2020 Fall Newsletter | XXXVI Edition

William Guy Forbeck Research Foundation



Cancer's Leading Thinkers. Together in one room.

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Thinking Ahead

This fall marks the 35th anniversary of the Foundation and we had big plans to recognize its founders, participants, and so many others for their awe-inspiring work over the decades. We are extremely saddened that this can no longer safely be done in person, but this just gives us more time to plan the 36th Anniversary Gala. In the meantime, the annual golf outing has been rescheduled to Friday, September 25th, and the Foundation is getting ready for the busiest year it will ever have in 2021.

While we are seemingly frozen in time waiting for the new normal, cancer is continuing to kill approximately 10 million people (about half the population of New York) per year. Resources are being diverted from every aspect of life towards COVID-19 related research and issues. While this is incredibly important so life can resume sooner rather than later, our most complex foe, cancer, is not getting any closer to a cure.

It is going to take countless resources and strategizing to counter the damage done from the stay at home orders, recreating research that had to be destroyed, lost funding, etc. Organizations like the Forbeck Foundation will be more important than ever. Also, the small intimate format has always been more productive than the large meetings but will be more feasible than the large convention formats. The Foundation has prided itself on running highly influential and respected Forums for the scientific community. This format does not lend itself to going virtual. It is all about discussion, unpublished research, and innovative ideas. There is a sense of comfort and privacy that is achieved when meeting in person which is not possible with virtual events. Consequently, almost all of the 2020 meetings have been delayed until 2021. We are also planning for the new challenges that come with inperson meetings and including social distancing, masks or any other considerations for the comfort and safety of all participants.

While the Foundation has paused programming this year, 2021 will make up for the lack of programming in 2020. We will need our supporters more than ever to help keep up with this monumental year and attempt to fast track the reignition of cancer research. Our costs have only been delayed. We will have less time for fundraising with the 16 meetings in 2021, so please keep us in mind this year.

Our current focus is on Sponsoring our Scholars. Although they cannot meet in person; the Scholar Program is still going strong. In fact, we have more scholars than we have ever had before! We are considering alternative ways to help this group and we continue to accept scholars for the 2021 programs. We especially want to be there to support this group so that cancer research does not lose brilliant minds or progressive research done by the younger generation.

Sponsor a Scholar Program

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. We invite you to read all about our newest group of scholars on pages 8 and 9. All of these scholars need to be sponsored. Please help us ensure they can attend the retreats which will further their careers. They are the future of cancer research.

Please contact admin@wgfrf.org for more information.



Science in a time of COVID

In collaboration with Clark Chen, MD, PhD, Jamie Collins, Aaron Jesser, and Lauren Roadman

With COVID-19 consuming the entirety of our consciousness, containment has emerged as an absolute priority for our nation. With exception to COVID-19 related research, containment efforts translated into an once-in-a-generation laboratory shutdown and hibernation in research activities. The consequence of such a historical measure is palpable throughout the scientific community. To better understand this impact, the William Guy Forbeck Research Foundation surveyed its Awardee and Scientific Advisory Board, with researchers spanning five continents, a broad spectrum of disciplines, and different stages in career path.

The survey included the following questions. How have you been able to carry out your research? How have you and your colleagues been impacted? What do you see as the long-term consequences?

The responses portrayed a bleak outlook with recurring concerns for diversion of research funds toward efforts that narrowly focused on COVID-19. These concerns were magnified by the anxiety of economic downturn and its impacts on philanthropy, industry, and government research funding. Echoed in the responses was escalating stress suffered as scientists were being deprived from their pursuit of life sustaining passion. Worries of revision deadlines, lost reagents and anguish of stalled momentum were also expressed. Senior investigators repeatedly expressed concern for the vulnerability of new investigators who had begun their maiden scientific voyages in this challenging environment. As death from COVID-19 continues to escalate and is now the number two cause of death in the U.S., we believe that it is absolutely appropriate that research be directed toward COVID-19. However, it is equally important to keep in mind that the ailments that previously affected our human condition remain constant. Cancer, trauma, and other diseases continue to kill and devastate. It is fair to assume that every reader of this article is either suffering from these diseases or is touched by them through a loved one. As such, we simply cannot afford to have research funds diverted from progresses in these fields. In this context, it is essential that the research community engage the political dialogue to compel increased research funding.

In a recent interview, Dr. Anthony Fauci, a national leader in infectious disease and a voice of reason in this uncertain time, foresees a path toward return of professional sports, such as the NFL. If there is a pathway forward in an arena where contacts are inevitable during this containment period, there must be a way for us to continue our scientific endeavors in a deliberate and safe manner. If there are daily news cycle with counts of human sufferings from cardiac, cancer, and other diseases, analogous to how COVID-19 death are tallied, our citizens will not tolerate anything but a timely resumption of research in these fields. Thus, it is critical that the leaders in our scientific community deliberate and issue guidelines for how we can rationally and safely resume research in a timely manner.

As segments of our population are at higher risk for morbidities related to COVID-19, our young investigators will be particularly vulnerable to this COVID-19 pause in laboratory research. They are also most sensitive to the many subsequent funding challenges. Arguably, one of the most devastating consequence of this containment will be the loss of brilliant scientific minds who sought out alternative career paths - minds from which the next generation of cures will emerge. Support of our young investigators is imperative in this context. More than ever, policies and framework for institutional support, faculty mentoring, and peer support of junior faculty will be needed.

Viruses have been and will remain a part of our ecosystem and our human experience. The likelihood of viral pandemics will increase as the world more and more becomes one. Improving the human conditions in parts of the world that need help the most is no longer an altruistic pursuit. It is a requisite for the safety of our family and our loved ones. As it is difficult to determine the nature

Remembering Anthony Terlato

This year we mourn the tragic loss of Anthony Terlato. He was a great friend to so many including the Forbeck Foundation. He was a legend in the wine industry and known for being "The Father of Pinot Grigio" and his impeccable taste. We personally knew him as a kind face that was always willing to support the Foundation.



toward a better tomorrow.

of the next viral outbreak and the form

that mitigation will take, a fundamental

and broad understanding of the biologic

processes that underlie our human

physiology is no longer an abstraction. It

is a requisite for the next diagnostic tool or

therapeutic intervention that will enable us

The COVID-19 pandemic has exposed

the fragility of our research infrastructure

and the incredible need to fortify it. Our

emergence from this pandemic will be

entirely dependent on the fruits of our past

research...antibody testing, PCR diagnostics,

DNA sequencing, patient tracking ... every

aspect of our current measure against COVID-19 is built on accomplishments of our

scientific predecessors. Viewed in this light,

COVID-19 is a painful reminder for the need

for our society to re-focus on the support for

The World will be forever transformed by the

COVID crisis, and it is our responsibility as

global citizens to shape this transformation...

fundamental scientific investigations.

to better address the next epidemic.

Anytime we reached out to Tony or his family to facilitate a wine tasting at the Blue Jean Ball, or provide wine for an event, there was never a question of their support. We will greatly miss Tony and will keep the Terlato family in our thoughts and hearts.

It's a really fascinating and unique meeting structure, in my experience. We have a small group of scientists who are thinking together about a problem from many different aspects, and you're really here to think not just to listen or to learn, which you hope you do at meetings... in a very different way. It is unique, and it is, I think, extremely stimulating and likely to affect the work of most of us. -- Judy E. Garber, MD, MPH, Harvard University

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Biomolecular Condensates in Cancer

January 28 - 31, 2021 Danfeng Cai, PhD, HHMI Janelia Research Campus Alex Holehouse, PhD, Washington University in St. Louis Tanja Mittag, PhD, St. Jude Children's Research Hospital

Large-scale cytological changes are a classical hallmark of cancer, although the molecular etiology of these changes has historically been poorly

understood. A cell can be organized through membrane-bound organelles such as the endoplasmic reticulum and Golgi, or through membrane-less biomolecular condensates such as nuclear bodies and stress granules. While membrane-bound organelles are well-studied, the biology of membrane-less biomolecular condensates is less well-understood. It has recently been found that biomolecular condensates form through phase separation - a demixing phenomenon in which the associated acromolecular components form a dense liquid-like assembly. In addition to large well- defined membrane-less organelles, there exist a growing number of examples in which smaller biomolecular condensates mediate key biological processes. Of particular recent interest, much of the transcriptional machinery appears to consist of biomolecular condensates. The meeting is timely since the field of biomolecular condensates in various aspects of biology has grown exponentially in recent years, and the roles of biomolecular condensates in diseases such as cancer are just beginning to be uncovered.

Mutational Signatures in Pediatric Cancer

January 28 - 31, 2021 Stephen J. Chanock, MD, National Cancer Institute Adam Shlien, PhD, The Hospital for Sick Children Jinghui Zhang, PhD, St. Jude Children's Research Hospital

The signatures of mutation in cancer genomes are markers of past mutagen exposure or intrinsic DNA repair defects. Recent studies in childhood

cancer have shown that mutational signatures can uncover a tumor's origin - including whether it arose due to inherited mutation – and point to therapeutic vulnerabilities. In this meeting, we will discuss the enormous potential utility for mutational signatures in childhood cancer, in predicting future outcomes and eligibility to clinical trials, mechanisms for therapy-induced drug resistance, as well as performing "reverse genetics" – understanding the tumor's past evolutionary trajectory and possible etiology. Knowing that childhood cancers are unique, this meeting's participants will propose mutational signatures approaches that have been adapted for these patients specifically. This will include focusing on thresholds for detecting underlying mutational signatures, such as mismatch repair and homologous recombination, as well as signatures involving structural rearrangements, which are frequent drivers of childhood cancer malignancy.

Immunotherapies and Mechanisms of Immune Escape

February 11 - 14, 2021 Esra Akbay, PhD, UT Southwestern Medical Center Monica Guzman, PhD, Weill Cornell Medicine

Modern immunotherapy concepts have changed cancer treatment paradigms, such as immune checkpoint blockade, chimeric antigen receptor (CAR) T cells, and antibodybased therapies have made rapid progress in the past 10 years and are currently used to treat

many cancers. Immune checkpoint inhibition circumvents tumor-induced suppression of cytotoxic T cell function. Adoptive transfer of T cells expressing chimeric antigen receptors (CARs) are currently approved treat CD19-positive cancers. However, only a fraction of patients responds, especially in the field of solid tumors as, for example, the immune microenvironment plays a critical role in disease progression and outcomes as it can induce immune suppressive signals that prevent immune-surveillance and allow malignant cells to persist. Therefore, combination treatments using different kind of immunotherapies targeting multiple checkpoints are necessary for more effective responses. Discussing all the components that are known to play a role in the mechanisms of immune escape and the emerging concepts for therapeutic interventions in a multidisciplinary setting will lead to the implementation of rationally designed trials to better harness immune system for the benefit of cancer patients.

"I love coming to a group like this because they think outside the box. They think big. They're not satisfied with things like 'remission' and 'response.' They're only satisfied with big words, big concepts like 'cure.'" -- Chad Pecot, MD, The University of North Carolina









Dynamic Histone Methylation and Chromatin Organization in Tumor Suppression

March 4 - 7, 2021 Kimryn Rathmell, MD, PhD, Vanderbilt University Brian D. Strahl, PhD, University of North Carolina

A multitude of activities and functions have arisen around the enzymes that provide specific methylation marks on histones, and which are perturbed in cancer. These enzymes, for example SETD2, are no longer considered as simple factors in the specificities of methylation. Recent data suggests that spatial distribution, dynamic activity, structural composition, as

well as non histone targets adds to the complexity of these interactions as they pertain to cancer. The recent explosion of mutations in the histone tail residues as well as methyltransferase domain mutants has led to potential for siloing as the eld grapples with the diverse functions of these proteins. A meeting of this nature has potential to advance the science of histone biology in cancer suppression and to bridge in the many additional targets and factors involved, bringing investigators approaching this problem from a diverse array of angles.

The Genesis and Function of Extrachromosomal Oncogene Amplifications in Cancer

March 18 - 21, 2021 Peter C. Scacheri, PhD, Case Western Reserve University Chia-Lin Wei, PhD, The Jackson Laboratory

The purpose of this meeting is to explore the formation and function of extrachromosomal DNA (ecDNA) amplifications in cancer. In recent years, driven largely by advances in genomics and DNA sequencing technologies, we have learned that extrachromosomal DNA is far more prevalent in tumors than previously appreciated. ecDNA is present in 20-40% of all human

cancers and is particularly common in aggressive cancers notoriously difficult to treat, such as brain cancer. Moreover, our inability to effectively treat cancer is due to an innate ability of ecDNA cancers to evolve in response to environmental stressors. We have further come to learn that circular ecDNAs incorporate active gene enhancer elements that provide a selective growth advantage beyond that of the oncogene alone. This highlights an additional layer of regulatory complexity in canonical cancer driver events that will need to be considered to maximize targeted cancer therapies for patients.

Cellular Reprogramming and Metastatic Disease

March 18 - 21, 2021 Sarah- Maria Fendt, PhD, VIB-KU Leuven Center for Cancer Biology Raul Mostoslavsky, MD, PhD, 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.

Aneuploidy in Cancer Development, Prognosis/Treatment

April 15 - 18, 2021 Uri Ben-David, PhD, Tel Aviv University Elsa Logarinho, PhD, Institute for Molecular and Cell Biology Stefano Santaguida, PhD - Univ. of Milan/European Institute of Oncology

We propose a meeting, entitled "Aneuploidy in cancer development, prognosis and treatment", that will bring together researchers from the cancer aneuploidy community. In particular, we envision a platform of creative thinking and scientific cooperation, gathering scientists with

expertise and interests in studying how chromosome imbalances arise during tumorigenesis and how they can be targeted for cancer therapy.









Diet and Metabolic Therapeutics in Cancer

April 22 - 25, 2021 Navdeep Chandel, PhD, Northwestern University, Heather Christofk, PhD, UCLA, and Jason Locasale, PhD, Duke University

Nutrition exerts profound effects on health, and dietary interventions are commonly used to treat diseases of metabolic etiology. Although cancer has a substantial metabolic component, the development of principles

that define whether nutrition and new metabolic therapies may be used to influence tumor outcome are in the beginning stages. Nevertheless, it is also established that targeting metabolic pathways with longstanding chemotherapy agents or radiation can sometimes lead to controlled therapeutic outcomes. In contrast, whether specific dietary interventions could influence the metabolic pathways that are targeted in standard cancer therapies is not well known. This meeting will bring together current and emerging leaders whose work bridges the nutrition-cancer interface to discuss their work in the new interdisciplinary field of 'Nutrition and Cancer Metabolism' and to explore its potential as a novel area for therapeutic intervention.

Neuroendocrine Cell Fate in Development and Cancer

April 22 - 25, 2021 Cory Abate-Shen, PhD, Columbia University and Julien Sage, PhD, Stanford University

Neuroendocrine cancer is an umbrella term for a group of tumors in which cancer cells have the particularity of displaying traits similar to those of both nerve cells and hormoneproducing cells. Neuroendocrine tumors can occur in many tissues and organs in the human body, including the lung, the pancreas, the prostate, or the skin. Some neuroendocrine

tumors are very rare, others are more frequent; some are indolent, while others are extremely aggressive and fatal. Historically, neuroendocrine tumors have been studied and treated in the context of the specific site in which they were found. More recently, however, accumulating evidence has indicated that neuroendocrine tumors from various sites shared a number of important features, especially in terms of oncogenic drivers. A major goal of the meeting will thus be to discuss the biology and clinical aspects of neuroendocrine tumors to understand the differences and similarities between these tumors in different organs and tissues. We hope to arrive at a consensus about how to distinguish and define this group of tumors, and hopefully identify a framework to develop future therapeutic strategies that may help all patients with neuroendocrine cancer.

Uncovering New Mechanisms of LKB1

May 13 - 16, 2021 Russell Jones, PhD, Van Andel Institute and Reuben Shaw, PhD, 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 23 - 26, 2021 Steven G. Dubois, MD, Dana-Farber Cancer Institute and 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

October 28 - 31, 2021 Joan Brugge, PhD, Harvard Medical School, Charles Sawyers, MD, Memorial Sloan Kettering, and 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 11 - 14, 2021 Janell Ayres, PhD, Salk Institute and 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.

Graft vs Host Disease

November 11 - 14, 2021 Bruce Blazar, MD, University of Minnesota and 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.

"I think it's important to attend the Forbeck meetings because they are unique. Most meetings that we go to have a lot of people there. People are very reluctant to talk about unpublished research, and that is not the case with the Forbeck meetings. The idea is to talk purely about unpublished research and also only in front of a small group of scientists that are from the same field but don't necessarily have the same interests and follow the same direction. So there's a lot of diversity and a lot of communication." -- Jan Karlseder, PhD, Salk Institute







Emerging Strategies to Overcome Heterogeneous Resistance Mechanisms



Kirsten Bryant, PhD - University of North Carolina at Chapel Hill

Dr. Kirsten Bryant completed her postdoctoral studies in 2018 at UNC- Chapel Hill where she currently is a researcher and assistant professor. Her current research involves establishing strategies that target autophagy (when the body consumes its own tissue during metabolic processes in starvation and certain diseases) inhibition. Recently Bryant has addressed the role of a KRAS, a protein that facilitates a signaling pathway related to cell growth, death, and mutation. The KRAS protein displays self-devouring activity that supports the growth of pancreatic cancer. Many of Bryant's studies have led to the initiation of clinical trials at the Anderson Center and UNC-Chapel Hill. Bryant shares the Forbeck Foundation's vision of breaking down barriers between researchers and hastening progress through collaboration.



Cihangir Duy, PhD - Weill Cornell Medicine

Dr. Cihangir Duy received his PhD in Molecular Biology/ Immunology from The University of Southern California. He then began his post-doctoral program at Cornell focusing his research on cancer epigenetics where he uncovered that chemotherapy is not effective at eliminating leukemia cells. He discovered that it in fact pauses the regeneration process of the cells only to resume this process once the treatment is finished. Duy's post-doctoral studies were interrupted by a cancer related family emergency, that continued to motivate his research and studies. He finished his post-doctoral work in 2019 and received a position as an instructor at Cornell. Currently his research is focused on examining the biological mechanics relating to the leukemic cells and their

regeneration pathways. He hopes that attending the Forbeck Forum will help him discuss and design creative strategies against resistant mechanisms, and that he will be given the opportunity to initiate fruitful collaborations with the aim to improve the survival of cancer patients.



Peter Winter, PhD - Massachusetts Institute of Technology

Dr. Peter Winter is a postdoctoral researcher at MIT. His current work is focused on drug resistance. One goal of his research is to eliminate Minimal Residue Disease (MRD) in pursuit of a solution, Winter and his group found that mature cells are more resistive to treatment and are more vulnerable to mutations. Additionally, the group is establishing features of the transcription process in cancer cells that do not respond to chemotherapy treatment specifically in pancreatic cancer cells. Winter aims to establish an independent research group that studies mechanisms of drug resistance in cancer. He hopes the Forbeck Forum will enable him to engage with experts in cancer biology, refine his avenues of scientific investigation and give him the connections he needs to begin his career as an interdisciplinary scientist.

New Insights into the Role of Microbes in Cancer



Semir Beyaz, PhD - Cold Spring Harbor

Dr. Semir Beyaz received his Ph.D. in Immunology from Harvard in 2017. During that time, he studied epigenetics and metabolic mechanisms that control cellular fate during differentiation which could be in response to diet. Meaning he assessed how different metabolic and nutritional factors resulted in why certain cells become the cells they do. For example, what factors make normal cells turn into mutated cancer cells. Metabolic reactions are chemical reactions that take place in our body and are essentially the scientific explanation of how living things live. Different environmental factors such as tobacco use or obesity can negatively affect a normal metabolic state resulting in cell mutations and possibly cancer. Beyaz takes a detailed look at how different

environmental and external variables or interruptions disrupt immunity, impact inflammation, and promote tumor growth and additionally whether these factors can be reversed. He currently is a principal investigator at Cold Spring Harbor Lab in New York.



Camille Jacqueline, PhD - University of Pittsburgh

Dr. Camille Jacqueline received her Ph.D. in Health Biology from the Univ. of Montpellier France. She is currently in a postdoctoral position studying Immunology for the Univ. of Pittsburg. Jacqueline's work is focused on a new group of tumor-associated antigens (TAA) that are successful cancer immunosurveillance proteins. Meaning that these TAA's are able to essentially monitor the body for possible bacteria, viruses, or cancer/ pre cancer cells. There is an association between the presence of TAA's and a lowered lifetime risk of cancers. Jacqueline has been able to test and confirm that infection and inflammation related changes have been reported as a result of TAA being present. This type of research is crucial to the advancement of cancer prevention/treatment. The preventative implications of this work, finding something naturally in our bodies that is able to supervise our cells and prevent tumor growth, would be monumental.



Defne Bayik, PhD - Cleveland Clinic

Dr. Defne Bayik received her PhD in Molecular Biology and Genetics. She was a postdoctoral fellow for the National Cancer Institute working on a Cancer and Inflammation Program and now Bayik is a postdoctoral research fellow at the Lerner Research Institute in Cleveland Ohio. Her research is focused on sex-specific therapy resistance used to treat glioblastoma, which is a rapidly growing and spreading tumor found on the brain or spine. Bayik has discovered a genetic difference in males and females that results in only females being responsive to a specific type of cancer immunotherapy. Furthermore, she is assessing the degree to which alterations in the microbiome, meaning changes to the genetic information of the good natural bacteria and viruses inside us, correlate to the

degree and mechanism of immunosuppression in glioblastoma. Studying and understanding more about perturbations in our bodies regulatory systems is an important part of understanding cancer prevention, causation, and treatment.

Targeted Therapies in Pediatric Cancers



Dr. Adam Durbin received both his PhD and his MD from the University of Toronto. He went on to do his Residency in pediatric medicine at Boston Children's Hospital. Durbin is currently a clinician- scientist studying pediatric cancer epigenetics, and as an Instructor in pediatric hematology/ oncology. His research is focused on high-risk neuroblastoma a type of cancer that results in tumor growth in specific parts of the nervous system. According to the National Cancer Institute, most of the demographic inflicted with this cancer are children under the age of five. Durbin states unfortunately that "40% of those inflicted with this cancer end up relapsing and dying". In his studies Durbin has focused on a group of DNA transcription factors called core regulatory circuitry (CRC's) that

when disrupted result in cell death. This means that one could purposefully disrupt the CRC in order to program certain cells to die instead of divide, which could possibly stop the growth of the cancer.



Jessie Brown, PhD - Columbia University

Adam Durbin, MD, PhD - Dana Farber Cancer Institute

Dr. Jessie Brown is currently a postdoctoral research fellow at Columbia University. She received her PhD in Medical Sciences while studying Molecular Oncology and Tumor Immunology at the NYU School of Medicine. Her current focus is on chemoresistance in pediatric Acute Lymphoblastic Leukemia (ALL). This form of cancer is "one of the most common types of leukemia accounting for 30% of all pediatric cancer" (Children's Hospital of Philadelphia). ALL influences the premature production of white bloods cells, which results in the loss of full function. This means the white blood cells affected are unable to properly fight infection. Brown's research has theorized that these mutations in white blood cell production arise from genetic mutations, and additionally

that a conglomerate of cell regulating restrictions may result in chemotherapy resistance. Brown and her group have been able to identify the master regulators associated with drug resistance and self- renewal. This information can then go onto be applied to new cancer treatment therapies, which can hopefully increase the success and treatment of this destructive pediatric cancer.



Loretta Li, MD - Ann & Robert H. Lurie Children's Hospital of Chicago

Dr. Loretta Li received her medical degree from Harvard, which she followed up with a High Impact Cancer Research program also with Harvard. She served as clinical fellow in Pediatric Hematology/Oncology at Boston Children's Hospital and additionally served as research fellow studying Leukemia Biology again with the Dana Farber Cancer Institute. Currently Dr. Li is an assistant professor at Northwestern University, and she is an Assistant Professor/ Attending physician at the Lurie Children's Hospital of Chicago. Dr. Li's work is related to leukemia and the inhibition of a certain gene called the Janus Kinase 2 (JAK 2). This gene is associated with proteins that control the production of blood cells. When this gene is mutated the blood cells can become mutated and

carcinogenic. Currently Dr. Li is committed to building a research program focused on defining mechanisms of resistance, which will inform strategies to prevent/overcome resistance, genetic translation of an inhibitor that contributes to stopping the growth of tumors (meaning figuring how to clinically induce the production of an inhibitor for JAK2) and developing proteins that could be used to degrade JAK2.

- All bios researched and written by Emily Faul

"Forbeck Forums meet a very important need of the pediatric oncology and cancer community and that is bring together experts for several days to talk in a very focused way and really be unencumbered in not having all the large meetings and other requirements and pressures to do other things." -- Stephen J. Chanock, MD, National Cancer Institute

www.wgfrf.org

In Memory of

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