Founded in 1958

KARIN GRUNEBAUM
cancer research foundation

January 2026 - Volume 22

From the Chair

Dear Friends of the Karin Grunebaum Cancer Research Foundation:

Time marches on….

As some of you may know, the Foundation has been in existence for 66 years. During that extended time period, we have been blessed to be guided by a Board of Trustees comprised both of highly respected cancer-related medical and research professionals on the faculties of Harvard Medical School and Boston University School of Medicine, as well as a number of Karin's descendants (myself included, as her oldest child). In fact, after founder Fritz Grunebaum passed away in 1992, leadership of the Foundation passed to Daniel Federman, M.D. – Harvard's Dean for Medical Education. Karen Antman, M.D., a renowned cancer researcher and Boston University's Provost and Dean of the Medical College also served as an active member of the Board of Trustees for many years.

I have often said that our small family foundation's greatest asset is the recognition and imprimatur of serious achievement that our highly respected professional medical/research Trustees endow to the Foundation. So, it is with heartfelt reverence and regret that we recently accepted the resignations of three of our professional Trustees: Michael Droller, M.D.; Michael Gimbrone, Jr., M.D.; and Robyn Karanauskas, Ph.D. Each, in their own way, contributed significantly to the Foundation's long-term success.

Michael Droller, as a third-year medical student at Harvard Medical School, was the first recipient of a Karin Grunebaum cancer research grant in 1966. He went on to become the Katherine and Clifford Goldsmith Professor of Oncology, and Chairman Emeritus of the Department of Urology at The Mount Sinai Medical Center in New York.

In 1967, Michael Gimbrone was ironically the second recipient of a Karin Grunebaum cancer research grant. He was also a third-year medical student at Harvard Medical School, and became the Elsie T. Friedman Professor of Pathology at Harvard Medical School, and Director of the Center for Vascular Excellence at Brigham and Women's Hospital in Boston.

Robyn Karnauskas, Ph.D., has spent her professional life as a well-respected biotechnology analyst on Wall Street, specializing in analyzing new cancer drugs and treatments. She has worked at Deutsche Bank, and most recently as Managing Director at Truist Securities.

We will surely miss these long-serving Trustees, and we thank them for their many contributions to the Foundation.

On the much brighter side, we welcome our newest Trustees: Hee-Young Park, Ph.D. and Elisabeth Battinelli, M.D., Ph.D., who were both unanimously elected at the Foundation's annual meeting in October 2025.

Hee-Young Park is the Dean *ad interim* at Boston University Chobanian & Avedisian School of Medicine (BUCASM); the Carolann S. Najarian, MD Professor and Chair of Medical Sciences and Education; and Professor of Dermatology. Her election continues the Foundation's tradition of having Dean-level representation from both funded institutions (Trustee David Golan, M.D., Ph.D., is Harvard's Medical School Dean for Basic Science and Graduate Education). In a staggering coincidence, Hee-Young was mentored years ago by Michael Gimbrone!

Elisabeth "Beth" Battinelli is Chief, Mass General Brigham (MGB) Division of Classical Hematology; Clinical Director MGB Special Coagulation Laboratory; and an Associate Professor at Harvard Medical School. Beth continues the tradition of elevating former Karin Grunebaum Fellows to Trusteeship, as she was a selected Karin Grunebaum Fellow in 2012.

And so, the 66-year-old Foundation renews itself with these stellar additions to our Board. We look forward to many new and exciting achievements going forward.

Welcome aboard!

Steven

From Boston University Medical School

Julie Palmer, M.P.H., ScD
Karin Grunebaum Professor in Cancer Research, Boston University Chobanian and Avedisian School of Medicine, Director Slone Epidemiology Center; Co-Director, Boston University-Boston Medical Cancer Center

Dr. Julie Palmer, the Karin Grunebaum Professor in Cancer Research at Boston University Chobanian & Avedisian School of Medicine, recently had her work published in Environmental Research journal.

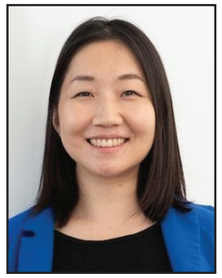
Summary
Research on lung cancer leads to changes in national guidelines for screening eligibility

Early detection of lung cancer through low-dose computed tomography (LDCT) screening is one of the most promising strategies to reduce lung cancer mortality in high-risk individuals. LDCT screening is recommended by the United States Preventive Services Task Force (USPSTF), the American Cancer Society (ACS), and the National Comprehensive Cancer Network (NCCN) for individuals who meet certain age and smoking history criteria. The USPSTF guidelines were updated in 2021 to expand the eligible age group from 55-80 to 50-80 and to reduce the required number of pack-years of smoking from 30 to 20. Dr. Julie Palmer, the Karin Grunebaum Professor in Cancer Research at the Boston University Chobanian and Avedisian School of Medicine and Dr. Chi-Fu Jeffrey Yang, a thoracic surgeon and lung cancer researcher from the Massachusetts General Hospital and Harvard Medical School have been working together to assess how well the revised guidelines work in the Black population. Our most recent report on this topic, published in the Journal of Clinical Oncology in 2024, was based on data from both the Black Women's Health Study and the Southern Community Cohort Study, a study from Vanderbilt University. We showed that using a 20-pack-year smoking history cutoff as a selection criterion for lung cancer screening excluded many study participants who had been diagnosed with lung cancer and led to a marked racial disparity in screening eligibility between Black versus white individuals, with a higher proportion of white patients identified as high risk. Applying a 20-year smoking duration cutoff (instead of pack-years) increased the proportion of lung cancer patients who would qualify for screening and, importantly, eliminated the racial disparity in screening eligibility. In the paper, we further noted that a guideline of 20 years duration of smoking would be easier for clinical use. Following publication of our paper, the NCCN revised their guidelines for lung cancer screening in October 2024. Under the new guidelines, individuals who are age 50 or older and have smoked for at least 20 years or have 20 pack-years of smoking are deemed eligible for screening by low-dose CT scan.

Potter AL, Xu NN, Senthil P, Srinivasan D, Lee H, Gazelle GS, Chelala L, Zheng W, Fintelmann FJ, Sequist LV, Donington J, Palmer JR & Yang CJ. Pack-Year Smoking History: An Inadequate and Biased Measure to Determine Lung Cancer Screening Eligibility. J Clin Oncol. 2024 Jun 10;42(17):2026-2037. doi: 10.1200/JCO.23.01780. Epub 2024 Mar 27. PMID: 38537159; PMCID: PMC11191064.

Incoming Fellow

Ang Cui, Ph.D.
Assistant Professor, Harvard Medical School



Deciphering lymph node cytokine activities in immunotherapy for melanoma

Despite advances in immunotherapy, many cancer patients, including those with melanoma, do not yet respond to immune checkpoint blockade such as anti-PD-1, and there is a significant challenge in understanding the mechanisms of resistance and predicting which patients are more likely to benefit from such therapies. Cytokines, extracellular signaling molecules of the immune system, play a critical role in immune activation, potentially mediating an effective or ineffective immune response to immunotherapies. However, due to the large number of cytokines and cell types, as well as the complex cellular responses to diverse cytokines, it has been challenging to systematically decode cellular responses to cytokines in responders vs. non-responders in cancer patients. The lymph nodes are key organs where anti-tumor immunity develops and are routinely biopsied for cancer staging purposes. Therefore, understanding cytokine activities in lymph nodes holds tremendous potential for deciphering mechanisms of anti-cancer immunity and predicting treatment response. Recently, we built the Immune Dictionary (Cui et al., Nature, 2024), where we systematically measured the single-cell transcriptomic responses of >17 immune cell types in response to each of 86 cytokines in murine lymph nodes in vivo, providing a large-scale compendium of cell-type-specific cytokine signatures for >1,400 cytokine-cell type combinations. Based on the dictionary, we created its companion software, Immune Response Enrichment Analysis (IREA), enabling automated assessment of cytokine activities from gene expression data. This creates a unique opportunity to understand cytokine responses in lymph nodes in cancer immunotherapy, which may offer unprecedented mechanistic and predictive insights into cancer immunotherapy responsiveness. My Karin Grunebaum Cancer Research Foundation Faculty Fellowship project aims to leverage our Immune Dictionary to investigate cytokine activities in tumor-sentinel lymph nodes in responder and non-responder populations to anti-PD-1 therapy in proof-of-concept mouse tumor models of melanoma, and serves two basic and translational goals: 1) it sheds light on cytokine pathways that mediate an effective anti-cancer immunity in conjunction with anti-PD-1, and 2) it establishes a novel framework for predicting cancer patient responses to anti-PD-1 based on their lymph node biopsies at the time of diagnosis.

Incoming Fellow

Mollie E. Barnard, ScD
Assistant Professor, Department of Medicine, Section of Hematology & Medical Oncology, Chobanian & Avedisian School of Medicine, Boston University

Characterizing the immune response to breast cancer in Black women

Background: A primary goal of my research program is to improve racial equity in breast cancer outcomes. In the United States (US), White women are slightly more likely to develop breast cancer, yet Black women are 40% more likely to die from the disease. This disparity is partly due to differences in the prevalence of metabolic health conditions (obesity is 48% more common, and diabetes is 75% more common among Black women compared to White women); however, the biologic pathways through which obesity and diabetes influence breast cancer aggressiveness have not been well described.

To characterize biologic pathways that may explain why obesity and diabetes influence tumor aggressiveness, our team has been working to (a) characterize differences in gene expression among breast cancer cases with and without obesity and type 2 diabetes, and (b) leverage gene expression data to compare tumor immune cell abundance among breast cancer cases with and without obesity and type 2 diabetes.

Proposed Research: As a next step, I am eager to investigate how metabolic health influences the spatial distribution of immune cells in tumors from Black women with breast cancer. My long-term goal in pursuing this line of work is to develop a deeper understanding of how tumor–immune cell interactions within the breast tumor microenvironment drive breast cancer progression. This is both biologically interesting and has broad clinical implications, since the strength of the immune response has the potential to influence breast cancer outcomes. It is also an area that requires methodologic innovation because there is no gold standard approach to the spatial analysis of multiplex immunofluorescence (mIF)-stained tumor tissue cores. Therefore, as a Grunebaum Faculty Research Fellow, I will work toward two key goals:

Goal 1: Estimate the associations between metabolic health and breast cancer immune cell abundance, as measured by mIF staining of specific immune cell types (e.g., CD8+ cytotoxic T cells).

Goal 2: Develop a spatial analysis pipeline for tumor and immune cell data derived from tissue microarray-based mIF staining of tumor cells, B cells, T cells, and macrophages. This pipeline will take as input, cell-level data from the HALO® image analysis platform. It will provide as output spatial statistics that indicate how immune cells and tumor cells interact within the breast tissue.

Significance and Impact: On completion of these goals, our team expects to better understand how metabolic health influences the type and quantity of immune cells in the breast tumor microenvironment. We will also be well positioned to evaluate how metabolic comorbidities influence the relative locations of tumor and immune cells—an important step toward further personalization of cancer care.

References:

1. Giaquinto AN et al. Breast cancer statistics 2024. CA Cancer J Clin. 2024;74(6):477-495.

2. Saka AH et al. Cancer statistics for African American and Black people, 2025. CA Cancer J Clin. 2025;75(2):111-140.

3. Moore JX et al. Metabolic Syndrome Prevalence by Race/Ethnicity and Sex in the United States, National Health and Nutrition Examination Survey, 1988-2012. Prev Chronic Dis. 2017;14:E24. Epub 20170316. doi: 10.5888/pcd14.160287.

4. National Center for Health Statistics (U.S.), National Center for Health Services Research. Health, United States. 1975-1976/77, 1979: DHEW publication no (HRA). Rockville, Md., Washington, D.C.: U.S. Dept. of Health, Education, and Welfare, Public Health Service, Health Resources Administration. For sale by the Supt. of Docs., U.S. G.P.O.; 1976.

5. Palmer JR et al. Type II Diabetes and Incidence of Estrogen Receptor Negative Breast Cancer in African American Women. Cancer Res. 2017;77(22):6462-9.

6. Zhang F et al. Type 2 Diabetes Mellitus and Clinicopathological Tumor Characteristics in Women Diagnosed with Breast Cancer: A Systematic Review and Meta-Analysis. Cancers (Basel). 2021;13(19). Epub 20211005. doi: 10.3390/cancers13194992.

7. Yao S et al. Breast Tumor Microenvironment in Black Women: A Distinct Signature of CD8+ T-Cell Exhaustion. J Natl Cancer Inst. 2021;113(8):1036-43.

Outgoing Fellow

Thomas L. Clarke, Ph.D.
Assistant Professor, Pathology and Laboratory Medicine, Chobanian and Avedisian School of Medicine, Boston University

I would first like to thank the trustees of the Foundation for your generous contribution to my research program by supporting me as the 2024-2025 Karin Grunebaum Faculty Research Fellow at Boston University Chobanian & Avedisian School of Medicine. The fellowship has been vital to establishing our research team and program and has directly contributed to the success of several projects. These projects include: 1) understanding the role of ZNF280A, a newly identified DNA repair factor, in DNA double-strand break repair and the etiology of a human developmental syndrome, 22q11.2 distal deletion syndrome 2) Identifying additional uncharacterized zinc finger proteins with roles in DNA damage repair.

Thanks to the foundation's support, project one has been accepted for publication at Nature Cell Biology, one of the world's leading cell biology journals. In addition, preliminary data generated for other ongoing projects will form the basis of large multi-million-dollar grant applications to the National Institutes of Health in the coming months. The fellowship has helped set the trajectory for our team's future success in discovering novel molecular mechanisms of genomic instability and DNA damage repair in cancer and human developmental disease.

Background:
The American Cancer Society projects that there will be over two million new cases and over 600,000 cancer-related deaths in the United States in 2025. Therefore, understanding the molecular mechanisms driving cancer development and therapy resistance is absolutely critical. DNA damage and genomic instability are recognized as fundamental hallmarks of cancer. The mission of my laboratory is to elucidate fundamental mechanisms of DNA damage repair, and leverage these newly uncovered mechanisms to aid the development of novel cancer therapeutics (3). We use a series of complementary and orthogonal approaches across molecular biology, cell biology, genomics, and proteomics to further our understanding of how defects in DNA damage repair contribute to genome instability and lead to human developmental disease, premature aging, and cancer. My laboratory is particularly interested in identifying novel chromatin-interacting proteins that are involved in DNA damage repair. A long-term goal of our research program is to understand how chromatin re-establishment following DNA damage repair is initiated and determine whether this process can be targeted as a new therapeutic strategy in cancer.

Current Work:
Most recently, my laboratory has been investigating the structure, function and regulation of novel chromatin associated proteins in the context of genomic integrity maintenance. We are currently exploring important questions related to the biological function and mechanistic regulation of the zinc finger 280 protein family. Zinc finger proteins are encoded by as much as 5% of the human genome, however the majority of zinc finger proteins remain uncharacterized, despite their implication in a range of human diseases including human developmental syndromes, neurodegeneration, and several different types of cancer.

During the course of my Karin Grunebaum Faculty Fellowship, my laboratory has uncovered key mechanisms that help to understand how a previously uncharacterized protein, ZNF280A, orchestrates DNA damage repair. Specifically, we have shown that ZNF280A is critical for repairing a type of DNA lesion known as a DNA double-strand break. DNA double-strand breaks are among the most toxic of DNA lesions, and if unrepaired can result in mutations that are known to drive human disease including cancer. Interestingly, one copy of ZNF280A is lost in a human developmental syndrome called 22q11.2 distal deletion syndrome. Patients with this syndrome present with clinical features including, microcephaly (abnormally small head circumference), short stature and growth defects, global developmental delay, immune deficiency, cognitive impairments and an increased risk of developing cancer. Using cells derived from these patients, we were able to demonstrate that these patients have more DNA damage in their cells because they are defective in DNA damage repair mechanisms. Therefore, we have been able to uncover a potential mechanism driving the development of this human developmental syndrome. This work has been accepted for publication at Nature Cell Biology (Clarke et al, Nature Cell Biology – In press).

In addition to this work, we have also uncovered another uncharacterized zinc finger protein with important roles in DNA damage repair. Importantly, the expression of this protein is significantly reduced in breast cancer patients, particularly those with one of the most aggressive and difficult to treat forms of the disease, triple negative breast cancer. By understanding the molecular mechanisms that this newly uncovered protein is involved in, we are hoping to develop novel treatment strategies for breast cancer patients.

In closing, support from the Karin Grunebaum foundation has been critical for the development of my new laboratory and has provided crucial seed funding to initiate several new exciting avenues of research. Thank you again for your invaluable support.

Outgoing Fellow

Charlie Evavold, PhD
Ragon Fellow
Mass General, MIT, and Harvard

Synthetic killer mRNAs as novel innate immune inspired cancer immunotherapies

Background:
Our research program integrates synthetic biology, immunology, and systems-level analysis to explore how innate immune signaling and programmed cell death can be leveraged for cancer immunotherapy. We focus on cell-intrinsic death pathways, especially gasdermin-mediated pyroptosis, and their consequences for tumor clearance and immune activation. Pyroptosis represents a lytic, inflammatory form of cell death capable of releasing damage-associated molecular patterns (DAMPs) and tumor antigens that can trigger potent immune responses when properly directed. However, cancer cells and tumor-resident macrophages frequently develop resistance to both intrinsic and immune-induced death mechanisms.

To overcome these barriers, we engineer synthetic killer mRNAs encoding cell death executioners and deliver them to tumor cells and suppressive myeloid populations using lipid nanoparticles (LNPs). These synthetic biology tools enable temporal and cell type-specific control of cell death. Our lab's core innovation lies in combining these synthetic payloads with metabolic sensitization strategies to unlock immunogenic cell death in otherwise resistant tumors. By characterizing how metabolic and trafficking pathways govern cell death susceptibility, and by comparing distinct cell death modalities (e.g., apoptosis vs pyroptosis), we aim to create programmable immunotherapies that rewire tumor-immune communication.

Research Goals:
Tumor resident myeloid cells and cancer cells are strong candidates for manipulation by cell death pathways and inflammatory modules. By achieving the following goals, we will uncover both intrinsic and extrinsic mechanisms by which cell death pathways influence tumor progression and anti-tumor immunity. Our work will enable development of new strategies to target resistance mechanisms or susceptibility targets within immunosuppressive macrophages and cancer cells while also promoting productive antigen presenting activities of DCs to improve cancer therapies through principles of innate immunity and synthetic biology.
Goal 1: Define regulation of innate immune and cell death pathways in myeloid and cancer cells.
Goal 2: Determine antigen presentation and adaptive immune consequences of cell death pathways.

Progress on Research Goals:
We have made substantial progress toward identifying regulators of gasdermin-mediated cell death and building inducible models to study the immune consequences of synthetic induced cell death in cancer models.
Rabgef1 is a broad regulator of pyroptosis. Rabgef1-deficient macrophages (sgRNA targeted) have increased cholesterol content compared to wild type (WT) macrophages (Figure 1A). Using synthetic mRNAs encoding the pore-forming N-terminal (NT) domains of five gasdermin family members (murine GSDMA3 and human GSDMB-E), we demonstrated that Rabgef1 licenses increased pyroptosis across this protein family in murine macrophage immune cells (Figure 1B-F). Rabgef1-deficient macrophages have decreased membrane permeability assayed by live cell imaging of propidium iodide staining following NT gasdermin encoding mRNA transfection, suggesting this protein is a key trafficking adaptor that licenses gasdermin activity at the plasma membrane (Figure 1B-F).

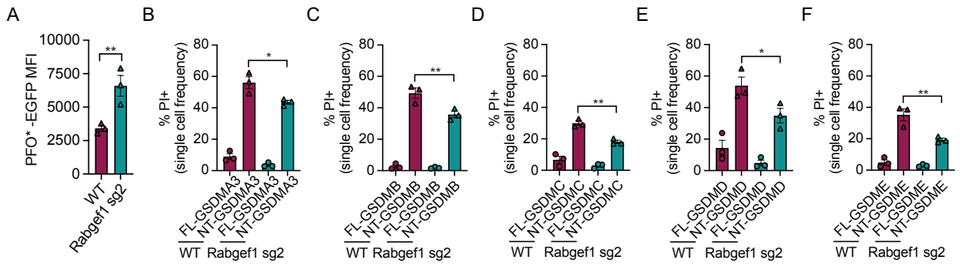


Figure 1: Rabgef1 is a general regulator of mammalian gasdermin pore forming activity in macrophage immune cells. (A) WT and Rabgef1-deficient murine immortalized bone marrow derived macrophages (iBMDMs) stained for plasma membrane cholesterol content with cholesterol biosensor PFO*-EGFP by flow cytometry. (B-F) WT and Rabgef1-deficient macrophages transfected with mRNA:LNPs encoding the pore forming fragment (NT) or inactive, full-length (FL) form of mouse GSDMA3 and human GSDMB-GSDME looking at pore forming activity via propidium iodide (PI) staining that indicates membrane permeability by live cell imaging.

Cholesterol depletion sensitizes tumor cells to gasdermin-mediated pyroptosis. We discovered that reducing plasma membrane cholesterol using methyl-β-cyclodextrin (MCD) (Figure 2A) sensitizes B16F10 melanoma cells to pyroptosis driven by gasdermin NT for all 5 human gasdermin family members known to form pores (Figure 2B-F). This generalizes prior findings on GSDMD in macrophages and shows that membrane lipid and sterol composition is a central regulator of lytic pore formation. Similar sensitization was observed in LLC1 lung cancer, PDAC pancreatic cancer, and MB49 bladder cancer models, supporting the generality of this approach and potential for therapeutic action using metabolic sensitization combined with cell death mRNA:LNPs.

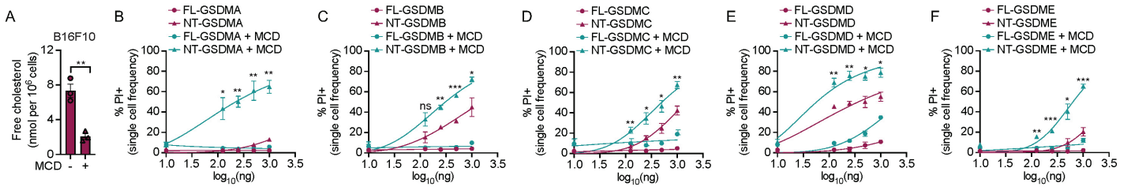


Figure 2: Plasma membrane cholesterol depletion sensitizes tumor cells to mammalian gasdermin pore forming activitiy in cancer cells. (A) MCD depletes cholesterol from B16F10 murine melanoma cells assayed by Amplex Red free cholesterol quantification kit. (B-F) B16F10 melanoma cells transfected with mRNA:LNPs encoding the pore forming fragment (NT) or inactive, full-length (FL) form of human GSDMA-E across a dose curve of mRNA concentrations looking at pore forming activity via propidium iodide (PI) staining that indicates membrane permeability by live cell imaging.

GSDME pyroptosis requires metabolic priming in resistant cancers. We noted that with mRNA:LNP transfection alone that B16F10 melanoma and PDAC cells are resistant to NT-GSDME mediated plasma membrane pore formation even at high mRNA doses (Figure 2F). This resistance was reversed when plasma membrane cholesterol was depleted with MCD (Figure 2A, 2F). To further investigate cholesterol enhancement for future in vivo tumor studies, we created a doxycycline-inducible NT-GSDME expression model in B16F10 melanoma and PDAC organoids that can be depleted for plasma membrane cholesterol using the DHCR7 inhibitor AY9944 (Figure 3A, B, E, F). Despite robust transgene induction, cell lysis only occurred when cholesterol was simultaneously depleted for both melanoma and pancreatic cancer cells (Figure 3A-H). This highlights an inherent resistance to pyroptosis in certain cancers and confirms that metabolic context dictates the efficacy of synthetic cell death induction.

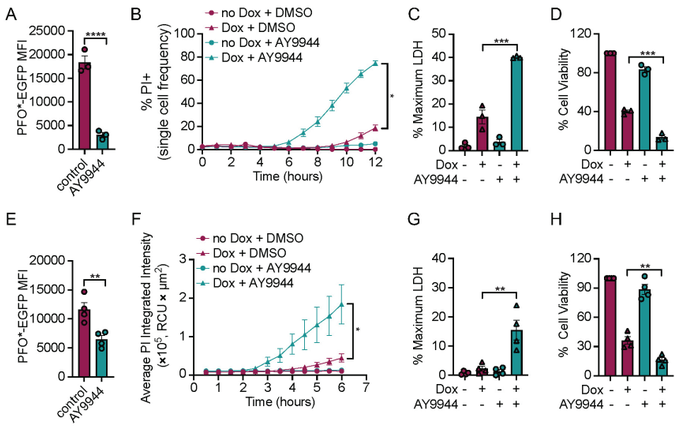


Figure 3: GSDME pyroptosis requires cholesterol depletion in resistant cancers. (A, E) Treatment with the DHCR7 inhibitor AY9944 results in diminished plasma membrane cholesterol staining with the biosensor PFO*-EGFP by flow cytometry in B16F10 murine melanoma cells (A) and murine pancreatic adenocarcinoma cancer (PDAC) organoids (E). (B, F) Propidium iodide (PI) staining for membrane permeability after Doxycycline-inducible NT-GSDME with or without AY9944 cholesterol depletion in melanoma (B) and PDAC (F). (C, G) Lactate dehydrogenase (LDH) release for cell lysis after Doxycycline-inducible NT-GSDME with or without AY9944 cholesterol depletion in melanoma (C) and PDAC (G). (D, H) Cell viability assayed by ATP quantification after Doxycycline-inducible NT-GSDME with or without AY9944 cholesterol depletion in melanoma (D) and PDAC (H).

tBID and NT-GSDMD allow for synthetic models of apoptosis and pyroptosis in cancer cells. To study how different forms of death impact dendritic cell (DC) activation and T cell priming, we engineered bladder cancer cell lines (MB49, UPPL1541, BBN963) with inducible synthetic death circuits. In the UPPL1541 model, Doxycycline-inducible NT-GSDMD induced propidium iodide uptake indicating membrane perforation consistent with pyroptosis (Figure 4A), whereas Doxycycline-inducible tBID induced Annexin V staining indicating phosphatidylserine exposure consistent with apoptosis. These controlled systems will allow head-to-head comparisons of cell death outcomes in immune co-culture and in vivo tumor models.

Outgoing Fellow

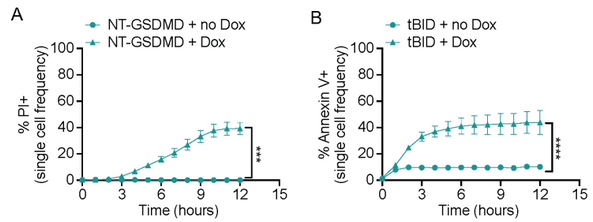


Figure 4: Synthetic models of apoptosis and pyroptosis in engineered cancer cells. (A) UPPL1541 model of murine bladder cancer engineered to express Doxycycline-inducible NT-GSDMD as a model of pyroptosis seen by PI staining of membrane permeability by live cell imaging. (B) UPPL1541 model of murine bladder cancer engineered to express Doxycycline-inducible tBID as a model of apoptosis seen by Annexin V staining of phosphatidylserine exposure by live cell imaging.

Continuing Studies:
We are expanding our synthetic cell death models to additional cancer types to assess how different forms of cell death affect antigen uptake, processing, and presentation by DCs. Co-culture studies with primary DCs and antigen-specific T cells are underway to evaluate how apoptosis versus pyroptosis influences adaptive immune activation. We are also characterizing cell-intrinsic and metabolic resistance mechanisms in cancer and myeloid cells to optimize delivery and efficacy of mRNA-encoded synthetic executioners.

Significance and Impact:
Our work defines key regulators of lytic cell death and reveals how cholesterol and intracellular trafficking pathways shape pyroptosis susceptibility across diverse tumor models. By combining metabolic perturbation with synthetic induction of lytic cell death, we are pioneering a new class of innate immune-inspired cancer therapies. These studies lay the foundation for next-generation immunotherapies that exploit programmed cell death to break immune tolerance and drive durable anti-tumor immunity.

Acknowledgements & Shout Outs

- **Thank you to Dr. Michael Droller and Dr. Michael Gimbrone,** our first and second KGCRF Fellows, respectively. Their dedication and service on our Board of Trustees have helped shape the foundation into what it is today. We deeply appreciate their insight—not only as distinguished medical professionals, but also for their personal understanding of the foundation's origins—which has ensured that we remain true to our original mission.

- **Welcome to Dr. Elizabeth Battinelli, MD, Ph.D., M.Sc. and Dean Hee-Young Park, PhD** as our newest members of our Board of Trustees! Dr. Battinelli was a former KGCRF fellow (2012-13) and is currently an Associate Professor at Harvard Medical School. Dean Park is the interim dean of Boston University's medical school. She is also the Chair of Medical Sciences & Education, Professor of Dermatology, and Associate Dean of Faculty Affairs.

- **Congratulations to Dr. Genevieve Boland '16** for her new role as Vice Chair of Research for the Integrated Department of Surgery at Mass General Brigham.

- **Dr. Russell Jenkins '19** recently had a paper published in Cell Reports Medicine, referred to as, "Overcoming resistance to immunotherapy by targeting CD38 in human tumor explants". It was published by both Dr. Jenkins and Or-Yam Revach, PhD, a postdoctoral research fellow.

Congratulations to all our past Karin Grunebaum Cancer Research Fellows!

Karin Grunebaum Cancer Research Foundation January 2026 Volume 22

Professional Development Curriculum, Cancer Biology Program at Harvard

The Cancer Biology Program at Harvard provides curricular and extracurricular activities both to teach graduate students about cutting-edge research and to hone professional skills. As a result, the Program fosters a collaborative community of researchers that is both stimulating innovation and paving the way for future discoveries. Diverse programming enriches students' educational experience – from designing and executing courses highlighting the forefront of cancer research, to organizing seminars and symposia that bring together the brightest scientific minds, to creating opportunities for mentoring and networking where students interact with faculty, industry leaders, and professionals from other cancer-related fields.

Professional development activities and resources available to the students include the Karin Grunebaum Cancer Research Foundation poster competition, attendance at national and international meetings, conferences, courses and workshops in cancer biology and related topics, society memberships, computer supplies, and books. As future leaders, the graduate students share their knowledge by teaching, mentoring, and inspiring undergraduate students. Their commitment to research is pushing the boundaries of what is known about cancer, and their discoveries are disseminated in publications and conferences around the world.

The Foundation's generous gift to the graduate training at Harvard directly supports professional development of our students. The funds are used for KGCRF Poster Competition and Professional Development Awards and the KGCRF Career Catalyst Awards.

Karin Grunebaum Cancer Research Foundation Professional Development Awards

These are monetary prizes in the amount of $1,000 each that go towards professional development and are awarded for best posters during the Annual Spring Symposium and KGCRF Poster Competition. In 2025, we awarded:

Two KGCRF Rising Young Scientist Awards
(up to and including G3s)

- Alanis Carmona, G3, Biological Sciences in Public Health, Jessalyn Ubellacker Lab
  Poster title: *Characterizing and targeting extracellular matrix proteins that contribute to breast survival in the lymph node*

- Olivia Lavidor, G2, Chemical Biology, Brian Liau Lab
  Poster title: *Targeting the Regulatory Domains of DNMT3A to Rescue Loss of Function Phenotypes in AML*

Two KGCRF Early Career Investigator Award
(G4s and up)

- Alice Bertocchi, G5, Immunology, Judith Agudo & Stephanie Dougan Labs
  Poster title: *Eosinophils alter metastatic spread in pancreatic cancer*

- Rachel Hongo, G4, Biological and Biomedical Sciences, Leonard Zon Lab
  Poster title: *Investigating extracellular factors driving neural crest state reactivation in melanoma initiation*

For more information about the 2025 KGCRF Poster Competition awardees, please see document 2025_KGCRFPosterCompetition-Awards.

KGCRF Rising Young Scientist Awards
(graduate years – G1, G2, and G3)

ALANIS CARMONA | G3
Biological Sciences in Public Health
Jessalyn Ubellacker Lab

Characterizing and targeting extracellular matrix proteins that contribute to breast survival in the lymph node

Breast cancer is the most prevalent cancer among women in the United States, representing approximately 30% of all new female cancer cases annually. This year alone it is anticipated that over 310,000 new cases of invasive breast cancer will arise and result in over 42,000 deaths. Most cancer-related deaths result from metastasis, for which there is no treatment. Metastatic spread can occur through the circulatory and/or the lymphatic system. Prior work from our laboratory has shown that the lymph node tumor microenvironment protects metastasizing cancer cells from ferroptosis, which is a mode of cell death caused by unchecked lipid oxidation. Within the context of the lymph node microenvironment, in this project we sought to understand the contribution of cancer associated fibroblasts (CAFs) in the lymph node in secreting extracellular matrix (ECM) factors that could also contribute to differences in lipid oxidation levels of cancer cells in lymph nodes. The ECM is comprised of an intricate mixture of proteins including proteoglycans and adhesive glycoproteins, such as collagens and laminins. Here, we discovered that specific ECM components in lymph nodes provide protection from lipid oxidation and ferroptosis-inducing agents in vitro. We next tested conditioned media from human CAFs generated from patients with and without breast cancer detected in the lymph nodes to determine the extent to which the CAF-conditioned media, as well as CAF-derived ECM components, protect against lipid oxidation. We found that CAF-conditioned media from patients with, but not without, breast cancer cells in the lymph nodes protected breast cancer cells from ferroptosis in vitro.

OLIVIA LAVIDOR | G2
BChemical Biology
Brian Liau Lab

Targeting the Regulatory Domains of DNMT3A to Rescue Loss of Function Phenotypes in AML

DNA methyltransferase 3A (DNMT3A) is an epigenetic writer protein responsible for deposition of suppressive methylation marks on cytosine nucleobases in DNA. Because of its genome-wide, essential function, DNMT3A is often mutated in developmental and hematological disorders. The most common mutation is the dominant-negative R882H, which accounts for about two thirds of DNMT3A mutations in AML. DNMT3A is active as a tetramer, and ongoing debate in the field has left two hypotheses of the mechanism of R882H loss of function; Either it breaks a central protein-protein interface to form dimers, or it strengthens this interface resulting in macro-oligomers. Using techniques such as sucrose gradient ultracentrifugation, hydrogen deuterium exchange mass spectrometry, and DNA-encoded library screens, we aim to (1) lend evidence toward the oligomerization hypothesis, (2) explore the role of understudied regulatory domains of DNMT3A, and (3) potentiate the rescue this phenotype with a first-in-class small molecule activator for DNMT3A.

KGCRF Early Career Scientist Awards

(graduate years – G4 and up)

ALICE BERTOCCHI | G5
Immunology
Stephanie Dougan & Judith Agudo Labs

Eosinophils alter metastatic spread in pancreatic cancer

Pancreatic ductal adenocarcinoma (PDAC) is the third most common cause of cancer death in the US with a 13% 5-year survival rate. A key contributor of this dismal prognosis is early and frequent metastatic spread with limited treatment options for patients. In the primary tumor reactive stroma, we found increased eosinophils in both human and mouse PDAC compared to the normal pancreas. We used the ddGata mice, which lack eosinophils, to study their role during metastatic spread using a mouse model of resectable metastatic PDAC. We found that mice lacking eosinophils have increased metastasis to the lung in our spontaneous model, but no difference is seen in establishment of IV injected tumor cells, suggesting eosinophil control at the primary tumor site. Single cell sequencing of tumor cells and staining find alterations in their EMT state. When we investigated the anti-metastatic role of eosinophils, we observed production of IL-4 in human and mouse PDAC tissues. We showed that IL-4 treatment correlates with decreased metastasis to distal organs and increased epithelial state of the cancer cells in our spontaneous model of resectable PDAC, independently of IL-4 signaling in immune cells. We conclude that eosinophils in PDAC inhibit the metastatic process, in part, through control of tumor cell state through secretion IL-4.

RACHEL HONGO | G4
Biological and Biomedical Sciences
Leonard Zon Lab

Investigating extracellular factors driving neural crest state reactivation in melanoma initiation

Melanoma is the deadliest form of skin cancer, but it is curable with surgical resection if diagnosed early, making improved strategies for early detection and prevention critical. In a zebrafish melanoma model where melanocytes express human BRAFV600E in a p53 null background, we observe single melanocytes reactivating an embryonic neural crest program (crestin:EGFP+) that expand to form melanomas. This neural crest state reactivation marks a key transformative step in melanoma initiation. To understand what drives this transition, we are investigating the role of the local extracellular microenvironment. We performed proteomics on crestin:EGFP+ regions versus cancerized field skin and identified 498 upregulated proteins, including 13 ligands and matrisome components. Genetic overexpression of candidate ligands, such as midkine, in zebrafish melanocytes accelerated neural crest reactivation in vivo, suggesting that secreted factors in the microenvironment can promote melanoma initiation. Ongoing studies using zebrafish knockouts, single cell RNA-sequencing, and spatial transcriptomics aim to define ligand-receptor interactions and their cellular sources. These findings support a model in which local microenvironmental factors cooperate with oncogenes to trigger melanoma, providing insights into how tissue context influences cancer initiation

KGCRF Career Catalyst Awards for Fall 2025

Awardee: Isabella DelPriore
Graduate Program: Biological and Biomedical Sciences
Lab: Alex Toker

KGCRF CCA Award: To support attendance and presentation at the annual AACR 2025.

Awardee: Emma Lecky
Graduate Program: Biological and Biomedical Sciences
Lab: Alex Toker

KGCRF CCA Award: To support attendance and presentation at the annual AACR 2025.

Awardee: Wafa Malik
Graduate Program: Biological and Biomedical Sciences
Lab: Aaron Hata

KGCRF CCA Award: To support attendance at the Gordon Research Conference on Decoding the Cancer Genome and Epigenome.

Awardee: Sreekar Mantena
Graduate Program: Bioinformatics and Integrative Genomics
Lab: Soumya Raychaudhuri

KGCRF CCA Award: To support attendance at the 2025 Cold Spring Harbor Systems Immunology Conference

Awardee: Isabella DelPriore

"As a third-year graduate student in Alex Toker's Laboratory, my projects broadly focus on uncovering novel mechanisms by which the PI3K/AKT pathway regulates cellular metabolism. The PI3K/AKT pathway is frequently hyperactivated in breast cancer, leading to dysregulation of cellular proliferation and metabolism. Through pharmacological perturbation of AKT with degraders and inhibitors and a CRISPR/Cas9 screen, I have identified ribosome biogenesis as a point of regulation within this network that is altered in cancer cells. **My current project aims to characterize the regulation of ribosomal homeostasis by AKT and test the efficacy of co- targeting ribosome biogenesis alongside PI3K/AKT inhibition in pathway mutant breast cancer, eventually informing therapies for this population of cancer patients.**

The motivation for this project is rooted in discovering novel aspects of cancer biology that will ultimately improve treatment strategies for cancer patients. Given this translational goal and cancer focus, it will be of great benefit to my research to participate in the American Association for Cancer Research (AACR) annual meeting. This meeting brings together researchers and clinicians across several institutions to present and discuss cancer at all levels, from basic, to translational, to clinical research. The opportunity to be immersed in and interact with this diverse community working towards the common goal of understanding and treating cancer will complement my research and provide opportunities for personal and professional growth.

By participating in the AACR annual meeting, **I will have the opportunity to practice my science communication and presentation skills. The ability to refine and effectively convey the story of my research is an extremely valuable and essential skill as a scientist.** Participating in a meeting such as AACR will allow me to practice speaking about my science in both formal and informal settings, with peers and those more advanced in their careers. **Beyond practice in communication skills, the benefits of these interactions include receiving feedback from experts that I otherwise would not have the chance to speak with. This is especially important considering that the ribosome biogenesis component of my work is a new focus for the lab, and thus I would benefit from discussing my findings with those whose specialty lies in this field.** Furthermore, any connections that I make at the meeting will be valuable for potential future collaborations.

Beyond interacting with other scientists and researchers, there are several different sessions that will be held at AACR covering a range of topics, some of which are directly relevant to my research. For example, I plan to attend "Protein Degraders: Progress to the Clinic," as I am utilizing AKT degraders in my work; therefore, this session will be beneficial in considering the clinical potential of the drugs I am working with and what obstacles remain. Additionally, my project centers around alterations in cellular metabolism, and thus the "Metabolism and Organelle Biology in Cancer" session will be informative for my research.

In addition to topics that will directly support my work, **exposure to research outside of my focus area will provide new perspectives and insights that will expand my thinking.** Not only will this inform potential future directions, but also help put my work into context, emphasizing how my research fits into the larger picture of cancer research as a whole. **The clinical aspects of AACR will help ground the day-to-day work that I am conducting in the lab, bridging the gap between basic discovery science and medicine.** This provides personal motivation and will keep me informed of current obstacles that exist in cancer treatment and patient care from a broader perspective beyond the research that I am exposed to within the Harvard scientific community. To further make connections outside of the Boston science network, I will attend the "Cancer and Biomedical Research Career Fair," which will help me begin thinking about potential career paths within the field of cancer research, as I plan to continue to work in this space after completing graduate school."

KGCRF Career Catalyst Awards for Fall 2025
(continued)

Awardee: Emma Lecky

"I am applying for the Karin Grunebaum Cancer Research Foundation Career Catalyst Award to support my attendance at the 2025 American Association for Cancer Research (AACR) Annual Meeting in Chicago, IL. **My research is focused on characterizing a novel antibody-drug conjugate (ADC) to target human epidermal growth factor 2 positive (HER2+) breast cancer.**

HER2+ breast cancer is driven by overexpression of the HER2 receptor, leading to aggressive tumor growth. Targeted therapies, including trastuzumab and ADCs such as trastuzumab-deruxtecan, have improved patient outcomes by blocking HER2 signaling and delivering chemotherapeutics directly to cancer cells. However, there remains a pressing need to improve ADCs to increase efficacy, reduce toxicity, and overcome resistance mechanisms. We have developed a novel ADC with an ultra-high drug-antibody ratio (DAR) of 50-100 drug molecules per antibody, enabling conjugation of trastuzumab with targeted therapies including PI3K and MEK inhibitors. **By targeting these otherwise toxic agents directly to cancer cells, we aim to improve tolerability and mitigate resistance, marking a promising advance in cancer therapeutics. I have submitted a first-author abstract to the 2025 AACR meeting which highlights our findings that our ADC undergoes internalization within HER2-positive cancer cells, effectively inhibiting cell proliferation and suppressing PI3K and MEK signaling.**

Attending the AACR meeting offers **a valuable opportunity to gain insights into the latest advancements in cancer therapeutics research that will directly complement my work.** This year, AACR is holding a symposium on Advances in Diagnostics and Therapeutics, with a session on challenges and opportunities in the development of next generation antibody drug conjugates. Through sessions such as this one, I will be exposed to innovations in ADC development, including target selection, payload optimization, and clinical trial design. Learning from leading experts in ADC research will broaden my understanding and potentially inspire refinements and improvements to our approach. Additionally, **I plan to attend skill-building workshops on emerging experimental techniques to strengthen the technical aspects of my research. Notably, AACR is offering a workshop on Chemistry to the Clinic, which will provide insight into cancer drug design and development processes.**

The AACR meeting will also provide opportunities to develop my presentation skills and expand my professional network, both essential steps as I work toward my goal of becoming a principal investigator of my own research group. **Presenting my work will enable me to refine my communication techniques, receive constructive feedback, and practice articulating complex data to a diverse scientific audience.** This experience will improve my ability to present research confidently and effectively, a critical skill as I progress in my career. Moreover, the AACR meeting offers opportunities to network with other researchers, clinicians, and industry leaders in the field of cancer therapeutics. Engaging with peers and experts will not only broaden my perspective on the latest innovations in ADC research but also foster collaborations, mentorship, and potential postdoctoral opportunities. Engaging with experts addressing similar challenges will introduce alternative approaches, methodologies, and technologies that could further my research."

Awardee: Wafa Malik

"Over the course of my graduate studies in the Biological and Biomedical Sciences (BBS) program and research in the Krantz Family Center for Cancer Research at Massachusetts General Hospital, **I have focused on identifying mechanisms that drive drug resistance in lung cancers treated with targeted therapies.** My research, along with the work of others, has increasingly implicated epigenetic rewiring in the adaptive response to suppression of oncogenic signaling by targeted therapies. Moreover, novel agents that can alter epigenetic rewiring are entering the clinic, providing potential opportunities to translate these basic findings to patients. I am applying for the Karin Grunebaum Cancer Research Foundation Career Catalyst Award **to deepen my training in the field of cancer epigenetics by enabling me to attend the Gordon Research Conference on Decoding the Cancer Genome and Epigenome.** This opportunity will allow me to acquire critical knowledge and build valuable connections that will directly support the advancement of my research.

While I have a foundational understanding of cancer biology, my background in the specific mechanisms of epigenetics—particularly as they relate to chromatin remodeling, transcriptional regulation, and cellular adaptation to therapeutic pressures—is still developing. To fill this gap in my training, one key avenue for expanding my proficiency in this area will be through attending the Gordon Research Conference on Decoding the Cancer Genome and Epigenome. This conference brings together top scientists and clinicians who are at the forefront of research in cancer epigenetics and related fields. **Participation in this event will offer me the chance to engage with cutting-edge research, learn about emerging techniques and technologies, explore the latest findings related to epigenetic regulation in cancer.** Specifically, I seek to achieve the following goals: **1) To enhance my understanding of technical approaches for studying epigenetic regulation in cancer cells that I can employ in my own research, 2) identify potential collaborators to enhance and expand my research directions, and 3) build my professional network to gain scientific and career mentorship from leading experts in the field.** The insights I gain will directly inform my work on novel therapeutic targets for overcoming drug resistance in lung cancer, while exposure to the latest advancements will shape my future research directions, ensuring my contributions remain at the forefront of cancer epigenetics.

In summary, the Karin Grunebaum Cancer Research Foundation Career Catalyst Award will enable me to gain proficiency in cutting-edge epigenetic techniques that will be foundational to my future research endeavors, as well as expert mentorship to guide my career development. Ultimately, **the resources provided by this award will better position me to make significant contributions to the identification of novel therapeutic targets and the development of combination therapies aimed at overcoming drug resistance in KRASG12C-mutant lung cancers."**

Awardee: Sreekar Mantena

"Throughout my first two years of medical school, I cared for many patients with complex solid tumors and hematological malignancies. I saw firsthand how developing a deeper understanding of the human adaptive immune system could empower us to develop entirely new classes of therapies that advance their care. As I read more and more immunology literature, I became fascinated by T cell receptors (TCRs) — surface proteins on T cells that enable them to detect and respond to threats. TCRs exhibit extraordinary inter- and intra-individual diversity: each of us has trillions of TCR sequences that are nearly completely distinct from those found in other people. Recently developed technologies have, for the first time, made it possible to sequence TCRs at scale. My graduate school research is dedicated to developing principled statistical methods to interrogate these TCR sequences, illuminating fundamental principles of T cell biology that can be used to develop next-generation therapeutics.

Currently, I am building computational methods to study a subset of T cells that express two T cell receptors, called dual TCR T cells. My preliminary results suggest they may achieve uniquely potent anti-tumor activity and could be harnessed to develop new types of personalized cancer treatments. I am hoping to validate these results by partnering with systems immunologists and cancer immunologists. The 2025 Cold Spring Harbor Systems Immunology Conference would enable me to build such collaborations. Many cancer immunologists whose work I have long admired, including Mark Davis, will be present at this meeting. Conversing with faculty and trainees at the conference would enable me to build the collaborations needed to experimentally validate the statistical signals we are observing in the dual TCR T cell population. Furthermore, the attendees of this conference have pioneered novel experimental and computational approaches to interrogate the TCR repertoire - learning about these new techniques and ideas would also significantly accelerate my graduate school research.
Finally, as an MD/PhD trainee, my long-term career aspiration is to use insights from TCR sequencing to improve the treatment of hematopoietic malignancies. I have never had the opportunity to attend a scientific conference before, and I would be very grateful to be able to build lasting relationships with the faculty and peers that will be present at the 2025 Systems Immunology Conference. I'm excited to learn from them, and I'm sure these relationships will propel my future career aspirations."

Karin Grunebaum Cancer Research Foundation January 2026 Volume 22

KGCRF Student Summer Research Fund

Summer 2025 Impact Report

August 14, 2025
Steven Wallach
Karin Grunebaum Cancer Research Foundation
Dear Steven and Trustees of the Karin Grunebaum Cancer Research Foundation,

On behalf of the Medical Student Research Program at Boston University Chobanian & Avedisian School of Medicine, I wanted to thank you for your generous support of our medical student cancer researchers. The stipends provided to 4 students this summer made it possible for them to work full-time with our faculty on cancer research projects. Our medical student research advisory committee selected the following outstanding students to engage in cancer research training with our faculty.

- Brittney Anderson worked with Dr. Gerald Denis, on a project that explored "The effects of adipose derived exosomes on triple negative breast cancer cells' susceptibility to systemic therapy"
- Joshua Choi trained with Dr. Christopher Heaphy and Dr. Joshua Campell on a project that evaluated "Th17+ cells as prostate cancer biomarkers and their correlation with ancestral and environmental factors"
- Isabelle Joy performed research with Dr. Thomas Clarke (Karin Grunebaum Cancer Research Foundation Fellow) on a project that explored "The importance of ZNF280D for DNA damage repair and the etiology of endometrial cancer"
- Sabrina Mellinghoff engaged in research training with Dr. Mollie E. Barnard and Dr. Julie Palmer on a project that examined "Breast Cancer Recurrence among Black Women"

It is amazing to see the depth and breadth of the cancer research projects. The students are studying different cancer types using a variety of experimental approaches from basic science (wet lab) to clinical research and public health.

I would like to point highlight in more detail two students: Isabelle Joy and Sabrina Mellinghoff. Isabelle is working with Tom Clarke's group, and it is terrific to see the generational impact that your foundation had for both mentor and trainee. Over the spring and summer, I met with Tom to discuss research, share strategies for navigating academia, and mentoring students He has told me how meaningful it has been having Isabelle work in his lab and shared these inspirational words that he asked to pass along:

> *"Thanks to the generous support of the Grunebaum Foundation, first-year medical student Isabelle Joy was able to spend her summer in my laboratory, launching an exciting new project on endometrial cancer. Isabelle is studying a gene of unknown function whose loss is common in advanced disease and is linked to significantly poorer outcomes, especially among patients of African ancestry. With the Foundation's sponsorship, she successfully created specialized endometrial cancer cell lines in which this gene's expression is reduced, giving us a powerful new model to investigate its role. Beyond these important research advances, your support has given Isabelle the opportunity to develop hands-on molecular biology skills, deepen her passion for scientific discovery, and lay the groundwork for a career that blends patient care with academic research. The impact of the foundation's support will continue to grow as she carries this project forward throughout her medical training."*

I would also like to mention Sabrina Mellinghoff, who reached out to Dean Antman and then to me this spring after her highly competitive summer research program at the National Institutes of Health was abruptly cancelled. Sabrina expressed a strong interest in cancer biology, and as I sometimes see my role as a "research matchmaker", and we were able to connect Sabrina to Dr. Palmer and Dr. Barnard. It was clear that this would be a terrific match, and it has been gratifying to see Sabrina's research project progress over the summer. At the end of our summer program we ask students to write up a research progress report. Sabrina wrote what I believe to be a heartening note about her training and how meaningful the support of the Grunebaum foundation has been for her, which affirmed her passion for being an oncologist.

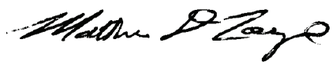
> *"I cannot thank my mentors Drs. Barnard and Palmer enough. I'm very inspired and motivated by their high standards and rigorous conduct of research- it truly is an honor to be working with them and I'm learning a tremendous amount. I also thank the organizers of MSSRP and the Karin Grunebaum Cancer Research Foundation for making this work possible. It is my dream to become an oncologist, and this ongoing research experience has made me more sure of and passionate about my career path. While we learn about the medical and scientific drivers of cancer in the classroom, it has been very meaningful to learn about the epidemiological perspectives and apply a population-level lens to the disease, seeing its course play out in many hundreds of individuals."*

It is through your support that enables our students to engage in world-class cancer research training. I am confident that these early training experiences will motivate and mold our students' paths as they become the next generation of clinicians and cancer researchers.

I am also excited to see how far their research has come and if you are available, I would like to invite you to our 2026 Medical Student Research Symposium on February 13, 2026. I am certain that you will be as impressed with our students' research accomplishments as I am.

On behalf of the Boston University Chobanian & Avedisian Medical School Research Program, thank you for your continued support and would be delighted to discuss our programs further.

Best wishes,
Matt



Matthew D. Layne, PhD
Assistant Dean of Research
Associate Professor of Biochemistry & Cell Biology
Boston University Chobanian & Avedisian School of Medicine
72 E Concord St
Boston MA 02118 mlayne@bu.edu

Boston University Chobanian and Avedisian School of Medicine
Karin Grunebaum Cancer Research Foundation Student Summer Research Fund

The stipends provided to 4 students this summer enabled them to work full-time with our faculty on cancer research projects. Our medical student research advisory committee selected the following outstanding students to engage in cancer research training with our faculty.

- Carly Batt trained with Dr. Gerald Denis on a project that investigated the intersection of metabolic dysfunction and breast cancer.
- Kaina Chen investigated the mechanisms of telomere shortening in pediatric osteosarcomas with Dr. Rachel Flynn.
- Christopher Lietz trained with Dr. Hui Feng and sought to identify novel drugs to enhance cisplatin efficacy in patients with HPVnegative head and neck squamous cell carcinoma using bioinformatic approaches.
- Sienna Wang also trained with Dr. Hui Feng and worked to develop a zebrafish model of HPV+ head and neck squamous cell carcinoma.

To note, Drs. Flynn and Feng are previous Grunebaum fellows, and it is terrific to see how your foundation supports the training of multiple generation of scientists. Our students are continuing in their research projects into the fall, and we look forward to learning more about their discoveries at our January 2025 Research Symposium.

It is the generosity of our supporters that allows the Karin Grunebaum Cancer Research Foundation to continue funding researchers who are working to eradicate all forms of cancer.

Please donate using the QR code

KGCRF Student Summer Research Fund
Summer 2025 Impact Report

Note of Thanks from Brittney Anderson (MED'28)

Dear Karin Grunebaum Cancer Research Foundation,

My name is Brittney Anderson, and I am a first-year medical student at the Chobanian & Avedisian School of Medicine. I am writing to express my heartfelt gratitude for your generous support through the Karin Grunebaum Cancer Research Fellowship. Your investment in aspiring physician-scientists like me has a lasting impact, not only by advancing cancer research but also by making it possible for students to pursue meaningful work without financial burden.

This summer, I had the rewarding opportunity to investigate the role of adipocyte-derived exosomes in triple-negative breast cancer (TNBC). Delving into how these exosomes influence the behavior of the MDA-MB-231 cell line was both intellectually stimulating and personally fulfilling. Through this work, I developed valuable technical skills, deepened my understanding of cancer biology, and became even more inspired by the potential translational applications of this research. Contributing to the pursuit of safer, more effective therapies for cancer patients has been an incredibly motivating experience that will shape my future in medicine.

Attending the Karin Grunebaum Foundation meeting on June 13th was a true highlight. It was inspiring to learn more about the foundation's long-standing commitment to fostering impactful cancer research. Hearing about the remarkable projects you support gave me a renewed appreciation for the collective efforts being made to transform cancer care. I am honored and humbled to now be part of that legacy.

Sincerely,
Brittney

Note of Thanks from Isabelle Joy (MED'24,'28)

Dear Grunebaum Foundation Members,

My name is Isabelle Joy, and I am a medical student at the Chobanian & Avedisian School of Medicine. I'm originally from Wayland, Massachusetts, where I was raised in a community that emphasized service and learning values that continue to guide me in my journey toward becoming a physician. I'm incredibly grateful to have my summer research funded by the Grunebaum Foundation, and I sincerely thank you for your generous support.

I chose to attend the Chobanian & Avedisian School of Medicine for its strong commitment to underserved communities and its integration of public health and social justice into the curriculum. One of my most meaningful experiences so far has been my cancer research, where I've had the opportunity to explore mechanisms driving disease progression in endometrial and breast cancer. This work has deepened my interest in bridging basic science and clinical care.

In the coming year, I look forward to continuing this research and pushing myself to integrate molecular medicine into the clinical setting. Your support has not only eased financial burdens but has also affirmed my passion for research and medicine. I am truly grateful for your investment in my education and future.

Thank you again for your support and encouragement.

Warmest regards,
Isabelle Joy

Note of Thanks from Joshua Choi (MED'28)

Hello,

My name is Joshua Choi and I am now entering my second year of medical school. Having been able to spend this past summer researching cancer development and racial disparities has been a greatly rewarding experience and I am extremely grateful for the Summer Scholarship Stipend graciously provided by the Karin Grunebaum Cancer Research Foundation for making this possible.

This past summer, I spent my time studying immune responses to prostate cancers. I was able to investigate whether or not the presence of Th17+ cells, a subset of immune cells, can be used as a prognostic biomarker for prostate cancer across different races. Coming from an immigrant background, exploring racial differences and possible explanations for the disparities in outcomes across different races has been a subject close to heart. As I pursue a future in oncology, being able to research cancer outside of the classroom has been a fundamental experience in integrating my preclinical curriculum, academic advancements, and my hopes for future clinical treatment.

As I move into my second year of medical education, I am excited for the next steps. Continuing the research that I began in the summer, I'm eagerly looking forward to sharing our findings with the rest of the academic community (our initial results seem very promising!). Thank you again for your financial support and enabling me to pursue that which is important to me during this critical period of my medical training.

Sincerely,
Joshua Choi

Note of Thanks from Sabrina Mellinghof (MED'28)

Dear Donors of the Karen Grunebaum Foundation,

My name is Sabrina Mellinghoff, and I am the medical student whose research you sponsored this summer. I was raised in Bronxville, a small town suburb of New York City, although my father is from Germany and my mother from Hong Kong. I went to Yale for college where I studied History of Science and Medicine and ran the 200m dash on our track and field team. Now I am going into my second year at BU medical school.

I so greatly appreciate the Karen Grunebaum Foundation, not only for your generous funding of my summer project, but for your overall mission of battling cancer. Cancer is a truly formidable and terrifying opponent- I know this in part because my father is a brain cancer doctor and researcher. He specifically treats glioblastoma patients, and right now that is cancer without a cure- this is what he is working toward in his lab.

I feel that it is also my calling to become an oncologist, and of course it is a long road to get there (internal medicine residency, fellowship in hematology and oncology, and then potentially further specialization from there). I am not sure what type of cancer I want to treat, but this summer I did a deeper dive into breast cancer through my project with Drs. Julie Palmer and Mollie Barnard at the Slone Epidemiology Center. We studied recurrence of breast cancer in the Black Women's Health Study and are working to write a paper that would hopefully set the standard in imputing recurrence date from death data.

Thank you so very much!
Sabrina

Your Support is Vital to our Mission

The KGCRF relies soley 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.
You can make your contribution via check or please use our website to donate via PayPal at https://www.grunebaumfoundation.org/html/SupportContributions.asp

I enclose my gift of:

❑ $50 ❑ $100 ❑ $150

❑ $250 ❑ $500 ❑ $1000

❑ Other: $_____

Name: _____

Street Address:_____

City, State, Zip:_____

Country: _____

Telephone: _____

Email: _____

Please return to: **KGCRF**
64 Dodge St, Unit 15
Beverly, MA 01915

You may also donate online at:
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