderful opportunity for the scholars to discuss their work in a form outside of their institution and to gain insights into new ideas to progress their studies.

The enthusiasm of the scholars to attend the meeting, present their work and interact with each other is simply

2006 ATTENDEES

SENIOR INVESTIGATORS

Gerard Evan, PhD	University of California	San Francisco, CA
Ed Harlow, PhD	Harvard Medical School	Boston, MA
John Kemshead, PhD	Baxter Cellular Therapies	Manchester, UK
Jean Wang, PhD	University of California	San Diego, CA

2002 SCHOLARS

Alison A. Bertuch, MD, PhD	Baylor College of Medicine	Houston, TX
Jan Karlseder, PhD	The Salk Institute	La Jolla, CA
Masashi Narita, MD, PhD	Cold Spring Harbor Lab	Cold Spring Harbor, NY

2003 SCHOLARS

James F Amatruda, MD, PhD	Univ Texas Southwestern Medical Ctr	Dallas, TX
Christopher Bakkenist, PhD	University of Pittsburgh	Pittsburgh, PA
Elsa Flores, PhD	Univ. Texas M.D. Anderson Cancer Ctr	Houston, TX
Norman Sharpless, MD	Univ. North Carolina	Chapel Hill, NC

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	University 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

wonderful to see. The scholars have requested that the Trustees of the Foundation extend their tenure for attending the program from three years to four and through the support of the Scientific Advisory Board this will take effect from 2007 onwards.

I cannot think of any activity that is more worthwhile for the Foundation, both in the short and long term. This type of investment in the scholars who will become leaders in the field of cancer research in the future is something that can only bring benefits in fighting a group of diseases that we all want to see eliminated.

As we move from 2006 into 2007, on behalf of the scholars, I would like to thank our Retreat chairperson *Ed Harlow* and the other 2006 mentors who willingly gave their time to attend the Forum and look forward to this years meeting that will be chaired by *Jean Wang* from the Dept of Medicine, Moores UCSD Cancer Center, La Jolla, California.

"This year's forum was even better than last year's forum from my perspective. We scholars knew each other and this promoted greater cross talk between us. I do think that relationships are being built between scholars that will last throughout their careers. The contacts with the mentors are also useful. Gerard and I have been in touch this week. I really like him and I don't

think I would have gotten to meet him outside this forum. He has had a great impact on my thinking already."

Chris Bakkenist



I can't thank the Jr. Board enough for allowing me to participate in the Scholar Retreat again this year. I must say, it was an exceptional event. The mentors were excellent; in fact, the ability to spend a few days with such great minds was truly an extraordinary opportunity. I left incredibly motivated. Also, I felt part of a special class of young investigators. It was great seeing everyone again and learning about their progress. Overall, I think that in just two short years the Jr. Board has met its goals with this retreat.

With sincere appreciation and best wishes to you and all the members of the William Guy Forbeck Research Foundation,

Alison Bertuch



"I would like to thank you for the wonderful Scholar Retreat at Lake Geneva. From my point of view, the meeting format is as good as it could be in terms of opportunities to learn, opportunities to interact with peers and role models, and opportunities to have fun. Again, big time thanks to your foundation and our retreat mentors for this terrific experience."

Ingo Mellinshoff

Update: 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.

In the 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 cancer study groups around the world.

Since that meeting, this group has reached a consensus on the INRG criteria, collated and analysed statistical data on 8,800 children with neuroblastoma, published a number of papers in the *Journal of Clinical Oncology*, made two keynote talks at the International Society of Pediatric Oncology (SIOP) in Geneva, Switzerland, and the INRG abstract has been selected for oral presentation at the American Society of Clinical Oncology (ASCO) meeting to be held in Chicago in June.

FOUNDATION

DVD/VIDEO AVAILABLE

A video presentation showing the history, purpose and activity of the Foundation is available by contacting the Foundation. Please specify DVD or VHS video format.

WEB SITE - www.wgfrf.org

New this year is the Foundation's video using streaming technology. The Foundation web site contains general information, a summary article on each of the Forums organized by the Foundation, information about "Focus on the Future" awards and grants, Junior Board activities, and several web site "pointers" for more information on cancer.

"FOCUS on the FUTURE"

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.

2006 SCHOLARS

The Foundation received a number of very qualified applications for the 2006 Forbeck Scholar Award. The Scientific Advisory Board selected four outstanding young scientists to attend the 2006 Forum and receive this award. The Foundation was pleased to present this year's Scholar Award to four candidates.



Benjamin L. Ebert, MD, PhD is a post-doctoral fellow in the lab of *Dr. Todd Golub* at the Broad Institute of Harvard and M.I.T. His research projects include dissecting the molecular basis of myelodys-

plastic syndromes by taking the first ever functional approach to identifying the gene on chromosome 5q responsible for the disorder. *Dr. Golub* says "I can confidently say that Ben is among the very best - perhaps the most talented person to pass through my lab. He has a voracious appetite for science, and is highly inquisitive and interactive." Ben received his PhD at Oxford University as a Rhodes scholar.



Catriona Jamieson, MD, PhD is recently recruited as an assistant professor at the University of California, San Diego, Department of Medicine. Previously, she was a post-doctoral fellow

in *Dr. Irv Weissman's* lab at Stanford University. *Catriona* discovered candidate cancer stem cells involved in the progression of chronic Myelogenous leukemia (CML) to acute leukemia, and the molecular events involved in enhancing CML cancer stem cell proliferation and self renewal. Her

research continues in this area, with an ultimate goal of designing and testing novel cancer stem cell diagnostic and therapeutic strategies. *Dr. Dennis Carlson*, says "Catriona brings an outstanding capability and intelligent passion to her research. For this reason, I have entrusted her to coordinate a Stem Cell Work Group comprised of researchers from the research-rich environment represented in La Jolla."

"Thank you so much for putting together such a thoughtful symposium. It was a very important forum and I was certainly honored to receive such a prestigious award that represents everything that I believe in. I look forward to future retreats." Catriona Jamieson



Carla F. Bender Kim, PhD has been working in *Dr. Tyler Jacks* group at MIT. Carla has identified, purified and begun to characterize a novel cell type in the adult lung (BASCs) with properties

of stem cells. Moreover, she has made a series of observations implicating this cell as the cell of origin in a model of non-small cell lung cancer. *Dr. Jacks* says Carla "is a very bright and highly inquisitive individual... and her research has been touted by others in the cancer biology and stem cell fields as a highly significant advance." She received her PhD from the University of Wisconsin.

"It was a joy to participate in the forum this weekend. It certainly was very helpful for me to interact with all the scientists, and I have some new ideas to test right away." Carla Kim



Joseph C. Wu, MD, PhD was nominated by *Dr. Sanjiv Sam Gambhir*, MD, PhD from Stanford University School of Medicine. Joseph is a clinical instructor in the department of Radiology and

Cardiovascular Medicine. *Dr. Gambhir* says Joseph's main research "interests are on stem cell therapy for cardiovascular applications. He has nicely combined molecular imaging techniques to this field and has demonstrated the utility of tracking stem cell survival, proliferation, migration, and misbehavior in living subjects."

"I had a great time, both intellectually and personally. I look forward to continuing to participate at the Forbeck Scholars program in upcoming years."

Joe Wu

FORUM PLANNING

2007 Forum: MicroRNA & Cancer

The 2007 Forbeck Foundation Forum will focus on the topic of microRNA. MicroRNA, as the name implies, are small RNA that have been identified in human cells. Despite their small sizes, microRNA have big roles in human biology. Recently, scientists have discovered that microRNA are involved in cancer, and that microRNA will be useful in the prediction, the diagnosis and the treatment of cancer. The



2007 Forum chairmen:
Dr. Carlo Croce & Dr. Greg Hannon

Forbeck Forum on MicroRNA and Cancer will be a catalyst for expanding this forefront of cancer research.

What are microRNA and why are they important?

To answer these questions, we need to recall the central dogma of biology established in the second half of 20th century based on the double-helical DNA structure and the studies of simple organisms such as bacteria. In this central dogma, DNA is the genetic repository. DNA is transcribed into three types of RNA- messenger (mRNA), transfer (tRNA) and ribosomal (rRNA). Together, these three types of RNA assemble proteins from amino acids and the proteins then carry out the biological functions. This central dogma has spun tremendous progress in biology, leading to the identification of mutated human genes that encode defective or dangerous proteins to cause various diseases including cancer.

After the human genome sequence was completed at the beginning of this century, biologists were surprised to find that only 2% of the genome coded for proteins.

Some of the genome sequences are used to build the infrastructure for gene expression and chromosome maintenance, but this infrastructure does not take up 98% of the genome. Most recently, biologists have realized that our genome encodes RNA other than mRNA, tRNA and rRNA. Among these new types of RNA are the microRNA. At the moment, computational methods have predicted that there are about 1000 microRNA genes in the human genome. The exact number will probably not be known for a while.

So far, biologists have found microRNA to function as an inhibitor of mRNA. MicroRNA bind to mRNA with two consequences, either causing mRNA to be degraded, or preventing mRNA from being translated into proteins. A microRNA can bind to several different mRNA. Conversely, more than one microRNA can target a single mRNA. In fact, two human microRNA have been found to bind an mRNA that codes for a protein, which cancer cells depend on for their survival in the body. Patients with defects in those two microRNA genes end up with too much of this protein and therefore are at a high-risk for cancer.

The Forbeck Forum will bring together leaders in the microRNA field. The organizers of this Forum will be *Dr. Carlo Croce*, who has discovered microRNA mutations in a large number of human cancers, and *Dr. Greg Hannon*, who has created microRNA to block genes that cause cancer. Because microRNA are small, scientists believe that they can be efficiently delivered into cancer cells in the body. Therefore, microRNA holds the promise for being a brand new class of medicine that can be used to treat cancers that are difficult to eradicate with the currently available therapies.

2008 Forum: Immunotherapy and Breaking Tolerance

The immune system in our bodies keeps us safe from our environment that is full of bacteria and viruses. When we catch a cold it is our immune system that fights the infection. When we cut ourselves our immune system ensures that foreign invaders do not take over our bodies.

For the last thirty years scientists and clinicians have tried to exploit the bodies immune system to treat patients with specific forms of cancer. Antibody therapy for diseases such as lymphoma and even breast cancer have been shown to be effective in many instances and these reagents are sold today for main line therapy. In academia efforts continue to exploit the cellular arm of the immune system and we are closer than ever to making an impact on specific cancers. Responses have been seen in patients but they remain unpredictable. Nevertheless the potential has been identified and the 2008 forum is intended to bring together a group of academics and clinicians to discuss manipulation of the immune system to assist in the treatment of cancer. This is the second time the Foundation has focused on this area of medicine indicating its importance in the development of new treatments for cancer and how quickly the field is moving. The formal title for the meeting is "Immunotherapy and breaking tolerance" and this will be chaired by Drs. Dr James Allison and Dr. Stan Riddell, both of whom are recognized as world leaders in this field.



2008 Forum chairmen:
Dr. James Allison & Dr. Stan Riddell

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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, 2006 thru mid-March, 2007)

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Scientific Advisory Board Report

Continued from Page 1

Forums have been held on anti-angiogenic agents, bacterial agents, viral oncolysis, targeting of cyclic-dependent kinases and tyrosine kinase receptors, antisense approaches and gene therapy. In addition, the Foundation has held meetings on the use of immunotherapy in cancer, which includes the use of cytokines, monoclonal antibodies, cancer vaccines and immunogene therapy. For example, Melanoma is a type of skin cancer where there has been a strong focus on immune therapies employing cancer vaccines monoclonal antibodies, dendritic cells, and gene therapy. In 2008 we will return to this topic as it is now clear that cancer treatments based on the manipulation of the immune system are turning out to be effective in the treatment of specific malignancies.

Antisense oligonucleotides are synthetic nucleic acids that have been in clinical trials for cancer for some time now. Small interfering molecules of ribonucleic acid (siRNAs) can be targeted to tumors and have been used with limited success. The 2007 Forum will focus on exploiting the discovery of the existence of hundreds of tiny RNAs, known as microRNAs (miRs), which regulate gene expression in animal and plant cells. This discovery has led to exciting new investigations into the role of miRs in hematology and hematological malignancies.

As indicated above, progress in cancer treatment is happening on all fronts. Whilst the Foundation focuses on the science underlying cancer development and treatment it must not be forgotten that, to some extent, we are masters of our own destiny. Life style choices such as smoking impact on our general health. The incidence of lung cancer in men is dropping. However, as women tended to take up smoking later than men the incidence of the disease continues to rise.

There is a role for the individual, the academic cancer centre, the pharmaceutical industry and in particular the Foundation in finding a cure for cancer. The Foundation is bringing together academics and clinicians to discuss developments in particular fields and creating an environment for interactions that would not normally exist. Out of these meetings we expect that new ideas will surface to shorten the cancer treatment cycle. Looking at the topics we have focused on in the last decade and matching these with current new approaches to cancer treatment we know we have been at the forefront of developments and the scientific advisory board will strive to keep the Foundation at the cutting edge. My thanks go to all who continue to support the Foundation's efforts.



John T. Kemshead
Chairman, Scientific Advisory Board

2006 FINANCIAL REPORT

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 Junior Board, the "Scholar Retreat." The Trustees continue to aim at a very high mark - that 90% of the total expense goes directly to support scientific programs. In 2006, 92.1% 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 2006, the Junior 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

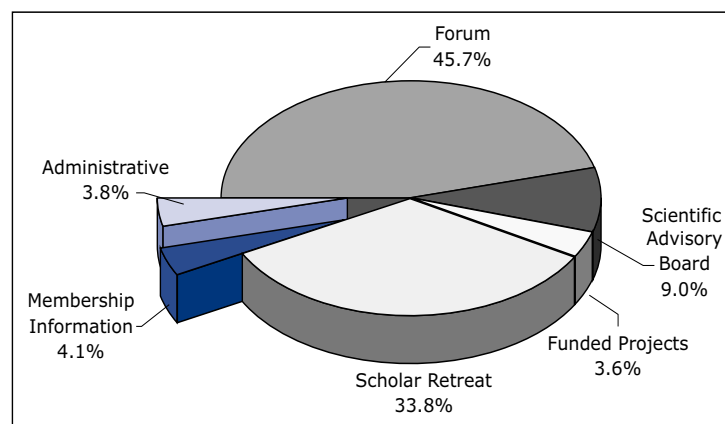
Historically, 85%-90% of the total expenses go directly to supporting the annual Forum and Foundation projects.

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

The Foundation has no paid employees, and the 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.

Projects funded during 2006 included four Scholar Awards and the second Scholar Retreat.



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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 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.