OCTOBER 2026

BIOMEDICAL GENETICS SECTION

QUARTERLY NEWSLETTER | ISSUE 20

New Hires!



Emerson Lillie– Core lab

"Hi, I'm Emerson! I am originally from Grand Rapids, Michigan and attended Michigan State University where I earned my degree in Medical Laboratory Science. I worked as a generalist in a clinical laboratory before moving to Boston and starting my position here. I am hoping to get my master's degree here at BU in the future. Outside of work I enjoy running, reading, and trying new restaurants."

Section Updates

New publication from Dr. Lei Hou, PhD in Nature Communications:

Hou, L., Li, Y., Xiong, X. et al. Genotype epigenome phenotype integration reveals peripheral immune contributions to type I bipolar disorder. Nat Commun (2026). *https://doi.org/10.1038/s41467-026-72543-0*

Abstract: Immune dysfunction has been increasingly implicated in bipolar disorder, but the underlying mechanisms remain unclear. To address this, we profile 833 genome-wide chromatin immunoprecipitation sequencing datasets spanning five histone marks in peripheral blood immune cells from 88 Type I bipolar disorder patients and 92 controls, integrating them with whole-genome sequencing and clinical data. We identify disease-associated *cis*-regulatory elements and genetically influenced regulatory elements, revealing immune signatures and pathways involving calcium signaling and endoplasmic reticulum transport. By integrating genetic risk variants, differential and genetically influenced regulatory elements, and regulatory element—gene links, we prioritize 39 driver genes, 28 of which are exclusively supported by blood evidence. We further stratify patients into five epigenomic subtypes with distinct clinical features and genetic risk profiles and identify compounds that reverse disease-associated epigenomic signatures. Here, we combine immune epigenomics with genetics and clinical traits to identify driver genes, patient subtypes, and therapeutic candidates, highlighting immune contributions to type I bipolar disorder pathogenesis.

New publication from Dr. Huiping Zhang, PhD:

Koo JS, Zhang H. Stem Cell Models for Elucidating Cellular Mechanisms of Substance Use Disorders and Advancing Addiction Pharmacology. Stem Cells Int. 2026 Aug 7;2026:1717710. doi: 10.1155/sci/1717710. PMID: 42571089; PMCID: PMC13451643.

Abstract: Substance use disorders (SUDs) impose major global morbidity and mortality, yet the cellular mechanisms linking genetic risk to neural vulnerability, disrupted neurodevelopment, and drug-induced neuroadaptations remain poorly understood. Human pluripotent stem cell (hPSC) technologies, including embryonic and induced pluripotent stem cell (ESC/iPSC)-derived neurons, three-dimensional (3D) brain organoids, and organoid-on-a-chip platforms, provide scalable, human-relevant models to address this gap. ESC/iPSC-derived neuronal cultures enable interrogation of genetic and epigenetic determinants of drug susceptibility and response; cerebral organoids recapitulate tissue architecture and emergent network dynamics; and microfluidic organoid-on-a-chip systems facilitate maturation, enhance reproducibility, and enable controlled exposure paradigms. In this review, we synthesize recent stem cell-based

studies of alcohol, opioid, and stimulant exposure, highlighting insights into neurodevelopmental disruption, synaptic and signaling alterations, neuroinflammation, and network-level dysfunction. We critically evaluate limitations of stem cell-based studies, including ethical concerns, inter- and intra-line variability, incomplete cellular maturation, and difficulties in modeling complex circuitry and comorbid conditions. We propose strategies to enhance translational relevance, including standardized differentiation protocols, addition of patient-derived cells and vascular and immune components, and integration of multi-omics approaches (transcriptomics, epigenomics, and proteomics) with functional readouts to map molecular pathways underlying drug vulnerability and resilience. Finally, we outline the therapeutic and precision-medicine potential of stem cell platforms for target discovery, predictive toxicology, and individualized treatment modeling. Despite remaining challenges, stem cell-based approaches offer a powerful and increasingly tractable path from genetic association to mechanistic insight and therapeutic innovation in addiction research.

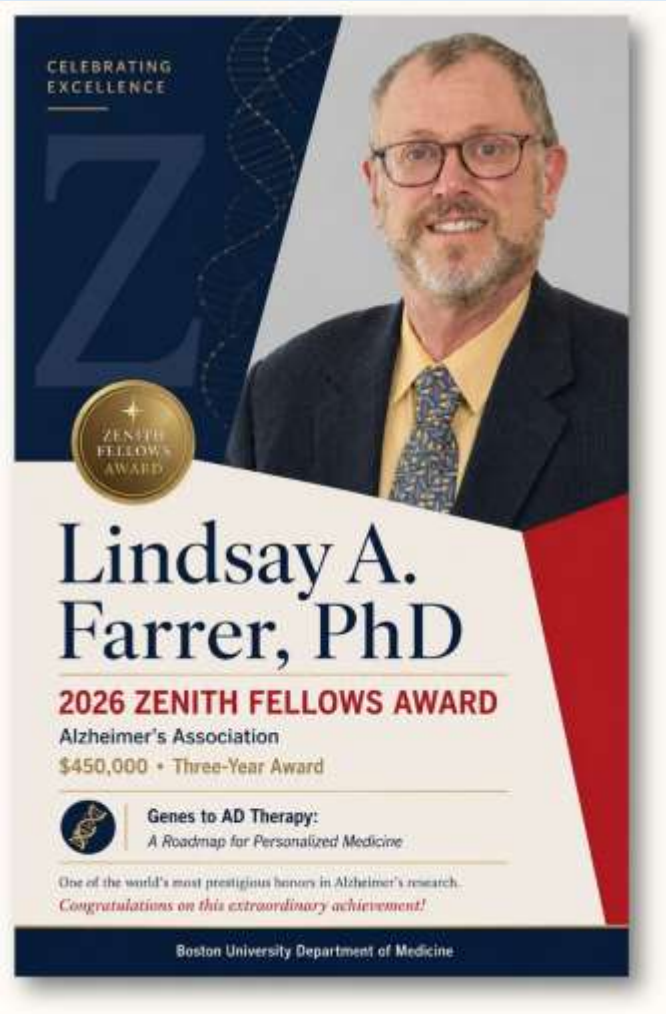
Another new publication from Dr. Huiping Zhang, PhD:

Ngo AL, Qu X, Alkhansari NG, Nguyen L, Nguyen UT, Zhang H. Dynamic m6A Methylation in Neurodevelopmental and Neurodegenerative Disorders. Epigenomes. 2026 Oct 3;10(4):62. doi: 10.3390/epigenomes10040062. PMID: 42924721; PMCID: PMC13692518.

Abstract: N6-methyladenosine (m6A), the most prevalent internal modification in eukaryotic RNA, has emerged as a key epitranscriptomic regulator in the central nervous system, coordinating gene expression programs that govern neural development, synaptic plasticity, and neuronal adaptation throughout the lifespan. This review synthesizes emerging evidence that m6A dysregulation represents a shared epitranscriptomic mechanism linking neurodevelopmental and neurodegenerative disorders, highlighting a mechanistic convergence that bridges these traditionally distinct disease categories. Dysregulation of m6A-mediated control of neurogenesis, synaptic maturation, and dopaminergic development has been implicated in neurodevelopmental disorders, including autism spectrum disorder, attention-deficit/hyperactivity disorder, and intellectual disability. In neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and Huntington's disease, m6A dysregulation mediates multiple interconnected pathogenic processes, including neuroinflammation, mitochondrial dysfunction, tau hyperphosphorylation and aggregation, α-synuclein dysregulation, and impaired neuronal repair. Environmental stressors and metabolic perturbations modulate m6A modification patterns, suggesting that gene–environment interactions contribute to disease susceptibility through epitranscriptomic mechanisms. m6A modification functions as a dynamic molecular switch governing neural plasticity and circuit function across early development, adulthood, and aging, and its dysregulation predisposes individuals to neurological disease manifestation throughout the lifespan. Advances in cell-type-specific and circuit-resolved m6A profiling will deepen our understanding of m6A dysregulation and strengthen its translational potential as both a diagnostic biomarker and a therapeutic target

in neurological diseases. Furthermore, we evaluate pharmacological and molecular strategies targeting m6A regulatory machinery, including the writers METTL3/METTL14, the erasers FTO/ALKBH5, and reader proteins, as promising therapeutic approaches to restore aberrant neuronal circuit function in neurodevelopmental and neurodegenerative disorders.

Huge congratulations to Dr. Farrer for receiving the Zenith Fellows Award and being named as a William Fairfield Warren Distinguished Professor! Click the link here for the BU article about this announcement, and here for an article about the Zenith Fellows Award.



Here are some photos from our surprise party for Dr. Farrer:







Here's a photo from the Evans Day reception!



This quarter, our section was awarded a few grants. Here they are:

- U19 AG068753 Precision Monitoring and Assessment in the Framingham Study: Cognitive, MRI, Genetic and Biomarker Precursors of AD & Dementia 08/01/2026-07/31/27 PI Lindsay Farrer
- R01AG048927 Alzheimer Disease Genetic Architecture in African Americans 09/01/26-05/31/27 PI Lindsay Farrer
- Genes to AD Therapy: A Roadmap for Personalized Medicine 11/1/26-10/31/29 Agreement with Alzheimer's Association, PI Lindsay Farrer
- Development and Validation of an AI Care Service for Dementia Prevention Based on a Large-Scale Brain-Omics-Language Foundation Model (Data Processing, Standardization, and Analysis - Agreement with Chosun University 05/01/26-11/30/27 PI Lindsay Farrer
- 2P30AG072978 Boston University Alzheimer's Disease Research Center 09/01/26-06/30/27 M-PI: Dr. Jun and Dr. Farrer

If you would like more information, please email Emma at emsteele@bu.edu.

BUMD News and Events



The Staff Advisory Council is putting on another Staff Appreciation Fair at BUMC on **October 29, 2026, in the Hiebert Lounge** on the 14th floor:

"You're invited to the Staff Appreciation & Resource Fair on the BU Medical Campus! Enjoy some fall drinks & snacks, revel in the jams of the BU Bands jazz trio, all while you learn more about the amazing resources available to BU staff. We will be holding both a warm coat drive and a canned food drive in support of the BU community. There will be donation drop-off bins at the event. We encourage you to find more information about what to donate at the above links."

Register for the event here.

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!

Have a great fall!

