KARIN GRUNEBAUM
cancer research foundation

BOARD OF TRUSTEES

November, 2014 • Volume 11 Founded in 1958

From the Chair

Dear Friends of the Karin Grunebaum Cancer Research Foundation –

This year's letter will introduce you to our newest Trustees (and say farewell to others), and also highlight the achievements and recognitions recently awarded both to our Research Fellows and Trustees. In a small Foundation such as ours, the secret to success is to have a stellar Board of Trustees to show the world the extremely high caliber of research being conducted under our auspices, and to have Research Fellows whose outstanding performance brings them (and us) national recognition. Thus, it is a sad day when we have to say good-bye to any of these medical luminaries.

Since last year's letter we have regretfully said farewell to **Trustee Frank Hsu, M.D.,** who was a Karin Grunebaum Fellow in 1986 -1987, and was the third former Fellow to serve as a Foundation Trustee. Frank moved to California to become Chief Medical officer at Zyngenia, Inc., and found it impractical to continue commuting to Boston as a Trustee. We wish him the very best, and thank him for his years of service to the Foundation.

In a small Foundation such as ours, the secret to success is to have a stellar Board of Trustees to show the world the extremely high caliber of research being conducted under our auspices, and to have Research Fellows whose outstanding performance brings them (and us) national recognition.

We also lost the very valued services of **Trustee Karen Antman, M.D**. due to her selection as Chair-elect of the Council of Deans of the American Association of Medical Colleges, (an organization of US and Canadian medical schools). As she simultaneously continues her duties as Provost of the Medical College and Dean of the Boston University School of Medicine, she felt that she had to part company with the Foundation due to the extra time requirements of her new position. Karen added so much invaluable creativity and insight to the Board of Trustees, that her resignation left us all with a heavy heart. We are however happy to report that at the urging of the rest of the Board, she has decided to stay on as Trustee Emeritus and offer her services when and where she is able. We also wish her great success in her new position and thank her for her years of service to the Foundation.

On the brighter side, the Foundation was privileged to welcome **Edward "Ed"**

(continued on page 4)

From the Fellows

Boston University School of Medicine

Neil Ganem, Ph. D. '14
Howard Hughes Medical Institute,Department of Pediatric Oncology
Dana-Farber Cancer Institute, Children's Hospitaland Department of Cell Biology, Harvard Medical School

I am writing to express my sincerest thanks for choosing me as a 2014-2015 Karin Grunebaum Cancer Research Fellowship recipient. I am truly honored by the selection. Your gift will no doubt have a significant positive impact on both the research, and the people, in my lab. I would also like to apologize for having to miss the meeting with you in Boston, and to let you know that I fully intend on being present at the next get-together at Harvard Medical School in October. I look forward to meeting you all then.

I also wanted to tell you a little bit about myself, as well as the research you gift is helping to support in my lab. I received my PhD in Biochemistry from Dartmouth Medical School in 2006 and completed my post-doctural training in the Department of Pediatric Oncology at the Dana-Farber Cancer Institute at Harvard Medical School in December 2011. I was then promoted to Instructor of Hematology and Oncology at Dana-Farber and held that position until I moved to Boston University in September, 2013. My wife of 7 years, Amity Manning, is also an alumnus of Dartmouth and is currently an instructor of Genetics at Massachusetts General Hospital. We have three boys, Caelan, 5, Evan, 3, and Declan, 5 months.

My lab studies the causes and consequences of a phenomenon known as genetic instability, which is a hallmark of nearly all solid tumors. Genomic instability is characterized by the presence of both structural and numerical changes in chromosomes and is known to promote tumor progression, relapse, and chemotherapeutic resistance. Consequently, highly genomically unstable tumors have a very poor prognosis. One major focus of my research program is to uncover the cellular defects that generate genomic instability in human cancer cells, and one of our specialties is the use of high-resolution microscopy to directly visualize chromosome dynamics in living cancer cells. I'll make sure to show you some very cool movies of cells undergoing division at our meeting in October.

A second major focus of my group is to identify whether genomic instability represents a 'point-of-weakness' that can be therapeutically exploited. My lab has recently developed an innovative screening approach to comprehensively identify the proteins and molecular pathways required for genomically unstable cancer cells to survive and proliferate. Ultimately, our long-term scientific goal is to develop new strategies to therapeutically target these pathways in order to specifically inhibit the growth of abnormal, genomically unstable cancer cells while sparing the normal cells from which they originated. I'm very excited about this work, and some very promising leads have already emerged.

Aside from the science, your gift is also helping to support many people in my lab. Currently, my lab has 2 PhD students, Elizabeth Shenk and Amanda Bolgioni; an MD/PhD student, Sanghee Lee; a Master's student, Hatim Mustaly; and a research technician, Allison Matthews. I'm very fortunate that so many great people have decided to join my lab, and we certainly have our share of fun while doing science! I've included some photos so you can put faces with names.

Like the Grunebaum family, and so many others, my life was seriously affected by a debilitating disease to a loved one. When I was 12, my father was diagnosed with Parkinson's disease, and by the time I was 15, he was gone. Personally, I can think of few things in life that would be more gratifying than starting and organizing a foundation to support basic Parkinson's research in honor of my father. For that reason, I would like to congratulate the Grunebaum family for sustaining the legacy of Fritz Grunebaum by maintaining his foundation for more than 50 years. The charitableness is truly remarkable, and the Grunebaum family should be immensely proud.

I want to assure you that I will work to the absolute best of my abilities to maximize the return on your selfless investment.

Sincerely,
Neil J. Ganem, PhD

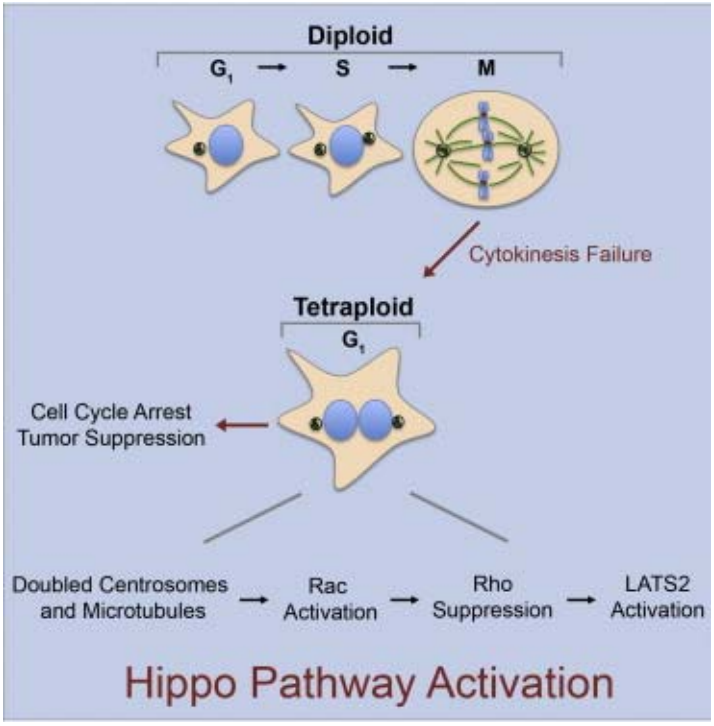
Boston University *(continued from previous page)*

Cytokinesis Failure Triggers Hippo Tumor Suppressor Pathway Activation

The following is the Summary of the article, by Neil Ganem, et al, published in Cell magazine, August, 2014.

Genetically unstable tetraploid cells can promote tumorigenesis. Recent estimates suggest that 37% of human tumors have undergone a genome-doubling event during their development. This potentially oncogenic effect of tetraploidy is countered by a p53-dependent barrier to proliferation. However, the cellular defects and corresponding signaling pathways that trigger growth suppression in tetraploid cells are not known.

Here, we combine RNAi screening and invitro evolution approaches to demonstrate that cytokinesis failure activates the Hippo tumor suppressor pathway in cultured cells, as well as in naturally occurring tetraploid cells invivo. Induction of the Hippo pathway is triggered in part by extra centrosomes, which alter small G protein signaling and activate LATS2 kinase. LATS2 in turn stabilizes p53 and inhibits the transcriptional regulators YAP and TAZ. These findings define an important tumor suppression mechanism and uncover adaptive mechanisms potentially available to nascent tumor cells that bypass this inhibitory regulation.



Rachel L. Flynn, Ph.D. Awarded Peter Paul Professorship

in Faculty Spotlight
September 19th, 2014

A Peter Paul Professorship was awarded to Rachel Flynn, a School of Medicine assistant professor of pharmacology and experimental therapeutics,

University trustee Peter Paul (GSM'71) created the professorships named for him in 2006 with a $1.5 million gift, later increased to $2.5 million. Jean Morrison, BU provost, and President Robert A. Brown select recipients from faculty who are holding their first professorship, have arrived within the last two years, and have been recommended by deans and department chairs.

"It is a privilege to witness the development of talented young scholars into outstanding teachers and researchers," says Morrison. "From the discovery of novel new cancer treatments and effective approaches to the HIV epidemic to improving conditions for an aging workforce, [the recipients] are fulfilling—and in many ways exceeding—the promise we saw in them when they joined the BU community. We are enormously proud of the important work they're performing and excited to help advance their research careers."

Flynn, who earned a doctoral degree in cancer biology from the University of Massachusetts Medical School, has been at BU since June 2013. She studies the role telomeres, repetitive DNA sequences that cap the ends of chromosomes, play in cancer development. Each time a cell divides, Flynn says, it loses a chunk of telomere instead of more essential genes further upstream. When telomeres get too short, cells either stop growing or die.

"That is the aging process," she says. But cancer cells have a way to "highjack this mechanism. When a telomere starts to get shorter, cancer outsmarts it" by reactivating the mechanism that keeps it growing forever.

Telomeres maintain their length using two pathways. Flynn's lab studies the pathway used by osteosarcoma and glioblastoma—rare and lethal cancers of the bone and brain—and hopes to identify novel treatments that would target this highjacked pathway to better manage the cancers.

So far, Flynn has seen promising results. One compound she's testing in vitro doesn't just stop cancer cells from growing, but completely obliterates them—and with minimal effects to surrounding healthy cells. The next step is to test the compound in mouse models.

"If it works as well as it does in a dish, it'll be amazing," she says.

Flynn will use the award to hire lab personnel and to buy reagents. "It's a tremendous opportunity to represent Peter Paul and have money to build my lab," she says, "but the real goal is to raise the bar, to elevate cancer research at BU."

From the Chair *(continued from page 1)*

Harlow, Jr., Ph.D. and **Robyn Karnauskas, Ph.D.** to the Board of Trustees. Ed is Professor of Biological Chemistry and Molecular Pharmacology at Harvard Medical School, while Robyn is a Biotechnology Analyst and Director at Deutsche Bank. We are sure that they will add new dimensions in thought and action for the Board to consider in the coming years. A very warm welcome to both of you!

Both of our 2013-2014 Research Fellows achieved high professional recognition this year for their research sponsored by the Foundation. **Fellow Hui Feng, Ph.D.** was awarded the Ralph Edwards Career Development Professorship, which recognizes outstanding junior faculty researchers, and she was asked to present her research findings at the national Zebrafish Disease Models Conference. **Fellow Nancy Cho, M.D.** was selected to present her work both at the American College of Surgeons conference and at the Academic Surgical Congress. Congratulations to both of our Research Fellows on these outstanding accomplishments!

Kudos also flow to **Trustee Douglas V. Faller**, **M.D., Ph.D**. the Karin Grunebaum Professor in Cancer Research at Boston University School of Medicine (BUSM) who was awarded the Marta Marx Award by the Scleroderma Foundation for receiving the top score of all proposals reviewed by the scientific peer-review committee.

We continue to be blessed with the stellar performances of our Fellows and Trustees. We appreciate the time and effort they dedicate to their research and to the Foundation.

Many thanks to all of you….
Steven Wallach, Chairperson

New Karin Grunebaum Foundation Trustee

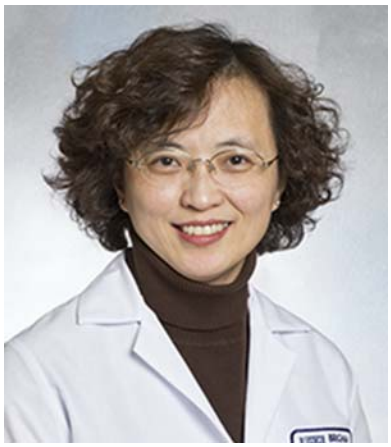
Ed Harlow, PhD
Ludwig Professor of Cancer Research and Teaching
Professor of Biological Chemistry and Molecular Pharmacology
Harvard Medical School

Dr. Harlow and his laboratory study the mechanisms that underlie the early stages of cancer development. He is best known for advances in our understanding of how cells determine when it is appropriate to divide. In 1988, his lab discovered how some viruses alter cell proliferation by using viral proteins to interact with and inactivate negative regulators of proliferation. Loss of these "brakes" on proliferation leads to cell division at inappropriate times. This model is widely applicable to cancer, and the discovery of how these viruses subvert cell regulation led to major advances in our knowledge of how cells control cell division. These contributions have been recognized by many awards including the Sloan Prize from the General Motors Research Foundation, Bristol-Myers Squibb Award for Distinguished Achievements in Cancer Research, and Medal of Honor from the American Cancer Society. Dr. Harlow was elected to the National Academy of Sciences in 1993 and the Institute of Medicine in 1999. He is currently a Professor of Biological Chemistry and Molecular Pharmacology at Harvard Medical School where he holds the Virginia and DK Ludwig Chair for Cancer Research and Teaching.

Dr. Harlow did his undergraduate training at the University of Oklahoma and received his Ph.D. from the Imperial Cancer Research Fund Laboratories (London, England) in 1982. Much of his early work on the function of viral proteins was performed while on staff at Cold Spring Harbor Laboratory from 1982 to 1990. In 1990 he moved to Boston to become the Scientific Director of the Massachusetts General Hospital Cancer Center. From 1995 until 1998 he led the planning efforts for the nation's cancer research efforts while an Associate Director at the National Cancer Institute, and he currently serves as a Senior Advisor to the Director of the National Cancer Institute. From 2009 to 2011, while on leave from Harvard, Dr. Harlow was the Chief Scientific Officer of Constellation Pharmaceuticals, a biotechnology company in Cambridge, MA, that is developing small molecule inhibitors for cancer therapeutics. He is also co-author of one the most widely used manuals for biology research, entitled *Antibodies, A Laboratory Manual,* and he has published extensively in distinguished peer-reviewed journals.

Karin Grunebaum Cancer Research Foundation Faculty Research Fellowship 2014
Christine G. Lian, MD

EPIGENETIC REGULATIONS AND MELANOMA



Our Dermatopathology Laboratory at Brigham and Women's Hospital focuses on making discoveries of new avenues for early diagnosis, prevention and therapeutic intervention for melanoma, as a prototype of aggressive human cancers. Melanoma is one of the very few human cancers with the steadily rising incidence worldwide. In the United States, melanoma is the fifth most common type of new cancer diagnosis in men and the seventh most common type in women. The National Cancer Institute estimates that in 2012 there will be 76,250 new cases and 9,180 deaths in the United States due to melanoma. Thickness of the primary tumor measuring by ruler remains the most important determinant of prognosis for localized disease, for which surgery remains the mainstay of therapy. Once melanoma metastasized, the prognosis remains dismal with a 7-month median survival and a 5-year survival rate of 15%. In addition, metastatic melanoma has been one of the most therapeutically challenging malignancies, with significant chemotherapy resistance. There is a pressing need for novel biomarkers focused on both genetic and epigenetic defects that will better define the malignant potential of primary lesions, predict clinical outcome and forecast therapeutic responses.

The initiation and progression of melanoma involves both genetic and epigenetic events, which are heritable changes in gene expression that are not due to any alteration in the DNA sequence. DNA methylation, histone modification and miRNA regulations are fundamental to the genesis of cancer (Figure 1). Among all the epigenetic mechanisms in cancer biology, DNA methylation, which occurs at the carbon-5 position of cytosine to form 5-methylcytosine (5-mC) is most well studies. However, the mechanisms underlying DNA demethylation are much less understood. A breakthrough discovery was made in 2009 and identified that the first and most critical step of this reaction involves oxidation of 5-mC to 5-hydroxymethylcytosine (5-hmC), which is performed by the Ten Eleven Translocase (TET) family dioxygenase enzymes (Figure 2). Understanding the precise cellular function of the TET family enzymes and the biologic significance of 5-hmC loss and dysregulated DNA demethylation is currently a high priority in cancer biology research. Our recent work demonstrates that global loss of 5-hydroxymethylcytosine (5-hmC) as an epigenetic hallmark in melanoma and reveals the very first genome-wide 5-hmC distribution in cancer (Cell, 2012). Most importantly, by re-introducing TET2 / isocitrate dehydrogenase 2 enzymatic activities and thus rebuild 5-hmC levels in melanoma cells, tumor growth is suppressed and tumor-free survival is enhanced in animal models. More studies by us and others further demonstrated the clinical implications of 5-hmC (Figure 3 and Summary). This landmark publication in epigenetic regulation of DNA

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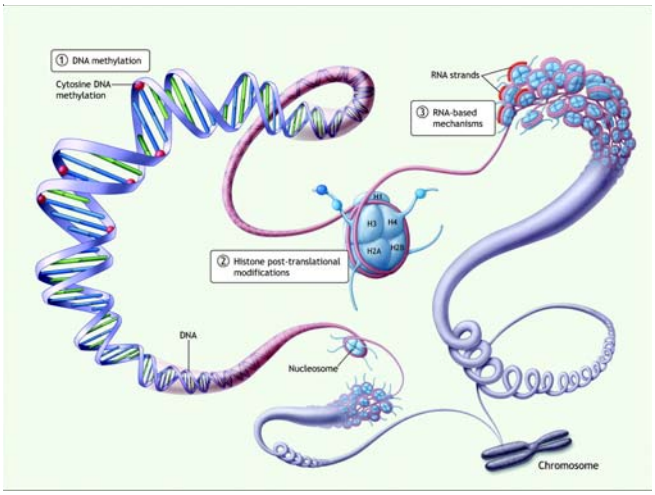

Figure 1. Summary of the three primary epigenetic mechanisms. (1) DNA methylation. (2) Histone post-translational modifications. (3) RNA-based mechanisms, including miRNAs and lncRNAs. Note: this diagram does not illustrate its mechanisms of binding and silencing mRNAs. From Matouk and Marsden (2008), reprinted with permission from Lippincott Williams & Wilkins.

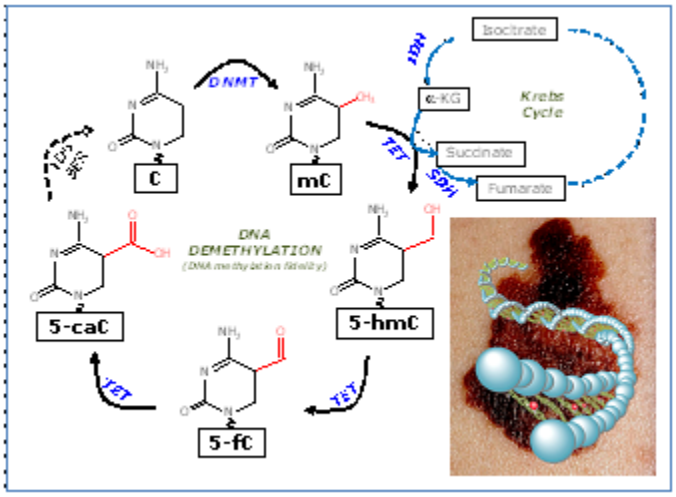

Figure 2. The pathway involved in the TET-dependent generation of 5-hmC, an epigenetic mark that is lost from the melanoma genome (pictorially rendered to lower right).

Epigenetic Regulations and Melanoma *(continued from previous page)*

hydroxymethylation received multiple positive reviews and comments, such as Dr Santiago Uribe-Lewis, an epigenetics expert based at the Cancer Research UK Cambridge Research Institute commented "Evidence is mounting that many different types of tumor, including brain and bowel cancers, have depleted levels of 5-hmC. This latest study is very interesting, as it shows that it's possible to replenish 5-hmC levels in melanoma cells, and that this can help to slow the growth of tumors in animal models. The challenge now is to translate this early-stage lab work into an effective treatment for people – this is no easy task, but this work is a step towards that goal." "This work is a prime example of how basic research on mechanisms of epigenetic regulation can yield clinically significant insights that hold great promise for diagnosing and treating cancer." said Anthony Carter, PhD, of the National Institutes of Health's National Institute of General Medical Sciences, which mainly funded the research. Subsequently, studies from us and others further demonstrate the clinical implications of 5-hmC with the potential to be the first epigenetic mark that can be used immunohistochemically for diagnosis and for evaluation of melanoma virulence.

With the support of Karin Grunebaum Cancer Research Foundation Faculty Research Fellowship, we will focus on the research project of targeting epigenetic aberrations of DNA hydroxymethylation in chemoresistant melanoma stem cells.

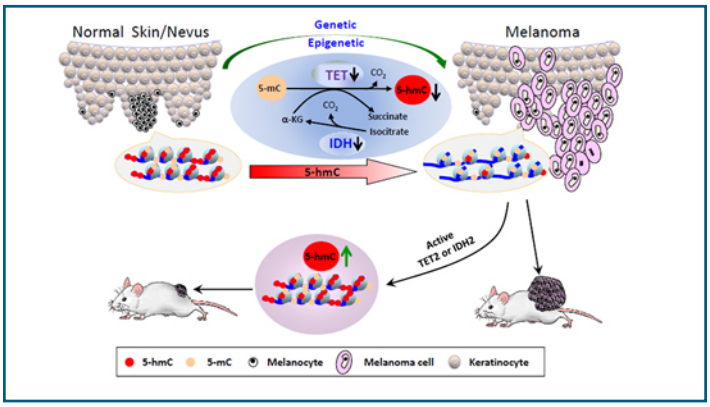

Figure 3. Loss of 5-hydroxymethylcytosine is an epigenetic hallmark in melanoma. Reprint from Cell.

With the support of Karin Grunebaum Cancer Research Foundation Faculty Research Fellowship, we will focus on the research project of targeting epigenetic aberrations of DNA hydroxymethylation in chemoresistant melanoma stem cells. The project will link the two most timely concepts of epigenetic regulation and cancer stem cells to develop novel strategies for melanoma treatment, with the ultimate goal to develop novel therapeutic strategies to sensitize chemotherapy-resistant melanoma by targeting melanoma stem cells via epigenetic modulation of the 5-hmC pathway. More importantly, given the reversible nature of epigenetic regulation, such epigenetic studies could lead to new strategies for cancer therapy. Moreover, new insights into the epigenetic landscape that will permit the use of more patient-specific therapeutic agents could provide important inroads into personalized medicine.

Summary

DNA methylation at the 5 position of cytosine (5-mC) is a key epigenetic mark that is critical for various biological and pathological processes. 5-mC can be converted to 5-hydroxymethylcytosine (5-hmC) by the ten-eleven translocation (TET) family of DNA hydroxylases. Here, we report that "loss of 5-hmC" is an epigenetic hallmark of melanoma, with diagnostic and prognostic implications. Genome-wide mapping of 5-hmC reveals loss of the 5-hmC landscape in the melanoma epigenome. We show that downregulation of isocitrate dehydrogenase 2 (IDH2) and TET family enzymes is likely one of the mechanisms underlying 5-hmC loss in melanoma. Rebuilding the 5-hmC landscape in melanoma cells by reintroducing active *TET2* or *IDH2* suppresses melanoma growth and increases tumor-free survival in animal models. Thus, our study reveals a critical function of 5-hmC in melanoma development and directly links the IDH and TET activity-dependent epigenetic pathway to 5-hmC-mediated suppression of melanoma progression, suggesting a new strategy for epigenetic cancer therapy.

http://www.cell.com/abstract/S0092-8674(12)01012-4

Karin Grunebaum Cancer Research Foundation Faculty Research Fellowship 2013 – 2014 Final Report

Nancy L. Cho, M.D. Brigham & Women's Hospital

The KGCRF Faculty Research Fellowship award has been an invaluable source of support over the past year and has enabled me to focus on understanding mechanisms of desmoid tumorigenesis in order to develop targeted therapies for this disease. Following is a report of the laboratory's accomplishments during the grant period.

Mesenchymal Stem Cells (MSCs) and Desmoid Tumorigenesis

Aim 1: To demonstrate that bone marrow-derived MSCs play a key role in DT etiology. In the past year, we have made excellent progress with this aim including the following accomplishments:

- Performed immunohistochemistry on over 30 DTs and showed that all tumors expressed stem cell markers, whereas matching normal stromal tissues were uniformly negative.
- Established 15 different desmoid-derived mesenchymal cell lines from individual patients with both sporadic and genetically inherited forms of desmoid tumors.
- Performed subcutaneous implantations using MSC lines in xenograft models to investigate whether MSCs are the progenitor cells of DTs.
- Initiated generation of GFP+Apc1638N mice and are currently genotyping pups for experiments.
- In future experiments, we will perform bone marrow transplantations using GFP+Apc1638N donors and Apc1638N and WT recipients to determine whether bone marrow-derived MSCs are recruited to sites of DT formation. We will also irradiate the bone marrow of these mice and transplant them with WT bone marrow to determine whether this process alters DT formation in these mice.

Aim 2: To induce terminal differentation in desmoid-derived MSCs with dysregulated Wnt signaling. Using our DT-derived cell lines, we have made significant progress in targeting MSCs with pharmacological agents that inhibit oncogenic pathways involved in desmoid tumorigenesis:

- Performed in vitro experiments investigating mechanisms of sorafenib activity in DTs. Preliminary results show that sorafenib significantly decreases proliferation and invasion in DT-derived cells relative to control fibroblasts.
- Investigated downstream signaling effects of sorafenib, suggesting that antitumor activity is mediated via ERK, Akt, and mTOR pathways.
- Performed immunohistochemistry/ELISA demonstrating that hyaluronic acid (HA) expression is increased in DTs and DT-derived cell lines relative to controls.
- Demonstrated that treatment with the HA synthesis inhibitor 4-Methylumbelliferone (4-MU) decreased proliferation in DT-derived cell lines relative to controls. 4-MU also inhibited overall expression of HA as well as HAS2, a HA synthesizing protein, in cells.
- In future experiments, we will examine the synergistic effects of sorafenib with mTOR, Hedgehog, and HA inhibitors. Results from in vitro experiments will be validated using the Apc1638N mouse model to study tumor formation and response to drug treatment. These studies will be of particular relevance in understanding mechanisms of resistance that inevitably occur in many patients following drug therapy.

Abstracts/Presentations/Manuscripts

As a result of the Fellowship, our laboratory has presented our work at national meetings, received prestigious recognition, and given invited lectures. We also have several manuscripts in preparation based upon experiments performed during the award period.

1. L. Rosenberg, M. Bertagnolli, N. Cho. Targeting mesenchymal stem cells in desmoid tumors. 2013. Poster presentation to the Harvard Surgery Research Day Symposium, Boston, MA.
2. N. Cho. "Mesenchymal stem cells and desmoid tumorigenesis." 2013. Oral presentation to the Karin Grunebaum Cancer Research Foundation, Boston University Medical Center, Boston, MA.
3. N. Cho. "Targeting Mesenchymal Stem Cells in Desmoid Tumorigenesis." 2013. Oral presentation at the American College of Surgeons Annual Meeting, Washington, D.C.
4. L. Rosenberg, M. Bertagnolli, N. Cho. Sorafenib suppresses desmoid tumor growth and invasion via inhibition of ERK signaling. 2013. Poster presentation to the Massachusetts Chapter of the American College of Surgeons, Boston, MA. Commission on Cancer Award.
5. L. Rosenberg, M. Bertagnolli, N. Cho. Sorafenib suppresses desmoid tumor growth and invasion via inhibition of ERK signaling. 2014. Oral presentation to the Academic Surgical Congress, San Diego, CA.
6. L. Rosenberg, C. Yoon, M. Bertagnolli, N. Cho. Sorafenib suppresses desmoid tumor growth and invasion via inhibition of ERK signaling. 2014. Poster presentation to the American Association for Cancer Research, San Diego, CA.
7. L. Rosenberg, M. Bertagnolli, N. Cho. Sorafenib suppresses translation in desmoid tumors via inhibition of mTOR signaling. 2014. Poster presentation to the Harvard Surgery Research Day Symposium, Boston, MA.
8. L. Rosenberg, C. Yoon, M. Bertagnolli, N. Cho. Sorafenib suppresses translation in desmoid tumors via inhibition of mTOR signaling. 2014. Abstract accepted for oral presentation to the American College of Surgeons Annual Meeting, San Francisco, CA.
9. A. Briggs, L. Rosenberg, J. Buie, H. Rizvi, A. Carothers, M. Bertagnolli, N. Cho. Targeting hyaluronic acid and CD44+ mesenchymal stromal cell interactions in desmoid tumors. Manuscript in preparation.
10. L. Rosenberg, C. Yoon, M. Bertagnolli, N. Cho. Antitumor activity of sorafenib in desmoid-derived mesenchymal cells. Manuscript in preparation.

Your Support is Vital to our Mission

The KGCRF relies solely on private donations. In order to continue the fight we ask for your support and hope that you will give what you can.

Your tax-deductible contribution will directly help fund the cancer research effort, since all our Officers and Trustees are unpaid volunteers, and the Foundation has no paid employees.

I enclose my gift of:

❑ $50 ❑ $100

❑ $150 Other $ _____

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Street Address: _____

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Email: _____

Please return to: KGCRF, 85 Sherman Street, # 8, Cambridge, MA 02140

You may also donate online at:

http://www.grunebaumfoundation.org/html/SupportContributions.asp

The Foundation's Mission and its chosen path to Mission Accomplishment.....

Because Karin Grunebaum died at age 39 from an unknown primary site malignancy, the overriding objective of the Karin Grunebaum Cancer Research Foundation is the eradication of all types of cancer. The Foundation's original Declaration of Trust, written in 1958, mandates that the Foundation's funds be exclusively used for "...aiding research in and study of the cause, treatment and cure of cancer."

The Foundation's Trustees firmly believe that the eradication of cancer will only occur through successful research accomplishments which are followed by successful practical/commercial application. Thus, the Foundation has chosen to invest its funds directly in dedicated cancer researchers in hope of helping them achieve significant accomplishments to eliminate all types of carcinomas and thereby eradicate each and every type of cancer.

KARIN GRUNEBAUM
cancer research foundation