WILLIAM GUY FORBECK
RESEARCH FOUNDATION
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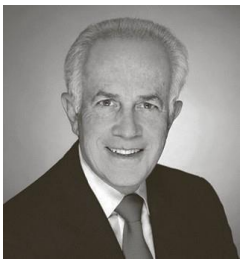
FROM THE CHAIRMAN'S DESK



Charles H. Jesser, CPA
President, Board of Directors

The William Guy Forbeck Research Foundation has come a long way in its thirty-two years. It started out with a vision to honor a young man who bravely fought a deadly cancer and comforted others in the same position as he. It began with the concept that "Doctors don't talk." Since its inception, the Foundation has been about getting doctors from all over the world with different backgrounds and skill sets to work together to figure out how to treat this disease.

One of the most significant achievements has been the recent funding and development of a database, the International Neuroblastoma Research Group Database (INRGdb). Doctors use the database to research and compare the symptoms and treatments in various stages of neuroblastoma and determine the best treatment path to follow.

I have been fortunate to witness this evolution from the beginning. Meeting the doctors, listening to the discussions, interacting with other families who want to duplicate our meetings for the benefit of a family member. To the best of my knowledge, no one has been able to successfully mirror what we have accomplished, especially our worldwide recognition in the medical research community.

With all that we have accomplished, we still have a long way to go. Our Foundation is financially sound but constantly cultivating additional funding opportunities so we can grow the high functioning initiatives that we support. We are always looking for skilled directors who can contribute their talents to our Board of Directors as well. With an Executive Director, Administrator and Fundraising Director, we are well on the way to fulfilling our short and longer term goals while keeping our focus on the fact that we need to grow financially and operation-wise in order to keep up with the ever expanding demands that accompany medical research.

Please feel free to reach out to me with any questions, suggestions or recommendations that you may have that will serve to help us to maintain our financial and operational strength in continuing to fulfill our mission.

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Multiply Your Donation!!!

$200k to WGFRF = $100k to University of Chicago

Time is running out.
We need your help!

The Forbeck Foundation received a challenge grant to raise its annual contribution level to $200k to help meet growing funding needs. If the Forbeck Foundation meets the goal, the anonymous challenge grant organization will donate $100k to the University of Chicago for the International Neuroblastoma Research Group database (INRGdb), an extremely important program to WGFRF and children with neuroblastoma around the world.

FORUM 2015: IMMUNOTHERAPY

William Guy Forbeck Research Foundation XXXIst Annual Forum
Hilton Head Island, South Carolina
November 12-15, 2015

CHAIRMEN

Catherine Bollard, MD
George Washington University
Washington, D.C.

Daniel Powell, PhD
University of Pennsylvania
Philadelphia, PA

The 2015 Forum was focused on Cancer Immunotherapy. Major topics of the forum included (i) cellular therapies using antigen-specific and gene-modified T cells for targeting leukemia and solid tumors; (ii) overcoming hurdles and barriers with regard to immunogenicity, immune escape, and the role of tumor microenvironment; (iii) vaccine strategies and antigen presentation; (iv) the role of immune cells in allogeneic transplantation; and (vi) current antibody/combination approaches for the treatment of pediatric malignancies. During the past decade, major advances have been made in improving the efficacy of these modalities and regulatory hurdles have been taken. Nevertheless, there is still a long way to go to fully exploit the potential of immunotherapeutic strategies to improve the cure of children and adolescents with malignancies. This forum supported new collaborations and insights for further translational and clinical immunotherapy studies.

Session I: TILs and Tracking T cells in vivo

Chris Klebanoff, MD, National Institute of Health. The first speaker Dr. Klebanoff discussed how there is excitement in the field with the adoptive transfer of gene-engineered T cells expressing chimeric antigen receptors (CARs) targeting CD19, that some of the oldest and most durable successes have been utilizing tumor infiltrating lymphocytes (TILs) for melanoma and how this field has dramatically improved our knowledge regarding the basic immunobiology of cancer and how this information will aid in the development of newer T-cell therapeutics for solid tumors. Dr. Klebanoff also noted the increased focus of the field towards more highly personalized immunotherapy approaches.

2015 Forbeck Forum Participants

Subject: Immunotherapy

Session I:
TILs and Tracking T cells in vivo

Chris Klebanoff, MD
National Cancer Institute
Patrick Hwu, MD
MD Anderson Cancer Center
Dirk Busch
University of Munich
Stanley Riddell, MD
Fred Hutchinson Cancer Center

Session II:
Overcoming Immune Evasion Strategies

Catherine Bollard, MD
George Washington University
Barnara Savoldo, MD, PhD
University of North Carolina
Chris Hourigan, MD
National Institute of Health

Session III:
Are all CAR T cell Therapies Created Equal?

Terry Fry, MD
National Cancer Institute
Steve Gottschalk, MD
Texas Children's Hospital
Daniel Powell, PhD
University of Pennsylvania
Michael Jensen, MD
Fred Hutchinson Cancer Center

Session IV:
Therapeutic Strategies to Combat Metastasis

Jennifer Wargo, MD
MD Anderson Cancer Center
Leslie Kean, MD, PhD
Seattle Children's Hospital
Mark Dudley, PhD
Novartis

Patrick Hwu, MD, MD Anderson Cancer Center. Dr.. Hwu then provided his broad experience using TILs for melanoma and how this therapy has been greatly enhanced using combination immunobiologics, most critically the checkpoint inhibitors. Given the "off the shelf" nature of such inhibitors, it should be possible one day for the vast majority of patients to benefit from immunotherapy. Dr. Hwu also discussed the use of biomarkers that could be used to identifity which patients are more likely to respond to TIL therapy and checkpoint inhibitors. Dr Hwu emphasized the fact that the systematic and methodical optimization of manufacturing the most effective T-cell products would be required for this approach to become scalable for the treatment of common cancers. Finally, Dr. Hwu stressed the need for a return to studies of vaccines for the treatment of cancer.

Dirk Busch, University of Munich. Dr. Busch provided more basic insights into the power of the T cell. His laboratory has demonstrated that a defined T cell subset (central memory T cells) is essential for robust and long-term responses after infusion. This finding explains why even very low numbers of infused T cells can be sufficient (in the extreme, a single T cell) to reconstitute protective immunity against cancer and/or viruses. His work is now identifying which T cell markers can be used to select for T cell use in therapy that have the greatest potential to eradicate disease. Dr. Busch provided reports from several ongoing clinical trials using purified T cell products for the treatment of infections after allogeneic stem cell transplantation or after genetic modification for the treatment of cancer.

Stan Riddell, MD, Fred Hutchinson Cancer Research Center. Dr. Riddell then provided new information demonstrating remarkable remissions in heavily treated patients with acute lymphoblastic leukemia even in the adult population. He showed that T-cell products should ideally be derived from the central memory T cell population and should have a fixed CD4:CD8 T cell ratio. He therefore provided convincing data that determining the characteristics that are desired in the starting T cell population, and analyzing the persistence, migration, metabolic fitness, function and fate of transferred T cells, is critically important when monitoring patients who have received T cell therapies so that we can learn what makes a T cell the most effective therapy for killing cancer cells.

Session II Overcoming Immune Evasion Strategies

Catherine Bollard, MD, The George Washington University. Under the selective pressure of a competent immune response, cancers and viruses are prone to become resistant to the immune system. One straightforward approach to avoid tumor or virus induced immune escape is administration of T-cells very early in the disease course, while targeting multiple virus- and tumor-associated antigens can reduce the risk of escape due to loss of antigen presentation. Dr Bollard, therefore, highlighted various clinical trials utilizing these approaches for hematologic malignancies and virus-associated diseases.

Barbara Savoldo, MD, PhD, University of North Carolina. Dr. Savoldo then highlighted several gene therapy approaches to overcome tumor immune evasion with a specific focus on Hodgkin's Lymphoma, CLL and neuroblastoma. Dr. Savoldo discussed that chimeric antigen receptor (CAR) T cells are sensitive to immune checkpoint inhibitors. Given this, genetic approaches might be applied to render T cells for therapy less sensitive to checkpoint inhibition.

Chris Hourigan, MD, National Institute of Health. Dr. Hourigan provided an overview based on his unique experience in Acute Myeloid Leukemia (AML) demonstrating that AML is oligoclonal even within an individual patient, with the clone predominant at first presentation not necessarily the same as the one responsible for post treatment disease relapse and death. He highlighted how AML may be the ideal disease to study tumor evasion strategies and that while there is no single antigen suitable for immune targeting in every AML patient, that every AML patient has at least one antigen that can be targeted with immune therapy. He rationalized that the best targeted immunotherapies for AML

should, therefore, target multiple antigens, not a single antigen.

Session III Are all CAR T cell Therapies Created Equal?

Terry Fry, MD, National Cancer Institute. Dr. Fry then expanded on the CAR T cell therapy initially discussed by Dr Riddell. Most critically he highlighted other targets being evaluated such as CD22 for pediatric ALL as well as the development of cytokine receptors with functional importance for the leukemic cell.

Steve Gottschalk, MD, Texas Children's Hospital. Dr. Gottschalk then expanded on the use of the CAR technology for solid tumors such as pediatric brain tumors and osteosarcomas. He highlighted the fact that the success of CARs in the setting of solid tumors has been hampered by a lack of unique tumor associated antigens, inefficient homing of T cells to tumor sites and the immunosuppressive microenvironment and discussed the strategies that have been and should be undertaken to improve outcomes for pediatric patients with solid tumors.

Daniel Powell, PhD, University of Pennsylvania. Dr. Powell then developed this field further identifying other strategies to improve CAR T cell therapies for solid tumors. He discussed the identification of new "safe" tissue-specific antigens that represent "druggable" and effective antigens expressed by the tumor, the tumor vasculature or immunosuppressive cellular elements in the tumor microenvironment. Dr. Powell discussed the building of multivalent T cells that address tumor antigen heterogeneity and antigen loss and the development of in vivo model systems that mimic these challenges in order to design and test new strategies for the creation of potent and safe CAR T cell therapy for common solid cancers.

Michael Jensen, MD, Seattle Children's Hospital. Dr. Jensen then discussed his laboratory's work which focuses on T-cell genetic modification for re-directing antigen specificity to tumors utilizing CARs as well as the evolution of multifunctional cytoplasmic signaling domains developed for these chimeric antigen receptors (CARs) that provide dual activation and costimulatory signaling. He also discussed the increasingly broad array of genetic manipulations including not only transgene insertion, but also targeted gene knock out using engineered targeted nucleases such as TALEN's and ZFN. He highlighted the fact that the next decade of advances in this arena will depend on iterative bench-to-bedside back-to-the-bench translational studies capable of sustaining the evolution of these technologies in the context of clinical parameters relevant to the pediatric oncology patient population.

Session IV Combination Strategies and Commercialization

Jennifer Wargo, MD, MD Anderson Cancer Center. In the final session, Dr. Wargo discussed how to better identify predictors of response to immune checkpoint blockade (and other forms of cancer immune therapy). She stressed that the longitudinal collection of tumor specimens from donors was critical to the discovery of predictors of response. Dr, Wargo pointed out that genomic and immune heterogeneity can influence tumor growth and response to therapy. She then effectively showed how insights gained through these investigations will inform studies in patients with earlier stage cancer. Finally, she discussed novel strategies to enhance responses to immune therapy such as through modulation of the gut microbiome.
Leslie Kean, MD, PhD, Seattle Children's Hospital. Dr. Kean then built on her wealth of expertise in the biology of hematopoietic stem cell transplantation (HCT) and graft-versus-host disease (GVHD). She addressed three critical questions in the field: (1) What are the mechanisms that drive breakthrough T cell allo-activation and tissue damage despite current immune suppression strategies?

"We see it as a huge honor to be able to attend such a meeting and certainly it is well-known that the ideas that are generated from this unique meeting are extremely important for advancing the treatment of our patients with cancer."
Catherine Bollard, MD, George Washington University

(2) Can we design treatment strategies to directly target these mechanisms? and (3) What are the necessary components of a GVHD-prevention strategy that will safely produce longterm immune tolerance? To answer these questions, and thus to uncover the mechanisms driving the pathogenesis of T cell activation and tissue invasion, Dr Kean's group has undertaken a major initiative to use a systems biology approach to define the T cell-centric GVHD transcriptome. The Non-Human Primate GVHD Transcriptome Project has allowed her to identify novel molecular pathways active in GVHD, many of which represent "druggable" targets for which candidate interventions are immediately available. Her results are providing a comprehensive map of the gene expression networks that control T cell activation, antigen recognition and tissue invasion. Importantly, the lessons from these GVHD-inducing T Cells 'Gone Wrong' can be used to harness T cell activation and survival pathways for the right reason: effective T cell immunotherapy for cancer eradication. These lessons might also aid in our understanding of the mechanism accounting for toxicity, such as cytokine release syndrome, that is sometime observed in patients receiving adoptive T cell therapy.

Mark Dudley, PhD, Novartis. In the final session Dr. Dudley provided a commercial perspective of how to move the novel therapeutics discussed during the course of the Forum from Phase I clinical trials to commercialization.

The transition from Phase 1 to commercial manufacturing usually requires substantial increase in the quality of reagents and equipment, establishment of reliable and harmonized release (potency) assays, introduction of efficiencies into the manufacturing workflow, and reduction of the cost of goods. Sponsors are incentivized to demonstrate manufacturing consistency, scalability and profitability, while maintaining product efficacy. Introducing process changes into an established cell product can be challenging because critical quality attributes or key process parameters may be altered or poorly characterized. Additionally, if potency assays are not predictive of clinical outcome and suitable animal models are not available for safety testing, then major process changes need to be vetted through clinical trials in patients. Therefore, he emphasized that the strategic use of small cohort "pilot" studies for discovery and development can enable efficient target discovery, process change and commercial product scale out.

For more information on Forbeck Forum and past Forum reports, please visit www.wgfrf.org

Summary

The Forbeck Forum offered a unique environment to openly discuss the latest advances as well as the challenges associated with the development of novel immunotherapies for pediatric cancer. Based upon the recent developments and the clinical data that was shared, it was surmised this is an era of profound hope for pediatric and adult patients with cancer. Since the last Forbeck Forum focused on Cancer Immunotherapy gathered in 2008, the field has witnessed 90% cancer remission rates following CAR T cell therapy in patients with childhood and adult acute lymphoblastic leukemia, the reproducibility of adoptive T cell therapy induced cancer regressions in melanoma, synovial sarcoma and lymphoma, and the FDA approval of immune checkpoint inhibitors as well as cancer vaccines. Even lung cancer has joined the cadre of immune responsive cancers. Still, not all patients respond to immune therapy and it was agreed by the group that there was much work remaining to be done if these dramatic cancer remissions are to be achieved more reproducibly and in other cancer types. Discussions were spirited and launched new collaborations aimed at answering some of the important questions in the field.

"The Forum is a place to discuss, to argue, debate and to move the fields forward in a particular area of cancer research. It's important because even though people know each other's literature they don't always get a chance to be in one room and to have provocative conversations and really think about how to move the fields forward, the important questions that need to be answered, and some of the real controversies in the field."
Kristina Cole, MD, PhD, Children's Hospital of Philadelphia

FORUM CONSENSUS AND CONCLUSIONS

Adoptive T cell therapy: T cell persistence in vivo following infusion is important for long term clinical response.

Strategies to improve T cell persistence include:

- Receptor engineering. But the lingering question now is – can we overdo it?
- Lymphodepletion is also critical to achieve T cell expansion and persistence in vivo but evidence now suggests that the specific agents used to achieve adequate lymphodepletion are important.
- It is key to comprehensively characterize and understand the cell product being transduced and/or infused to the patient.
- Combination therapy (e.g. with vaccines) may improve T-cell persistence. But when is the best time to vaccinate? Pre versus post T cell infusion or both?

Immune checkpoint inhibitors: Some patients with melanoma, renal cell cancer and lung cancer respond to checkpoint inhibitors – will this work in other cancers?

- Increased intratumoral T cells or high tumor mutation levels may be good predictive markers for responsiveness to therapy.
- Checkpoint inhibitors should be used in combination with other therapies.
- Can we better control or predict immune side effects from checkpoint inhibitors?
- Are there other (better) checkpoints?

Hematologic Malignancies

(i) Adoptive T cell therapy can work but tumor immune evasion is a major barrier to the success of immune based therapies.

- Antigen loss as seen with CD19-CAR T cell therapies underscore the importance of targeting multiple tumor antigens.
- Combination therapies such as combining T-cell therapies with checkpoint inhibitors and antibodies offers a unique multimodel targeted approach to overcome tumor immune evasion.
- Gene modification strategies can also be utilized to render adoptively transferred T cells resistant to the immune suppressive tumor microenvironment.

(ii) Another challenge for the field is when and how to monitor for response especially in the hematologic malignancy setting.

(iii) Finally, it is clear that more clinical trials are needed. But the question is, how early can we or should we treat especially when considering the vulnerable pediatric cancer population.

Solid Tumors

Lively debate was made as the collective group discussed the curative potential of cancer immunotherapy in patients with solid tumors. It was clear that the immune systems plays a role in the control of solid cancer and that for some patients, the patient's own immune cells could be stimulated to induce tumor regression, particularly with the use of checkpoint inhibitors and adoptive TIL therapy. However, unlike hematologic malignancies, solid tumors are difficult to permeate and have an immunosuppressive core.

Given these challenges, the following conclusions and questions were drawn:

Adoptive T cell therapy for solid tumor:

- Conditioning the hostile immunosuppressive tumor microenvironment will be necessary. Lymphodepletion may also be critical to provide "space" for transferred T cells.
- T cell access to solid tumor is limiting. The barrier of the tumor stroma will need to be addressed.
- Combination therapy will be key (e.g. with checkpoint inhibitors) and may improve adoptive T cell potency. Will it be safe? Will it be more effective?

TILs recognize neo-antigens, which may be the "tumor rejection" antigens. Strategies to specifically expand these TILs hold significant promise – but is it achievable?

- Are neo-antigens hope or hype?
- Efficiency in timing from neo-antigen discovery to clinical application may be a challenge.
- With advanced methodologies now available to enrich even rare tumor-reactive TILs to therapy, will TIL therapy work in other cancers - how many and which ones?
- Are neo-antigens necessary for the efficacy of TILs and checkpoint inhibitors?
- CAR technology: is it a "one trick pony"?

Will it only work in hematologic cancer? If it is to work in solid tumors, we must consider the following:

- Find exceptional antigens. CAR antigens must be expressed uniquely on cancer cells and not on cells of normal organs; at least not on "essential" organs - can we do it?
- Solid tumor antigens have heterogeneous tumor antigen expression. Multiple antigen targeting may be necessary to achieve maximum response.

Longitudinal tumor tissue collection is an issue. Study of the immunobiology of resected/collected tissues is key to the identification of tumor immune evasion mechanisms and the development of therapies to thwart these barriers to the successful immunotherapy. With available tissue we can do the following:

- Better identify predictors of response to immune checkpoint blockade.
- Learn the intrinsic and extrinsic mechanisms of tumors to evade immune recognition.
- Eludicate the immune properties of rare tumor reactive T cells in human cancer and develop approaches to bolster their activity.
- Mine tumor cells for new target antigens.

During the past decade, major advances have been made in improving the overall efficacy of cancer immunotherapy modalities. The FDA has approved multiple immune checkpoint inhibitors and cancer vaccines, and CART cell therapy is expected to be approved within the next year based in part on the 90% response rates seen in pediatric patients with ALL. Still, there is much work remaining to be performed in order to fully exploit the potential of immunotherapeutic strategies to improve the cure of children and adolescents with cancer. This year's forum was successful in bringing together talented physicians and scientists to discuss the challenges of creating more effective immunotherapies for childhood cancer and supported new collaborations and insights for further translational and clinical immunotherapy studies.

Past Forbeck Forum Immunotherapy Meetings

Immunotherapy and Breaking Tolerance - 2008
Chaired by: James Allison, PhD, Memorial Sloan Kettering Cancer Center and Stanley Riddell, MD, Fred Hutchinson Cancer Center

Allogeneic Stem Cell Transplatation - 2000
Chaired by: Frederick Appelbaum, MD, Fred Hutchinson Cancer Center

Gene Therapy and Tumor Vaccines - 1992
Chaired by: Drew Pardol, MD, PhD, Johns Hopkins University

For more information on Forbeck Forums, please visit www.wgfrf.org.

SCIENTIFIC ADVISORY BOARD

John T. Kemshead, PhD
Chairman
Scientific Advisory Board

2015 represented a series of "firsts" for the Foundation. This year was the first time we have awarded funding to allow our scholars to collaborate on scientific projects. This program is open to all current and past scholars. Funding is awarded upon a competitive basis, and in 2015 the Foundation awarded two such projects to our past scholars. This represents a significant financial commitment for the Foundation as both clinical and basic research are expensive to fund.

These funding projects are initiated by Forbeck Scholars, but collaborators may be outside the Scholar Program. This condition helps the Foundation with quality control and created a reasonable pool of applicants. As it was, the SAB had to review eleven applications and a considerable debate ensued as to pick the best two. The two projects will run for two years with a possible extension for a further year if the work is going exceptionally well. Each project will be reviewed after one year to ensure that the scientists are sticking to their documented goals. This is not a fundamental change to the focus of the Foundation, as we still wish to fund the "think tank" type meetings that have proven so valuable in the past. The collaborative research program is meant to add to the repertoire of our activities rather than change the primary focus.

Another first for 2015 was the number of scholars that applied for our program enabling them to attend the Foundation's annual meeting in Hilton Head, SC. This year we had over 100 applicants which made the job of the SAB particularly hard as the quality of the applicants is very high. This does raise the question as to if and how we could increase participation of scholars in the meeting, but at the present time all feel that to do this puts the purpose of the meeting at risk.

In 2015, Jean Wang, also retired from the Scientific Advisory Board. Jean's support and hard work with the Foundation is greatly appreciated and she will be missed. However, this has allowed us to invite new members to the SAB. The Foundation has a well-balanced SAB that covers the majority of the disciplines needed as well as reflecting both basic science and clinical expertise.

The commitment to pediatric oncology remains. The Foundation directly supports the International Neuroblastoma Study Group database, where there are over 10,000 patients listed from all over the world. This makes the study of a rare disease so much easier, and I cannot stress the importance of this work for collating information and ultimately making good judgments on the clinical care of these children.

A final first for 2015 was the Board of Directors having to curb the enthusiasm of the SAB to extend our research funding program. The Board, while keen to do what they can to further the mission of the Foundation, had to say "no" at this time to show appropriate financial prudency. This in itself has brought to the fore the need for enhancing our fundraising activities, so that we can grow and continue to go from strength to strength.

Collaborative Research Program

The Forbeck Foundation is now accepting applications for the Collaborative Research Project funding program!

Funding is only available to past and current Forbeck Scholars who collaborate on research. Funding of research projects will be based upon collaborations between two laboratories/fellows/scholars/MDs/ PhDs in their early years of scientific development. At least one of the collaborators must be either a past or current Forbeck Scholar.

The funding will be for two years, with the option for one additional year subject to review by the Scientific Advisory Board. Preference will be given to Scholars who are developing their own laboratories and need funding to enable them to establish themselves as independent investigators capable of receiving major funding from alternative sources.

Applications due in April of each year!

SCIENTIFIC ADVISORY BOARD MEMBERS

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New Scientific Advisory Board Members

Kristina Cole, MD, PhD is a former Forbeck Scholar, Kristina has remained involved with the Foundation, collaborated on research with other Forbeck Scholars and is chairing the 2016 Scholar Retreat.

James Amatruda, MD, PhD is a former Forbeck Scholar. Jim has remained involved with the Foundation, collaborated on research with other Forbeck Scholars and attended the 2016 Focus Meeting.

FORUM 2016: Chromosomal Instability/Aneuploidy

Large-scale sequencing efforts have provided unprecedented insight into the genomic changes that occur during tumorigenesis. We now understand that structural and numerical chromosomal aberrations are an almost universal feature of cancer and that tumors are karyotypically heterogeneous, constantly evolving ecosystems. The challenges we are faced with now is to explain what drives this genetic plasticity and to find ways to exploit this hallmark of cancer for therapeutic intervention. The development of new single cell analysis tools and the development of ever more sophisticated cell and animal models of genome instability have opened up new ways to tackle these longstanding questions.

The Forbeck Foundation meeting will bring together the leading researchers studying cancer genomes and their evolution with scientists who seek to understand the mechanisms underlying genome instability. By bringing researchers together from these diverse fields, we are hoping to develop hypotheses and approaches to describe in molecular detail how genome instability mechanisms shape the cancer genome and how the condition fuels tumor evolution.

The goal is to bring together leaders from cancer genome and chromosome instability fields to discuss the state of their respective fields and ways forward to translate this information to the clinic.

Angelika Amon, PhD
Koch Institute for Integrative Cancer Research at MIT

David Pellman, MD
Dana-Farber Cancer Institute at Harvard Medical School

FORUM 2017: MYC/RAS

Karen Cichowski, PhD
Dana-Farber Cancer Institute

Gerard Evan, PhD
University of Cambridge

Myc and Ras are not only two of the most commonly deregulated genes in human cancer but also downstream effectors for a host of other oncogenic mutations in many different tissue types. Myc and Ras also potently cooperate with each other in tumor development, a synergy that seems to imply that each are necessary yet qualitatively distinct. Nonetheless, despite intensive study of these pivotal oncogenes for more than 25 years, we still have no clear understanding of how these oncogenes work, how they cooperate, or how to convert our insights into cancer therapies.

This Forbeck Foundation meeting will bring together thought leaders from both the Myc and Ras "fields" and beyond to address these critical questions and think outside the conventional biopharmacological box. Can we directly target Myc or Ras pharmacologically?

If so, how? Or should we instead target their more pharmacologically tractable downstream effectors? If so, what are they? And while Myc and Ras drive cancer, can we conversely expect that their inhibition will kill cancer cells? Alternatively, does aberrant Myc and/or Ras activity expose novel vulnerabilities that might be exploited to specifically target cancer, but not normal, cells? How and why might these vulnerabilities arise, since both Myc and Ras participate in the growth and survival of all normal cells? Finally, knowing that most targeted cancer drugs "fail" because cancers evolve around them, why should Myc and Ras be any better as targets than many, more druggable, molecules?

The goal will be not only to discuss the current state of each field but also how to apply that knowledge to the development of effective and broadly applicable cancer therapies.

FORBECK FOUNDATION SCHOLAR RETREAT

Lake Geneva, Wisconsin
October 1-4, 2015

Kimryn Rathmell, MD, PhD
Vanderbilt University
Nashville, TN

October 1, 2015 brought another phenomenal group of energetic and accomplished young investigators together at the Geneva National Resort for the 2015 Forbeck Scholar Retreat.

It was my privilege to chair this year's retreat, an annual pilgrimage that marked the first 5 years of my own faculty career path, and which I have to credit with shaping my career as a physician scientist. I was incredibly honored to chair this meeting.

The Forbeck Foundation has integrated several key experiences into this exciting event. Together they have the effect of profoundly shaping the lives and careers of young scientists at this critical juncture as they develop an independent research program which will sustain their careers for the next 30+ years. These elements are: 1) a structured venue for discussing new research in a way that allows for extended open discussion, 2) an atmosphere that supports candor, with a relaxed environment so that scholars and mentors can achieve an intense scientific discussion that is both challenging and immensely fun, and 3) a sequestered environment in which scholars and mentors are given the time to get to know each other. The setting allows all to be comfortable with being open and critical and to establish relationships that allow for more than superficial conversations about career and scientific issues.

The 2015 Scholars came from a fairly broad range of topic areas in cancer biology: Drug Resistance Mechanisms, Tumor Metabolism, Epigenetics, and Invasion/ Metastasis. Mentors included former Forbeck Forum attendees Dr. Chi Dang, University of Pennsylvania and Dr. Jeff Rathmell, Vanderbilt University, as well as Dr. Cheryl Walker, Texas A&M, and Dr. Nav Chandel, Northwestern University. We all learned new details and ways to think about how these processes affect cancer.

The event was kicked off by an inspiring keynote by Dr. Chi Dang, Director of Abramson Cancer Center at the University of Pennsylvania. Dr. Dang spoke to the scholars with a candid and poignant style about the privilege of this career, as well as the responsibilities associated with it, from his perspective as a physician-scientist, chemist, cancer biologist, and leader in the academic medicine community. His talk covered a broad range of issues that are encountered, or will be encountered, by the scholars in public perceptions of science, value-based decision-making, publication biases and credit issued for publication, issues involved in large team projects. Importantly, he reminded us all that facts have half- lives, and our search for "truth" is a never-ending quest.

The opening session on **cancer epigenetics** set the stage with energized talks by Grant Challen on epigenetic modifier mutations that shed light on emerging resistance and aging; Alvaro Rado-Iglesias, who shared a novel finding from a non-cancer syndrome Bronchio-oculofacial syndrome (BOFS) illustrated the way positional enhancer regions in the genome can affect gene expression; Chris Vakoc had a first slide that led to a heated discussion of what constitutes an oncogene. Having resolved that issue, his discussion of screening strategies to determine the key functional domains of epigenetic regulators led us to consider new ways to nominate the active portions of these enigmatic proteins; Julie Losman described the first Forbeck funded collaborative grant between herself and scholar Cory Johannessen to use an unbiased saturation mutagenesis technique to anticipate mutations in genes that will be likely to produce resistance. I finished the morning session with one more epigenetics talk discussing our newest foray into how the disruption of histone methylation may contribute to tumorigenesis.

The afternoon session turned to **metabolism**. Led by Katy Wellen, this talk transitioned us from epigenetics with links from metabolism to histone regulation and DNA repair. Cory Johannessen helped us sort out the signal from the weeds with saturation mutagenesis strategies to reveal key protein domains that would be likely to be most sensitive for drug targeting. Kris Sarosiek explored the issue of "priming" and how the potential for proliferation was a key feature in the sensitivity of cells to signals inducing cell death. Mentor, Dr. Navdeep Chandel of Northwestern University championed the mitochondria as the center of the known universe. With these ideas all bouncing around our heads, we retired to the bar and dinner where we knew already the weekend would be a success based on the chatter amongst the scholars and the mentors about the new ideas that had been explored in the conference.

Day two brought a group energized to push the envelope even further. We started off with an **ACC Challenge:** Kris Wood (Duke) examined ways of rapidly screening for potential therapeutic options for melanoma, and Chad Pecot (UNC) showed us that there are many ways to imagine [and track] how a cancer cell makes it's way from the primary tumor to sites of metastatic growth. Mentor Cheryl Walker challenged my histone-centric view of the world with ideas that epigenetic modifiers might have more to do than their day job. The afternoon brought Mario Shields sharing his fascinating ways of "seeing" as well as manipulating the environment of the cancer cell. Mentor, Jeff Rathmell reinforced our impression that metabolism is really complicated. Louise van der Weyden showed us that accidents of nature can in fact be used to reveal novel facets of cancer; and finally Mentor Chi Dang brought us full circle with exciting new things that Myc continues to reveal about the underpinnings of cancer.

Our final afternoon of scientific sessions continued around a cozy fireplace with an informal (and spirited) discussion starting on the topic of navigating team science, but also including lab management, grantsmanship, and topic prioritization. From there we proceeded to the Blue Jean Ball, and enjoyed the wine tasting. This lovely event was a nice chance for us all to finalize those connections, meet the generous supporters of this important organization, and give us all a chance to remember again why we do this—because individuals and families continue to be profoundly affected by this disease.

FOCUS ON THE FUTURE: SCHOLAR AWARDEES

Esra Akbay, PhD

Dana-Farber Cancer Institute

Sponsored by:
David & Dorcas Collins

Dr. Akbay earned a Bachelor of Science degree in Molecular Biology and Genetics from Bilkent University in 2005. She then entered the Cancer Biology program at the University of Texas in Dallas. Her studies focused on understanding the development of Type I and Type II Endometrial Cancers to develop new treatment strategies. After receiving her PhD degree in 2010, Dr Akbay began her postdoctoral fellowship focusing on generating clinically relevant genetically modified mouse models of lung cancer at the Dana-Farber Cancer Institute. She works on developing strategies to overcome resistance mechanisms to targeted agents and immunotherapies and developing novel treatment strategies that utilize host defense mechanisms in the treatment of lung cancer.

Annette Kunkele, MD

University Hospital Berlin

Dr. Kunkele did her thesis at the German Cancer Research Center in Heidelberg in the field of cancer immunology. In 2011 she received a grant from the German Government that enabled her to join a research lab abroad. Since her German lab focuses on neuroblastoma, she joined Dr. Jensen's lab at the Ben Towne Center for Childhood Cancer Research where she could combine research in the field of neuroblastoma and in the field of immunotherapy. The main focus of her work was on optimizing T cell therapy for neuroblastoma. Within a trial at Dr. Jensen's lab, we now treat children with neuroblastoma using the CAR-T cell product she helped to develop. At the end of 2014 she returned to Germany with the goal to initiate a clinical CAR-T cell trial for children with neuroblastoma.

Shannon Maude, MD, PhD

Children's Hospital of Philadelphia

Dr. Maude is an Attending Physician in the Division of Oncology at The Children's Hospital of Philadelphia and an Assistant Professor of Pediatrics at the University of Pennsylvania Perelman School of Medicine. Dr. Maude received her MD and PhD degrees from the University of Pennsylvania School of Medicine as part of the Medical Scientist Training Program. She subsequently completed her Residency in Pediatrics and Fellowship in Pediatric Hematology/Oncology at The Children's Hospital of Philadelphia. Dr. Maude has a special interest in novel therapies for acute lymphoblastic leukemia, particularly targeted therapy approaches and engineered T cell therapy. Dr. Maude focuses on mechanisms of relapse and novel approaches to overcome resistance to CAR-modified T cell therapy.

Stefani Spranger, PhD

University of Chicago

Dr. Spranger's pre-doctoral research focused on fundamental human immunology with an orientation towards immunotherapy for acute myeloid leukemia. Her training was supervised by Professor Dr. Dolores Schendel at the Helmholtz Center Munich. To further train in the field of tumor immunology she joined the laboratory of Dr. Thomas F. Gajewski at the University of Chicago. Since joining the laboratory, she has enhanced her technical and academic skills related to tumor immunotherapy and the use of genetically engineered mouse models for cancer development. She has made the novel discovery that has recently been published in Nature.

Scholar Award

Forbeck Scholar Award recipients are invited to participate in the Foundation's Scholar Retreats for the subsequent four years. This gives them further opportunities to interact with their peers and meet with different groups of international cancer experts. Existing awardees have found these experiences invaluable in developing collaborations as well as opening up a variety of career opportunities. Joining this group also offers awardees the opportunity to apply for other programs sponsored by the Foundation that are only open to Forbeck Scholars. During this four-year period, the Foundation will pay all expenses to attend the meetings and an honorarium to individuals at the end of their tenure periods.

Important Scholar Dates

Scholar Award
Applications due April 15, 2016

Collaborative Research Program
Appliations due April 15, 2016

Upcoming Retreats
October 13-16, 2016
October 12-15, 2017
October 11-14, 2018
October 10-13, 2019
October 8-11, 2020

Focus Meetings

Focus meetings give Scholar Awardees the opportunity to host their own think-tank with a topic and participants of their choosing. These meetings are a vehicle to further the research and professional development of Forbeck Scholars. Scholars have used these meetings as a way to delve deeper into a topic they want to learn more about or to develop a paper. The Foundation will assist with logisitics, but otherwise it is the Scholar's meeting.

For information on sponsoring a scholar, please contact admin@wgfrf.org.

Focus Meetings

2016: Renal Medullary Carcinoma

April 21-23, 2016 - Nashville, TN

Renal medullary carcinoma (RMC) is a rare but aggressive and highly understudied cancer that specifically affects individuals carrying one or more of the sickle cell genes. Moreover, the disease strikes at a peculiar age distribution window—typically between the ages of 10 and 30. Thus, this disease is disproportionately represented in the African-American population, and because the age of onset straddles typical pediatric and adult oncology boundaries, the experience with this rare cancer is further diluted.

In addition to the rarity of this cancer contributing to a wide spectrum of diagnostic and therapeutic interpretations, this cancer remains one of the most lethal. Currently, only 10% survive past 2 years and fewer than 5% survive 3 years. To date, none has survived past 5 years including those who presented with stages I/II disease. These contemporary statistics cast doubt on benefit of screening of sickle cell trait carriers (e.g., using U/S of the kidneys) since the presumed window between detectability and lethality is presently too narrow.

Kimryn Rathmell, MD, PhD
Vanderbilt University
Nashville, TN

Nizar Tannir, MD
MD Anderson Cancer Center
Houston, TX

2017: Precision Cancer Medicine by Functional Biomarkers

May 18-20, 2017 - Boston, MA

Anthony Letai, MD, PhD
Dana-Farber Cancer Institute
Boston, MA

With the ever-growing number of targeted cancer therapies comes the growing need for predictive biomarkers to assign these therapies to the patients who will most benefit from them. This is the task of precision medicine. While the precision medicine in cancer is often equated with cancer genomics, there are important and increasingly appreciated limits on how well genomic information can serve as a precision medicine tool in cancer. An emerging alternative strategy that is very important to my laboratory and others is to put drugs of interest into contact with the patients' actual tumor cells and measure the effect. Exactly what is measured and how it is measured differs depending on the approach.

There are two main anticipated outcomes at the meeting. First, this is be the first such meeting ever of investigators explicitly focused on a functional predictive biomarker approach in cancer. As such, it will provide a unique opportunity to compare challenges and opportunities different investigators have confronted in the effort to provide a functional precision medicine tool. Second, Dr. Letai anticipates using this meeting to generate an outline for constructing a MATCH-like trial in cancer based on functional biomarkers.

Collaborative Research Program

Immune Cell Genomic and Metabolic Profiling in Renal Medullary Carcinoma

Kimryn Rathmell, MD, PhD
Vanderbilt University
Nashville, TN

Benjamin Vincent, MD
University of North Carolina
Chapel Hill, NC

Our Forbeck funding involves a collaboration between Dr. Ben Vincent, MD, University of North Carolina at Chapel Hill, and the laboratories of Dr. Kim Rathmell, MD, PhD and Dr. Jeff Rathmell, PhD at Vanderbilt University. We are working together to better understand the immune microenvironment in clear cell renal cell carcinoma. Working on the gene expression front, Dr. Vincent has observed a unique pattern of immune repertoire gene expression in renal tumors and has recently completed the expression analysis in pre- and post-treatment specimens from a clinical trial led by Dr. K Rathmell. His work points to a robust infiltrate of T cells in these tumors, and future work will examine the restriction pattern of these T cells for specific neoantigens.

The T cells themselves have been examined from freshly isolated tumor specimens in the Rathmell laboratories, where functional studies are revealing new insights into the metabolic features that govern the ability of these cells to activate in response to stimulation. Our work to date has demonstrated a clear distinction between resident T cells in the tumors and T cells that are detected elsewhere—both by gene expression and immune and metabolic studies. The objective of this work is to understand the immune cell composition of clear cell renal cell carcinoma, with the goal to modify the tumor microenvironment such that a larger proportion of patients can experience a favorable response to immune modulatory therapy.

Comprehensive Structure-Function Analysis of Mutant IDH

Cory Johannessen, PhD
Broad Institute
Boston, MA

Julie-Aurore Losman, MD, PhD
Dana-Farber Cancer Institute
Boston, MA

For over 100 years it has been known that cancer cells have profoundly altered metabolic states, and it has long been hypothesized that this altered metabolism plays a central role in tumorigenesis. However, it is only recently that metabolic enzymes have been directly causally linked to oncogenic transformation. The discovery of mutations in the Isocitrate Dehydrogenase (IDH) family of metabolic enzymes in cancers as varied as glioma, chondrosarcoma, cholangiocarcinoma and Acute Myeloid Leukemia (AML), has refocused attention on the idea that oncogenes can promote cancer by metabolically rewiring cells.

With the support of the Forbeck Foundation, we are using a novel mutagenesis technique, MITE (Mutagenesis by Integrated Tiles) to comprehensively, functionally map mutant IDH1 and mutant IDH2. Our goal is to identify the domains in mutant IDH1 and mutant IDH2 that regulate transforming activity, enzymatic activity, protein stability and subcellular localization. We expect that, by fully characterizing the structure and function of mutant IDH, we will improve our understanding of how mutant IDH mediates transformation and how mutant IDH can be more effectively targeted therapeutically.

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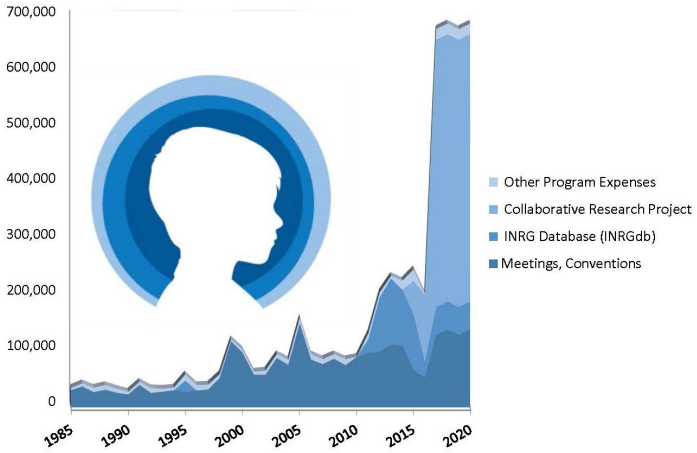
Program Growth

Matthew M. Gerdes
Director/Treasurer

Since the inception of the William Guy Forbeck Research Foundation (WGFRF) in 1985, there has been a committed effort to implement its mission to promote advances in cancer research through collaboration. The mission has been executed by hosting a multitude of collaborative meetings with some of the leading cancer researchers throughout the world.

In the most recent decade WGFRF has increased program funding. One new program provides funding to the International Neuroblastoma Risk Group (INRG) to develop a database, maintaining patient data for neuroblastoma to clinicians and researchers around the world. The second new program began in 2015, the Collaborative Research Program. This program gives Forbeck Scholars the opportunity for funding of projects based on collaborative research.

WGFRF aspires to magnify its footprint in cancer research by increasing its collaborative funding. This is being done with the purpose to fulfill the vision to see cures for all cancers.



We thank you for your continued support of our mission and assisting us with our vision.

FROM THE PARTICIPANTS

"There is essentially no overlap in what we are doing and yet there is no way either of us could do this project without the other. So it's the perfect collaboration."
Julie-Aurore Losman, MD, PhD on the Collaborative Research Program

"I would say the scholar meeting was the biggest thing that has affected my career."
W. Kimryn Rathmell, MD, PhD on being a 2004 Forbeck Scholar

They have really provided an opportunity to catalyze some of the research that we are doing that in the absence of the Forbeck community we wouldn't have been able to do."
Cory Johannessen, PhD on the Collaborative Research Program

"I have never actually had an experience like that before, where you are stuck in a room with some of the smartest people in the field, and you just talk about science."
Julie-Aurore Losman, MD, PhD on the Scholar Retreat

"The Forbeck Forum really draws upon and attracts the highest quality researchers in their fields in scientific cancer research programs."
Michael Jensen, MD on Forbeck meetings

"Rather than these meetings with 500 people. Sometimes you benefit more from being with 20 in how well you can interact."
Alvaro-Rada Iglesias, PhD on Forbeck meetings

IN APPRECIATION

From the beginning, Billy's family has been very greatful for all the people who have worked to make the activities of the Foundation a success. They are greatful 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. Special appreciation goes to the Foundation Directors, the Scientific Advisory Board and volunteers whose thoughtfulness, time and energy have done so much for the success of the Foundation and the Forums. Most importantly, many thanks go to the hundreds of donors, individuals businesses and foundations, whose financial support assures the continued work in Cancer research.

Sincere Thanks,
The Forbeck Family

WGFRF.org
William Guy Forbeck Research Foundation

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Hilton Head Island, South Carolina 29926

Save the Dates

WGFRF GOLF OUTING

GOLF OUTING
FRIDAY, MAY 20th, 2016

11:00 am Shotgun Start
Lakewood Golf Club
Lake Geneva, Wisconsin
Register under About Us/Golf Outing at wgfrf.org

Blue Jean Ball

BLUE JEAN BALL
SATURDAY, OCTOBER 15th, 2016

6:00–10:00 pm
Big Foot Country Club
Lake Geneva, Wisconsin
Purchase tickets under About Us/Blue Jean Ball at wgfrf.org

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