KARIN GRUNEBAUM
cancer research foundation

November, 2013 • Volume 10

Founded in 1958

From the Chair

Dear Friends of the Karin Grunebaum Cancer Research Foundation:

A sad event this year made me think that our readers might want to become better acquainted with our world-renowned group of medically trained Trustees who help guide the Foundation. I am very proud of having them as Trustees, (and friends) and cannot thank them enough for their incredible knowledge, talent and time which they freely donate to the Foundation.

The precipitous event was the loss of our dear friend (and member of the Board of Trustees since 1961), **Merrill Feldman, M.D., D.M.D.**, to cancer. Dr. Feldman graduated undergraduate, medical and dental school from Harvard, and was Chairman Emeritus of Boston University School of Medicine's Radiation Department – making him ideally situated to help establish the long relationships between Harvard Medical School, Boston University School of Medicine and the Foundation. His wit and wisdom at Board meetings (especially his incisive financial wisdom and guidance) will be sorely missed.

Karen Antman, M.D. is the Provost of the Medical College and Dean of the Boston University School of Medicine. A renowned cancer researcher, she came to her present position after being Deputy Director for Translational and Clinical Sciences at the National Cancer Institute of the National Institutes of Health (NIH), and before that was Wu Professor of Medicine and Pharmacology and Director of the Herbert Irving Comprehensive Cancer Center at her *alma mate*r, Columbia University College of Physicians and Surgeons, where she co-directed the cancer care service line at New York Presbyterian Hospital.

Michael Droller, M.D. graduated from Harvard Medical School, where he was the first Karin Grunebaum Fellow. From that time onward, he rose to be Chairman Emeritus of the Mount Sinai Medical Center's Department of Urology – holding Professorships in both Urology and Oncology.

Douglas Faller, M.D., Ph.D. is the first (and current) Karin Grunebaum Professor in Cancer Research and Director of the Cancer Center at Boston University School of Medicine. With his medical degree from Harvard Medical School and his Ph.D. from MIT, he has been appointed Professor of Medicine, Pediatrics, Biochemistry, Microbiology, Pathology and Laboratory Medicine, plus Vice-Chairman, Division of Medicine at Boston University School of Medicine.

Michael Gimbrone, Jr., M.D. became the second Karin Grunebaum Fellow, having graduated from Harvard Medical School. Dr. Gimbrone is Chairman Emeritus of the Department of Pathology and serves as Professor of Pathology and Director of the Center for Excellence at Brigham and Women's Hospital.. Among his awards, is being a co-recipient (with Dr. Judah Folkman) of the J. Allyn Taylor International Prize in Medicine, recognizing his contributions to the establishment of the field of Vascular Biology.

(continued on page 7)

From the Fellows

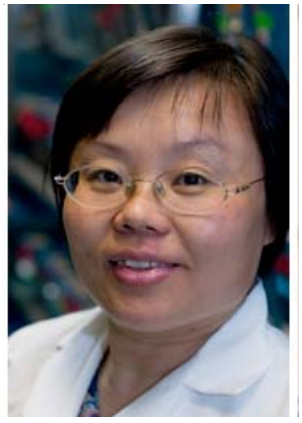
Boston University School of Medicine

The Laboratory of Zebrafish Genetics and Cancer Therapeutics

TARGETING VULNERABILITIES OF MYC-RELATED CANCERS

Cancer takes the lives of millions each year and over 90% of cancer-related deaths result from tumor metastasis. Therefore; there is an urgent need to develop novel strategies that can block tumor metastasis and kill cancer cells efficiently and specifically. *MYC*, aptly referred to by Gerard Evan as the oncogene (cancer-causing gene) from hell, is aberrantly activated in nearly all human cancers, including leukemias, lymphomas, neuroblastoma and carcinomas. Cancers with aberrant *MYC* activity are often aggressive, rapidly spreading to distant tissues. These facts underscore the need to improve understanding of the regulatory molecules involved with MYC-induced tumor initiation and progression, thus allowing the researchers to discover and exploit the vulnerabilities of the cancers, in order to develop novel and effective drugs targeting MYC-related cancers.

The zebrafish offers many unique advantages as a cancer model: easy monitoring of tumor development *in vivo*, due to its transparency and the ability to differentially fluorochrome-label tumor cells and vasculature; a high degree of genetic similarities with humans; simple techniques for functional studies of candidate genes identified through ongoing human genomic analysis; and the feasibility of conducting genetic and chemical screens to dissect the molecular pathways of tumorigenesis and identify promising lead compounds. The research in Feng laboratory uses the zebrafish model in combination with human cell line and patient sample analyses, (i.) to discover molecules needed for MYC-induced tumor initiation and progression and (ii.) to evaluate their potential as therapeutic targets.



Hui Feng, M.D., Ph.D.
KGCRF Fellow
2013-2014

One major project in Feng laboratory is to dissect molecular pathways that regulate the initial step of tumor metastasis - *intravasation* - the entry of tumor cells into vascular and/or lymphatic vessels. Improved understanding about tumor cell intravasation is the key for future therapeutic developments to block the spread of tumor cells from their primary site. Through interrogating human cancer genomic information with the imaging and genetic capacities of the zebrafish system, researchers in Feng laboratory focuses on identifying novel "intravasation genes" that promote the intravasation of *MYC*-driven tumor cells. Using this combined approach, the Feng laboratory has successfully identified multiple promising genes. Genes identified through these studies should represent promising targets for therapeutic intervention to block MYC-associated metastasis. As the next step of the research, their therapeutic potential will be evaluated using human cancer cell lines.

The second major project in Feng laboratory is to exploit the genetic capacity of the zebrafish system to identify genes that, when inactivated, can inhibit or delay tumor development. Researchers in Feng laboratory have identified a gene critical for energy production and macromolecule synthesis - dihydrolipoamide succinyltransferase (*DLST*) - ~50% reduction of DLST delayed Myc-induced T-cell leukemia but did not influence fish development. This finding is consistent with MYC's important roles in cancer metabolism. In the short-term, researchers in Feng laboratory will focus on identifying and characterizing genes and pathways important for the initiation, maintenance, and/or progression of MYC-induced T-cell lymphoma, neuroblastoma and breast cancer, and testing the potential of these genes or related pathway regulators as novel therapeutic targets to treat these diseases. In the long-term, researchers in Feng laboratory will extend these

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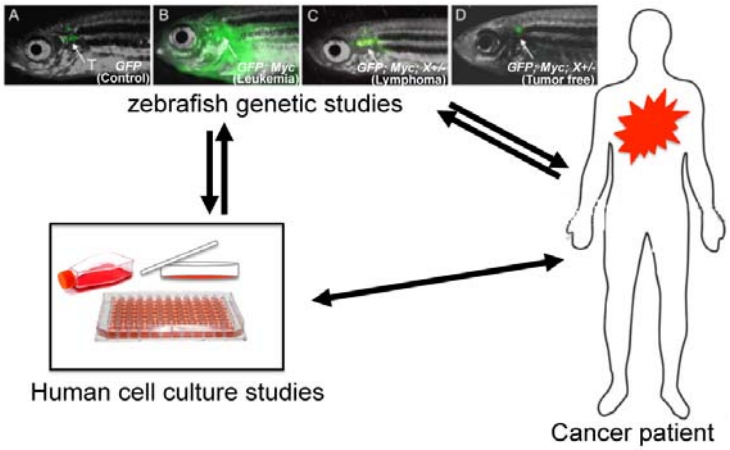
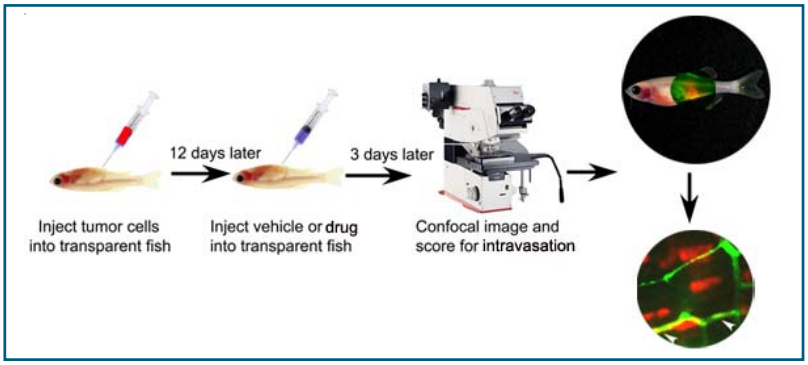


Research strategy used by Feng Laboratory to identify effective therapeutics:
The research strategy is to combine the zebrafish genetic studies with the analyses of primary patient samples and human cell culture system. Zebrafish genetic studies will identify novel target genes that, when inactivated, can delay Myc-induced tumorigenesis and/or disease progression. The human relevance and therapeutic potential of these genes will be validated using human cell culture system. Alternatively, candidate target genes will be discovered by analyzing primary patient samples and then validated through zebrafish genetic studies and human cell culture system.

Boston University *(continued from previous page)*

studies to include other MYC-dependent cancers.

The Feng laboratory possesses the expertise to conduct the research described above. Dr. Feng is an expert in using zebrafish to study human cancers and has made seminal contributions to the understanding of human T-lymphoma/leukemia and neuroblastoma. Her research led to over 12 publications in top-tier journals, such as *Nature Cell Biology, Nature, Cancer Cell,* etc. In addition, Dr. Feng has established collaborations with multiple investigators (Drs. Travis Denton, Guanglan Zhang, Rebecca Chin, Sam Thiagalingam, and Anurag Singh), who will provide additional expertise in medicinal chemistry, bioinformatics, 3-dimensional cell culture, and functional genomic analysis.



Research procedures to study intravasation:
Tumor cells labeled with red-fluorescence are injected into transparent fish that express green fluorescent protein in the vasculature. After tumor transplantation is established in the fish, small molecules (drug to be tested) or a vehicle control will be injected into the tumor site. The fish will then be examined with a high-magnification microscope to visualize and quantify the ability of a drug to block intravasation of tumor cells.

The financial support from the Karin Grunebaum Cancer Research Foundation will help researchers conduct the above research to find therapeutics that are more specific and less toxic to patients with cancer.

More about Dr. Feng and Her Work

Hui Feng spends a lot of time staring through zebra fish. Through because these vertebrates, which have a great deal of genetics in common with humans, are transparent. In fact, one particular breed, called Casper—after the Friendly Ghost—is so phantasmal that Feng says that "you can read newspapers through this fish."

Feng doesn't read the news through them, though. The School of Medicine assistant professor of pharmacology and medicine is more interested in tracking the pathways of dyed tumor cells as they metastasize through the zebra fish's vasculature, which is tinted a contrasting color. In the less than two years since her tank-filled lab opened, she has identified genes that, when blocked with targeted treatments, could prevent the metastasis of certain types of cancer, like the most stubborn forms of leukemia.

In recognition of her groundbreaking work, Feng was awarded the Ralph Edwards Career Development Professorship, which recognizes MED researchers. The award was made possible this year by the estate of obstetrician and gynecologist Ralph Edwards (MED'52).

Feng, director of the Laboratory of Zebrafish Genetics & Cancer Therapeutics, says the honor reminds her that University officials appreciate faculty research and they want to support it. "It's not just about the money," she says. "The spiritual or mental support really means so much to us."

Karen Antman, MED dean and Medical Campus provost, recalls the researcher's discoveries early in her career, which found their way to top-tier research journals, including *Nature*, *Cell Biology*, *Cancer Cell*, the *Journal of Experimental Medicine*, and *PNAS*. A graduate of Beijing Medical University, Feng completed a master's in cardiovascular pharmacology at Peking Union Medical College and a doctorate in cellular biology at the University of Georgia.

"Since joining the School of Medicine faculty," Antman says, "Dr. Feng has demonstrated an exceptional level of scholarship, mentorship, teaching, and collegiality and quickly established herself as an independent research scientist, effectively and efficiently setting up a robust research program."

Feng is one of three assistant professors who were given career development awards, which recognize junior faculty who have been at the University for less than two years and have held no prior professorships. Cornel Ban, a College of Arts & Sciences assistant professor of international relations, received the inaugural Stuart and Elizabeth Pratt Career Development Professorship, dedicated to CAS scholars. And Nachiketa Sahoo, a School of Management assistant professor of information systems, was awarded the Reidy Family Career Development Professorship, which has recognized faculty members in SMG and the College of Engineering in alternating years since 2010.

Excepted from original article authored by Leslie Friday and posted on BU Today 13 September 2013.

Boston University School of Medicine

In B-cell chronic lymphocytic leukemia (B-CLL) deletion of the 17p13 chromosomal region, encompassing the *GPS2* (G-protein suppressor 2) locus, is often associated with a particularly poor prognosis and with refractoriness to conventional therapies. Based on the observation that GPS2 expression has been reported as significantly downregulated in patients carrying the 17p deletion, we have hypothesized that GPS2 may play a significant role in the pathogenesis of B-CLL. Thus, we proposed to investigate GPS2 role as a tumor suppressor gene and to address whether GPS2 presence in B cells may be required to regulate the balance between pro-survival and pro-apoptotic responses.

Chronic inflammation triggered by infections is often involved in the initiation and progression of several chronic lymphoid malignancies of B-cell type and Toll like receptors (TLRs) have been show to play a key role in the antigen recognition process (Bertilaccio et al., 2011; Booth et al., 2011; Crampton et al., 2010; Lanzavecchia and Sallusto, 2007; Lau et al., 2005; Muzio et al., 2009). Because our preliminary data suggested that GPS2 might be able of regulate TLRs signaling, we proposed to focus on investigating whether GPS2 inhibitory activity may be relevant for Ubc13-dependent TLR signaling in primary mouse macrophages and primary mouse B-cells. Characterization of GPS2 role in TLR response was first investigated in bone marrow-derived macrophages by using specific ligands for each receptor, namely LPS (to investigate TLR4 response), Pam3CSK4 (to investigate TLR1/2 response) and CpG (to activate TLR7/9)(Dunne and O'Neill, 2003; Saitoh and Miyake, 2009). Macrophages from wild type mice were compared to macrophages from aP2-GPS2 transgenic mice that overexpress GPS2 by gene expression profiling. Preliminary results confirm the hypothesis that TLR signaling is impaired by GPS2 overexpression. We are currently performing similar experiments in B cells prior to extending these studies genome wide by performing RNA-seq (high-throughput sequencing of the B-cell transcriptome) in primary murine B-cells to investigate the effect of GPS2 overexpression or downregulation on the B Cell transcriptome.

Valentina Perissi
KGCRF Fellow
2012-2013

Our long-term goal is testing the general hypothesis that absence of GPS2 causes constitutive activation of the NF?B signaling pathway and resistance to apoptosis in a model mimicking the loss of GPS2 in human B-CLL cells. To this end, we have generated a B-cell specific null animal model by crossing $GPS2^{fl}$ conditional knock out mice with the $CD19^{+}$-Cre mice (Jackson Laboratories). This is a powerful tool that will allow investigating *in vivo* GPS2 potential role as a tumor suppressor gene. Preliminary results have confirmed GPS2 specific deletion in the B cells compartment and seem to indicate that cytokines production (measured by looking at mRNA expression by qPCR) in response to TLR response is impaired. Further experiments will be required to determine the lymphocytes immunophenotypic profile and to investigate whether these mice can recapitulate the leukemic phenotype.

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Mesenchymal Stem Cells (MSCs) and Desmoid Tumorigenesis

Karin Grunebaum Cancer Research Foundation Faculty Research Fellowship

Desmoid tumors (DTs), or aggressive fibromatosis, are rare, mesenchymal tumors that can occur sporadically or in the context of the heritable cancer syndrome, familial adenomatous polyposis (FAP) **(Figure 1)**. Although rare in the general population, DTs are infiltrative tumors that can produce disfigurement, functional deficits, and are a leading cause of death in FAP patients. Treatment often involves surgical excision, which is associated with high recurrent rates. Little is known about the molecular biology driving desmoid tumorigenesis, and **effective systemic therapy for DTs remains elusive**. Our laboratory seeks to characterize the biological requirements necessary for DT formation and identify novel targeted therapies for this disease.

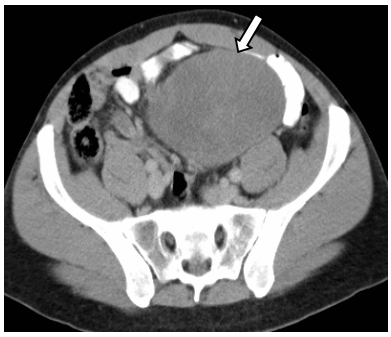


Figure 1. Intra-abdominal desmoid tumor.

Recently, a growing body of literature has focused on the role of mesenchymal stem cells (MSCs) in contributing to the development of pediatric and adult mesenchymal tumors. We have shown that MSCs are abundant constitutents of DTs and may play a critical role in DT formation and maintenance **(Figure 2).** Our study was the first to isolate and characterize desmoid-derived MSCs from a human patient. We are currently expanding several MSC lines obtained from patients with FAP or sporadic tumors. To our knowledge, no other groups have been successful in establishing and maintaining MSCs obtained from DTs under these conditions, which is a significant limitation in the advancement of this field. These cell lines will be an indispensable tool for accelerating preclinical drug testing and characterizing genetic variations between heritable and sporadic cases.

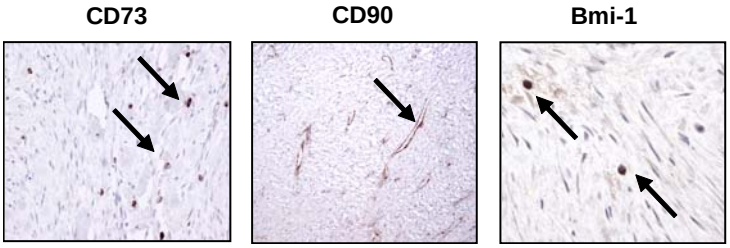


Figure 2. Representative staining of stem cell marker expression in DTs. Sixteen archived DT samples were stained for the stem cell markers CD73, CD90, and Bmi-1 demonstrating the presence of MSCs (black arrows) within the tumors. Original magnification was 10X.

Whereas conventional therapies may successfully treat differentiated fibroblasts that comprise the tumor bulk, resistant cancer stem cells can persist and provide the basis for tumor initiation, progression, and relapse. Alternate therapies are needed that not only target differentiated cells but also cancer stem cells, resulting in a more durable and complete response. During the Karin Grunebaum Fellowship, our laboratory will test the hypothesis that **MSCs play a critical role in desmoid tumorigenesis and provide a novel therapeutic target for patients with unresectable disease.** We will study the role of bone marrow-derived MSCs in DT etiology using the *Apc*^{1638N} mice as an animal model of human DTs. We will also target desmoid-derived MSCs for terminal differentiation with pharmacological agents that inhibit the Wnt pathway as a novel therapeutic strategy. These experiments will address major gaps in our current understanding of desmoid pathophysiology and the role of MSCs in driving tumorigenesis. Our results will provide key mechanistic insights into tumor-stroma interactions and identify novel targets for therapy in patients with DTs and Wnt-dependent cancers.

Nancy Cho
KGCRF Fellow
2013-2014

As a clinical referral center for patients with desmoid tumors, we are uniquely situated to study these rare tumors and have access to human tissue that will enable us to uncover the cellular requirements necessary for DT formation. We also have the ability to expeditiously translate benchside discoveries to clinical trials, which would have a significant impact on patients afflicted with these life-threatening tumors. The data generated from our aims will enhance our understanding of how MSC deregulation stimulates desmoid tumorigenesis and other Wnt-related cancers and will be a novel contribution to the field. As such, the overall biological and clinical impact of this work is very high.

Nancy Cho delivered a presentation on her research at the American College of Surgeons in October, 2013 and has been invited to present an abstract at the 2014 Academic Surgical Congress.

More about the Harvard Medical School Program

The Cancer Biology Area of Concentration within the Biological and Biomedical Sciences Graduate Program at Harvard Medical School offers innovative training opportunities to a growing number of Harvard graduate students. As a direct result of generous sponsorship by the Karin Grunebaum Cancer Research Foundation, the Cancer Biology Area of Concentration has experienced a period of remarkable growth. KGCRF funding has allowed the concentration to offer stronger and more varied pedagogical training to our predoctoral scholars, and to foster an integrated community of Cancer Biology faculty and trainees. We recently commenced another year under the strong leadership of Professors Karen Cichowski (BWH), Andrea McClatchey (MGH), Rosalind Segal (Dana-Farber Cancer Institute), and Curriculum Fellow Megan Mittelstadt (HMS).

On September 25, 2013 the Cancer Biology Area of Concentration faculty and trainees welcomed 25 new graduate students to Cancer Biology at our annual Fall Welcome event. At this event, new students learn about our curricular and paracurricular training opportunities, and actively network with the faculty and peers with whom they could potentially train. The Cancer Biology Area of Concentration community has grown rapidly to more than 80 graduate students, through partnership with a dedicated and proactive student steering committee.

Cancer Biology trainees also regularly attend the Seminars in Oncology at the Dana-Farber Harvard Cancer Center. The seminar speakers for this series are invited, nationally recognized cancer biology researchers. With support from KGCRF, the graduate students are able to partake in a Graduate Student Lunch Series with these speakers. Each of these educational events allows 8-10 students the opportunity for dynamic intellectual interactions with each DFCI Seminar speaker. This casual forum also allows our students to receive external feedback and advice on various aspects of their graduate training and careers. Through this series, students begin thinking about future research projects and long-term career goals as they hear about the latest research in cancer biology. Third year PhD student Ilana Kelsey "cannot emphasize enough how important the networking opportunities have been at these events for forming new collaborations and gaining insight into others' research."

Thanks to the Karin Grunebaum Cancer Research Foundation's broad-reaching vision and deep, ongoing commitment to the education of new generations of cancer researchers, we are able to support pedagogical training and intellectual community for an increasing number of Cancer Biology graduate students.

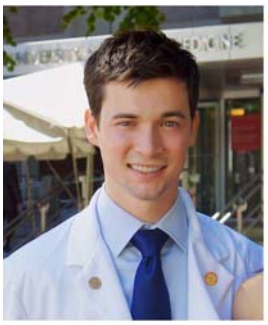
Funds from KGCRF have also been instrumental in propelling the Cancer Biology Student Data Club. This monthly series provides an opportunity for students, at all stages of their graduate work, to present their research to their peers. In this forum, junior students gain practice giving a presentation, brainstorm their research ideas, and receive candid advice from senior students. We recently expanded this training initiative with the addition of a trainee-led "Current Topics in Cancer Research" journal club. This journal club provides a venue for senior graduate students to meet in a round-table format to discuss leading and cutting edge technologies in cancer biology research published in recent high profile papers.

Perhaps the most exciting and innovative training opportunity for Cancer Biology graduate students is "The Epidemiology and Molecular Pathology of Cancer," a hands-on, two-week intensive skill-building experience, which HMS is able to offer thanks to funding from the KGCRF. In this course, our students study multiple types of cancer and begin to master essential molecular pathology techniques, state-of-the-art image analysis of human biomarkers, tissue processing, and tumor histology. This course is one of our most popular resources, as the faculty members who design and deliver the curriculum are leaders in the fields of cancer pathology and epidemiology.

Thanks to the Karin Grunebaum Cancer Research Foundation's broad-reaching vision and deep, ongoing commitment to the education of new generations of cancer researchers, we are able to support pedagogical training and intellectual community for an increasing number of Cancer Biology graduate students. Perhaps one of our trainees, Cancer Biology MD-PhD student Jennifer Yeh, says it best: "I enjoy being part of the Cancer Biology Area of Concentration for the opportunity to meet other students passionate about cancer research and because it provides a supportive graduate student community."

As we look to the future, and the expansion of Cancer Biology training across the entire Harvard University landscape, sustained funding from KGCRF will ensure that a vibrant community of scholars is ready to tackle future challenges in this important field of research.

Thank you to the Foundation



Dear Mr. Wallach,

I would like to extend my sincerest thanks to you and the Karin Grunebaum Cancer Research Foundation for your support of my research experience this past summer.

My project focused on the epigenetic silencing of the tumor suppressor, ARHI, in breast and ovarian cancer. I investigated the ability of the new class of epigenetic anticancer drugs, histone deacetylase inhibitors, to re-express this tumor suppressor and induce apoptosis in cancer cells. Specifically, I developed an assay to determine the location within the ARHI gene at which histone deacetylases bind and negatively regulate transcription. This work is valuable in the context of the current dearth of understanding regarding the mechanisms of epigenetic therapies, and will hopefully help make these therapies more effective and eventually more widely available to patients in need.

As a medical student, this summer research experience was not only immensely intellectually challenging and satisfying, but also incredibly valuable for my professional future. This work has enhanced my understanding of cancer biology and afforded me the opportunity to contribute to the field in which I one day may practice as a physician. As I move forward in my career and begin to use this experience in the service of patients, please know that I will be continually grateful for the generosity and support of this foundation.

With many thanks and best regards,
Garrick Horn
BUSM Class of 2016

Dear Mr. Wallach,

Thank you for funding my summer research project at Boston University School of Medicine. My project analyzed the impact of demographic factors such as race, marital status, income, insurance, primary language, etc. on mortality rates in prostate cancer patients at Boston Medical Center. The results have been interesting and should contribute to greater understanding disparities in cancer patient outcomes. This project has helped me to lay the groundwork in further assessing the disparities by looking at differences in treatment choice and delays across the groups.

I will never forget the value of this experience and the role that the Karin Grunebaum Cancer Research Foundation played in it.

I am now a second-year medical student and am currently working towards a career in radiation oncology. I am hoping to present and publish this project and a few others in nationally-known medical journals. Currently, I am a chair of the Radiology Interest Group, a co-founder of the disabilities group, a Human Behavior in Medicine tutor, and a researcher for the radiation oncology department at BUSM in my spare time.

I am truly grateful for the opportunity that you gave me to remain in Boston and further my medical research this past summer. I will never forget the value of this experience and the role that the Karin Grunebaum Cancer Research Foundation played in it.

Sincerely,
Alexander Rand
BUSM Class of 2016

From the Chair *(continued from page 1)*

David Golan, M.D., Ph.D. is the Dean of Graduate Education at Harvard Medical School, which continues the Foundation's more than 50-year tradition of having a Dean of Harvard Medical School on the Board of Trustees. Dr. Golan received his Ph.D. in molecular biophysics and biochemistry and his M.D. degree from Yale University. He has been appointed Professor in the Harvard Medical School Department of Biological Chemistry and Molecular Pharmacology.

Frank Hsu, M.D. is the third former Karin Grunebaum Fellow currently on the Board of Trustees. After graduating from Harvard Medical School, he served as Assistant Professor (Oncology) at Yale Medical School, and then worked in the pharmaceutical industry as Senior Medical Director (Oncology) at Genzyme, and currently as Chief Medical Officer at Zyngenia, Inc.

Edward ("Ed") Harlow, Jr., Ph.D. received his Ph.D. from Kings' College in London through the Imperial Cancer Research Fund Laboratories. He has served as Chair of the Harvard Medical School Department of Biological Chemistry and Molecular Pharmacology, and currently is the Virginia and D.K. Ludwig Professor of Cancer Research and Teaching at Harvard Medical School as well as being Senior Advisor to the Director, National Cancer Institute.

Robyn Karnauskas, Ph.D. earned her Ph.D. from the University of Chicago in the field of cancer biology. She has focused her career on evaluating the efficacy of existing and proposed treatments for cancer and is currently the Lead Biotechnolgy Analyst and Director for Deutsche Bank.

A mighty impressive group….! Thanks to each of you.

Steven Wallach
Chairperson
www.grunebaumfoundation.org

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Thank you for your generous part in the Karin Grunebaum Cancer Research Foundation program.

We are committed to continue the fight against this terrible disease.

KARIN GRUNEBAUM
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