WILLIAM GUY FORBECK
RESEARCH FOUNDATION
wgfrf.org

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Making a Difference

John T. Kemshead, PhD
Chairman
Scientific Advisory Board

The world has significantly changed over the last 30 years. Back in the mid-1980s, mobile telephones looked like briefcases and the internet was in its infancy. Communication was clearly not what it is today, and this had a major impact on the treatment of patients with rare cancers.

In different countries, pediatric oncologists set up their own working groups so that they could study rare diseases. Neuroblastoma, a horrible childhood cancer with a devastating prognosis if it presents in its worst form, is such a disease with around 100 patients diagnosed per year in a population of 50 million. Study groups in the major Western and Asian countries established their own clinical trials and presented results at international conferences. Small but significant differences were noted in outcomes in different geographies. Was this due to inherent differences between ethnic groups or due to the way the patients with the disease were classified and hence treated?

George and Jennifer Forbeck founded the Foundation realizing that, without standardization and enhanced communication, progress in treating the disease would be significantly impaired.

They brought together leading clinicians from all over the world, and out of this meeting came the International Neuroblastoma Staging System (INSS) – also known as the 'Forbeck Criteria'.

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2017 Golf Outing

Where: Lakewood Golf Club
Lake Geneva, WI
When: Friday, May 19th
Time: 10:00am
Tickets: $150 per player

Thank you to our 2016 raffle contributors:
Bull Valley Golf Club
The Glen Club
Hawk's View Golf Club

The 2017 Golf Outing is sponsored by...

POLSINELLI

SAVE THE DATE!

May | 3 | 2017

Cheer on the Chicago Cubs for a cure for cancer!

Where: 3637 N. Sheffield, Chicago

Time: Rooftop @ 6:05pm
Game Time @ 7:05pm

Tickets: $150 per person
*Ticket price includes food, drink and the game

Pubraiser

Join us for food, drinks and good company at

The Fainting Goat in Denver, CO

Wednesday
March 22nd, 2017
6 - 10pm

The Fainting Goat is teaming up with WGFRF to raise funds and awareness for cancer research. For every dollar spent during the event, a percentage will be donated back to WGFRF!

BLUE JEAN BALL

The Blue Jean Ball is the culmination of the Scholar Retreat, which it directly funds. We would like to thank the 2016 chairs, Jennifer Keefe, Tricia Forbeck, and Mary Nicholson as well as Manuel De Moya, manager of Pier 290. They made a truly special event. Jeff Trudell donated his services by playing for the event. Everyone had a great time dancing, watching the Cubs, tasting wines, bidding on auctions and all for a good cause! Looking forward to 2017!

Join us this year at the annual Blue Jean Ball on

October 14th, 2017

Please visit www.wgfrf.org for additional details and to register for the event!

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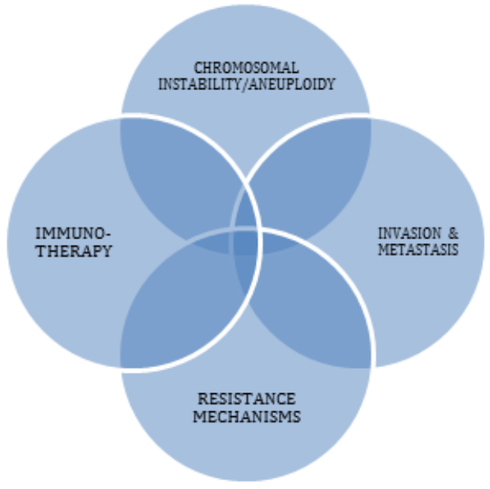
2017 SCHOLAR RETREAT

Lake Geneva, Wisconsin
October 12-15, 2017

Nabeel Bardeesy, MD, PhD
Retreat Chairman
Massachusetts General Hospital

Sixteen Forbeck Scholar awardees, who are outstanding junior clinical or post-doctoral fellows, are invited to Lake Geneva, WI, for a weekend scientific gathering. The Scholars focus on new and innovative ideas. The point of this meeting, is to foster collaboration among different institutions and different areas of science in order to advance cancer research. This has become an extremely fruitful meeting and past scholars have attributed much of their research to ideas and collaborations that have come from this meeting.

"The breadth and the depth of the scientific discussion at this retreat are both impressive and extraordinary. The unique presentation format provides a forum for consequential dissection of the central questions of the various fields, affording cross-fertilization of the ideas and concepts as invitees build on a critique of the ideas of each other...without a doubt, the ideas and talents cultivated by this Retreat will play key roles toward scientific advances that ultimately benefit humanity."
Clark Chen, MD, PhD, University of California



2017 Retreat Topics

The cross fertilization of ideas at the Scholar Retreat consistently initiates new ideas and new research in the fight against cancer.

FOCUS ON THE FUTURE: SCHOLAR AWARDEES

Uri Ben-David, PhD

Broad Institute

Research Interest – Works on understanding the cellular origins, functional consequences and potential vulnerabilities of aneuploidy and chromosomal instability in cancer. Characterizes the landscapes of chromosomal aberrations in cancer models and compares them to those observed in human tumors.

Lilian Kabeche, PhD

Massachusetts General Hospital

Research Interest – DNA damage is vital to repair damaged DNA to prevent mutations; several DNA damage repair proteins, including ATR have been shown to be important for mitotic progression in DNA damage. Her work focuses on understanding how DNA damage repair proteins work, both canonically, in repairing DNA damage and non-canonically, working in mitosis, to maintain genome stability.

Mia Levine, PhD

University of Pennsylvania

Research Interest – Focuses on the causes and functional consequences of chromatin protein evolution, also know as DNA packaging. DNA packaging is the folding of an organism's DNA molecule into a compact, orderly structure that fits within the limited space of a cell or virus particle.

Stefano Santaguida, PhD

Massachusetts Institute of Technology

Research Interest – Focuses on several aspects of the consequences of an unbalanced chromosome number, a condition known as aneuploidy, on cellular functions. This is an important focus in basic biology as well as highly relevant to human disease, since aneuploidy is strictly associated with cancer.

Neil Umbreit, PhD

Dana-Farber Cancer Institute

Research Interest – Studies how errors in chromosome segregation during mitosis can predispose cells to acquire mutations, including large-scale rearrangements of the genome, which are the hallmark feature of many cancers. Dr. Umbreit is especially interested in understanding how genomes continue to evolve over time after these initial mutagenic events, as it has important implications of tumor development and potential therapies.

Jason Sheltzer, PhD

Cold Spring Harbor Laboratory

Research Interest – The Sheltzer lab applies in vitro, in vivo, and in silico analyses to understand the genetic changes that underlie cancer development and progression.

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 14, 2017

Collaborative Research Program
Applications due April 14, 2017

Sponsor a Scholar

Your pledge of $1,000 each year for four years, the duration of the Scholar commitment, will directly support a Scholar's participation in the annual Retreat. An individual Scholar will be identified with your pledge. As a sponsor you will be invited to the Keynote dinner with the scientists and receive two tickets to the annual Blue Jean Ball for each year you sponsor.

For a list of Scholars available for sponsorship, please contact admin@wgfrf.org.

2017 FORUM

MYC and RAS

MYC and RAS are genes that make proteins that regulate how cells divide, differentiate and die. These are key attributes of cancer cells. Finding ways to interfere with the function of these proteins may open up new strategies to treat cancers.

Gerard Evan, PhD
University of Cambridge

Karen Cichowski, PhD
Brigham and Women's Hospital

In the last decade there has been a significant increase in the number of targeted cancer therapies. These are treatments that have been developed from basic research programs that have worked out differences between normal and cancer cells. They are designed to destroy the malignant cells, leaving normal cells unaffected. The MYC and RAS proteins are known to be key gates in pathways that are implicated in cell division differentiation and death. To date, it has been difficult for scientists to interfere with these proteins to kill cancer cells selectively. This meeting is planned to try and develop further insights into ways in which these proteins can be manipulated. It is also proposed that a discussion of clinical trial design will take place to see whether these can be designed to take into account the level of these proteins that are expressed in different cancer types.

Cancer research encompasses many different activities that range from basic research to multi-million dollar clinical trials that are primarily run by the pharmaceutical industry. You cannot get to a clinical trial without the ingenuity of basic researchers and clinicians, perhaps most importantly collaboration. The Foundation focuses on driving collaboration through its Think Tank Forums and Collaborative Research Grants to shorten the time frame needed to develop new cancer treatments.

2017 FOCUS MEETING

Precision Cancer Medicine by Functional Biomarkers

Chaired by: Anthony Letai, MD, PhD, Dana-Farber Cancer Institute

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 Dr. Letai's 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. Dr. Letai expects two main outcomes of the meeting. First, he thinks this will be the first such meeting ever of investigators explicitly focused on a functional predictive biomarker approach in cancer. As such, it would 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 would anticipate using this meeting to generate an outline for constructing a MATCH-like trial in cancer based on functional biomarkers.

COLLABORATIVE RESEARCH PROGRAM

Investigating the Causes of Blood Borne Cancers

The Collaborative Research Program is dedicated to promoting collaboration between scientists and institutions.

Collaborative research funding applications are due Friday, April 14th, 2017!

Chris Vakoc, MD, PhD
Cold Spring Harbor Lab
2011 Forbeck Scholar

Grant Challen, PhD
Washington University
2011 Forbeck Scholar

Dr. Challen and Dr. Vakoc will be working together to identify the genetic mutations that can lead to the development of certain blood borne malignancies. The DNA that makes up our genetic material is the blueprint that controls cell division and enables cells to perform their specific functions. During our lifetime, cells divide over and over again. Exposure to various toxic agents throughout our lives damages our DNA: these changes are called mutations. The presence of mutations in the genome can have serious medical consequences, such as the development of malignancies. Cancers are more common in older people, possibly due to the accumulated burden of genetic changes. Damage to our DNA may be a random event or it may occur at specific sites within the cell's genome. If one or more mutations affects the production of key proteins that control cell division, this can lead to the uncontrolled production of cells, which is a simplified description of cancer. It is hoped that the mutated proteins identified will become targets for novel drug-based therapies. If their research is successful, they may be able to devise new therapeutic strategies for patients with these life-threatening cancers.

Our supporters have affected all areas of cancer research with their generous support.

2017 Meeting Schedule

FOCUS MEETING
Precision Cancer Medicine by Functional Biomarkers
May 18-20, 2017
Boston, MA

FORUM
MYC and RAS
November 9-12, 2017
Lake Geneva, WI

SCHOLAR RETREAT
October 12-15, 2017
Lake Geneva, WI

Collaborative Research Projects

Comprehensive structure-function analysis of mutant IDH
W. Kimryn Rathmell, MD, PhD
Vanderbilt University
&
Benjamin Vincent, MD
University of North Carolina

Immune Cell Genomic and Metabolic Profiling in Renal Cell Carcinoma
Cory Johannessen, PhD
The Broad Institute
&
Julie-Aurore Losman, MD, PhD
Dana-Farber Cancer Institute

ANNUAL FORUM: *Chromosomal Instability & Aneuploidy*

Angelika Amon, PhD
Massachusetts Institute of Technology

David Pellman, MD
Dana-Farber Cancer Institute

2016 Participants

Peter Campbell, *Wellcome Trust Sanger Institute*
Lucia Carbone, PhD, *Oregon Health and Science University*
Daniela Cimini, PhD, *Virginia Tech*
Lucca Comai, PhD, *University of California*
Evan Eichler, PhD, *University of Washington*
Emily Hatch, PhD, *The Salk Institute*
Michael Lampson, PhD, *University of Pennsylvania*
Laura Landweber, *Columbia University*
Harmit Malik, PhD, *Fred Hutchinson Cancer Center*
Cynthia Morton, PhD, *Harvard University*
Zuzanna Storchove, *Max-Planck Institute*
Jan van Deursen, PhD, *Mayo Clinic*
Beth Weaver, PhD, *University of Wisconsin*

The 2016 Forbeck Foundation meeting provided an exciting venue for researchers in different fields to come together, for the first time, to discuss new advances in understanding the structure of cancer genomes, with potentially important implications for novel therapeutic strategies in cancer.

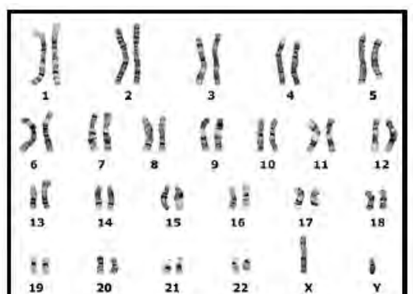
Much like the evolution of a new organism, cancer genomes evolve from normal ones by a series of DNA alterations, enabling all of the manifestations of the disease to develop. It is common to quote Shakespeare's Tempest for the insight that "What's past is prologue;" this insight was the theme of the 2016 Forbeck meeting. Knowing the past history of a cancer genome can help us identify the "drivers" of uncontrolled cancer cell division. Such drivers are important drug targets. Knowledge of the evolutionary history can also tell us about trade-offs made during cancer evolution, trade-offs that could lead vulnerabilities that might also be "druggable."

The problem with cancer is that we don't see the entire evolutionary history but only the final product of this evolution—the genome of the mature tumor. We, therefore, have to infer the evolutionary history of the cancer based on the DNA sequence of the cancer cell at the time of diagnosis and our knowledge of the "ways" that genomes can change.

This is a very similar challenge faced by evolutionary biologists who track the development of new species. Despite the conceptual similarities, these communities of scientists rarely interact. In this meeting, cancer geneticists and evolutionary biologists were able to discuss cutting-edged methods for defining the evolutionary history of complex genomes, with the specific goal of achieving a better understanding of cancer. An important focus was on strategies to identify DNA "signatures" of events that altered the genome. These signatures can be thought of as being similar to the fossil record that gives us insight into organismal evolution. To better define these signatures, the meeting also included molecular geneticists trying to recreate and better define these signatures in the laboratory.

Our chromosomes define who we are –

Like all living beings, humans are built from cells – approximately 37 trillion. Each of these 37 trillion cells harbors the same 23 pairs of chromosomes (46 total)



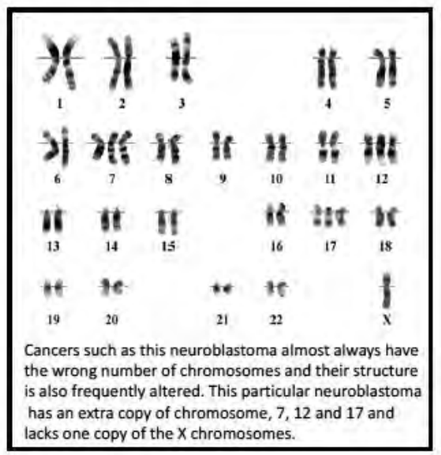
Our human genome is made of 23 chromosome pairs. All the information to make a human is contained within these chromosomes.

that are unique to us humans. Contained on these chromosomes, collectively called our genome, is all the information necessary to build a human from a fertilized egg.

The making of a human from a fertilized egg requires cell division during which each cell's chromosomes are duplicated and then evenly divided between the two daughter cells together with the cell's other content. The process of chromosome duplication and division is incredibly accurate. Cells make a mistake, incorrectly dividing up a chromosome pair, only once every 1000 to 10,000 divisions! The chromosome copying process is even less error prone. However, given that humans are made up of 37 trillion cells, even with such a low error rate, some cells in our bodies do end up with an incorrect chromosome number or errors in the information contained on chromosomes. The condition where a cell carries too few or too many chromosomes is called aneuploidy. Perhaps the most famous aneuploidy is Down Syndrome (also known as Trisomy 21). Individuals with Down Syndrome carry three copies of chromosome 21 instead of the normal two in each of their cells.

Chromosome number and structure change in cancer –

Aneuploidy is not only the cause of Down Syndrome, it is also a hallmark of cancer. More than 90 percent of solid tumors and 75 percent of blood cancers such as Leukemia and Lymphoma harbor too many, often twice to four times the normal number, and in rare cases too few chromosomes. We are all aware how dramatic the effects of Down Syndrome are on a person's intellectual abilities, health, and life expectancy. Changing the chromosome number by only a little, 47 instead of 46 chromosomes, has a dramatic impact on human health. How would gaining dozens of chromosomes and changing their makeup affect cancer cells? The goal of the 2016 Forbeck meeting was to understand how cancer cells end up with the wrong number and make-up of chromosomes and how these changes affect cancer formation, development and response to treatment.



Cancers such as this neuroblastoma almost always have the wrong number of chromosomes and their structure is also frequently altered. This particular neuroblastoma has an extra copy of chromosome, 7, 12 and 17 and lacks one copy of the X chromosomes.

"I think that the Forbeck Foundation is an amazing organization that targets the right scientific population."
Martine Roussel, PhD

New technologies, known as high throughput DNA sequencing technologies, have provided unprecedented insight into how the cancer cell's genome changes as cancers develop. We now understand that not only chromosome structures and number are altered in cancer, we also know that within a tumor not all cancer cells are alike. Cells within the same tumor differ in their chromosomal make-up and are said to be heterogeneous and constantly evolving. The challenges we are faced with now is to explain what drives this plasticity and to find ways to exploit this hallmark of cancer for therapeutic intervention.

Another major goal of the meeting was to gain insight into how cancer genomes develop by considering what is known about how the genomes of new organisms evolve. Evolutionary biologists Evan Eichler, Lucia Carbone, Lucca Comai, Harmit Malik, and Laura Landweber described how humans, apes, fruit flies, plants and single celled animals known as protozoa use unusual strategies to shape their chromosomes and genomes.

Much discussion occurred around the topic of whether cancer genome evolution occurs one-step-at-a-time or rather in sudden bursts. This has important implications for how fast cancer develops and could have therapeutic implications if fast and slow evolving tumors have different properties. Dr. Comai's talk revealed an example of sudden genome evolution in plants that is strikingly similar to a

FORUM CONSENSUS AND CONCLUSIONS CONTINUED...

mechanism of cancer genome evolution discovered by Peter Campbell called "chromothripsis." Plants provide unique tools for studying this phenomenon. Significant discussion and cross-fertilization also occurred about methodology. Dr. Eichler emphasized the value of technical approaches that could "read" long sequences of DNA continuously to detect complex alterations of chromosomes. Peter Campbell presented a new method to define DNA sequence "signatures" that would indicate that the genome had been altered in specific ways.

Lively discussions and the development of new hypotheses were formulated surrounding questions as to what types of mechanisms are at play that facilitate the generation of abnormal cancer genomes. Jan van Deursen discussed the importance of centrosomes, key components of the chromosome division machinery in cancer evolution. Michael Lampson described that chromosome shape and structure can affect how chromosomes are divided during cell division, with specific alterations such as fusion between two chromosomes making them more susceptible to faulty partitioning. Emily Hatch and David Pellman discussed how chromosomes that find themselves isolated from the rest of the chromosomes can become damaged because their duplication becomes less efficient and accurate. David Pellman also discussed new work on abnormal duplication of the DNA and advanced a hypothesis that might explain some of the "signatures" described by Peter Campbell.

Another focus of the discussions was how cells react to having the wrong number of chromosomes. Daniela Cimini, Jason Sheltzer, Zuzanna Storchova and Angelika Amon discussed the wide-reaching effects that aneuploidy has on the state and function of normal cells and cancer cells. They proposed that an incorrect chromosome copy number can lead to further damage of chromosomes and changes in chromosome structure and number. Input from Dr. Eichler and others with expertise on genome evolution lead to ideas about how to test the contribution of this further damage to cancer genome structure.

A major topic of discussion was whether altered number of whole chromosomes could promote or inhibit tumor growth. Uri-Ben David presented data supporting the hypothesis that specific aneuploidies promote tumor development. Angelika Amon and Jason Sheltzer highlighted countervailing examples where chromosome number changes were not advantageous, arguing that the majority of chromosome abnormalities decrease cancer cell fitness but that some specific rare karyotypes do promote tumorigenesis.

An unexpected, yet exciting outcome of the meeting was the realization that not only did cancer biologists learn from evolutionary biologists but the reverse was also true. Lively discussions surrounded questions critical to both disciplines, such as how can we best infer evolutionary history from genome analysis data?

Is the evolution of new species and of cancer a gradual process or a sequence of defined, punctuated catastrophic events? And to what extent do errors in the chromosome division process shape the architecture of cancer genomes and define the development of new species? At the end of this meeting, it was clear that the evolutionary processes shaping new species have much in common with the development of cancer and that, if we understand one process, we will likely understand the other.

CONCLUSIONS AND OUTLOOK

The 2016 meeting on Chromosomal Instability and Aneuploidy was unique in that, for the first time, it brought together evolutionary biologists and scientists who seek to understand how our genomes change during the process of cancer development. Cross-fertilization as facilitated by the 2016 Forbeck meeting are critical to push the field forward. They generate new ideas and approaches that would have otherwise not occurred. The success of the 2016 meeting is perhaps best illustrated by the fact that it, already, has led to collaborations. David Pellman and Nabeel Bardeesy will work together to understand the complex processes that pancreatic cancer cells undergo to reshuffle their genomes. Uri Ben-David and Angelika Amon have initiated a collaboration to understand why certain chromosome gains and losses are highly prevalent in specific cancers.

MAKING A DIFFERENCE CONTINUED...

This meant that there was, for the first time, a way of staging patients that is universally available and that clinical trial results could be compared internationally. The Foundation continued to support the development of the INSS, and as more was learned about Neuroblastoma, the staging system, it grew to include information about the biology of the disease. With the inclusion of this information into the staging system, this then became known as the International Neuroblastoma Risk Grouping (INRG).

With such a rare disease, progress in understanding the reasons why it occurs in young children and how new treatments can be developed and tested is still limiting. Realizing this, in 2011 the Foundation supported the development of a database of patients with Neuroblastoma; the (INRGdb). Information about the patients clinical history, the biology of their disease and their treatment is coded in such a way that individuals cannot be identified so it can be shared with all.

Currently, details of over 18000 patients are in the database, which is quite an achievement. The database is housed at the University of Chicago and now also interfaces directly with the Children's Oncology Group Tumor Bank in Columbus, Ohio.

Funding this effort in its entirety is beyond the capabilities of the Foundation, and others have now stepped in to assist in the growth of the database that is available for all to query and learn more about the disease.

I challenge all of you reading this to find an instance where a small Foundation has made such a fundamental contribution to driving progress in treating a devastating cancer such as Neuroblastoma. Furthermore, the model we developed for setting up a common staging system for Neuroblastoma has been copied by other Foundations supporting other disease types.

Quite an achievement for a small Foundation, and we are not finished yet!

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"There are a lot of Foundations out there and they do a lot of great things to raise money for research and raise awareness. This Foundation is very unique and of course its altruistic but it is designed to bring researchers together and to challenge them. And that is what I find most unique about this Foundation that I really admire."
Chad Pecot, MD
University of North Carolina

"It's a legacy that we need to continue to build because until cancer is gone, before it's only in the medical history books, we need these young scientists to be empowered to do their work because it takes a village. It's a big village of researchers, and the Forbeck Foundation has really helped this village be more vibrant and more productive."

Michael Jensen, MD
Director, Ben Town Center for Childhood Cancer Research, Seattle Children's Research Institute

IN MEMORY OF THOMAS A. GELDERMANN

The Forbeck Foundation lost a Founding Member and dedicated friend to the Foundation. Thomas A. Geldermann, also known as TAG, passed away at age 90. Tom led an incredible life filled with love, family, success, adventure and charity. He helped to found the Forbeck Foundation after Billy Guy died in 1985 and launched the idea of the Founding Chairs. The funds brought in by this initial venture sustained the organization scientific think tanks and secured its success for over 30 years. All those who knew him were blessed and cherished the wisdom and strength he imparted.

Thank you to those who support the Forbeck Foundation in Tom's memory...

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IN MEMORIUM OF ALEX SHELDON

The Foundation had a very disheartening loss this year. Alex Sheldon had been diagnosed with cancer, treated, and was in remission. Then he relapsed, and his disease came back with a vengeance. Even with access to the best research in the country, his battle was lost at age 30. Alex's grandfather, Hayden Leason, and his uncle, Michael Leason, have both served on the Board of Directors of the Forbeck Foundation.

Seeing another healthy, strong, and athletic young person taken by this dreadful disease made us question again--why are we still losing to cancer? At one of the Foundation meetings this year, we heard one doctor ask a fellow scientist "Why haven't we cured cancer?" The answer was cancer is very complicated and cancer cells are incredibly smart and are able to evolve.

The loss of Alex will remind us all to renew efforts to outsmart this disease. Alex was a skier, golfer and entrepreneur and he was loved by his family and friends. He will be missed but will remain an inspiration to all those at WGFRF to work harder.

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CHICAGO CUBS EVENT
WEDNESDAY, MAY 3rd, 2017

6:05pm Rooftop, 7:05pm Game Time
3637 N. Sheffield, Chicago
Register under AboutUs/Cubs Event at wgfrf.org

WGFRF GOLF OUTING

GOLF OUTING
FRIDAY, MAY 19th, 2017

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 14th, 2017

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

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