October 2025 | Issue 17

Biomedical Genetics Section

Quarterly Newsletter

Section Updates

Congrats on your new R01 grant, Xiaoling Zhang!

Project Title: ***tRNA modifications regulating mitochondrial function and the mitochondrial proteome in AD/ADRD***

This NIH R01 grant provides $781,008 for the first year of support, with a potential total budget of ~$3.7 million over five years.

This study aims to identify AD-associated genetic variants and tRNA modifications across multi-ancestry datasets and human brain samples. Top candidate tRNAs and modifying enzymes will then be functionally evaluated in a 3D human assembloid model to uncover mechanisms of neurodegeneration and potential therapeutic targets.

Meet our Genetics Core Lab Technicians

We receive, store, and process human samples (blood, saliva, brain, placenta, and feces) to support research from various BU-affiliated projects. These include HOPE, which focuses on genetic and lifestyle risk factors for Alzheimer's-related dementias (ADRD), the New England Centenarian Study, which aims to understand the role genes play in determining exceptional longevity in humans, and the Black Women's Health Study, which seeks to create a better understanding of illness rates in medically under-represented populations. We store tens of thousands of unique DNA, plasma, and serum samples in our lab and receive over a dozen additional samples each week.

Miriam

I graduated from Vassar College in 2022 with a B.A. in Biology and am currently working on PhD applications to pursue a higher degree in Biostatistics! In the meantime, I have been taking math classes through BU's tuition remission program and am interested in programming and method development. Working at the Gencore lab has given me opportunities to explore both through maintaining our various online databases and optimizing the many procedures we follow for sample processing. Before working at Gencore, I worked for a pharmaceutical development company, and before that, I worked in Costa Rica studying frogs!

Maria

I graduated from Hobart and William Smith Colleges in 2022 with a B.S. in Biology, and a B.A. in English. I am currently enrolled part-time at Boston University's M.S. Biostatistics program in the School of Public Health. Before working at the Gencore lab, I was a research technician at MGH in the Molecular Pathology department studying different lung and pancreatic cancers. Working at the Gencore lab has allowed me to build on my research interests, and I take pride in the implications of the work we do at Gencore!

A Farewell to our Postdoc

In August, we sent off our postdoctoral associate, Moonil Kang, who has been part of our Genetics section since 2020. We are excited that he will continue collaborating with Boston University as he begins his new role as Assistant Professor at Sookmyung Women's University.

A Message from Moonil

The days in Boston are some of the best times of my life. I miss you all and the time in Boston already.
It has been an honor and a pleasure to work with you all over the past five years. I've learned so much from you, and I thank you for all your support. Wishing you all the very best for the future. Please keep in touch!

Sincerely,
Moonil Kang

September 18th: We Welcomed Our Guest from Rutgers University, Dr. Mark Gluck

Thank you to those that came to hear Dr. Gluck's talk on **Pathways to Healthy Aging:** ***How Lifestyle, Genetics, and Environment Interact to Promote Resilience Against Alzheimer's Disease in Older African Americans.***

https://brainhealth.rutgers.edu/

An Opinion Piece In the News By Shoumita Dasgupta

The Trump administration's approach to autism is tangled up with ableism, eugenics, and pronatalism

Eliminating neurodiversity is a dangerous goal

Oct. 3, 2025

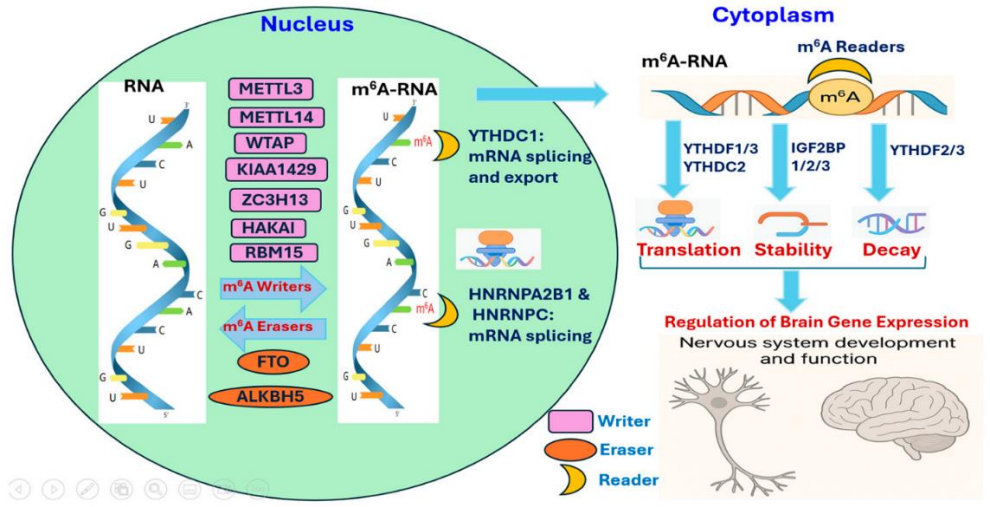
President Trump and health secretary Robert F. Kennedy Jr. have characterized the rise in autism diagnoses in recent years as an epidemic requiring emergency intervention.

This approach is factually wrong: The broadening definition of autism and the improvement in diagnosis in the same time period is largely responsible for the rise. But it's worse than a simple factual error. Using this framing dehumanizes autistic people in ways that echo the eugenic policies of the early 20th century and is just the latest iteration of politicizing and codifying harmful eugenic frameworks.

Read More

Some Recent Publications

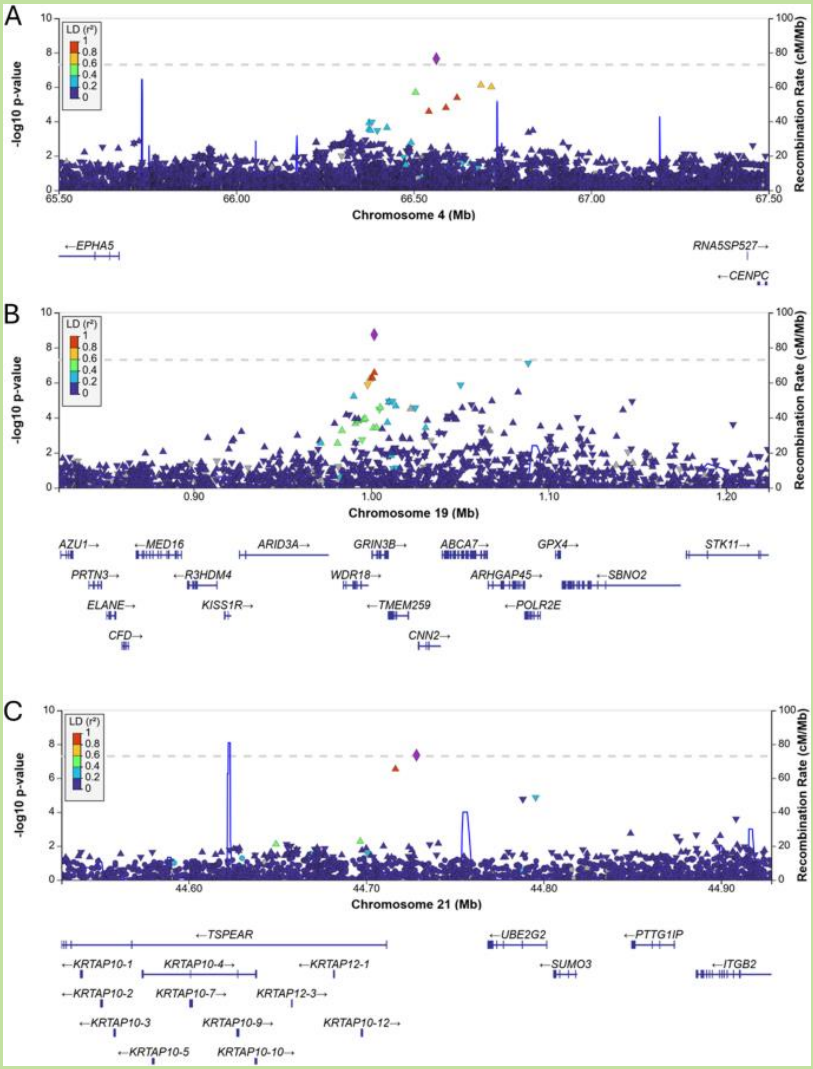
Koo JS, Zhang H. Decoding the role of m6A RNA methylation regulators in psychiatric and substance use disorders. Neurochemistry International. 2025; 190:106035. PMID: 40876784. Doi: 10.1016/j.neuint.2025.



Abstract

N^6-methyladenosine (m^6A) is the most prevalent internal modification in eukaryotic messenger RNA (mRNA) and plays a vital role in post-transcriptional gene regulation. In recent years, m^6A has emerged as a pivotal epitranscriptomic signal involved in neural development, synaptic remodeling, and the molecular pathophysiology of neuropsychiatric disorders. In this review, we summarize the mechanisms underlying the deposition, removal, and recognition of m^6A by dedicated methyltransferases, demethylases, and RNA-binding proteins. We further explore how these dynamic modifications influence neuronal differentiation and memory formation. Recent studies have linked aberrant m^6A regulation to psychiatric conditions such as depression, anxiety, schizophrenia, and bipolar disorder. Additionally, we discuss how pharmacological or genetic modulation of m^6A pathways may promote adaptive neural plasticity and enhance cognitive and emotional resilience. Despite these promising findings, significant challenges remain in achieving spatial and temporal specificity while minimizing off-target effects in the brain. Therefore, we advocate for more in-depth investigations into m^6A function within developmentally defined neural circuits to better understand its enduring role in maintaining neural homeostasis.

Sherva, R., Zhu, C., Zhang, R., Mez, J., Hauger, R., Merritt, V. C., Panizzon, M., Gaziano, J. M., Catanzaro, V., Schellenberg, G. D., Pericak-Vance, M., Haines, J. L., Wang, L.-S., Mayeux, R., VA Million Veteran Program, Farrer, L. A., Logue, M. W. Genome-wide association studies of Alzheimer's disease and related disorders stratified by sex, onset age, and Apolipoprotein E genotype reveal novel risk loci in African Americans. *Alz Res Therapy* 17, 171 (2025). https://doi.org/10.1186/s13195-025-01782-y.



Background

Alzheimer's disease (AD) risk variants have been identified in European ancestry cohorts that have stronger effects at certain ages, in individuals with a specific sex, or in those with specific isoforms of *APOE*, the strongest AD risk locus. However, sample sizes in African ancestry (AA) cohorts have been underpowered to perform stratified analyses.

Methods

We generated genome-wide association study datasets stratified by sex, age at onset (< 75 vs ≥ 75), and *APOE*-ε4 carrier status in AA cohorts from MVP and the Alzheimer's Disease Genetics Consortium (ADGC). Outcomes in MVP were AD and related dementias (ADRD; n = 4073 cases and 19,648 controls) and proxy dementia (i.e., reported dementia in a parent, *n* = 6216 cases and 21,566 controls) while ADGC analyses examined AD (*n* = 2425 cases and 5069 controls). The proxy dementia GWASs were included in the sex-stratified meta-analysis corresponding to the sex of the affected parent. The top genes were tested for differential expression in AA brain tissue.

Results

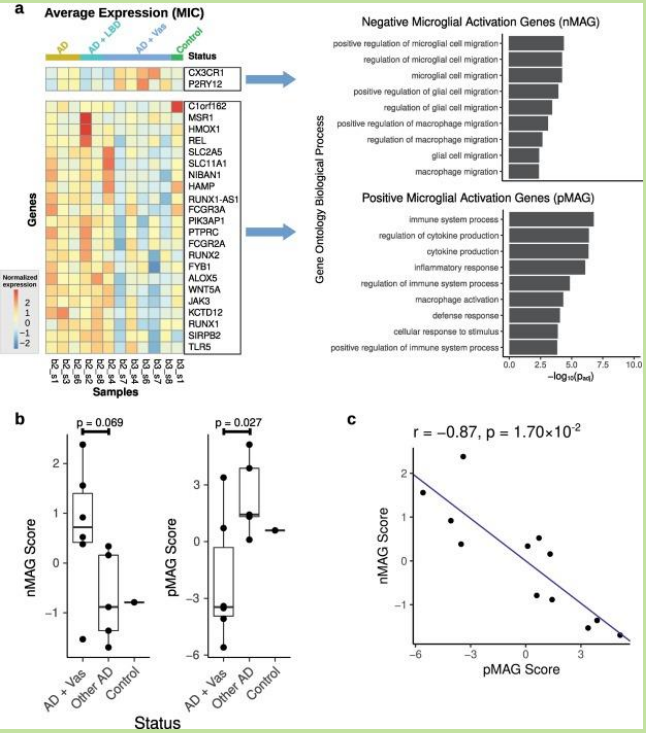
In addition to the *APOE* region, genome-wide significant associations were observed in an intergenic region near the *EPHA5* gene (rs141838133, $p = 2.19 \times 10^{-8}$) in individuals with onset < 75 years, in *GRIN3B* near the known AD risk gene *ABCA7* (rs115882880, $p = 3.83 \times 10^{-8}$) in females, and near *TSPEAR* (rs139130053, $p = 4.27 \times 10^{-8}$) in *APOE*-ε4 non-carriers. EPHA5 regulates glucose homeostasis, and ephrin receptors modify the strength of existing synapses in the brain and in pancreatic islets. It is unclear whether *GRIN3B* represents a locus distinct from *ABCA7*. Rs115882880 was

a significant eQTL for *GRIN3B* but not *ABCA7* in AA brain samples. *TSPEAR* regulates Notch signaling but has not been linked to neuronal function.

Conclusions

Age, sex, and *APOE*-stratified analyses of dementia in AA participants from two cohorts revealed potential new associations. Stratified analyses may yield critical information about the genetic heterogeneity underlying dementia risk and lead to advances in precision medicine.

Olayinka OA, O'Neill N, Empawi JA, Bock P, Hu J, Rickner H, Wong M, Stein TD, Wolozin B, Farrer LA, Zhang X. Single nucleus RNA sequencing unveils relationship between microglia and endothelial cells in mixed Alzheimer's disease and vascular pathology. Neurobiology of Disease. 2025; 216:107128. doi: 10.1016/j.nbd.2025.107128.



Abstract

Single-nucleus RNA sequencing (snRNAseq) allows for the dissection of cell type-specific transcriptional profiles. We evaluated differential gene expression using snRNAseq data generated from hippocampal region tissue donated by 11 Boston University Alzheimer's Disease Research Center (BU-ADRC) participants with neuropathologically confirmed Alzheimer's disease (AD) with or without co-existing pathology (AD only = 3, AD+vascular disease (Vas) = 6, AD+Lewy body disease (LBD) = 2). Expression of 19,893 genes was compared between AD+Vas and other AD groups for each cell type. Co-expression modules were identified in a set of 174 bulk RNAseq hippocampal samples from BU-ADRC. Modules enriched in differentially expressed genes (DEGs) were identified using Fisher's exact tests. The overlap between DEGs and co-expression modules was incorporated into quantitative gene set analysis. AD+Vas subjects showed decreased expression of genes related to immune activation in microglia ($t = -2.67$, $p = 2.72 \times 10^{-2}$). Expression of these genes was negatively associated with expression of receptors P2RY12 and CX3CR1 ($r = -0.87$, $p = 1.70 \times 10^{-2}$), which have been linked to microglial migration and activation, respectively. Expression of genes that negatively regulate angiogenesis in endothelial cells was decreased ($t = -4.84$, $p = 1.49 \times 10^{-3}$) and associated with expression of the microglial activation genes in the BU-ADRC dataset ($r = 0.68$, $p = 1.63 \times 10^{-2}$). This association and the finding of upregulation of P2RY12 in AD+Vas samples were replicated in 393 ROSMAP Study dorsolateral prefrontal cortex snRNAseq samples ($r = 0.34$, $p = 8.37 \times 10^{-12}$ and $z = 5.82$, pFDR = 8.73×10^{-6}, respectively). In summary, we found an expression profile in brain tissue from individuals with AD+Vas pathology that is associated with reduced activation and increased migration in microglia and angiogenesis in endothelial cells.

Ting-Ting Fu, Jawahar Mahendran, Bhavana Kapalli, Neha Rao, Lindsay Farrer, Lei Hou. Epigenetic regulatory network in microglia reveals context-dependent trans-regulatory programs in brain disease. European Neuropsychopharmacology. 2025; 99(Suppl 1):234. doi: 10.1016/j.euroneuro.2025.08.412.

Background

Genome-wide association studies (GWASs) have identified hundreds of loci across various brain diseases (i.e., Alzheimer's disease, AD). An increasing number of single-nucleus RNA-seq/ATAC-seq studies have revealed molecular alterations in AD and mapped with expression quantitative trait loci (eQTLs) at the cell-type level. However, how the interplay among genetic and non-genetic factors (i.e., inflammation) leads to these dysregulations remains elusive, hindering our understanding of molecular mechanisms and development of therapeutic strategies. The gap can be specified by asking three questions: 1. how does the epigenomic regulation drive cells transiting into disease associated state; 2. how are context-specific eQTL effects of disease related genetic variants modulated by transcription factors (TFs) acting downstream of non-genetic risk factors; 3. what are the regulatory programs with convergent effects of genetic and non-genetic risk factors? We hypothesize that there are limited dysregulated molecular programs under certain disease condition that might be shared across disease associated cellular contexts.

Methods

To address above questions, we build trans-regulatory programs characterizing the link among TF, cis-regulatory element (CRE) and target gene across disease-associated contexts focusing on microglia in AD. We curate ATAC-seq datasets covering human microglia under various perturbations and disease conditions related to AD. Then we calculate TF footprint score which quantifies TF binding profile across over 800 TFs for each context with TOBIAS and prioritize TF-CRE linking based on CREs with differential TF footprint scores between conditions and paired controls. Then we identify context-dependent epigenomic networks (cEpiNets) with clustering and embedding learning. Finally, we leverage cEpiNets to investigate epigenomic dynamics during microglia state transition in AD and dissect the effects of AD-associated genetic variants with contextual and functional annotations.

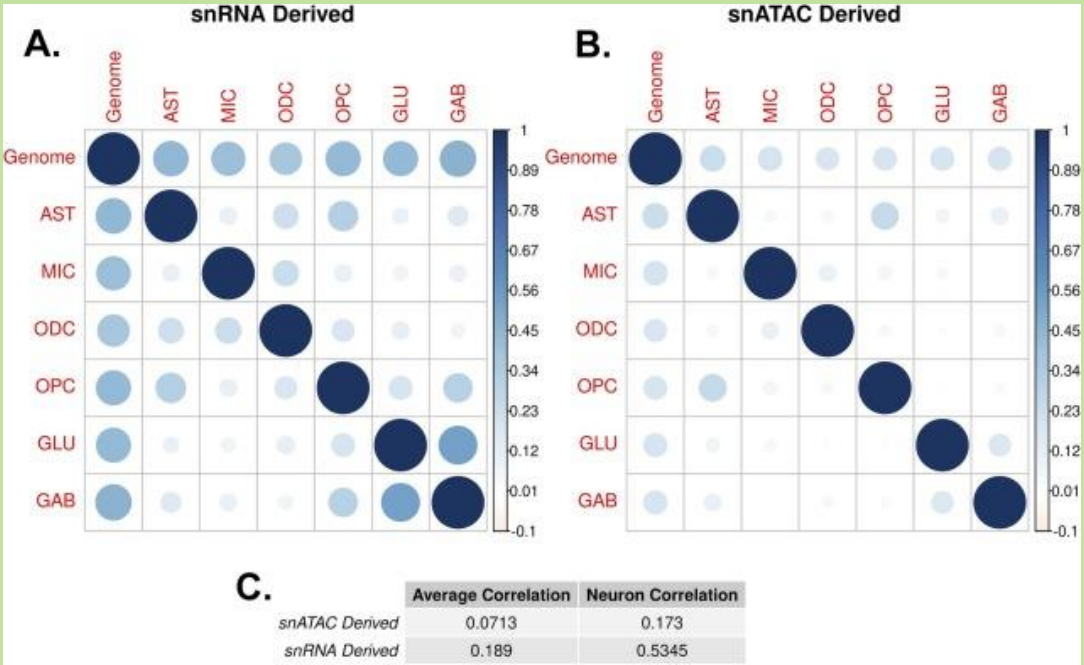
Results

We first confirm that TF footprint score can predict CRE activity and differential CRE signals well. Network modules derived from microglia cEpiNets enrich in AD-related functional pathways (i.e., beta-amyloid clearance, inflammatory response, neurotransmitter transport), validating the biological relevance of our approach. TFs like Fos- and Jun-related families show consistently increased binding overall in contexts such as AD microglia and sorted microglia with interferon (IFN)-β stimulation or AD risk variant (e.g., SORL1 KO, INPP5D) compared with control. CTCF shows decreased binding in AD but not in IFN-β-stimulated microglia. Furthermore, upregulation of TF-CRE-gene regulatory programs, including Fos-, Jun-related and other inflammation responsible TF families, drive the microglia transiting into inflammatory states, which are associated with AD in previous studies. By annotation for AD GWAS SNPs with cEpiNets, we further prioritize microglia state-specific regulatory programs which align with eQTLs enriched in AD GWAS signals, suggesting they alter effects of AD risk variants by differential TF binding.

Discussion

Overall, our work systematically chart context-dependent TF-CRE-gene networks for microglia under various contexts. This provides a comprehensive resource to dissect key regulators for functional AD risk variants, and explores the gene-by-environment interplay during pathogenesis of brain disorders.

O'Neill, N., Kurniansyah, N., Zhu, C., Olayinka, O. A., Mayeux, R., Haines, J. L., Pericak-Vance, M. A., Wang, L.-S., Schellenberg, G. D., Farrer, L. A., & Zhang, X. (2025). Multi-omic derived cell-type specific Alzheimer disease polygenic risk scores. *Neurobiology of Aging, 155*, 44–52. ISSN 0197-4580.



C.

	Average Correlation	Neuron Correlation
snATAC Derived	0.0713	0.173
snRNA Derived	0.189	0.5345

Abstract

Alzheimer disease (AD) polygenic risk scores (ADPRS) built from cell-type (ct) specific genetic variants can be used to infer cell-type contributions to AD. We derived two ct-ADPRSs using variants near single-nuclei RNA-seq (snRNA) derived cell-type specific genes or on single-nuclei ATAC-seq (snATAC) derived cell-type specific accessible chromatin regions. We generated a multi-omic ct-ADPRS for eight neuron subtypes using both single-nuclei datasets. SnATAC-derived ct-ADPRSs demonstrated considerably lower correlations among cell types (average r = 0.071) than snRNA-derived ct-ADPRSs (average r = 0.19), indicating their heightened cell-type specificity. The association of these ct-ADPRSs with AD endophenotypes was evaluated using logistic and linear regression models. Tau tangle burden was associated with astrocyte (AST) ct-ADPRS derived from snATAC (β=0.82, FDR=0.0013) and snRNA (β=0.60, FDR=0.045) as well as microglia (MIC) ct-ADPRS from both (snATAC: β=0.75, FDR=0.0047) (snRNA: β=0.63, FDR=0.028). AST ct-ADPRS was significantly associated with Mini-Mental State Examination score only when derived from snATAC data (β=-0.82, FDR=0.011). SST expressing GABAergic neuron ADPRS was strongly associated ct-ADPRS with neuritic plaque burden (β=0.087, FDR=0.0014) and the only neuron subtype ct-ADPRS significantly associated with AD endophenotypes. We investigated 1954 SNPs contributing to this ct-ADPRS and found the strongest association with variants upstream of the neuropeptide Y gene, *NPY*, particularly rs3940268 (β=-0.13, P = 8.2×10^{-5}). This association is significant even after adjusting for diffuse plaque (β=-0.12, P = 1.5×10^{-4}) or neurofibrillary tangle burden (β=-0.08, P = 3.9×10^{-3}). *NPY* was expressed in a small subset of neurons, and these findings suggest its strong impact on the association of SST+ GABAergic neurons with early AD pathology.

BUMC News

Vicki Jackson, MD, MPH Named New Chief and Chair of the Department of Medicine at Boston Medical Center and Boston University Chobanian & Avedisian School of Medicine

Dr. Jackson joins us from Massachusetts General Hospital, where she has served with distinction for more than two decades. She served as the Blum Family Endowed Chair in Palliative Care and the Chief of the Division of Palliative Care and Geriatric Medicine. Dr. Jackson was also a Professor in the Department of Medicine at Harvard Medical School, Co-Director of the Harvard Medical School Center for Palliative Care, and the founding Director for the Harvard Palliative Medicine Fellowship.

As a compassionate clinician and a visionary leader, Dr. Jackson brings a patient-centered approach, a strong academic foundation, strategic insight, and a deep personal commitment to equitable care that will greatly benefit the Department of Medicine and the broader BMC and BU communities.

New Application Structure for NIH Funded International Collaborations, Including Subawards

The National Institutes of Health (NIH) has announced in NOT-OD-25-155 a new application and award structure for NIH-funded projects with foreign components. This is a follow up to the NIH policy on Foreign Subawards previously released in NOT-OD-25-104.

For grants requesting NIH funding for one or more foreign components, NIH will require that competing applications submit applications to a Notice of Funding Opportunity (NOFO) that supports the new PF5 Activity Code for grants, new UF5 Activity Code for cooperative agreements, or another complex mechanism activity code that supports the International Project component type.

NIH will be developing and posting resources, including FAQs, as well as planning training on the new activity codes and application structure. We will share details as we receive them.

Please reach out to your Pre-Award Officer with any questions.

BOSTON UNIVERSITY
Office of the President

Important Financial Update from President Gilliam

July 7, 2025

Dear Members of Boston University's Faculty and Staff,

In the spring, we announced that we would need to make budget cuts throughout the University due to the hard reality that, like many of our peer institutions across the country, we continue to face financial headwinds. At that time, we took the preliminary step of pausing merit increases.

Recent and ongoing federal actions and funding cuts are affecting our research enterprise as well as day-to-day operations. Uncertainty remains about the full extent of potential challenges to our core mission of teaching, learning, and research. As we take part in a national renegotiation of how the country's higher education model will be funded, universities face other pressures such as rising inflation, changing demographics, declining graduate enrollment, and the need to adapt to new technologies.

Following that initial budget announcement, academic and administrative units participated in an exercise to reduce budgets as part of our Fiscal Year 2026 planning. This exercise is now complete: The University will implement an average 5% budget reduction across all units for FY2026. This will allow us to fulfill our mission at the highest level while reducing spending to align with revenue.

While we have worked hard to minimize impacts on our community, today we share the difficult news of layoffs. In coming days, the University will eliminate about 120 staff positions and about 120 vacancies. In addition, around 20 positions will undergo a change in schedule. Every effort has been made to limit the number of layoffs, and these actions will apply to approximately 1% of our employees. Still, we know that any reduction in staffing is painful.

Supervisors and Human Resources colleagues will meet with impacted individuals. Once this process is complete, schools, colleges, and unit leaders will inform their respective communities of the budget cuts and decisions made in each area.

Please know that these determinations were not made lightly and that we are committed to supporting our colleagues through this transition. Those leaving will be provided separation packages and career transitional services. We are profoundly grateful to all those involved and to the teams who are supporting them with care and respect during this transition. Resources for managers and staff are available through Human Resources business partners, the Faculty & Staff Assistance Office, and other campus programs.

This is a day of loss for all of us. There is no way around this. We know our community may need time to adjust to these difficult changes. Yet, it is also a necessary step in ensuring our future. Across our campuses, teams are already working hard to position Boston University to thrive. Over the coming months, there will be many efforts to reshape and reimagine the University in its most efficient and vital form.

Deans and unit leaders will soon share their FY2026 budgets, and while more uncertainty and challenges may come, we are optimistic that creativity and openness to new models will enable us to meet the current moment and our future.

We are grateful for the dedication, compassion, and integrity you bring to your work every day. We remain steadfast in our mission and confident in the collective talent and dedication of our community.

Thank you for all that you do to support Boston University and one another.

Sincerely,

Melissa Gilliam

BU TODAY
News, Research, Community

How Might the Federal Government Shutdown Affect BU?

Student aid will continue and researchers should work on existing grants, but new awards might be delayed. Read the article HERE.

BOSTON UNIVERSITY Office of Research

Remember: You can Find More Updates from Sponsored Programs to BU's research community HERE.

Boston University Chobanian & Avedisian School of Medicine

H-1B Visa Changes

The university has been receiving a high volume of questions around proposed changes to the H-1B visa system announced by the White House in September. The proclamation seeks to attach a $100,000 fee for new H-1B visa applications submitted after September 21, 2025, and to prioritize highly paid workers in the H-1B lottery.

There continues to be uncertainty about the proposed fee, including its applicability across various employers, and it is difficult to know exactly how this could impact the hiring landscape for the Class of 2026 and beyond. The proposed changes to federal regulations are still in flux, are subject to a public comment period, and may face legal challenges. Industry reaction is not uniform, with some expressing concerns about talent pipeline and others speculating that clearer rules could be preferable to the current lottery system. Because of this variability, it's impossible to predict how the hiring landscape may shift.

What we do know is that the uncertainty is adding stress for some international students. If you encounter a student with concerns related to their visa status, please refer them to the International Students and Scholars Office (ISSO) website and encourage them to contact their ISSO advisor. If you have a student whose career concerns are affecting their wellbeing, the Boston University Student Health Services website provides a comprehensive overview of available supports. In particular, the Faculty and Staff Guide to Helping Someone in Distress is a great tool for helping students navigate resources.

Some Helpful IS&T RCS Tutorials

Recordings and slides from past tutorials by RCS staff and vendors are available in the training section of the RCS website.

Tutorial topics include: *computer programming, data analysis, research computing basics, high performance computing, and domain specific topics.*

BUMC Upcoming Events

October 9th: World Mental Health Day

A campus-wide event and public health program that demonstrates our care for the BU community's mental health. The goals of World Mental Health Day are to call attention to the importance of checking in on your mental health, normalize mental health challenges, and remind the BU community of the many resources available to students, staff, and faculty. No registration required. Open to all BU students, staff, and faculty.

- **GSU Plaza (Charles River Campus)**
  11:00am to 3:00pm
  Rain location: GSU B03A
- **Talbot Green (Medical Campus)**
  11:00am to 2:00pm
  Rain location: INS 109 A/C

Both locations will offer:

- Free mental health screenings
- Screenings are free, confidential, and no insurance is required. Your information will not be shared with anyone unless there is a life-threatening emergency.
- The screening takes two minutes to complete. After you complete the screening and get your results, you may choose to speak with someone who will listen and direct you to relevant resources at BU and beyond.
- Resource fair - connect with resources from across campus
- Create your own self-care kit
- Engage in activities that spread positivity, encouragement, and kindness
- "Paws" & Reset with therapy dogs

Oct. 9th: BU Profiles Drop-in Q&A Session (Virtual)

BU Profiles allows you to network and collaborate with colleagues, find potential mentors, search for relevant panel members and advisory board expertise, evaluate research trends by investigator, and broadly survey the community at large while displaying your academic credentials, publications, research, and related career information to that same community.

Just drop in anytime during this Q&A session with your BU Profiles questions!

- How do grants and publications get onto my BU profile, and can I edit them?
- How do I use BU Profiles to find collaborators?
- Can you add this great new feature I thought of?
- I don't have a BU profile. How do I get one?

Date: Thursday, Oct. 9 | 2 p.m. - 3 p.m.

Zoom Link:
https://bostonu.zoom.us/j/99100702002?pwd=TTFOMmszcm9acGZMRkhjQVM0VjZIZz09

Contact: Jim Vlachos | jvlachos@bu.edu | (617) 358-1988

October 15th: ASHG 2025 Lessons from the Field

A Dialogue on Promoting Opportunity and Engagement in Human Genetics and Genomics

Wednesday, October 15: 11:45 am – 1:15 pm

Keynote speaker: Shoumita Dasgupta

This luncheon will feature interactive discussions on strategies for building a workforce where all individuals are encouraged to share their varied perspectives and experiences to enrich and benefit scientific knowledge.

Pricing: $45 ASHG member, $60 nonmember.

Registration: Ticket purchase required to attend. Includes a boxed lunch.

https://www.shoumitadasgupta.com/events/lessons-from-the-field-a-dialogue-on-promoting-inclusivity-in-human-genetics-and-genomics

October 16th: Shining Light on ARConvergence, Evans Days 2025

1:30pm – 2:30pm Keefer Auditorium

1:30-1:35pm **Introductory Notes**, Katya Ravid, DSc, Barbara E. Corkey Professor of Medicine, Founding Director, Evans Center IBR

1:35-1:50pm **Kenneth R. Lutchen, PhD, BU Convergence Science Initiative**

Senior Advisor to the President;
Dean of Engineering, Emeritus;
Professor (BME)

1:50-2:00pm **ARConvergence Approach: The Case of Precision Medicine for Alzheimer Disease**

ARC Directors: Lindsay A. Farrer, PhD, Rhoda Au, PhD, and Alice Cronin-Golomb

2:00-2:10pm ***David L. Coleman, MD, Junior Faculty Prize* Presentation**

The David L. Coleman, MD Junior Faculty Prize
is awarded to an assistant professor for special contributions to their colleagues, section and department, and in recognition of excellence in research and good citizenship as demonstrated by volunteer leadership, contributions to educational initiatives, mentorship and a commitment to diversity.

Department of Medicine Chair, 2006 – 2022

Winner to be presented by Drs. Katya Ravid and David Salant

2:10-2:30pm *Talk by* D. Coleman Prize Awardee (TBN)

Other Evans Days events (October 15-17th) can be found here:

https://www.bumc.bu.edu/medicine/research/evans-dept-of-medicine-research-days/

Resources

Don't Forget-- Submit an *Announcement Request Form* and share your news in the next issue of the Biomedical Genetics Section Newsletter!

Do you have exciting news that you want to share in the next issue of our quarterly Biomedical Genetics Section Newsletter? Submit an Announcement Request Form, located on the Resources page of our website!