Research Advancements New Faculty



Icahn SchoolThe Mindichof Medicine atChild Health andMountDevelopment Institute

MCHDI Developmental Outcomes

Research Advancements: Artificial Intelligence

Launching the Center for Artificial Intelligence in Children's Health

A rtificial Intelligence (AI) holds enormous promise for revolutionizing patient care, yet its journey from discovery to the bedside has repeatedly been impeded, limiting real-world impact. Many AI tools, developed both within and outside healthcare contexts, produce archives, multi-omics repositories, and real-time physiologic monitoring for this population. This infrastructure will serve as the cornerstone for interdisciplinary teams of researchers, clinicians, and data scientists to collaborate efficiently, accelerating the translation of AI models into practical clinical applications.

errors, embed biases, and/or generate unsafe recommendations when applied to actual patients. Rigorous development, including leveraging diverse datasets and multi-site, external validation, is essential to ensure both safety and efficacy. These challenges are amplified in pediatrics, where heightened ethical standards, more



The Center will initiate multiple prospective trials across pediatric specialties. A flagship project involves real-time video monitoring in Neonatal Intensive Care Units, where AI-driven analytics of video data from premature infants automatically detect early neurological and respiratory events, delivering actionable insights to caregivers in real time. In parallel, the Center will advance AI-enabled

Figure: Governance in Pediatric AI is intertwined with pediatric biodevelopment.

specialized FDA Software as a Medical Device (SaMD) regulations, and limited data availability present additional hurdles. In specialized environments like the pediatric intensive care units, critical data streams are all too often deprioritized for research, creating dangerous blind spots that undermine pediatric AI development

To address this gap, the Mindich Child Health and Development Institute and the Windreich Department of AI and Human Health have launched the Center for Artificial Intelligence in Children's Health. Under the leadership of Benjamin Glicksberg, PhD, the Center is committed to advancing pediatric AI research and translational science, specifically, conducting prospective clinical trials to evaluate AI-enabled tools for safety, efficacy, and seamless integration into specialized workflows before deployment.

Central to the Center's mission is the creation of a pediatric AI data hub- a unified platform integrating electronic health records, imaging

Benjamin Glicksberg, PhD

Founding Director, Center for Artificial Intelligence in Children's Health Associate Professor, Windreich Department of Artificial Intelligence and Human Health Mindich Child Health and Development Institute Hasso Plattner Institute for Digital Health at Mount Sinai The Charles Bronfman Institute for Personalized Medicine personalized medicine efforts by integrating multi-omic data collected via synergistic initiatives within Mount Sinai. These integrated insights can better facilitate early identification of rare pediatric diseases, drive novel drug target discovery, and support tailored treatment strategies based on each child's unique biological profile. Beyond clinical research, Al-driven health-economic analyses will identify care delivery gaps, inform resource allocation, and optimize operational performance within the pediatric health system.

Collaboration underpins every initiative, and this Center is no exception. The team will facilitate a vibrant community of stakeholders, clinicians, nurses, informaticians, geneticists, and industry partners, providing curated datasets, specialized software, and strategic mentorship to support innovation at all levels. By lowering barriers for both newcomers and experts, the Center will cultivate a vibrant innovation ecosystem, enabling promising concepts to progress swiftly yet safely from conception to clinical practice.

Mount Sinai's Center for Artificial Intelligence in Children's Health represents a decisive, bold step toward a future where Al-driven insights enhance diagnosis, personalize therapies, and improve outcomes for young patients. Through rigorous validation, regulatory excellence, and interdisciplinary collaboration, the Center aims to set a global standard for pediatric Al research and application, ultimately shaping a healthier future for children everywhere, starting with those who come through Mount Sinai's doors.

Mapping Ion Channel Isoforms in the Developing Brain

Peurogenesis, the process by which new neurons are generated and integrated into circuits of the developing brain, encompasses a complex choreography of precisely timed events. As we described in a recent review¹, newborn neurons in the embryonic cerebral cortex first emerge from the division of less specialized, immature cells called neural stem and progenitor cells (NSPCs). This process of neuronal differentiation proceeds sequentially and culminates with young neurons acquiring their final identities, which are comprised of a combination of individual properties, including gene expression and patterns of electrical activity. Disrupting any step in this elaborate sequence of developmental events can give rise to neurodevelopmental conditions, including intellectual disability and autism spectrum disorder (ASD).

NSPCs in the embryonic brain are influenced by factors in their environment, including electrical signals. We have shown that specific forms of a disease-associated ion channel – a protein at the cell membrane that converts electrical signals into long-lasting biochemical changes -inversely change their expression during neuronal differentiation in the cortex^{2,3}. We hypothesize that similar timed regulation of a broad array of ion channels allows immature cells to dynamically adjust their electrical properties as they are exposed to changing environmental signals during brain development. Mutations linked to neurodevelopmental disorders can disrupt this regulation, ultimately resulting in cellular phenotypes that underlie disease. We previously focused on an inherited developmental disorder called Timothy Syndrome (TS), characterized by abnormalities in many parts of the body, including the heart and brain^{2,4}. A majority of children affected by TS display features of ASD, including impairments in social interaction and delayed language development⁵. Classical TS (TS1) is caused by a mutation in the CACNA1C gene, which encodes the calcium channel Cav1.2. Like many other ion channel genes linked to neuropsychiatric disorders, CACNA1C can produce different versions of



Cav1.2 channels (i.e., isoforms) through RNA splicing. The TS mutation is present

Georgia Panagiotakos, PhD Associate Professor, Psychiatry and Neuroscience in one of two spliced exons (exon 8A) of *CACNA1C*, and we found that Cav1.2 channels containing this exon are more abundant in immature NSPCs compared to mature neurons. The presence of the TS mutation impairs splicing, causing channels containing the 8A exon (and thus also the TS mutation) to continue to be expressed at higher levels as NSPCs differentiate into neurons. This results in persistent calcium elevations that contribute to impaired differentiation in neurons from TS individuals.

More recently, our group has been working towards mapping the full spectrum of developmental splicing events in ion channel genes during neuronal differentiation using the developing mouse brain as a model. Our goal is to use these data to better understand when and where diseaseassociated mutations are likely to exert their effects. Using a labeling strategy in embryonic mice that allows us to collect neural stem and progenitor cells as well as young neurons, coupled with long- and short-read RNA sequencing approaches, we have begun to define specific forms of *Cacna1c* and other ion channel genes that change as NSPCs differentiate into neurons. We are now working towards characterizing the properties of the channels produced by these different *Cacna1c* isoforms and investigating how they might impact distinct aspects of neuronal differentiation.

References:

- 1. Arjun McKinney A, Petrova R and **Panagiotakos G**. Calcium and activity-dependent signaling in the developing cerebral cortex. *Development*. 2022 Sep 1;149(17):dev198853.
- 2. **Panagiotakos G,** et al. Aberrant calcium channel splicing drives defects in cortical differentiation in Timothy syndrome. *Elife.* 2019 Dec 23;8:e51037.
- 3. **Panagiotakos G** and Pasca SP. A matter of space and time: Emerging roles of disease-associated proteins in neural development. *Neuron.* 2022 Jan 19;110(2):195-208.
- 4. Splawski I, et al. Ca(V)1.2 calcium channel dysfunction causes a multisystem disorder including arrhythmia and autism. *Cell.* 2004 Oct 1;119(1):19-31.
- 5. Levy RJ, et al. A Cross-Sectional Study of the Neuropsychiatric Phenotype of CACNA1C-Related Disorder. *Pediatr Neurol.* 2023 Jan;138:101-106.

Pilot Project: 2025 Awardees

Project Title: Identifying Fibrogenic Drivers in Pediatric Cholestatic Disorders: Insights From Explanted Livers at Transplantation

Principal Investigators: Jaime Chu, MD (Communicating PI), Judy Cho, MD (Co-PI), John Hong, MD (Co-Investigator), and Jake Herb, MD/PhD Candidate (Co-Investigator)

Abstract: Cholestasis is impaired bile flow resulting from biliary cell (cholangiocyte) injury or dysfunction. In children, chronic cholestasis leads to progressive liver damage, liver scarring (fibrosis), and the need for liver transplantation. Despite advances in the diagnosis of genetic cholestatic disorders, our understanding of the pathologic drivers in these heterogenous conditions remain limited. A comprehensive dissection of the fibrogenic processes is urgently needed as hepatic fibrosis is the major determinant of mortality in these children. This pilot study aims to elucidate the fibrotic mechanisms underlying liver disease in three pediatric cholestatic disorders: biliary atresia (BA), progressive familial intrahepatic cholestasis (PFIC), and primary sclerosing cholangitis



(PSC) by directly examining end-stage livers that are removed from children at the time of their liver transplant here at Mount Sinai.

Jaime Chu, MD (Communicating PI) Associate Professor, Pediatrics Collaboration between Pediatric Liver Transplantation and Translational Genetics offers unparallelled and unique capabilities to investigate these life-threatening pediatrics disorders. Utilizing single-cell transcriptomic analysis, we will investigate diseasespecific drivers of hepatic fibrosis at the cellular level. We will perform single-cell RNA sequencing (scRNAseq) on liver explants collected from children at the time of liver transplantation, analyzing tissue from 15 patients (5 per group), and compare to scRNAseq datasets from normal liver. This analysis will allow us to identify unique fibrogenic landscapes, enriched pathways, and potential cell-cell interactions. Additionally, we will explore the role of cholangiocyte injury in activating hepatic stellate cells (HSCs) and driving fibrosis. By profiling the cholangiocyte secretome in BA and PFIC, we aim to understand how cholangiocyte-derived factors contribute to HSC activation and liver fibrosis.

This study represents the first comprehensive comparison of cholestasis-mediated fibrosis in pediatric liver disease through single-cell analysis. By investigating the interplay between cholangiocytes and HSCs, we hope to uncover novel therapeutic targets for antifibrotic treatments, ultimately improving outcomes for children with these debilitating disorders.

Project Title: Studying the Molecular Mechanisms of Human Beta Bell Replication in Insulinomas Using Single Cell Approaches

Principal Investigators: Esra Karakose, PhD (Communicating PI) and Robert Sebra, PhD (Co-PI)

Abstract: All forms of diabetes ultimately result from insufficient numbers of insulin-producing beta cells. Thus, beta cell regenerative drug therapies could provide a scalable and affordable approach for millions of people with diabetes. The most advanced beta cell regenerative therapies include inhibitors of the kinase, DYRK1A, exemplified by harmine, alone, or combined with GLP-1 receptor agonists (GLP-1RA's). On the other hand, intensive genomic, epigenetic, and transcriptomic analyses of insulinomas - benign pancreatic adenomas that grow and over-secrete insulin - compared to pure FACS-sorted human beta cells has revealed other potentially druggable targets and pathways for human beta cell regeneration. In-depth analyses on insulinomas has also revealed clear epigenetic misregulation, including altered enhancers and accessible chromatin. However, all these experiments have been performed on 'bulk' tissue. Insulinomas include complex mixtures of cell types that create substantial 'noise' in bulk sequencing. Thus, in Aim 1, "To Determine Epigenetic and Transcriptomic Profiles of Human Insulinomas Using Single Cell Approaches," I will study cell type-specific gene expression patterns and accessible chromatin regions in proliferating vs. quiescent human beta cells in insulinomas. In Aim 2, "To Validate Molecular Targets that Induce Human Beta Cell Proliferation," I will silence or overexpress the selected molecular targets from Aim 1 to understand their effect on human beta cell proliferation and function. These two aims will collectively be instrumental to identify novel druggable targets and pathways for the treatment of diabetes.



Esra Karakose, PhD (Communicating PI) Assistant Professor, Medicine

Pilot Project: 2025 Awardees

Project Title: The Roles of Pancreatic Sensory Circuits in Pancreatic Inflammation

Principal Investigators: Sarah Stanley, MB, BChir, PhD (Communicating PI), Hongzhen Hu, PhD, MS (Co-PI) and Kris Beaumont, PhD (Co-I)

Abstract: Pancreatitis -- inflammation of the pancreas -- affects a growing number of children, many of whom develop recurrent or chronic inflammation. Pediatric pancreatitis results in severe symptoms with significant effects on quality of life. Current therapies aim to reduce symptoms, such as pain and pancreatic enzyme insufficiency. However, treatment is often suboptimal and does not alter disease progression. Better therapies are needed for symptom control and to mitigate pancreatic inflammation.

The pancreas is densely innervated by vagal and spinal sensory



nerve fibers. Published studies suggest remodeling and hyperactivity of pancreatic sensory nerves contribute to pancreatitis symptoms and inflammation. Therefore, modulating sensory nerve

Sarah Stanley, MB, BChir, PhD (Communicating Pl) Associate Professor, Medicine and Neuroscience

activity may offer new approaches to treat pancreatitis symptoms and slow disease progression. However, our knowledge of the effects of pancreatitis on the architecture and gene expression of pancreasinnervating vagal and spinal sensory circuits is limited. Further, we do not know the precise contributions of pancreatic vagal and spinal sensory circuits to the symptoms and pathology in pancreatitis. Our proposal will address these gaps in our understanding of sensory nerve structure, gene expression and function in juvenile pancreatitis. Our hypothesis is that distinct pancreatic sensory neural circuits contribute to specific symptoms and pancreatic inflammation in pancreatitis. To test our hypothesis, we will determine the effects of pancreatitis on a) the 3D structure and gene expression of pancreatic vagal and spinal sensory and b) the function of pancreatic vagal and spinal sensory nerves to regulate inflammation, pancreatic function, pain and behavior in juvenile mice. Our multi-disciplinary team will use innovative imaging, spatial transcriptomic and targeted neuromodulation tools to generate a comprehensive understanding of pancreatic sensory nerve structure and function in pancreatitis. These studies will form a critical foundation for future work identifying therapies to treat the symptoms and delay disease progression in juvenile pancreatitis.

New Extramural Faculty

Benjamin Glicksberg, PhD

Benjamin Glicksberg, PhD is a leading researcher at the nexus of digital health, artificial intelligence, and clinical informatics. He develops advanced computational methods for large-scale biomedical data, including electronic health records (EHR), genomics, and physiologic waveforms to drive precision medicine and improve patient outcomes. Through interdisciplinary collaborations across academia, industry, and clinical settings, he bridges research and practice, translating AI breakthroughs into real-world tools. He holds numerous patents on multi-modal Al applications licensed by startups and public companies and created open-source software that leverages common data models to enhance EHR research generalizability. From his experience co-founding and advising health-tech startups, Dr. Glicksberg will also support entrepreneurial initiatives at Mount Sinai, facilitating the development and commercialization of Al-driven health technologies. Before joining Mount Sinai, he served as Vice President and Head of Data Science and Machine Learning at Character Biosciences, a genomics-based drug discovery startup. There, he derived digital biomarkers of disease progression from clinical and genomic trial data, identifying novel drug targets now entering clinical trials. Dr. Glicksberg has authored 190 peer-reviewed publications with over 18.000 citations and mentored medical and

Key Publications:

- Klang E, Apakama D, Abbott EE, Vaid A, Lampert J, Sakhuja A, Freeman R, Charney AW, Reich D, Kraft M, Nadkarni GN, **Glicksberg BS**. A strategy for cost-effective large language model use at health system-scale. *NPJ Digit Med*. 2024 Nov 18;7(1):320.
- 2. **Glicksberg BS**, Timsina P, Patel D, Sawant A, Vaid A, Raut G, Charney AW, Apakama D, Carr BG, Freeman R, Nadkarni GN, Klang E. Evaluating the accuracy of a state-of-the-art large language model for prediction of admissions from the emergency room. *J Am Med Inform Assoc*. 2024 Sep 1;31(9):1921-1928.
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- 4. Vaid A, Johnson KW, Badgeley MA, Somani SS, Bicak M, Landi I, Russak A, Zhao S, Levin MA, Freeman RS, Charney AW, Kukar A, Kim B, Danilov T, Lerakis S, Argulian E, Narula J, Nadkarni GN, **Glicksberg BS**. Using Deep-Learning Algorithms to Simultaneously Identify Right and Left Ventricular Dysfunction From the Electrocardiogram. *JACC Cardiovasc Imaging*. 2022 Mar;15(3):395-410.
- 5. Lee SJ, Cho L, Klang E, Wall J, Rensi S, **Glicksberg BS**. Quantification of US Food and Drug Administration Premarket Approval Statements for High-Risk Medical Devices With Pediatric Age Indications. *JAMA Netw Open*. 2021 Jun 1;4(6):e2112562.

New Extramural Faculty-Continued

graduate students, postdocs, engineers, and scientists. As Principal Investigator or Co-Investigator, he secured more than \$15 million in funding from industry, foundations, and government, including the National Library of Medicine. He received a bachelor's degree in neuroscience from Skidmore College, a PhD in neuroscience from the Icahn School of Medicine at Mount Sinai, and completed a postdoctoral fellowship at UCSF. At Mount Sinai, he is developing novel AI frameworks to streamline clinical workflows and advance precision - especially in pediatrics.

Benjamin Glicksberg, PhD

Associate Professor, Windreich Department of Artificial Intelligence and Human Health



Amy Rapp, PhD

Amy Rapp, PhD is an Assistant Professor in the Division of Tics, OCD, and Related Disorders in the Department of Psychiatry. A licensed clinical psychologist, she received her Ph.D. from the University of California, Los Angeles. Dr. Rapp completed a NIMH T32 postdoctoral research fellowship in the Center for OCD and Related Disorders at Columbia University Irving Medical Center/ New York State Psychiatric Institute and afterwards, was appointed to Assistant Professor. She joined the faculty of the Icahn School of Medicine at Mount Sinai in October 2024.

The overarching goal of Dr. Rapp's research is to mitigate the deleterious long-term impact of childhood-onset psychiatric disorders by leveraging the power of individual differences to inform personalized early intervention and prevention strategies that could be delivered during sensitive developmental periods (i.e., adolescence). Toward this goal, her research attempts to identify reliable and robust neural, cognitive, and environmental factors that are associated with transdiagnostic symptom dimensions of anxiety, compulsive, and related disorders. She strives to harness the clinical utility of individual differences by using precision-analytic research



methods like theory-driven computational modeling and trial-by-trial analysis of EEG. Future directions for her program of research include

Amy Rapp, PhD Assistant Professor, Psychiatry

Key Publications:

1. Rapp AM, Ashinoff BK, Baker S, Simpson HB*, Horga G*. Transdiagnostic anxiety-related increases in information sampling are associated with altered valuation. *Comput Psychiatr.* 2024 Nov 6;8(1):202-216. (**Denotes co-last authors*)

2. **Rapp AM**, Tan PZ, Grammer JK, Gehring WJ, Miller GA, Chavira DA. Cultural values influence relations between parent emotion socialization and adolescents' neural responses to peer rejection. *Res Child Adolesc Psychopathol.* 2022 Feb;50(2):255-267.

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identifying how and when neurocognitive alterations emerge in development and translating this information into targeted, individualized interventions for youth.

Ellerie Weber, PhD

Ellerie Weber is a Health Economist and an Assistant Professor in the Department of Population Health Sciences and Policy at the Icahn school of Medicine at Mount Sinai. She received her PhD in Business Economics from the University of Chicago's Booth School of Business, and completed a post-doctoral fellowship at RAND-University of Pittsburgh's Health Institute. Her research applies principles of microeconomics and econometric techniques to study the health sector, particularly how health policies affect prices, competition and health outcomes. She has published

Key Publications:

- 1. Weber E, Kim H, Ng A, Howell FM, Fox A, Janevic T. Medicaid Eligibility Gaps and Pandemic-Era Postpartum Insurance Rates. *JAMA Health Forum.* 2025 Mar 7;6(3):e250109.
- Janevic J, Birnie L, Belfon K, Glenn L, Maru S, Reynolds S, Fox A,
 Weber E. Immigrant Inequities in Uninsurance and Postpartum Medicaid Extension: A Quasi-Experimental Study in New York City, 2016–2021. *American Journal of Preventative Health.* May 2025.

on topics relevant to current health policies, including Medicaid, telehealth, price transparency, and hospital-insurer bargaining. Dr. Weber's current projects focus on maternal health and health equity, specifically looking at how Medicaid continuous coverage provisions granted to postpartum people under the Families First Coronavirus Response Act impacted beneficiaries' insurance coverage and heath care utilization. She is also an expert in cost analyses and helps support clinicians, community-based organizations, and other researchers assess the cost effectiveness of their particular practices or interventions. In addition to her PhD, Dr. Weber received an MBA the University of Chicago's Booth School of Business and a



Bachelor's of Science degree in Economics from

Ellerie Weber, PhD Assistant Professor, Population Health Sciences and Policy

- Weber E, Peskin MF, Markham CM, Shegog R, Baumler ER, Addy RC, Temple JR, Hernandez B, Cuccaro P, Thiel MA, Gabay EK, Tortolero Emery S. Economic Evaluation of an Intervention to Prevent Adolescent Dating Violence (Me & You). *J Interpers Violence*. 2023 Feb;38(3-4):2983-3010.
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- 5. Weber E, Floyd EJ, Kim Y, White C. Peering Behind the Veil: Trends in Types of Contracts Between Private Health Plans and Hospitals. *Medical Care Research & Review.* 78(3), July 23 2019.

the London School of Economics. A native New Yorker, she is a mom of three boys and enjoys spending time in nature with her family.

New Intramural Faculty

Prashanth Rangan, PhD

Prash Rangan, PhD earned his PhD in Biophysics from Johns Hopkins University and completed a postdoctoral fellowship in Developmental Biology. He began his academic career at SUNY Albany and joined Sinai as an Associate Professor three years ago. At Sinai, the Rangan lab focuses on unraveling the mechanisms that govern the formation of maternal contribution-a critical process for initiating new generations. Maternal contribution involves the transmission of crucial cellular components and regulatory molecules necessary for proper embryonic development. We utilize a multidisciplinary approach to explore how maternal components are generated during oogenesis and refined throughout oocyte maturation. Our primary goal is to understand how aged or inappropriate components are identified and removed during this maturation process. Additionally, we study the effects of any residual, uncleared components on fertility and childhood development. By combining cutting-edge genetic tools, advanced cellular biology, and state-of-the-art developmental methodologies, we are reshaping our understanding of oocyte maturation and the early stages of embryonic development. Our research not only deepens our insight into the basic biology of reproduction but also challenges previous paradigms by linking these molecular events to long-term fertility outcomes and overall developmental health. Our findings also have the potential to identify critical biomarkers for reproductive health and provide new therapeutic targets for addressing devel-



opmental anomalies, ultimately advancing our approaches to fertility restoration and childhood health interventions.

Prashanth Rangan, PhD

Associate Professor of Cell, Developmental, and Regenerative Biology and Pediatrics

Key Publications:

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- 3. Sarkar K, Kotb NM, Lemus A, Martin ET, McCarthy A, Camacho J, Iqbal A, Valm AM, Sammons MA, Rangan P. A feedback loop between heterochromatin and the nucleopore complex controls germ-cell-tooocyte transition during Drosophila oogenesis. *Dev Cell*. 2023 Nov 20;58(22):2580-2596.e6.
- 4. Martin ET, Blatt P, Nguyen E, Lahr R, Selvam S, Yoon HAM, Pocchiari T, Emtenani S, Siekhaus DE, Berman A, Fuchs G, **Rangan P**. A translation control module coordinates germline stem cell differentiation with ribosome biogenesis during Drosophila oogenesis. *Dev Cell*. 2022 Apr 11;57(7):883-900.e10.
- 5. Blatt P, Wong-Deyrup SW, McCarthy A, Breznak S, Hurton MD, Upadhyay M, Bennink B, Camacho J, Lee MT, **Rangan P**. RNA degradation is required for the germ-cell to maternal transition in Drosophila. *Curr Biol.* 2021 Jul 26;31(14):2984-2994.e7.

Tirtha K. Das, PhD, Keynote Speaker, Hunter College High School Inaugural Research Symposium, New York, NY, 23rd November 2024, "Using Fly Models to Explore Cancer and Therapies"

Son Duong, MD, NHLBI, K08, "Precision Prediction of Right Ventricular Size, Function, and Outcomes in Patients with Repaired Tetralogy of Fallot"

Esra Karakose, PhD, Breakthrough T1D (formerly JDRF) Career Development Award, "Exploring the Effects of Beta Cell Regenerative Drugs on Cell Fate Decisions in Human Pancreatic Islets" **Kaya Parikh**, Terra NYC STEM Fair in the Medicine and Health Sciences, "A Novel Method for Optimizing and Quantifying Dosage Efficacy of Breakthrough KRAS-G12D Inhibitor MRTX1133 using Drosophila melanogaster", 2nd place award

Amy M. Rapp, PhD, NIMH, K23, "Dissecting Neurocognitive Components of Compulsivity Using Computational Modeling and EEG"

Hugh Sampson, MD, Scholar GPS ranked #1 in Food allergy and #1 in Allergy in general (lifetime)

Scott Sicherer, MD, Scholar GPS, Ranked #2 in Food Allergy and #3 in Allergy in general (lifetime)

Faculty Highlights

Publications

Truong DT, Trachtenberg FL, Hu C, Pearson GD, Friedman K, Sabati AA, ... **Anderson BR**, ... Newburger JW. Six-month outcomes in the longterm outcomes after the multisystem inflammatory syndrome in children study. *JAMA Pediatr.* 2025 Mar 1;179(3):293-301.

Anderson BR, Herman PM, Whedon JM, Bradley R. Determinants of complementary and integrative health approaches: A cross-sectional study using the all of us research program. *J Integr Complement Med.* 2025 Mar 3.

Seo H, Cuddleston WH, Fu T, Navarro E, Parks M, Allan A, ... **Breen MS**, ... Humphrey J. Cytosine-to-uracil rna editing is upregulated by pro-inflammatory stimulation of myeloid cells. *bioRxiv*. 2025 Mar 17.

Xue Y, Hou X, Zhong Y, Zhang Y, Du S, Kang DD, ... **Brown BD**, ... Dong Y. Lnp-rna-mediated antigen presentation leverages sars-cov-2-specific immunity for cancer treatment. *Nat Commun*. 2025 Mar 4;16(1):2198.

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Roselli C, Surakka I, Olesen MS, Sveinbjornsson G, Marston NA, Choi SH, ... **Chami N**, ... **Loos RJF**, ... Ellinor PT. Meta-analysis of genomewide associations and polygenic risk prediction for atrial fibrillation in more than 180,000 cases. *Nat Genet.* 2025 Mar;57(3):539-47.

Li X, Chen H, Selvaraj MS, Van Buren E, Zhou H, Wang Y, ... **Chami N**, ... **Loos RJF**, ...Lin X. A statistical framework for multi-trait rare variant analysis in large-scale whole-genome sequencing studies. *Nat Comput Sci*. 2025 Feb;5(2):125-43.

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Hong JG, Trotman J, Carbajal Y, Dey P, Glass M, Sclar V, ... **Chu J**. Mannose reduces fructose metabolism and reverses mash in human liver slices and murine models in vivo. *Hepatol Commun*. 2025 Apr 1;9(4).

Crook S, Sanchez CM, Dragan K, Woo JL, Jiang P, Neidell M, **Anderson BR**. Days alive out of health care: A novel measure of health status after congenital heart surgery. *J Am Coll Cardiol*. 2025 Mar 4;85(8):851-62.

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Faculty Highlights

Publications, continued

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Chen X, Cunha Carvalho B, Sinha A, Pittman N, Benkov K, Lai J, **Lafaille MC**, **Dunkin D**. Irritable bowel syndrome with diarrhea in pediatric patients is associated with type 2 and type 9 t cells in the intestinal mucosa. *Cell Mol Gastroenterol Hepatol*. 2025 Feb 28:101488.

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