William Guy Forbeck Research Foundation

Cancer's Leading Thinkers. Together in one room.

Contents

Legacy of a Life

"A life may exist for just a short time. And yet can touch and affect many other lives. William Guy Forbeck lived this kind of life. One short life has led to many positive results."

Billy's life may have only been for 11 years but it has been making an impact on cancer research for 35 years. It is not always easy to quantify the impact of the Forbeck Foundation but one clear sign of its' impact and relevance can be seen by the demand from the scientific community for Forbeck Forums. The explosion of information in cancer research has amplified the need for these meetings. It is an exciting time for new therapies, less toxic treatments, new discovering of genetic markers every day. But the common theme at every meeting is the more we learn the more we realize we don't know.

During Forbeck Forums, brilliant researchers from around the world come together to learn, debate and collaborate. It is amazing to hear them say I have that piece that you need for your research. Or we did that experiment and it didn't work. Progress isn't just made in the lab. Over the years an intricate web of new relationships has been created and reach all across the globe making progress for all those afflicted by cancer.

Throughout the course of the Foundation's 35-year history, there have been:

- 55 Forums
- 14 Scholar Retreats
- 4 Intl. Neuroblastoma meetings with 6 publications from INSS/INRG
- Funding for the creation of the Interactive International Research Group database (iINRGdb).
- Global participation by over 700 scientists and clinicians from 22 countries in these meetings
- 2 Collaborative Research Projects
- Sponsor Advances in Neuroblastoma Research (ANR) seminal bi-annual Neuroblastoma meeting
- Countless papers, collaborations, and clinical trials

We hope that anyone who has been a part of this incredible journey for any of the past 35 years will join us in celebrating the Legacy of Billy Guy this October in beautiful Lake Geneva.

For more information, please see the enclosed save-the-date card or visit our website.

2019 Forum Reviews

3D Chromosomal Architecture and Nuclear Topology

Ari Melnick, MD, Cornell Medical College
Jane Skok, PhD, New York University

The symposium brought together basic mechanism of nuclear architecture from the computational and mechanistic perspective with studies of cancer genetics and cancer biology. The rationale for this symposium was related to the fact that we are starting to understand that the ability of cells to control the three dimensional architecture of their genomes plays a fundamental and essential role in coordinating and controlling gene expression and thus creating all the various cellular phenotypes in the human body. Along these lines, there are nascent data showing that mutations affecting either the proteins that mediate genomic architecture, or the non-coding genomic elements to which they bind are extremely common in human cancers. Moreover, emerging data show that the types of mutations that occur in cancer may be determined by the position of genes within cell nuclei. Hence it was critical to bring together experts in nuclear architecture with cancer biologists and physicians to see how this information can be merged together to understand and eventually target this fundamental property of cancer cells.

One aspect that came into focus is the lack of consensus and clear data to indicate the precise nature of architectural features. Part of this is due to the complexity of translating abstract representations into physical features such as loops, etc. There is also semantic confusion and variable interpretation due to different analysis algorithms and interpretation. The approaches for studying nuclear topology are highly complicated and involve very advanced technologies and expensive equipment and sequencing.

Some of the immediate outcomes of this meeting are:

- An expanded directive to sequence non-coding DNA elements in cancer patients, which will be actively pursued by several of the participants who perform cancer profiling studies.
- The application of novel technologies to visualize genomic architecture in cancer patients to primary patient specimens for the better identification of cancer causing mutations.
- The integration of novel and unpublished findings on architectural protein mechanism of action with cancer models – providing new insight into how tumors work.
- A major novel concept that arose through discussion was that of identifying therapeutic agents that could reverse aberrant nuclear topology in cancer – by bringing together technologies and discoveries from different laboratories. Several participants announced plans to use these technologies to screen for such therapies.
- The co-organizers of this meeting had not previously collaborated scientifically but through their interactions on setting up the meeting, submitted and obtained the first National Cancer Institute Program Project Grant on Nuclear Topology in Cancer. Several of the participants in the Forbeck Symposium agreed to serve on the external advisory board of this PPG, thus providing a forum for ongoing discussion and collaboration on this topic.
- Judging from the many vibrant discussions and group breakout discussions, we expect that many additional collaborative projects are being launched.

"Critical contributions were made by the three Forbeck Scholars who were invited to participate in the meeting:
Danfeng Cai, from the NIH
Peter Ly, from UT Southwestern
Kathleen Xie, from Harvard
This was an exceptional group of young investigators who participated actively and made important contributions."

Forbeck Forums:
Cancer Research throughout the Decades

This list does not include focus meetings and many other scientific events funded and organized by the Forbeck Foundation.

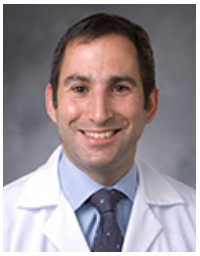
2021 – Mutational Signatures in Pediatric Cancer
Biomolecular Condensates in Cancer
Dynamic Histone Methylation and Chromatin Organization in Tumor Suppression
Diet and Metabolic Therapeutics in Cancer
Neuroendocrine Cell Fate in Development and Cancer
Fusion Oncogenes
2020 – Leveraging Synthetic Lethality to Treat Cancer
Cellular Reprogramming and Metastatic Disease
Graft vs Host Disease
Uncovering New Mechanisms of LKB1
Targeted Therapies in Pediatric Cancers
Emerging Strategies to Overcome Heterogeneous Resistance Mechanisms
New Insights into the Role of Microbes in Cancer
2019 – Cancer and Aging
Telomerase-Mediated Telomere Targeting in Cancer
DIPG Consensus Meeting
Leukemia Stem Cells, Heterogeneity, and Metabolism
3D Chromosomal Architecture and Nuclear Topology
2018 – Cancer Predisposition
2018 – Tumor Microenvironment
2018 – Epigenetic Therapy
2018 – Metabolic Signaling and the Epigenome
2017 – MYC/RAS
2016 – Chromosomal Instability/Aneuploidy
2015 – Cancer Immunotherapy
2014 – Invasion and Metastasis
2013 – Resistance Mechanisms
2012 – Tumor Metabolism
2011 – Epigenetics
2010 – Cancer Genomics
2009 – The Biology and Treatment of Primary Brain Tumors
2008 - Immunotherapy and Breaking Tolerance
2007 – Micro RNA and Cancer
2006 – Stem Cells
2005 - Innovations in Imaging in Cancer Research
2004 - Molecular Targets in Pediatric Malignancies
2003 - DNA Damage and Cancer Susceptibility Syndromes
2002 - Cellular Senescence and Cancer
2001 - Differentiation as Cancer Therapy
2000 – Allogeneic Stem Cell Transplantation

For more information about these topics and to learn about topics before 2000, please visit wgfrf.org.

Scientific Advisory Board

DIPG Consensus Meeting

Oren Becher, MD, Northwestern University
Cynthia Hawkins, MD, PhD, The Hospital for Sick Kids



The Forum was focused on a rare childhood brain cancer called DIPG, which stands for diffuse intrinsic pontine glioma. It is one of the most difficult cancers to treat due to its location and it's infiltrating nature, so it cannot be removed with surgery. This is why it is still incurable in 2019. We brought in a diverse group of physicians and scientists that normally does not meet. This included neuro-oncologists, neurosurgeons, neuropathologists and basic scientists that study chromatin biology, tumor microenvironment, the challenges of delivering drugs to the brain, as well as those developing innovative approaches for this tumor. We discussed what we currently know about DIPG, what questions remain to be tackled and what are barriers for the development of effective therapies and how to overcome them. The meeting was relevant now because there have been lots of scientific advances regarding the biology of DIPG, but this new knowledge has yet to yield improved therapies for patients with DIPG.

We are planning to write a review article on DIPG based on the discussions we had on some of the overarching questions in the field. The article will review the state of knowledge, address several important questions in the field highlighting areas of consensus or disagreement between attendees, as well as summarize some new approaches in development. In particular, we will attempt to address a critical question regarding new therapies: what scientific knowledge regarding a specific new therapy is needed to have in advance of initiating a clinical trial for children with DIPG. As currently there are too many clinical trials for children with DIPG and the scientific rationale for all of these is quite variable. DIPG Families with children with DIPG currently struggle with choosing specific clinical trials for their children. It is possible that the dissemination of our future article will be informative to some families, and, as a result, they will more critically assess the state of scientific knowledge of novel therapies to help them decide the best trial for their child.

Telomerase-Mediated Telomere Targeting in Cancer

Titia de Lange, PhD, Rockefeller University
Jerry W. Shay, PhD, UT Southwestern

The past meeting, held in Denver from October 17-19, was the second Forbeck meeting on telomere biology. Since the last meeting, 17 years ago, great progress has been made in the field and several cancer-relevant aspects of telomere biology as it relates to cancer were discussed vigorously. Relevant to neuroblastoma, a recent meeting in the UK addressing new drug development strategies for this disease as well as a recent publication, reported that patients with high telomerase activity have overall poorer outcomes. We had a detailed discussion on how to target telomerase in cancer, exploring the pros and cons of telomerase inhibitors and so-called telomerase poisons, one of which has already been shown to be effective in neuroblastoma cell lines. We also discussed the potential deleterious consequences of telomerase inhibition based on new clinical knowledge of inherited telomere diseases which mimic telomerase insufficiency. Several clinicians discussed the challenges of treating such patients as well as the implications of patient data for understanding the role of telomeres in cancer. An important aspect of our discussion focused on recent new findings that indicate that children born with overly long telomeres are predisposed to cancer. In addition, we discussed the mechanisms by which loss of telomeres drive changes in the cancer genomes and the potential of gleaning actionable information from the current NextGen cancer genome sequencing efforts. Finally, a considerable part of our discussion was focused on a second pathway for telomere maintenance, called ALT. The ALT pathway is highly significant to neuroblastoma since a substantial number of patients have cancers that employ this pathway. This pathway has been known for a long time but was poorly understood. Recent progress now has revealed the details of how ALT works and is beginning to suggest how ALT could be targeted in the cancer clinic.

As humans age, the ends of linear chromosomes, telomeres, become progressively shorter. Short telomeres elicit a DNA damage response and can induce either senescence or apoptosis both of which can halt tumorigenesis. Telomerase, the enzyme that elongates telomeres, can counteract telomeres shortening and prevent senescence and apoptosis. This enzyme is absent from most normal tissue but is often activated in cancer. Recently, it has become clear that telomerase activation is a highly selected aspect of tumorigenesis, often occurring through point mutations in the promoter of the gene encoding the limiting component of telomerase. While telomerase is not oncogenic per se, it is almost universally required to permit the indefinite growth that occurs as part of cancer progression. The recent data on mutational telomerase activation has confirmed the view that telomerase is a required aspect of cancer cell proliferation and further supports the view that inhibition of telomerase should be an effective cancer therapeutic. These and other new data on telomeres and the role of telomeres in cancer made the discussion in the context of the Forbeck forum highly appropriate and timely.

Leukemia Stem Cells, Heterogeneity, and Metabolism

Martin Carroll, MD, University of Pennsylvania
Craig T. Jordan, PhD, University of Colorado
Aaron Schimmer, MD, PhD, Princess Margaret Cancer Center



Although there have been recent advances leading to less toxic therapies for acute myeloid leukemia (AML), the survival of patients after a diagnosis of AML is still poor. This meeting allowed researchers to take a "deep dive" into two discreet area's of AML pathogenesis where there has been significant new recent information but also significant controversy. First, the investigator's considered the title theme of leukemic stem cells vs leukemic heterogeneity. It was previously thought that all leukemia cells from a given individual were highly similar to each other. However, it has become clear from numerous lines of evidence that there are significant differences between an individual's leukemic cells that create a barrier to cure. In one model, cells that are similar to blood stem cells but have leukemic properties are resistant to chemotherapy. Significant discussion was had on how these cells can be characterized, their defining features, whether they are the target of novel therapies, etc. In the stem cell model, these leukemic stem cells give rise to all other leukemic cells. In an alternative model, leukemic cells vary from each other randomly. This random variation may occur through changes in the DNA (genetic variation), the molecules that regulate gene expression (epigenetic variation) and others. Significant discussion of recently described genetic variation was had with proposals and discussions about how to target this variation before it leads to therapeutic resistance.

Significant conceptual change in the impact of studies of leukemic cell metabolism did occur. One of the recent surprises in leukemic therapy is the success of the drug, Venetoclax, when used in combination with drugs known as hypomethylating agents. Venetoclax is thought to act through inhibition of a cell survival protein called Bcl2. However, the biologic studies of the role of Bcl2 in AML biology have shown conflicting results. An exciting session occurred where three investigators showed work that suggests either that Bcl2 has a previously unknown function or that Venetoclax may work through a different mechanism than proposed. In particular, the three groups have data that Venetoclax may inhibit the leukemic cells machinery for energy production, a biochemical process known as oxidative phosphorylation. Data was presented that combining venetoclax with other agents that further cripple this process is even more effective at killing leukemic cells in the laboratory than combining it with hypomethylating agents. Ideas for how to translate this into clinical trials were discussed. Overall, a major impact of the meeting on this diverse audience was to increase the understanding of the critical role of altered cell metabolism to leukemic development. Additionally ,there was significant discussion about developing new methodologies for studying cell metabolism in primary human AML cells. We believe that these discussions will quickly lead to new combination treatment approaches that take advantage of this new insight. New collaborations have actually already been initiated based on interactions at the meeting.

How does this impact patients today? As noted above, the discussion of leukemic cell metabolism and studies of the mechanism of Venetoclax activity in AML are likely to be highly impactful. In general, cancer doctors work to develop therapies by combining agents that are toxic to cancer or leukemia cells without causing an increase in side effects. It was agreed that the great breakthrough of the Venetoclax-hypomethylating agent combination is that leukemic control or remission can be achieved without a high level of toxicity. Data at the meeting demonstrate that this is not likely to be curative, however, for the majority of patients. Thus, the focus on adding new agents to this recently developed and lower toxicity regimen is likely to lead to new approaches towards curative therapy. In fact, some such trials have already been opened at the researchers sites and ideas were discusses about how to interpret those trials as they are performed.

"The meeting was highly successful in bringing together not simply the known experts in the field, but individuals with backgrounds in leukemic research, basic cellular biochemistry, clinical medicine and others to discuss and challenge idea's in the field. The meeting was highly interactive and lively."

"Several developments are likely to result from this meeting. All meeting attendees express great enthusiasm for the open, interactive 'give and take' of the meeting format. Several of the attendee's organize larger meetings and there was discussion about how to bring this format into these other meetings."

Leveraging Synthetic Lethality to Treat Cancer


February 20 - 23, 2020
Carla Grandori, MD, PhD, SEngine Precision Medicine
Christopher Kemp, PhD, Fred Hutchinson

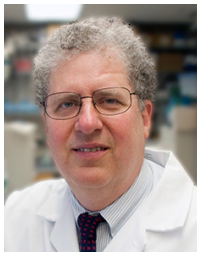
Two major roadblocks to improve cancer patient outcome are: 1) the shortage of available targeted agents and 2) lack of precision in assigning existing drugs to patients. Most cancer drug development efforts focus on a small number of genes that are commonly mutated in cancer, such as activated oncogenes. While there are obvious benefits to focusing on such targets and their downstream signaling components, a broader approach to target discovery will almost certainly provide increased opportunities. There are currently only ~ 100 cancer genes being targeted in the clinic, yet the human genome contains at least 22,000 coding genes. The purpose of this forum will be to highlight roadblocks to exploiting synthetic lethality for clinical benefit and strategies to overcome these.

Cellular Reprogramming and Metastatic Disease


March 19 - 22, 2020
Sarah- Maria Fendt, VIB-KU Leuven Center for Cancer Biology
Raul Mostoslavsky, Harvard University

We propose to explore current understanding of the molecular mechanisms that allow cancer cells to leave the primary site, disseminate, and grow as metastatic disease in a foreign tissue. We will also explore how these cells avoid/resist treatment as part of such metastatic disease progression. Despite major advances in cancer treatment, once tumors metastasize, and regardless of the tumor type, prognosis remains dismal. New knowledge suggest that beyond genetic mutations, cancers cells may acquire non-genetic adaptations (metabolism, epigenetics, cell cycle resistor phenotypes, immune modulation) that could confer unique advantage to these aggressive cells. We plan to discuss new models to study metastatic disease and current state of affairs in the field.

Graft vs Host Disease




April 23 - 26, 2020
Bruce Blazar, MD, University of Minnesota
Leslie Kean, MD, PhD, Harvard University

Allogeneic Hematopoietic Cell Transplantation (HCT) represents a powerful curative therapy for a broad range of both malignant and non-malignant diseases. However, alongside its powerful disease-modifying and graft-versus-leukemia effects, allogeneic HCT is also associated with multiple toxicities, which continue to significantly compromise long-term success. Amongst the most deadly of these complications are acute and chronic GVHD, which, together, develop in up to 80% of HCT patients. This conference will identify the new horizons in the biology and therapeutics for GVHD. We will look deeply into the growing molecular understanding of the pathogenesis of this disease and develop an agenda for the key questions the field must address in order to eliminate this deadly complication of HCT. Importantly, the identification of underlying mechanisms and therapeutic strategies to control GVHD is expected to have wide relevance to a number of diseases and therapeutic modalities, including the fields of autoimmunity and of T cell-based immunotherapy strategies.

Uncovering New Mechanisms of LKB1


May 14 - 17, 2020
Russell Jones, PhD, Van Andel Institute
Reuben Shaw, The Salk Institute

The tumor suppressor gene STK11, which encodes the serine/threonine kinase LKB1, is one of the most frequent hotspot mutations in human cancer. LKB1 plays multi- faceted roles in cancer, being implicated in both hamartomatous polyposis syndromes and malignant disease, including lung cancer (>30% of lung cancers display STK11 mutations). However, there has been limited progress in developing therapies to treat patients with LKB1- deficient tumors. Recent advances in understanding the tumor suppressor functions of LKB1, including regulation of inflammation, epigenetics, and anti-tumor immune responses, hold new promise for translating fundamental scientific discoveries to the clinic. The long- term vision of the meeting is to expand therapeutic options for patients with PJS or LKB1- deficient tumours by targeting novel aspects of LKB1-mediated tumor suppression.

Targeted Therapies in Pediatric Cancers

September 24 - 27, 2020
Steven G. Dubois, MD, Dana-Farber Cancer Institute
Martine F. Roussel, PhD, St. Jude Children's Research Hospital


Several novel targeted therapies based on rational scientific data hold promise to offer new and effective treatment not only for adult but also pediatric cancers. The new FDA pediatric legislative initiatives has galvanized pharmaceutical companies to extrapolating efficacy from adult data or other data to the pediatric population to streamline pediatric drug development and help to increase the number of approvals for pediatric use. Several clinical trials are ongoing for primary and recurrent and difficult to treat pediatric cancers that hopefully will advance children's care.

Emerging Strategies to Overcome Heterogeneous Resistance Mechanisms

November 5 - 8, 2020
Joan Brugge, PhD, Harvard Medical School
Charles Sawyers, MD, Memorial Sloan Kettering
Kris C. Wood, PhD, Duke University



Drug resistance limits the depth and duration of clinical responses to most anticancer therapies, including targeted therapies. This fact has motivated intense efforts in recent years to define the mechanisms of resistance to commonly used anticancer drugs based on the hope that by doing so, it will be possible to design new therapies that block these mechanisms and thereby circumvent resistance. In fact, the 2013 Forbeck Forum on Resistance Mechanisms, led by Joan Brugge, Ph.D. and Jeff Engelman, M.D., Ph.D. from Harvard (and attended by Dr. Wood), explored precisely this topic. Unfortunately, we now know that many diverse resistance mechanisms can exist for each drug, and these mechanisms often co-occur within individual patients. As a result, efforts to improve therapeutic responses by blocking individual resistance mechanisms have largely failed, while efforts to block combinations of resistance mechanisms have been limited by toxicities.

New Insights into the Role of Microbes in Cancer

November 12 - 15, 2020
Jannell Ayres, Salk Institute
Ken Cadwell, NYU Langone


Cancer biology and infectious disease research have a long history of cross-fertilization. Fundamental principles in cancer, such as the idea that a mutation in a gene can promote tumor formation, were established based on molecular virology experiments, and we now appreciate that many cancers have an infectious origin. The human papillomavirus vaccine and antibiotic treatment of Helicobacter pylori provide evidence that microbial triggers can be targeted to prevent cancer and save lives. More recently, ground breaking studies have identified the microbiome as a key variable in the development and treatment of cancer, including the efficacy of immunotherapy. Model systems and population studies are revealing diverse ways in which the collection of viruses, bacteria, and other transmissible agents that inhabit our bodies are impacting immunity. How do these microbiome members, either individually or as part of a microbe community, influence cancer immunity? We will discuss how our advanced understanding of host-microbe interactions can inform various steps of cancer biology, ranging from initiation to metastasis. The meeting will also include an exchange of ideas on whether infection-based cancers are more prevalent than previously appreciated, and how to leverage the power of the microbiome to improve patient outcome.

"The meeting was viewed as highly successful by all participants and several new collaborations were initiated at the meeting. The Forbeck scholars were all excellent and participated throughout the meeting in a very productive manner."

2019 Scholar Retreat in Review

The scholars of the 2019 retreat covered a diverse range of cancer topics, which included immune therapies, chromosome abnormalities, cancer predisposition and MYC genes. Although the scientists work on these seemingly disparate fields, it was amazing to see how many ideas each other had about how these problems can be inter-related. The forum also covered lots of mentorship from the more senior faculty, a "starting your laboratory 101" session for those who haven't begun their own laboratory, and, of course, plenty of spotted cow beer and wacky ping pong tournaments.

The cancer field has generated a tidal wave of new information over the past decade, especially in all of the fields covered by the scholars. However, most of the time we become very near-sighted in our own fields and fail to see the bigger picture. It was very helpful to stimulate tons of conversation about each other's topics, which generated lots of new ideas and collaborations.

There were many ideas generated for everybody's projects and everyone who presented went home with a long list of experiments and concepts to contemplate. There were also collaborations formed within several disciplines, and maybe more importantly, across different disciplines. As the chair of the meeting, having attended the meeting 6 years in a row, I can absolutely attest to this annual meeting leading to more thought provoking dialogue than any other time during my academic year.

Chad Pecot, MD
University of North Carolina

Leveraging Synthetic Lethality to Treat Cancer

Brooke Emerling, PhD - Sanford Burnham Prebys Medical Discovery Institute
Brook uses a genetic approach to elucidate the function of the PI5P4Ks (a kind of enzyme) in cancer metabolism. While trying to understand why cell growth was affected by this enzyme, she discovered another role this enzyme plays in metabolic processes call autophagy (consumption of the body's own tissue as a metabolic process occurring in starvation and certain diseases.) She hopes to find an Achilles' heel of p53 mutant tumors and to design therapeutic strategies for targeting the PI5P4Ks.

Mark Zimmerman - Dana-Farber Cancer Institute
During Mark's postdoctoral training at Dana-Farber, he has investigated how altered gene expression states are acquired and how these variations create vulnerabilities and opportunities for novel therapies. Mark is confident the Forbeck Foundation Forum will be the ideal setting for him to meet and hear from experts in this new and constantly evolving field, as well as forge relationships with other young investigators that may become future collaborators.

Andrew Venteicher, MD, PhD - University of Minnesota
During Andrews training, he gravitated toward projects that required innovative experimental approaches to address challenging questions in oncology. This required collaboration between a number of scientists with specific skill sets to efficiently plan, troubleshoot, and execute in new ways. Using a combination of genomic, proteomic, and single cell approaches, we seek to determine the shared and unique epigenetic regulators among IDH mutant cancers, which we anticipate will be vital to our success in designing tailored therapies for patients with these diseases.

Uncovering New Mechanisms of LKB1

Vajira Weerasekara, PhD - Massachusetts General Hospital
Vajira is a biochemist interested in LKB1 mediated organelle regulation and cancer. Vajira's work has implications for the pathogenesis and treatment of LKB1 mutant cancers and hamartomas, and broadly for LKB1 function. The highly focused, but multi-disciplinary framework for Forbeck meetings will provide Vajira with unprecedented opportunities to expand and critique the thought process behind these findings. Vajira is intrigued by the unique Forbeck presentation format, which pairs with his passion for discussion and debate.

Sponsor A Scholar

Each year, Forbeck Scholars are selected from an elite pool of up and coming scientists in the cancer research field to attend our four-year program. Your pledge of $1,000 each year (totaling $4,000) will directly support a scholar's participation in the program. An individual scholar will be identified with your pledge. You will be invited to the Kick-Off Dinner at the Scholar Retreat with the scientists and receive (2) tickets to the Blue Jean Ball each year you sponsor. This is your chance to participate in cancer research!

Please contact admin@wgfrf.org for more information.

Cellular Reprogramming and Metastatic Disease

Jatin Roper, MD - Duke University
Jatin's laboratory is interested in understanding the molecular mechanisms of stem cell function in the intestine and in colorectal cancer using innovative three-dimensional organoid and in vivo platforms. As a postdoctoral fellow with Omer Yilmaz at MIT, Jatin demonstrated that high fat diet-induced obesity activates peroxisome proliferator-activated receptor delta (PPARd) signaling in intestinal stem cells and progenitor cells, which increases stem cell regeneration and tumor initiation in the colon. …The overall goal of this research is to develop new treatment approaches for intestinal diseases such as inflammatory bowel disease and colorectal cancer..

Sharanya Sivanand, PhD - Massachusetts Institute of Technology
Pancreatic cancer, once metastasized, has a poor prognosis. It is currently unclear whether metabolic dependencies of tumors dictate where tumors metastasize. Given that most tumors display metastatic organotropism, I am currently studying the metabolic commonalities between primary and metastatic pancreatic cancers. This will shed insight into the role of tumor microenvironment in supporting the metabolic demands of cancers.

Elena Piskounova, PhD - Weill Cornell Medicine
Elena's lab focuses on the molecular mechanisms that underlie metastatic plasticity. As a postdoctoral fellow, Elena focused on the fundamental question of why metastasis is so inefficient that very few cancer cells can form metastatic tumors. As she is now developing her own independent research program, focusing on understanding other molecular adaptations that enable reprogramming and survival of metastasizing cancer cells, Elena believes becoming a Forbeck Scholar would be an incredible opportunity to discuss her work with colleagues, potential collaborators and learn from leaders in the field.

Graft vs. Host Disease

Armin Rashidi, MD, PhD - University of Minnesota
The questions Armin tries to answer his my research are highly cross-disciplinary, spanning a broad range of areas such as immunology, microbiology, computational biology, gastroenterology, and hematology/oncology. As a translational physician investigator, Armin is interested in linking the events occurring during anti-leukemia chemotherapy with acute GVHD. Understanding the risk factors of acute GVHD at earlier time points may permit development of novel prophylactic and therapeutic interventions targeting the inciting events before transplant. Such strategies may help reduce the risk of acute GVHD and provide new prognostic tools.

Tae Kon Kim - Vanderbilt University Medical Center
Tae is very interested in selectively inhibiting GVHD by manipulating co-inhibitory molecules in mouse BMT models. Tae is interested in the opportunity to be mentored to expand career development and receive critiques about future research from world renowned experts.

Melinda Biernacki, MD - Fred Hutchinson Cancer Research Center
Melinda is a hematopoietic cell transplantation (HCT) physician and scientist. Melinda's primary research focus is identifying cancer-specific antigens as targets to boost the graft-versus-malignancy (GVM) effect after allogeneic HCT or for stand-alone immunotherapy. Melinda has applied to the Forbeck Scholar Program because graft-versus-host disease (GVHD) is essential to consider when developing immunotherapy for hematologic malignancies. Mentorship by GVHD research experts, through the Forbeck Scholars Program, will be crucial for Melinda to frame and implement my immunotherapy research in the context of GVHD. Equally important is the close contact with other early-career investigators that the Program offers and the cross-disciplinary collaborations it will facilitate, providing new ideas to allow Melinda to contribute to accelerating improvements in HCT.

"Bringing researchers together from distinct scientific perspectives has great potential of generating new paradigms in cancer biology." - Andrew Venteicher, MD, PhD

In Memory of

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Shelly Beavers
Charlie Boehland
Jim Butschli
Anthony G. Forbeck
Billy Guy Forbeck
Kevin Patrick Forbeck
Jane McMillan Frackelton
William H. Frackelton, MD
Jean Marie Frick
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David & Dorcas Collins
Jennifer Forbeck
The Forbeck Family
Anne F. Sargent

In Celebration of

Don Cornelius

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